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An Array of Genetic Differences: How Your Body Influences Your Diet

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Abstract

Introduction
The debate on what one should and should not eat has grown increasingly heated in recent years, with nutritionists recommending competing diets. Although people try different diets for weight loss, clearer skin, cultural reasons, and overall health, the solution may be found at a more fundamental level. While many environmental and lifestyle factors influence health, one's genetic makeup plays a key role in determining what diets are best to follow.

Nutritional genomics, or nutrigenomics, examines the interaction between genes and nutrition, helping predict one's reaction to different nutrients [1]. Common genetic health issues, including lactose intolerance and gluten sensitivity, are well-documented and have already led to specialized dietary recommendations. However, a growing body of research suggests that many additional health risks related to nutrient consumption are rooted in individual genomic variations. Understanding these interactions may help explain why dietary recommendations are effective for some individuals but ineffective or even harmful for others. In recent years, large-scale genomic and nutritional studies have begun identifying numerous gene–diet interactions, highlighting the growing scope of nutrigenomics research. Using tools such as genome-wide association studies and large population nutrition datasets, researchers are now able to examine thousands of genetic variants linked to metabolic responses and dietary behavior.

Methods
To understand how genes influence dietary response, nutrigenomics research can be broken down into proteomics, metabolomics, transcriptomics, and epigenomics. When it comes to identifying specific genes, transcriptomics, the study of RNA expression, is extremely important. By analyzing RNA molecules, researchers can determine how genes are expressed, function, and evolve.

Scientists commonly examine single-nucleotide polymorphisms (SNPs), of which over 500,000 can be identified per individual [2]. They are able to compare SNPs, especially those involved in metabolic regulation and cell maintenance, and track their effects on gene expression. Researchers are now experimenting with the manipulation of nutrient intake to observe the body's reaction, including genomic-based changes. Scientists are currently proposing experiments that test individuals with the same diet and different genotypes and vice versa. However, these experiments will be challenging to regulate, and a large-scale project of
comparable size to the Human Genome Project will likely be necessary to obtain accurate results [2]. In the meantime, observation-based research will be conducted to identify associations between diet and genomics.

Results
Research has revealed several gene-diet interactions that significantly impact health. One well-studied example is the ACE gene, which codes for the angiotensin-converting enzyme. The enzyme converts the angiotensin I hormone into angiotensin II, a vasoconstrictor that can increase blood pressure. Certain dietary changes, such as increasing potassium, soy protein, alpha-linolenic acid, and reducing sodium intake, have been shown to decrease ACE activity, thereby reducing blood pressure. Specifically, the rs4343 polymorphism in exon 17 of the ACE gene exacerbates the effects of a high-saturated-fat diet on the angiotensin system [3]. Another mutation in the ACE gene, affecting susceptibility to hypertension, is known as the I/D polymorphism, located in intron 16. The D variant leads to higher sodium sensitivity, increasing the risk of high blood pressure [4]. As a result, high-sodium diets can be more harmful for individuals with this variant, contributing to hypertension.

Genetic differences and mutations can also contribute to our perception of taste. For example, certain genetic mutations may predispose someone to a more pronounced sweet tooth than others. A family of genes on the first chromosome called TAS1R is responsible for sweet taste reception. Specific differences in TAS1R3 can make an individual more sensitive to sweet tastes. A copy of the gene is inherited from each

parent, with more copies making the flavor more intense [5]. Without knowing this difference, sugary sweets may seem far more appealing to some than others. At first glance, this may not seem significant, but increased consumption of sugary foods can lead to conditions like type 2 diabetes or heart disease.

Furthermore, there are other genetic modifications that can leave people more susceptible to type 2 diabetes. One of these genes is the fat mass and obesity-associated (FTO) gene. Found on chromosome 16, this gene is also associated with obesity and breast cancer. People inherit one copy of the gene from each parent, but if they inherit two high-risk copies, they are about 70% more likely to become obese. Those with two high-risk copies have higher levels of the hormone ghrelin, the “hunger hormone," which stimulates appetite. These levels are especially elevated after eating, making people more prone to overeating because they feel hungry more quickly. If uncontrolled, there can be severe consequences, as consistent overeating can contribute to obesity. Using real-time brain imaging, some studies have even found that the FTO variant can change the way the brain responds to images of food [6,7]. These findings explain why individuals with the FTO variation tend to eat more and prefer foods that are calorically dense.

Conclusion

The biggest obstacle in understanding the genome’s response to nutrients is that each metabolic and gene-expressing pathway is unique, with genetic differences creating varied levels of risk for health conditions across individuals. A more comprehensive understanding of the human genome is required in order to develop highly personalized dietary recommendations. At the moment, nutrigenomic tests can be costly, at over $250. Some tests look at approximately 70 genes to provide a complete diet analysis [8]. Soon, tests could incorporate even more genes to provide the most accurate results at a cheaper cost. Nutrigenomics is critical in understanding how to proceed with diets, supplements, and lifestyle choices; and as research continues, there is potential for fully personalized, gene-based diets.

References

1. Cleveland Clinic, “How Nutrigenomics May Impact the Way You Eat,” Cleveland Clinic, May 10, 2022, https://health.clevelandclinic.org/how-does-nutrigenomics-work.
2. Mead, M. Nathaniel, “Nutrigenomics: The Genome–Food Interface,” Environmental Health Perspectives 115, no. 12 (December 2007), https://pmc.ncbi.nlm.nih.gov/articles/PMC2137135/.
3. Zambrano, Ana Karina, et al, “Genetic Diet Interactions of Ace: The Increased Hypertension Predisposition in the Latin American Population,” Frontiers in Nutrition 10 (October 26, 2023), https://doi.org/10.3389/fnut.2023.1241017.
4. Schüler, Rita, et al, “High-Saturated-Fat Diet Increases Circulating Angiotensin-Converting Enzyme, Which Is Enhanced by the rs4343 Polymorphism Defining Persons at Risk of Nutrient-Dependent Increases of Blood Pressure,” Journal of the American Heart Association 6, no. 1 (January 11, 2017), https://doi.org/10.1161/jaha.116.004465.
5. Fushan, Alexey A., et al, “Allelic Polymorphism within the TAS1R3 Promoter Is Associated with Human Taste Sensitivity to Sucrose,” Current Biology 19, no. 15 (August 2009): 1288–93, https://doi.org/10.1016/j.cub.2009.06.015.
6. Robertson, Sally, “Fat Mass and Obesity-Associated Gene (FTO),” News Medical, December 29, 2022, https://www.news-medical.net/health/Fat-Mass-and-Obesity-Associated-Gene-(FTO).aspx
7. UCL News, “How ‘Obesity Gene’ Triggers Weight Gain,” UCL News, July 15, 2013, https://www.ucl.ac.uk/news/2013/jul/how-obesity-gene-triggers-weight-gain.
8. Cleveland Clinic, “Nutrition,” Cleveland Clinic, https://my.clevelandclinic.org/departments/wellness/integrative/treatments-services/nutrition