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Role of Lifestyle, Gene Environment Interactions and Mutations in Multiple Genes in Obesity

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Introduction

Obesity is caused by a chronic energy imbalance in which a person consumes more calories from food and drink than their body requires fuelling its metabolic and physical activities. Obesity has become more common in recent decades, owing to an "obesogenic" society that provides easy access to high-calorie meals while limiting possibilities for physical activity. The obesity pandemic might be seen as a communal reaction to this situation. Obesity is a severe public health issue since it raises the risk of diabetes, heart disease, stroke, and other serious illnesses.

Every aspect of human physiology, development, and adaptability is influenced by genes. Obesity is no different. Yet, little is known about the exact genes that cause obesity, as well as the scope of so-called "genetic environment interactions," or the complicated interplay between our genetic makeup and our life experiences. Several unusual kinds of obesity are caused by single-gene mutations, known as monogenic mutations. Such mutations have been reported in genes that code for the hormone leptin, the leptin receptor, pro-opiomelanocortin, and the melanocortin-4 receptor, among others, which play important roles in appetite regulation, food intake, and energy balance [1].

Obesity is also a defining feature of some genetic disorders, such as Prader–Willi and Bardet–Biedl syndromes, which are caused by mutations or chromosomal abnormalities. Obesity is frequently associated with mental impairment, reproductive abnormalities, and other issues with these disorders.

Obesity-related genes can be found through genome-wide association studies: Hundreds of thousands of genetic markers are scanned across thousands of people' full sets of DNA in a genome-wide association research to uncover gene variants that may be linked to a certain illness. Gene variants that have a role in prevalent, complicated disorders like obesity can be discovered using these investigations. A mutation in only one little region of the DNA that encodes for a gene may often have a significant impact on the gene's function [2]. "Gene variants" or "single-nucleotide polymorphisms" (SNPs) are small DNA differences that are frequently linked to illness risk.

Obesity is seldom passed down in families in a clear inheritance pattern caused by alterations in a single gene. MC4R, which encodes the melanocortin 4 receptor, is the most usually implicated gene. A small percentage (5%) of obese persons in diverse ethnic groups have changes in MC4R that reduce its function. Children who are affected feel ravenous and gain weight as a result of their constant overeating (hyperphagia). Single-gene (monogenic) obesity has been linked to rare variations in at least nine genes thus far.

Gene-environment interactions: Obesity's fast expansion over the world is unlikely to be explained by genetic variations. This is due to the fact that the "gene pool"—the frequency of distinct genes in a population—remains relatively steady across many generations. New mutations or polymorphisms take a long time to propagate. So, if our genes have essentially remained the same throughout the previous 40 years of growing obesity rates, what has changed? The physical, social, political, and economic factors that determine how much we eat and how active we are are referred to as our environment. Changes in the environment that make it simpler for individuals to overeat and harder for them to obtain adequate exercise have played a crucial part in the recent rise in overweight and obesity.

Obesity-related gene-environment interactions are still in the early stages of research. Many people who inherit so-called "obesity genes" do not become overweight, according to the research thus far. Rather, it appears that eating a nutritious diet and getting adequate exercise might help to mitigate some of the obesity risk associated with genes [3].

Depending on their family history and ethnicity, most people have a genetic tendency to obesity. Changing one's diet, lifestyle, or other environmental circumstances to overcome a hereditary propensity to obesity is common. Some of those changes include the following:

1) Food availability at all hours of the day and in areas where food was previously unavailable, such as gas stations, pharmacies, and office supply stores;

2) A significant drop in physical activity during work, domestic activities, and leisure time, particularly among children;

3) An increase in time spent watching television, using computers, and engaging in other sedentary activities; and

4) The rise in popularity of highly processed foods, fast food, and sugar-sweetened drinks, as well as the pervasive advertising campaigns that promote them

Mechanisms through which genes regulate energy balance: The brain responds to signals from fat (adipose) tissue, the pancreas, and the digestive tract to control food intake. Hormones include leptin, insulin, and ghrelin, as well as other tiny molecules, send these messages. These impulses are combined with other inputs by the brain, which then sends instructions to the body: either consume more and use less energy, or the reverse. Small alterations in these genes can modify their levels of activity, which are the foundation for the signals and reactions that drive food intake.

Survival necessitates the use of energy. Rather than controlling weight increase, human energy management is designed to defend against weight reduction. To explain this finding, the "thrifty genotype" theory was presented. It implies that the genes that enabled our forefathers endure periodic famines are now being tested in areas where food is abundant all year [4].

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A greater knowledge of the genetic contributions to obesity—particularly common obesity—as well as gene-environment interactions would lead to a better understanding of the obesity causation pathways. Such data may one day lead to promising obesity prevention and treatment solutions. However, it's vital to note that genes have a little role in obesity risk, whereas our hazardous diet and exercise environment play a major one. "Genes may co-determine who gets fat, but our environment dictates how many become obese," one expert noted. That is why, in order to avoid obesity, we must change our environment to make healthy choices simpler for everyone.

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