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Customized Wellbeing and Sustenance Consumption of Diet and Health in Nutritional Genomics

M Caroline*

Department of Nutrition, University of London, London, UK

Corresponding author: M Caroline, Department of Nutrition, University of London, London, UK, E-mail: caroline.m@gmail.com

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Description

Nutritional genomics, also referred to as nutrigenomics, is a branch of science that investigates the connection between the human genome, diet and health. Through systems biology and relationships between single genes and single food compounds, experts in the field strive to comprehend how a food affects the entire body. The relationship that food has with inherited genes is called nutritional genomics or nutrigenomics. Several subfields, including nutrigenetics, nutrigenomics and nutritional epigenetics, are included in the umbrella term nutritional genomics.

Nutritional Therapy and Intervention

The mechanisms by which genes respond to nutrients and express particular phenotypes, such as disease risk, are explained in some detail in each of these subcategories. Nutritional genomics can be used for a variety of purposes, such as determining how much nutritional therapy and intervention can effectively be used for disease prevention and treatment. As other diseases closely related to diet (but not deficiency), such as obesity, became more prevalent, nutritional science expanded to include these topics as well. Nutritional science initially emerged as a field that studied individuals lacking certain nutrients and the subsequent effects, such as the disease scurvy, which is caused by a lack of vitamin C. Preventative measures are typically the focus of nutrition research, which aims to determine which nutrients or foods increase or decrease the risk of illness and bodily harm. For instance, an epigenetic pattern in which the maternal loci are inactivated by over methylation and the paternal copy in the chromosomal region is erroneously deleted has been specifically linked to Prader Willi syndrome, a disease whose most distinguishing feature is an insatiable appetite. However, while a few Single-Nucleotide Polymorphisms (SNPs) or other localized patterns may be associated with particular disorders, many more polymorphisms may be produced by population variation. Before the 20th century's food product globalization, the naturally occurring foods of Greece, Italy and Spain were referred to as the mediterranean diet. Whole grains, fruit, vegetables, olive oil, legumes and moderate amounts of red wine are all included in the diet. Dairy and foods high in fat are rarely consumed. Numerous studies on nutritional genomics have shown that the mediterranean diet is the best for nutrition. By providing anti-metabolic, anti-cardiovascular and anti-cancer agents, it has been shown to have a positive effect on mortality reduction. The abundance of dietary bioactive compounds found in mediterranean staples is the reason for these advantages. *Curcuma Longa* (turmeric), resveratrol, capsaicin, quercetin and the polyphenols in extra virgin olive oil are all examples of this. In order to stop angiogenesis and the onset of neurodegenerative disease, several of these bioactive compounds interact with the body's cellular and molecular function, gene expression and epigenome. There are numerous uses for nutritional genomics. Some disorders, such as diabetes and metabolic syndrome, can be identified through personalized assessment. Nutrigenomics can assist with customized wellbeing and sustenance consumption by evaluating people and make explicit dietary prerequisites. The prevention and treatment of specific genetic disorders are the primary goals. Obesity, Coronary Heart Disease (CHD), hypertension and diabetes mellitus type 1 are examples of genetically based disorders that respond positively to nutritional intervention. Spina bifida, phenylketouria and alcoholism are just a few genetic conditions that can frequently be prevented by the parent's diet.

Risk of Coronary Heart Disease

The body's sensitivity to food is how genes linked to nutrition show up. There is a correlation between the presence of two alleles at the E and B apolipoprotein loci and CHD in studies. Individual responses to consuming lipids are caused by these differences in loci. While others with different loci do not experience an increased risk of CHD and weight gain, some do. Across all populations, studies have demonstrated a direct link between a lower risk of coronary heart disease and a lower intake of lipids. In nutritional genomics, obesity is one of the most widely studied topics. Each individual may respond to diet differently due to genetic differences. The aim of this field is to suggest dietary changes that could prevent or reduce obesity by examining the interaction between dietary pattern and genetic factors. Some SNPs appear to increase the likelihood that a person will gain weight from a diet high in fat; when eating a diet high in fat or low in carbohydrates, those with the AA genotype in the FTO gene had a BMI that was higher than that

of those with the TT genotype. It was discovered that people with the GG homozygous genotype are more likely to have a higher BMI than people with the AA allele, whereas people with the A/G heterozygous genotype were found to be associated with obesity (in terms of BMI and waist circumference) and for people who eat a habitually high fat diet (>35% of energy intake). However, the group that consumes less than 35% of their energy from fat does not exhibit this difference. Single-nucleotide variants, or SNVs, are single nucleotide substitutions with an allele frequency of less than 1%. Any single nucleotide change in a DNA sequence, including common SNPs and uncommon germline or somatic mutations, can be referred to as a variant. As a result, cancer cell point mutations have been referred to as SNV. In molecular diagnostics applications, such as designing PCR primers to detect viruses, where the viral RNA or DNA sample may contain SNVs, DNA variants must also be taken into account. However, this nomenclature uses arbitrary distinctions and is not used consistently across all fields; there have been calls for a more consistent framework for naming differences in DNA sequences between two samples as a result of this disagreement. Single-nucleotide polymorphisms can occur in the intergenic regions (regions between genes), the coding sequences of genes, or the non-coding regions of genes. Due to degeneracy in the genetic code, SNPs within a coding sequence do not always alter the amino acid sequence of the protein produced. SNPs are not evenly distributed throughout the genome; SNPs are more likely to occur in non-coding regions than in coding regions or, more generally, in areas where natural selection is acting and fixing the allele of the SNP that represents the most favorable genetic adaptation (eliminating other variants). SNP density can also be determined by other factors, such as the rate of genetic recombination and mutation. The presence of microsatellites makes it possible to predict SNP density: Long repeat tracts typically occur in regions of significantly reduced SNP density and low GC content, making AT microsatellites particularly effective predictors of SNP density.