Perspective

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Investigation of Connection between the Human Genome, Diet and Health in Nutritional Genomics

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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 between food and inherited genes, also known as nutritional genomics or nutrigenomics, was first discussed in 2001. Several subfields, including nutrigenetics, nutrigenomics and nutritional epigenetics, are included in the umbrella term nutritional genomics. 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.

Risk of Illness and Bodily Harm in Nutrients

Preventive health nutritional science started out as a field that looked at people who were deficient in certain nutrients and the effects that had on them, like the disease scurvy, which is caused by a lack of vitamin C. As other diseases that are related to diet (but not deficiency), like obesity, became more common, nutritional science expanded to include these topics as well. 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. The naturally occurring foods that were native to Greece, Italy, and Spain prior to the 20th century's globalization of food products are referred to as the Mediterranean Diet. Fruit, vegetables, olive oil, legumes, whole grains, and moderate amounts of red wine are all part of the diet. Dairy and foods high in fat are minimally 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 applications for nutritional genomics. Some disorders, such as diabetes and metabolic syndrome, can be identified through personalized assessment. By assessing individuals and determining specific nutritional requirements, nutrigenomics can assist with personalized nutrition and health intake. 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 parents' diet.

Genetically based Disorders that Respond Positively to Nutritional Intervention

The body's sensitivity to food is how genes linked to nutrition manifest themselves in coronary heart disease. 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. Obesity In nutritional genomics, obesity is one of the most researched

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. Another variation that has to do with diet is 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. However, the group that consumes less than 35% of their energy from fat does not exhibit this difference. Phenylketonuria, more commonly referred to as PKU, is a rare autosomal recessive metabolic disorder that manifests itself postpartum but can be reversed with nutritional treatment. The 23 chromosome pairs in cell nuclei and a small DNA molecule in each mitochondrion make up the complete set of nucleic acid sequences that make up the human genome. These are typically referred to as the nuclear genome and the mitochondrial genome, respectively. Human genomes contain both DNA sequences that encode proteins and a variety of DNA types that do not. The latter is a diverse group that includes DNA those codes for non-translated RNA, such as ribosomal RNA, transfer RNA, ribozymes, small nuclear RNAs, and a number of different kinds of regulatory RNAs. It also includes DNA with structural and replicatory functions, such as scaffolding regions, telomeres, centromeres, and replication origins, as well as a large number of transposable elements, inserted viral DNA, pseudogenes that are not functional, and simple, highly repetitive sequences. Non-coding DNA is largely composed of introns. Non-functional junk DNA, like pseudogenes, makes up some of this non-coding DNA, but no one knows how much junk DNA there is in total. If the X chromosome is used, haploid human genomes, which are found in germ cells (the egg and sperm gamete cells that are created during the meiosis phase of sexual reproduction prior to fertilization), have 3,054,815,472 DNA base pairs, whereas female diploid genomes, which are found in somatic cells, have twice as much DNA. Even though there are significant differences between human genomes (on the order of 0.1% due to single-nucleotide variants and 0.6 percent when considering indels), these differences are much smaller than those between humans and their closest living relatives, bonobos and chimpanzees (1.1% fixed single-nucleotide variants and 4% when including indels). Basepair sizes can also vary; after each round of DNA replication, the length of the telomere decreases.