Genetic variation and dietary response in nutrigenomics.

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Abstract

Nutrigenomics is the study of how genes and diet interact. It uses methods like proteomics, transcriptomics, metabolomics, and lipidomics to look into the molecular interactions between nutrients and genes to determine how even small changes could potentially affect animal and human health/performance. Protein, fat, mineral, and vitamin supplementation changes are the main dietary modifications studied in livestock with a focus on health and production traits. For improved health, productivity, and the environment, nutrigenomics carefully chooses nutrients to fine-tune the expression of genes that fit animal/human genotypes. Nutrigenomics is a stride forward in our understanding of the function of food as an epigenetic component in the occurrence of various diseases because it merges nutrition, molecular biology, genomics, bioinformatics, molecular medicine, and epidemiology.

Keywords: Nutrigenomics, Supplement, Epigenetic.

Introduction

All health and disease are based on the interaction of heredity and environment, or nature and nurture. Environmental factors like nutrition and exercise determine who among those who are vulnerable to a disease or condition will actually get it. Genes dictate susceptibility to a disease or condition. A significant environmental aspect is nutrition. The study of inherited disease at the DNA level and the study of nutrition at the molecular level have been made possible by methodological developments in molecular biology and genetics. As a result of this research, (a) theories and research on the relationship between genetic variation and dietary response, known as nutrigenetics, have been developed. For example, individuals may respond differently to the same diet by achieving different levels of serum cholesterol and blood pressure due to genetic variation.

Nutritional genomics

"Nutritional Genomics" refers to the relationship between nutrients and cellular/genetic processes this phrase describes the intersection of human nutrition, molecular genetic knowledge, and biochemistry and genomics [1]. The following five principles might be used to summarise the conceptual framework for this genomic research:

1) Typical dietary substances influence gene expression and/or structure by directly or indirectly affecting the human genome.

2) Diet can provide a significant risk for a number of diseases in some people and under specific situations.

3) The onset, incidence, development, and/or severity of chronic diseases are likely to be influenced by a few diet-regulated genes (and their typical, common variations). Dietary chemicals indirectly regulate some of TFs. The sterol regulatory element binding proteins (SREBPs), for example, are activated by protease cleavage, an event regulated by low levels of oxy sterols and changes in insulin/ glucose and PUFAS

Nutritional epigenetics

Telomeres are nucleoprotein structures that cap the ends of chromosomes, and maintain chromosome stability. Degeneration of telomeres leads to whole chromosomal instability, and chromosomal fusion and therefore gene amplification, an important risk factor for cancer. Folate and nicotinic acid deficiency increased oxidative stress and telomere dysfunction [2].

Telomere and nutritional status

It is clear that even the small damages in the genome can cause crucial effects in whole human life [3]. DNA metabolism and repair is depending on a variety of dietary factors that act as cofactors or substrates. Nutritional requirements is important for the prevention of DNA oxidation (i.e. antioxidants such as carotenoids, Vit E and C), prevention of uracil incorporation into DNA (i.e., folate), maintenance methylation of CpG in DNA (methionine, cholin, folate and vitamin B12), as cofactors or as components of DNA repair enzymes (Zn, Mg), maintenance of telomere length (niacin, folate).

Genome damage and nutritional deficiency

As was previously noted, nutritional status affects the integrity of the genome, and a lack of several micronutrients can

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seriously harm the genome [4]. At least nine micronutrients, including vitamin E, calcium, folate, retinol, nicotinic acid, alpha-carotene, riboflavin, pantothenic acid, and biotin, have been found to have an impact on the integrity of the human genome *in vivo*. For DNA replication, repair, and maintenance of DNA methylation patterns, folate and vitamin B12 are essential. Studies conducted on human cells both in *vivo* and *in vitro* conclusively demonstrate that low levels of folate and vitamin B12, as well as high plasma homocysteine, are linked to the production of chromosomal fragile sites, chromosomal breakage, abundant uracil in DNA, and DNA hypomethylation. Niacin, commonly known as nicotinic acid, is essential for maintaining chromosome integrity and lowering cancer risk [5].

Conclusion

The relationship between nutrients, metabolic intermediates, and the mammalian genome is clarified by nutritional genomics. The response to bioactive food components depends on genetic background (Nutrigenetic effects), which might affect targets or sites of action for absorption and metabolism. The reaction to food ingredients is also influenced by DNA methylation and other epigenetic processes. The capacity of bioactive dietary ingredients to modify gene expression patterns (nutrigenomics effects) affects the total response as well. In addition, protein synthesis, degradation, and posttranslational modification may be affected by bioactive dietary ingredients. Understanding how human genetic variety, genome function, and dietary elements interact will make it possible to precisely manipulate genome function and stability over the course of a person's life for maximum health and illness prevention.

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