

Review Article

DEVELOPMENT AND PRINCIPLES OF NUTRIGENOMICS

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Genomics:

Genomics is the study of the functions and interactions of all the genes in the genome, including their interactions with environmental factors.

Nutrigenomics:

Nutrigenomics is the study of the relationship between what we eat and how our genes function and thus how healthy we are. The nutrigenomics aiming to achieve “personalized nutrition”. The recommendation of foods and supplements are based on a person’s genetic profile. The individual differences may be at the level of single nucleotide polymorphisms (variations in a single base pair) rather than at the gene level.

Generally speaking nutrigenomics is the study of how nutrients and genes interact and how genetic variations can cause people to respond differently to food nutrients.

Nutrigenomics examines the effects of nutrients on the expression of genes and how genetic makeup affects a person’s response to individual nutrients and combinations of nutrients.

Nutrigenomics is a new era in Nutrition research:

Understanding of human nutrition has followed developments in the sciences, primarily chemistry, biochemistry and physiology.

a) Naturalistic Era (400BC – 1750 AD)

In this era Hippocrates hypothesized about the body’s “innate heat”. During the next 500 years little happened in either the development of scientific knowledge or nutrition science.

b) Chemical – Analytical Era (1750 – 1900)

It is highlighted by Lavoisier’s calorimetric studies. He discovered how food is metabolized by oxidation to carbon dioxide, water and heat. He also invented the calorimeter.

c) Biological era (1900 – present)

It was founded on advances in chemistry, biochemistry and understanding of the metabolic pathways.

d) Cellular Era (Post 1955)

It focuses on understanding the functions of essential nutrients and the role of micronutrients (vitamins and minerals) as a cofactor for enzymes and hormones and their subsequent role in metabolic pathways. The roles of carbohydrates and fats in diseases such as diabetes and atherosclerosis were discovered and actual potential mechanisms have been uncovered.

Even in those observations of health and disease puzzle existed. Why can some individuals consume high fat diets and yet show no evidence of atherosclerotic disease? Genetic differences certainly were suspected. But elucidating and providing cellular, molecular and ultimately genetic level mechanisms in both healthy and unhealthy individuals provided to be a challenge.

With the continuing developments in tools that enable molecular level exploration of cause – effect phenomena, scientists have begun to develop hypothesis and conduct experiments to lay the foundation for a deeper level of understanding of gene-diet interaction. Today an emerging field of nutritional research focuses in identifying and understanding molecular level interaction between nutrients and other dietary bioactives with human genome during transcription, translation and expression, the process during which the proteins encoded by the genome are produced and expressed.

Genetic era or nutrigenomics:

Continuing and accelerating discoveries in genomics present possibilities for an ever more dynamic era of scientific investigation based on understanding the effects of nutrients in molecular level processes in the body as well as the variable effects of nutrients and non-nutritive dietary phytochemicals have on each of us as individuals. We call this the new era in nutritional science the **genetic era or nutrigenomics**.

Principles of nutrigenomics:

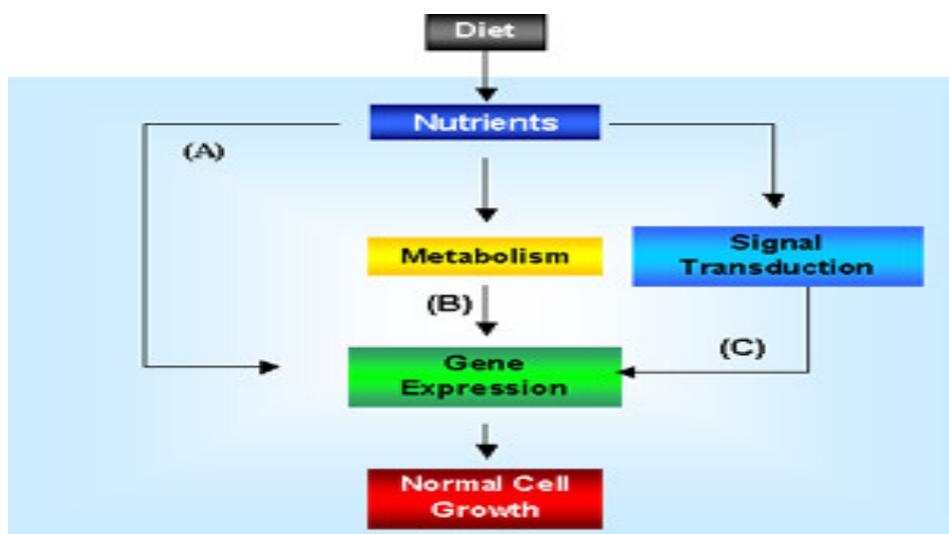
- Improper diets are risk factors for disease.
- Dietary chemical alter gene expression and / or change genome structure.
- The influence of diet on health and disease susceptibility depends upon an individual's genetic makeup.
- Genes regulated by diet play a role in chronic disease.

- “Intelligent Nutrition” – that is diets based upon genetics, nutrient requirements and status – prevents and mitigates chronic disease.

Link between DNA and Nutrition:

Dietary chemicals have been shown to alter gene expression in a number of ways for example they may

- Act as ligands for transcription factor receptors
- Be metabolized by primary and secondary metabolic pathways, there by altering Concentrations of substrates or intermediates or
- Serving as signaling molecules.



Genes, built from microscopic double – strands of DNA, direct the behavior of your body’s 60 trillion cells – they define your body’s outward physical features, such as the colour of your eyes, and hair and how efficiently it work on the inside. When your genes work well, they enable you live to a ripe old age with a low risk of disease. When genes don’t they can accelerate your body’s ageing process and increase your risk for cancer and other diseases.

The information in your genes is written in the chemical language of DNA. The letters in this alphabet are sequences of molecules called nucleotides. Each letter is constructed around one of four substances called DNA bases. Adenine (A), Cytosine (C), Guanine (G) and Thymine (T). Different arrangements of these four nucleotides spell different words and long strings of DNA (TACGACCTGA) from the genes that instruct cells to make specific enzymes and proteins. These enzymes in turn catalyze a host of other biochemical reactions and the proteins form the structure of hormones and tissue. All of this remarkable activity depends on nutrients. The DNA in the protein you eat is broken down and

reconstructed into your own distinctive DNA. Vitamins B₃ and B₆ are needed for thymine synthesis, folic acid for guanine and adenine and B₃ for cytosine. When these nutrients are lacking, DNA cannot be synthesized and its instructions cannot be carried out.

Diet and genetics interact in numerous ways to influence chronic disease risk. Genetics influence the absorption, excretion and metabolism of nutrients. Genetics also influences the human body's physiological responses to diet. Diet in turn may influence the expression (activation) of genes related to specific chronic diseases (Miller *et al.*, 1997).

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