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Volume 2 • Issue 3 • 2018

Scientia Ricerca

Editorial

Clinical Biotechnology and Microbiology

ISSN: 2575-4750

Nutrient and Nutraceuticals versus Modulation of Genetic Expressions in Cardiovascular Disease

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Received: May 29, 2018; **Published:** June 25, 2018

Volume 2 Issue 3 June 2018

The vital roles of genes in the life of a subject can be best understood through the total package of chromatin rather than individual gene [1]. Designer genes administer the barricading of cells and cellular recognition during development. There are tissue specific genes responsible for cellular differentiation and organogenesis. It is very likely that by changing nutritional environment, the activity and conformation of the chromatin may be changed, thus, may result in to genetic expression along with relaxation of chromatin. It seems that wild foods and nutraceuticals; w-3 fatty acids, antioxidants, vitamins and minerals are significant determinant of enzymes, hence these foods and nutrients can suppress the expression of harmful genes. Several enzymatic machineries; such as methyltransferases, histone deacetylases, histone acetylases, histone methyltransferases and methyl binding chromatin protein are under influence of chromatin complex [1].

In cellular function, a gene is made either awake or silent depending upon specific post translational modifications of histones on one side and methylation of cytosine of phenotype guanine (CpG) islands in the promoter region of a gene on the other side, resulting into a distinct trait for example cytosine of phenotype guanine (CpG) island methylator phenotype that are nucleotides in DNA. Several chromatin regulatory proteins are dynamic and are continuously recruited, bound and ejected which may be due to environmental factors like dietary proteins, antioxidants and vitamins.

Since nutrient and Nutraceuticals have interactions with genes, it poses the possibility that a genetic cause may explain the continued appearance of nutritional disease in the population by nutritional silencing of phenotype expression [2-4]. Polyunsaturated fatty acids (w-6 and w-3), milk, calcium, vitamin, iron, ascorbate and saturated fat have been found to modulate gene expression in various experimental studies [2-4]. The phenotypic expression for health or disease would depend on phenotype and environment, as well as on genotype and upon structural variations of genes [1]. There is a limited food supply such as in the rural population of developing countries and lower social classes in urban areas, which also have greater physical activity due to physically demanding occupations [2]. There is also in-utro undernutrition due to wide spread malnutrition during pregnancy common in developing countries [5,6]. Nevertheless these interactions predispose the biological mechanisms to adapt and develop survival gene which may modulate genotype for increased survival. In urban population of developing countries and immigrants from developing to developed countries, better food supply, usually western diet, may be associated with phenotypic expression for disease [7].

Citation: Sanjay Mishra. "Nutrient and Nutraceuticals versus Modulation of Genetic Expressions in Cardiovascular Disease". *Clinical Biotechnology and Microbiology* 2.3 (2018): 374-376.

The health status of gene, copy number variants (CNVs) or single-nucleotide polymorphisms (SNPs), whether single or polymorphic appear to be significant in the manifestation of health or cardiovascular disease (CVD), hypertension or diabetes and obesity [1,7,8]. Augmented intake of energy, may cause obesity due to expression of obesity genes, which is major cause of CVD. In one study [9], subjects were 383 consecutive patients with angiographically authenticated coronary artery disease (CAD) and 368 non-CAD subjects adjusted for age and BMI in the Japanese population. Single nucleotide polymorphisms (SNPs) in the adiponectin gene were determined by Taqman polymerase chain reaction (PCR) technique or a PCR-based assay for the analysis of restriction fragment length polymorphism. The plasma adiponectin concentration was analyzed by enzyme-linked immunosorbent assay. Among SNPs, the frequency of I164T mutation was appreciably higher in CAD subjects (3%) than in the control (1%, $p < 0.05$). The plasma adiponectin levels in subjects carrying the I164T mutation were considerably lower than in those without the mutation, and were independent of BMI. In contrast, SNP94 and SNP276, which are reported to be associated with an increased risk of type 2 diabetes [10], were associated neither with CAD prevalence nor with plasma adiponectin level. Subjects with I164T mutation exhibited a clinical phenotype of the metabolic syndrome.

Conclusions and Recommendations

In brief, this compilation concludes that nutraceuticals and wild foods that are rich sources of various nutraceuticals; w-3 fatty acids and antioxidants, can modulate genetic function and gene expression and may be important in the pathogenesis and prevention of chronic diseases of affluence. Further studies are required to demonstrate that a ratio of w-6/w-3 of 1:1 in the blood by nutraceutical supplementation can adapt the genes and provide further protection against CVD, diabetes and cancer. These manipulations according to time structure or as chronotherapy may be highly worthwhile.

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