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Editorial Open Access

Epigenetics, Diet or Exercise?

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Editorial

Genetics is the study of the effects of our maternal and paternal ancestry on our genetic codes, formulated from deoxyribonucleic acid (DNA). Genomics is the assessment of the genetic codes and their functions, and their combined effect on the development of the whole organism. Genetics identifies the functioning and composition of the single gene whereas genomics addresses all genes and their inter-relationships in order to understand their combined influence on the growth and development of the organism.

Epigenetics is the study of changes in gene expression or cellular phenotype, caused by mechanisms other than changes in the underlying DNA sequence [1]. These mechanisms can be one of, or a combination of, a myriad of environmental factors, such as diet, chemicals, exercise or ageing.

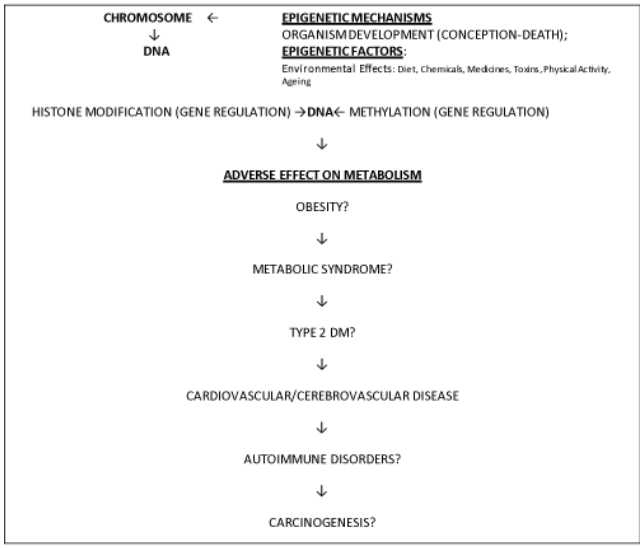

Figure 1: Epigenetic Mechanisms.

DNA Methylation

Addition of a methyl group to DNA nucleotides, Cytosine or Adenine, from dietary sources can activate or repress gene response.

Methylation modifications that regulate gene expression can cause genomic imprinting.

Histone Modification

DNA winds around Histone Proteins which can activate or repress gene response.

Histone is the main protein component of Chromatin (complex of macromolecules comprising DNA, Protein and RNA).

DNA = Deoxyribonucleic Acid

RNA = Ribonucleic Acid

Epigenetic marks are tissue specific and include DNA methylation and histone modifications which mediate biological processes such as imprinting (Figure 1). Many imprinted genes are regulators of gene expression controlling growth. Imprinting disorders often feature obesity as one of their characteristics [2].

Geneticists have identified genes such as the obesity gene (FTO) [3], a low fat gene (APOA5) [4], and that variations in the adiponectin gene (SNP 276 G Allele) can lead to hypoadiponectinaemia, which then results in insulin resistance, the metabolic syndrome, increased atherosclerosis and ultimately premature morbidity and mortality. Using exome sequencing a low-frequency coding variant in the SYPL2 that was associated with morbid identified [5].

There is an assumption that genetics, whether they are single or multiple nucleotide mutations could be responsible for polymorphisms resulting in the obesity epidemic that has developed in the latter part of the twentieth century and pervaded the current century. The question that requires an answer is, can we identify a specific genetic code which produces a variant lipid polymorphism which causes increased adiposity?

The significant beneficial effects of physical activity, appear to have been forgotten and replaced by a rapid rise in appetite suppressants and a demand for bariatric surgery. Such are the failings of an innate underactive society, who are desperate to lay the blame to a nucleotide sequence for their over-consumption of food, and alcohol.

Has the pharmaceutical industry become so authoritative and the surgical budgets so structured that balancing equations has deleted exercise, in the same way that scientists are attempting to delete rogue genes?

The adage "you are what you eat" would now seem to be more important than before, with a new emphasis on probiotics. The identification that the dietary ingestion of a bacterium that can decrease inflammatory responses of ageing, may have a dramatic influence on the management of obesity, without the side effects of traditional pharmacotherapies [6]. Indicators, typical of senescence

were restored to youthful levels, by ingestion of probiotics, comparable to using systemic administration of antibodies blocking pro-inflammatory cytokines, such as interleukin-17A. Interleukin 17 is a cytokine that acts as a potent mediator in delayed-type reactions by increasing chemokine production in various tissues to recruit monocytes and neutrophils to the site of inflammation, similar to Interferon gamma. Interleukin 17 as a family functions as a pro-inflammatory cytokine that responds to the invasion of the immune system by extracellular pathogens and induces destruction of the pathogen's cellular matrix. There is a correlation between probiotic consumption and IL-10 and IL-17 secreted by peripheral blood mononuclear cells in overweight and obese people [7].

Probiotics are live microorganisms which when ingested in adequate amounts; confer health benefits on the host. The intestinal microbiota plays a fundamental role in maintaining immune homeostasis [8]. Probiotics can influence the immune system by their metabolites, cell wall components and DNA. Products of probiotics are recognised by host cells with pattern recognition receptors [9]. Experimental studies have demonstrated the preventive effects of some bacterial strains on obesity [10]. Is it possible that our future health is in understanding our symbiosis with dietary probiotics and not looking for the golden bullet pharmaceutical? As to exercise, surely we should leave that to the obsessional athlete?

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