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Mini Review

Epigenetics: A Radiant Change in Dentistry

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Abstract

Epigenetics plays an important function in gene regulation, which either encourage or deteriorate human health.

Epigenetics is at the core of phenotypic variation in health and disease, it appears to be likely that understanding epigenome holds huge guarantees for preventing and treating diseases. It also offers a critical window to understand the role of the environment's interactions with genome in causing disease, which can help us to modulate those interactions to improve human health.

Moreover, other factors such as diet, smoking, stimuli, and inflammation can cause reversible change in epigenetically regulated gene expression which can make a contribution to the progression of certain diseases like cancer, hypertension etc.

Genetic risk is an important component in the model, influencing prenatal risk, the development of neuroendocrine dysregulation and negative parenting over time, and both early childhood and adolescent externalizing problems, as per quantitative genetic theory. It is worth noting here that this cross-disciplinary approach is becoming much more common place in a variety of fields.

Purpose of this paper was to establish a theoretical knowledge of gene-environment interactions for the early diagnosis of disease and to provide a possible preventive care for the betterment of human general and oral health.

Literature to fill the paper with healthy knowledge was gathered from Dr Radha Krishnan Central library, Jaipur and from various internet sites which were accessible for public domain and only in English dialect from year 1869 to 2017.

Keywords: Epigenetics; Phenotype; Genotype; Health; Disease

Introduction

This paper provides evidence that epigenetics plays an important function in gene regulation which eventually either encourage or deteriorate human health.

Epigenetics has been termed to define epigenesis, according to Conrad Waddington, it is how genotypes give rise to phenotypes during development [1].

Considering the role of plasticity genes, and the ways wherein composites of genes believed to code for openness to the environment (or may be disease) will assist to recognize other proposed mechanisms of improvement from a genetic perspective.

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Gene-environment Correlation

There are two factors responsible for biological variation - the nature of the organism and the nature of the conditions [2]. Darwin speaks to the possibility of genes and environment as being two components acting synergistic to outline our individual attributes.

In simplest form, gene-environment interaction means when two or more different genotypes respond to environmental variation.

In genetic epidemiology, gene-environment interactions are useful for understanding some diseases. Sometimes, sensitivity to environmental risk factors for a disease are inherited rather than the disease itself being inherited. Individuals with different genotypes are affected differently by exposure to the same environmental factors, and thus gene-environment interactions can result in different disease phenotypes [3].

In recent years, great progress in genotyping technology and cost control has enabled researchers to perform large-scale association studies, involving thousands of individuals genotyped on millions of markers. To date, Genome-Wide Association Studies (GWAS) have identified hundreds of genetic risk factors in complex diseases. However, the detected variants explain only a small part of the total heritability. Unexplained phenotypic variance may be partly attributed to undetected Gene-Environment (G×E) interactions [4].

It is a great deal of importance to know about these elements, genes and environment and their relationship.

The complex interplay of Gene and Environment: [5]

1. Virtually all-human diseases result from the interaction of genetic susceptibility factors and modifiable environmental factors, broadly defined to include infectious, chemical, physical, nutritional,

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and behavioral factors.

This is maybe the most vital reality in understanding the part of genetics and surrounding in the advancement of a disease. Many individuals have a tendency to define the reason for infection either as genetic or external environment. Without a doubt, some uncommon disease, for example, Huntington disease, might be due to a lack of a gene; however contribution of these diseases to human part is very little.

2. Variations in genetic makeup are associated with almost all disease.

Even so-called single-gene disorders actually develop from the interaction of both genetic and environmental factors. For example, Phenylketonuria (PKU) results from a genetic variant that leads to deficient metabolism of the amino acid phenylalanine; in the presence of normal protein intake, phenylalanine accumulates and is neurotoxic. PKU occurs only when both the genetic variant (phenylalanine hydroxylase deficiency) and the environmental exposure (dietary phenylalanine) are present.

3. Genetic variations do not cause disease but rather influence a person's susceptibility to environmental factors.

We do not inherit a disease state. Instead, we inherit a set of a susceptibility factors to certain effects of environmental factors and therefore inherit a higher risk for certain diseases. This concept also explains why the same environmental factors differently affect individuals. For example, some health-conscious individuals with "acceptable" cholesterol levels suffer myocardial infarction at age 40. Other individuals seem immune to heart disease despite smoking, poor diet, and obesity. Genetic variations account, at least in part, for this difference in response to the same environmental factors.

These days, it is notable that a large portion of the multifactorial human characteristics and diseases, for example, asthma, diabetes, cardiovascular infections, depression, rheumatoid arthritis, and tumour, result from an intricate interaction of the individual hereditary and different environmental variables.

Majorly, after the completion of Human Genome Project, a new dimension has opened up to define genetic variation at DNA-sequence level, enabling us to deepen our understanding in gene-environment interaction.

Moreover, some interaction can be explained without any molecular analysis, like fair-skinned humans are more prone to skin cancer due to strong effect sunlight when compared with darker skin.

And, others can be explained as a reproducible effect of an environmental exposure on a susceptible individual, like, the flushing response seen after alcohol ingestion in individuals with low-activity polymorphisms in the aldehyde dehydrogenase gene [6].

Regarding the statistical association between genes and environment, it is vital to carry out the study of gene-environment interaction as it helps to: [7].

- Obtain a better estimate of the population-attributable risk for genetic and environmental risk factors by accounting for their joint interactions.
- Strengthen the associations between environmental factors and diseases by examining these factors in genetically susceptible individuals.
- Dissect disease mechanisms in humans by using information on susceptibility (and resistance) genes to focus on the biological pathways that are most relevant to that disease, and the environmental factors that are most relevant to the pathways.
- Determine which specific compounds in the complex mixtures of compounds that humans are exposed to (such as diet or air pollution) cause disease.
- Use the information on biological pathways to design new preventive and therapeutic strategies.
- Offer tailored preventive advice that is based on the knowledge that an individual carries susceptibility or resistance alleles.

Epidemiological studies to analyse gene-environment interaction need to be very precise as the risk factors can only be uncovered after detailed examinations of various factors, i.e. genes and environmental factors.

In the development of preventive medicine, till today cohort studies on gene-environment interactions [8,9] have shown assuring results. Preventive medicine for genomic factors cannot be created as the germ lines remain same throughout the life but it can definitely be established for environmental factors including lifestyle habits like diet and exercise.

Essential preventive action against a disease, in any case, by enhancing individual way of life propensities will always be the concentration of preventive medicine, even in the genomic era.

In preventive medicine: Health Check + Genetic Information = Personal Preventive Measures

Relevant Opinions

Epigenetic explains hereditable changes that shows various phenotypic changes without any alternation in nucleotide sequence. Nature vs Nurture is a very interesting key point in the field of public health. How environment affects genes? It is very popular in the field of medical showing little attention to dentistry.

Although numerous studies have given relevant proof to epigenetic mechanism, demonstrating intense relationship between gene-environment in medical as well as in dental field. Genetic and environmental factors play a key role in the process of dental development.

Recent studies have been reported that environmental factors may increase the attachment loss and alter the virulence of pathogens in periodontal tissues [10].

For example, studies evaluating the methylation pattern of cytokine genes may have relevance for inflammatory diseases in which the expression of some cytokines is altered, such as in periodontal disease [11].

In individuals with periodontal disease, IL-6 gene is hypo-methylated in tissues when compared to healthy individuals. It

suggests an overexpression of this gene in tissues in an abnormal condition.

Since, this IL-6 gene is a key cytokine involved in bone resorption, which has also been detected in high levels in individuals with severe periodontitis, it is speculated that persistent inflammation and bacterial infection may also cause DNA methylation, which inactivates suppressors of cytokine signalling and contributes to exaggerate cytokine signalling [12].

Another study, V. R. N. Pradeep Bhagavatula Naga [13] stated that environmental and genetic factors both influence the development of dental fluorosis. In this study, from a total sample of 514 children, whose genotype and phenotype information were taken, 192 were selected for the study after excluding the subjects with indeterminate phenotypes and with indefinitive fluorosis. Analytic evaluations of gene-environment interactions were obtained based upon a case-only design.

Although findings were not statistically significant, but it provides some evidence for interaction between fluoride intake and a few genetic markers. These included:

- Tuftelin 1 marker cTUFT1rs6587597
- Osteopontin marker cSPP1rs10516800, and
- Osteocalcin marker D1S3737
- Haplotype analyses incorporating information from two markers within the tuftelin 1 gene cTUFT1rs3828054
- cTUFT1rs6587597, were also suggestive of gene-environment interaction.

This study concluded that multiple genes like Tuftelin 1, Osteopontin and Collagen 1 Alpha2 showed a positive association between fluoride intake and in the aetiology of dental fluorosis and suggest that further investigation into the role of genetics in the pathogenesis of dental fluorosis would be beneficial.

Some studies from medical background also give a knowledgeable insight into the epigenetic mechanism.

For example, Kate Northstone et al, [14] conducted a study in Avon, UK which shows an association between prepubertal onset of smoking in fathers and increased body mass index in their future sons. In this, of 9886 questionnaires distributed to fathers to inquire about their smoking habits, 5451 responded positively. Children were then measured from the age of 7 until the age of 17 by the study team and the results were significant.

To support the concluded of the study, the available genotypes were examined that are linked to the phenotypic variability in BMI.

- Variant rs9939609 in the FTO gene, related to BMI was considered.
- SNP rs1051730 at CHRNA5-CHRNA3-CHRNB4 that has been shown to interact with smoking to influence (decrease) BMI.

An adiposity allele scores comprising 32 SNPs associated with BMI [5].

This study showered a ray of light on epigenetic mechanisms (including DNA methylation, histone modification and miRNAs modification) which play a vital role in regulating gene expression by transferring information from one generation to another without altering the structure of DNA, also known as transgenerational effects.

One another paper on animals by S. Radhika et al, [15] summarizes recent epigenetic advances using rodent and primate (both human and nonhuman) models during in utero development and contributing to adult diseases later in life.

The authors investigated the molecular mechanisms underlying fetal programming and observed that epigenetic modifications to the fetal and placental epigenome accompany these reprogramming events. Based on several lines of emerging data in human and nonhuman primates, it was believed that modified epigenetic signature and the histone code in particular underlies alterations in postnatal gene expression and metabolic pathways central to accurate functioning and maintenance of health. Because of the tissue lineage specificity of many of these modifications, nonhuman primates serve as an apt model system for the capacity to recapitulate human gene expression and regulation during development [14].

Why Research in Epigenetics is Important?

Though studies focused on epigenetics in dentistry are still in paucity, there is increasingly more evidence for the association between epigenetic modifications and periodontal diseases, as well as inflamed dental pulp cells.

Epigenetics has effectively advanced into improving health at many levels. Epigenetic investigation is progressively being utilized to produce individual biomarkers, for example, the differential diagnosis and early acknowledgment of cancer.

Epigenetic also forms offer an approach to reach the advancement of new sorts of epigenetic drug substances. Some of these substances coordinated against DNA and histone modifications are already being effectively used to treat particular diseases.

As a general rule, epigenetic information and their understanding require exceptionally handle with care attitude. It is completely conceivable that epigenetic information reflect data about a man's way of life. Epigenomic information ought to in this manner be translated and assessed with mind so as to anticipate slander. Epigenetics and its concepts should be ranked higher in position when speaking on human lifestyle. It is imperative to watch out for the basic establishments of epigenetic information and the hypotheses derived from them.

As epigenetic mechanisms for human disease are identified and treatments are being developed or discovered. A few medications are utilized particularly due to their known effects for epigenome. Epigenetics is at the core of phenotypic variation in health and disease, it appears to be likely that understanding epigenome holds huge guarantees for preventing and treating diseases.

Epigenetics also offers a critical window to understand the role of the environment's interactions with genome in causing disease, which can help us to modulate those interactions to improve human health. Future researches are required to check out already done studies *via* which better understanding of the effect of epigenetics on dental health, and development of preventive medicines can be

approached for the better public health.

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