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Epigenetics In Action: How Environment and Lifestyle Change the Genome

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Abstract: Beyond genetics, overall human health is an integration of multiple environmental signals that begin at conception and act through epigenetic modifications. Environmental factors that influence epigenetics include behavior, nutrition, and chemicals and industrial pollutants. This review examines the impact of the environment on the epigenome, health, and disease, with a particular focus on cancer and neurodegenerative diseases.

Keywords: Epigenetics, DNA methylation, histone modifications, RNA methylation, environment, lifestyle, neurodegenerative diseases, cancer, toxins, nutrition.

Introduction: In today's world, with the increasing life expectancy of the population, neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD) are becoming a serious socio-economic burden. Their increasing prevalence and lack of effective treatment pose a challenge for future generations. Although various cellular mechanisms and genes involved in their occurrence and development have been studied, the exact molecular basis of these diseases remains unclear.

Epigenetics is the study of heritable changes in gene function that affect their expression and subsequent protein expression levels without altering the DNA sequence itself. Key epigenetic mechanisms include DNA methylation [4], histone modifications, non-coding RNAs, chromatin structure, and RNA methylation.

Environmental epigenetics studies how environmental factors influence cellular epigenetics and, as a result, human health. Epigenetic marks alter the spatial

conformation of chromatin, regulating gene expression without changing the DNA sequence.

Epigenetic mechanisms are actively involved in the development of the organism, starting in the prenatal period. Environmental influences at this stage can disrupt the epigenome of the developing organism, changing the risk of diseases later in life. On the other hand, bioactive components of food can induce protective epigenetic modifications throughout life, with nutrition at an early stage being of particular importance.

There is growing evidence that environmental neurotoxicants contribute to various forms of neurodegenerative and neurological diseases by inducing epigenetic changes and disrupting the epigenome. These pollutants include metals, pesticides, solvents, and other substances. Chemicals can regulate gene expression by affecting mRNA transcription, degradation, and translation. Abnormal changes in DNA or RNA methylation, non-coding RNA, and histone modifications may serve as biomarkers of neurotoxicity caused by environmental pollutants.

Research Methods

This article uses a literature review as the main research method. Scientific publications devoted to epigenetic mechanisms, their relationship with environmental factors and lifestyle, as well as their role in the development of oncological and neurodegenerative diseases are analyzed. Particular attention was paid to studies revealing the influence of DNA methylation, histone modifications and RNA methylation on gene expression in response to external stimuli. Data from both clinical trials and experiments on animal models, as well as on cell lines, were studied.

Results

Mechanisms of Epigenetics. DNA methylation is an epigenetic mechanism that involves the transfer of a methyl group to the C-5 position of cytosine, forming 5-methylcytosine (5mC). Methyl groups are added to DNA-by-DNA methyltransferase enzymes (DNMTs).

DNA methylation tends to act on promoters, causing gene silencing, while histone acetylation generally unwinds chromatin. RNA methylation – in addition to DNA modifications, various modifications of the nucleosides that make up the backbone of RNA (tRNA, rRNA, mRNA, etc.) have also been evaluated. N6-methyladenosine (m6A) in mRNA is the most studied mRNA modification, playing a role in modulating the processing and regulation of mRNA transcripts. Histone modifications – the histone proteins around which DNA is wrapped can be modified with various chemical groups (acetylation, methylation, phosphorylation, etc.). These modifications alter the spatial conformation of chromatin, either compacting it (preventing the binding of transcription factors) or opening it (allowing binding and activating cellular processes). In the course of studies using modern molecular genetic methods, the following vital functions of telomeres in the cell cycle were established: Mechanical: telomeres participate in the fixation of chromosomes to the nuclear matrix. At the zygotene stage of meiotic prophase, directed movements of the ends of chromosomes on the surface of the nuclear membrane occur so that the ends of homologous chromosomes close, and pairing (conjugation) of these chromosomes in strictly homogeneous sections begins from them. Stabilization: if a cell has telomerase activity, this provides an additional way to stabilize damaged chromosome ends. When a chromosome breaks accidentally, fragments without telomeric repeats at the ends appear. In the presence of telomerase, telomeric DNA joins the break sites, which stabilizes the fragments and allows them to function. Effect on gene expression: the activity of genes located near telomeres is reduced (repressed). This effect is often referred to as transcriptional silencing. With significant shortening of telomeres, the position effect disappears and the telomere genes are activated. "Counting" function: telomeric sections of DNA act as a clock device (the so-called replicameter), which counts the number of cell divisions after the disappearance of telomerase activity.

Environmental epigenetics and its impact. Active or repressive epigenetic marks depend on lifestyle and environmental factors. Environmental epigenetics studies how environmental exposures influence these epigenetic changes. Life experiences, habits, and environment shape us through their impact on our epigenome and health. For example, identical twins, although sharing the same genome, exhibit unique differences that result from differential gene expression under the influence of epigenetic factors.

Nutrition is one of the most studied and best understood epigenetic environmental factors. Adverse nutritional conditions during the prenatal period are associated with increased risk of disease later in life. For example, a birth cohort during the Dutch famine of 1944-45 showed that starvation during pregnancy led to an increased risk of type II diabetes, cardiovascular disease, metabolic disorders, and cognitive decline. Consistent epigenetic differences have been attributed

to lower methylation of a gene involved in insulin metabolism. Nutrients can act directly by inhibiting epigenetic enzymes (DNMTs, HDACs, or HATs) or by altering the availability of substrates required for these enzymatic reactions, influencing the expression of critical genes and our overall health.

Exposure to toxins and pollutants such as tobacco smoke, particulate matter, diesel exhaust, and ozone also have significant epigenetic effects.

* Tobacco smoke: causes DNA damage, oxidative stress, and inflammatory responses. Associated with hypermethylation of certain genes in animal models of lung cancer [Mathers et al., 2010]. In lung cancer cells, cigarette smoke extract induced aberrant expression of the synuclein gamma oncogene (SNCG) through demethylation of the promoter CpG island and inhibition of DNMT3B expression [Liu et al., 2007]. Smoking can also affect histone function, for example through downregulation of the Dickkopf-1 gene in lung cancer [Hussain et al., 2009]. Prenatal exposure to tobacco smoke is associated with gene-specific differences in DNA methylation patterns, which may lead to lifelong effects [2009].

* Particulate matter and other pollutants: Can alter the epigenetic landscape, affecting gene expression and contributing to disease development.

Epigenetic mechanisms in oncology. Cancer is a genetic and epigenetic disease. Mutations in genes that maintain tissue homeostasis, control the cell cycle, or regulate apoptosis are the genetic basis of cancer. However, cancer is also characterized by mutations in chromatin-remodeling enzymes and epigenome alterations resulting from abnormal attachment or removal of DNA marks or histone proteins. Accumulating evidence suggests that many adult diseases, including cancer, have an epigenetic origin. The increase in cancer incidence observed in developed countries since the 1950s may be due in part to exposure to endocrine disrupting chemicals (EDCs).

Epigenetic mechanisms in neurodegenerative diseases. Epigenetic dysregulation can cause the development of neurological disorders such as Parkinson's disease, Huntington's disease, and mood disorders (including depression and anxiety). Environmental neurotoxicants that because epigenetic changes play a key role in the development of neurodegenerative and neurological diseases.

Discussion

Human health is the result of a complex interaction between genetics and multiple environmental factors. Lifestyle factors, including diet, behavior, and exposure to toxins, play a key role in shaping our epigenome and, as a result, our health. This is especially evident in identical twins, whose phenotypic differences, despite having identical genomes, are due to epigenetic modifications.

Particular attention should be paid to diet as a powerful epigenetic factor. Not only individual nutrients, but also overall dietary patterns, such as the Western diet, can significantly influence the risk of developing chronic diseases, including cancer. These changes can occur early in development, shaping the epigenome and having long-term effects throughout life. Importantly, methyl-donor nutrients such as folate directly affect DNA methylation, and their deficiency can lead to hypomethylation of parts of the genome.

Chronic exposure to environmental toxins and pollutants such as tobacco smoke and industrial chemicals is a significant contributor to the increasing incidence of cancer and neurodegenerative disorders, with certain periods of development such as the prenatal period being particularly vulnerable. Understanding the effects of toxin combinations, doses, and critical windows of exposure is complex but critical.

The reversibility of epigenetic modifications opens up new perspectives in medicine. The development of epigenetic drugs that affect the enzymes that regulate these processes is a promising direction, especially in the field of oncology. Drug resistance, which in some cases is explained by epigenetic mechanisms, can also be overcome using targeted epigenetic therapy. This highlights the potential of epigenetics not only in understanding diseases, but also in developing innovative treatment strategies.

Conclusion

Epigenetics gives us a deep understanding of how our environment and lifestyle shape our genome, influencing our health and susceptibility to disease. Human health is the result of complex interactions between our genetics and multiple environmental factors. Epigenetic-driven changes in gene expression are shaped by our life experiences and habits, including diet, behavior, and exposure to environmental toxins.

We found that many environmental factors, including maternal diet and chemical pollution, can cause epigenetic changes as early as conception or later in fetal life. Chronic environmental exposures, in

particular, partially explain elevated rates of all types of cancer, affecting both those directly exposed and the developing fetus.

Although epigenetic marks are considered heritable, transgenerational epigenetic inheritance highlights the complexity of this process. Some of our traits, behaviors, diseases, and positive and negative life experiences leave epigenetic marks that can be inherited. However, these changes can be removed or modified by lifestyle changes. This opens up opportunities for preventive measures and the development of new therapeutic strategies based on the correction of epigenetic disorders.

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