BG Pulse Journal USA
ISSN:1947-2536 http://www.bgpulseusa.com/index.php/bg

THE IMPACT OF ENVIRONMENTAL FACTORS AND LIFESTYLE CHOICES ON OUR GENES

Roziqova Dilbar Abdurazzoqovna

Teacher of the “General.Sciences” department of the

Asia International University, Bukhara, Uzbekistan,

e-mail:roziqovadilbar@oxu.uz

Abstract: For decades, classical genetics posited that human phenotype and health outcomes were strictly determined by the inherited DNA sequence. However, emerging research in epigenetics demonstrates that environmental factors and lifestyle choices dynamically regulate gene expression without altering the primary genetic code.

Keywords: Epigenetics, DNA methylation, homeostasis, gene expression, lifestyle, chromatin, phenotype.

Introduction: For a long time, it was believed that our genetic destiny was predetermined at birth. Traditional biology suggested that human health, behavior, and disease risks were dictated solely by the rigid DNA sequence inherited from our parents, forming an unalterable phenotype. However, modern molecular biology has completely overturned this rigid view. Today, we know that our lifestyle, environment, and daily choices directly orchestrate how our genes behave without modifying the underlying genetic code

This dynamic layer of control is studied by the field of **epigenetics**. If the DNA sequence is compared to the computer hardware, epigenetic mechanisms act as the software that dictates which programs run (**gene expression**). To use another analogy, if DNA is a grand musical score, the environment and lifestyle serve as the orchestra conductor determining how the piece is played. Ultimately, these chemical adjustments allow our cells to adapt and maintain a stable internal balance known as **homeostasis** under changing external conditions.

The Blueprint of Lifestyle: Diet and Exercise

The Epigenetic Toll of Nutrition. Every molecule we consume acts as a biochemical command at the cellular level, influencing the structural state of chromatin—the complex of DNA and packaging proteins. The Power of a Healthy Diet: Natural, whole foods are rich in "epi-bioactive" compounds and DNA methylation modulators. These molecules attach chemical tags to DNA, effectively silencing harmful genes and awakening protective pathways. *Leafy Greens (Spinach, Broccoli, Kale):* High in folate (Vitamin B9), which serves as a crucial methyl donor. Folate stabilizes the DNA strand, prevents oncogenic mutations, and fuels DNA repair mechanisms. *Cruciferous Vegetables (Broccoli, Cauliflower):* Contain sulforafan, a compound that awakens *tumor suppressor* genes to arrest aberrant cell growth. *Citrus Fruits and Berries:* Packed with polifenols and Vitamin C, which structurally quiet pro-inflammatory genes, lowering the baseline risk for atherosclerosis and Type 2 diabetes.

Fatty Fish: Enhance the expression of neuroprotective genes in brain tissues, offering a genetic buffer against cognitive decline and depression.

The Structural Harm of Malnutrition**:** Conversely, a diet saturated with trans-fats, refined industrial sugars, fast food, and heavy preservatives induces systemic epigenetic stress.

Excessive Industrial Sugar: Triggers the chronic activation of the *NF-kB* genetic pathway. This sustained inflammation erodes insulin sensitivity and unlocks metabolic syndrome genes.

Trans-Fats and Fast Food: Over-activate genes regulating fat storage. Crucially, they imprint a stubborn "epigenetic memory" on cells, causing the body to resist weight loss even after entering a caloric deficit. *Pestitsids and Additives:* Can prematurely strip away protective methyl markers from DNA, uncoiling chromatin and exposing genes associated with accelerated aging

Epigenetic Memory and the In Utero Environment

The susceptibility of the epigenome to nutritional signals is never higher than during prenatal development. Under the Developmental Origins of Health and Disease (DOHaD) hypothesis, maternal malnutrition or metabolic stress forces the fetus to adapt to a perceived "hostile, nutrient-scarce" future. The fetal genome responds by epigenetically lock-programming its metabolism to maximize fat retention. Even if that individual maintains a highly conscious, healthy diet in adult life, their baseline genetic programming remains inherently biased toward obesity and cardiovascular pathology.

Congenital Heart Defects (CHD) and Environmental: Vulnerability Congenital Heart Defects (CHD) represent structural, anatomical abnormalities of the heart and its primary macro-vessels that manifest during the critical window of embryonic development (weeks 2 through 8 of gestation). Epigenetic and genetic disruptions during this vulnerable period severely compromise the precise orchestration required for cardiac morphogenesis.

Acyanotic (White) Defects: These present with normal skin perfusion because oxygenated blood continues to circulate somewhat effectively. Standard variants include atrial or ventricular septal defects (holes in the cardiac walls) and patent ductus arteriosus.

Cyanotic (Blue) Defects: These represent severe anatomical failures where deoxygenated venous blood mixes into the systemic arterial circulation. This induces systemic hypoxia, causing a distinct blue tint (cyanosis) on the lips and extremities. Prime examples include the Tetralogy of Fallot and the transposition of the great arteries.

Multifactorial Etiology: Nearly 90% of CHD cases are multifactorial, requiring both a baseline genetic susceptibility and an environmental trigger. High-risk triggers include maternal viral infections (rubella, influenza) during the first trimester, unmanaged maternal diabetes, maternal tobacco or alcohol consumption, and direct exposure to industrial radiational or chemical toxins during early embryogenesis.

Physical Activity, Chronic Stress, and Sleep: Physical Activity: Regular exercise dynamically remodels the epigenetic profile of muscle and neural tissues. High-intensity and hybrid training modes trigger chromatin remodeling that optimizes insulin sensitivity, downregulates systemic inflammation, and delays cellular senescence.

Chronic Stress and Sleep: Perpetual psychological distress floods the system with cortisol. This hormonal deluge introduces harmful methylation patterns that suppress immune response

genes and upregulate markers linked to clinical depression. Conversely, deep, restorative sleep acts as an epigenetic reset, initiating DNA repair, restoring cellular integrity, and stabilizing systemic.

Atmospheric Pollution, Heavy Metals, and Oncogenesis: The inhalation or ingestion of particulate matter, heavy metals, and industrial xenobiotics leaves a destructive trail of epigenetic scars on human DNA, sharply escalating the incidence of respiratory diseases and malignancies. Urban smog, industrial emissions, and secondhand tobacco smoke are highly concentrated with PM2.5 fine particulates and polycyclic aromatic hydrocarbons (PAHs).

Asthma and Hyper-reactivity: These fine particulates penetrate deep into pulmonary tissue, causing local oxidative stress that alters DNA methylation patterns in immune cells. This aberrantly trains the immune system to deploy an aggressive, chronic inflammatory response to harmless stimuli like cold air or pollen, cementing chronic asthmatic phenotypes.

Breaking the Pulmonary Defense: Toxic inhalants silence the local detoxification genes meant to neutralize ksenobiotiks. Furthermore, heavy metals such as cadmium, arsenic, and lead act as aggressive epigenetic disruptors. They enforce hyper-methylation on the promoter regions of vital *tumor suppressor* genes (such as *p53*), rendering them silent. Stripped of these molecular guardians, cells undergo rapid, unchecked replication, paving the way for aggressive pulmonary and systemic.

Conclusion

Our genetic code is a flexible framework, not an unyielding prison sentence. While we cannot change the inherited base pairs of our DNA, our everyday habits—the food we eat, the air we protect ourselves from, and how we manage stress—have the absolute power to dictate our physical fenotip. Environmental vigilance and conscious living are our most powerful tools to silence disease and optimize longevity at the deepest molecular level.

References

1. Alegría-Torres, J. A., Baccarelli, A., & Bollati, V. (2011). *Epigenetics and lifestyle.* Epigenomics, 3(3), 267-277.
2. Baccarelli, A., & Bollati, V. (2009). *Epigenetics and environmental chemicals.* Current Opinion in Pediatrics, 21(2), 243-251.
3. Godfrey, K. M., et al. (2011). *Epigenetic mechanisms mediating the long-term effects of maternal nutrition on child development.* Frontiers in Genetics, 2, 42.
4. Hoyo, C., et al. (2012). *Prerequisition of DNA methylation patterns in early development and susceptibility to environmental exposures.* Environmental Health Perspectives, 120(7).
5. Lim, U., & Song, M. A. (2012). *Dietary and lifestyle factors of DNA methylation.* Methods in Molecular Biology, 863, 359-376.
6. Nieto-Pellegrino, E., et al. (2022). *Epigenetic Effects of Healthy Foods and Lifestyle Habits.* Nutrients, 14(19), 3953