

How the environment influences human biology

Posted on September 21, 2018

View this essay online:

<https://academic-master.com/how-the-environment-influences-human-biology/>

Introduction

Human biology develops through continuous interaction between inherited characteristics and the environments in which people live. Genes provide biological instructions, but they do not operate independently of nutrition, infection, chemical exposure, stress, physical activity, climate, social conditions, and developmental experience. Environmental influences can act directly by damaging tissue, altering hormones, changing immune responses, or modifying behavior, and they can also affect how genes are regulated. Epigenetics is one important part of this interaction. The National Human Genome Research Institute explains that epigenomic marks help determine when genes are turned on or off and can change in response to environmental exposures or disease (National Human Genome Research Institute [NHGRI], n.d.). These mechanisms include DNA methylation and chemical modifications to DNA-associated proteins. Environmental biology should not be reduced to epigenetics, however, because health also changes through infection, inflammation, injury, social deprivation, and access to care. Human outcomes emerge from the combined effects of genes, exposure, timing, dose, tissue, behavior, and social structure.

Genes, Epigenetics, and the Exposome

The concept of gene-environment interaction explains why genetic variation does not create a fixed biological destiny. A person may inherit susceptibility to a disease without inevitably developing it, while people with different genetic backgrounds may respond differently to the same exposure. Smoking, nutrition, medication, occupation, infection, and physical activity can therefore modify the probability that an inherited vulnerability becomes clinically important. Epigenetic research provides one mechanism for this interaction because environmental exposures may alter patterns of gene regulation without changing the underlying DNA sequence. Reviews of environmental epigenetics describe associations between air pollution, metals, tobacco smoke, nutrition, and altered DNA methylation, although researchers must distinguish causal effects from consequences of disease, tissue differences, and confounding variables (Bollati & Baccarelli, 2010; Martin & Fry, 2018). The broader exposome concept extends this reasoning by examining the totality of exposures across life, including pollution, diet, occupation, stress, housing, climate, and internal biological responses. People rarely encounter one factor in isolation, so understanding human biology requires studying

mixtures and cumulative exposure.

Nutrition, Pollution, and Chemical Exposure

Nutrition, air quality, and chemical exposure illustrate how the environment can influence development and disease through several pathways at once. Nutrients provide energy and materials for growth, immune function, hormones, and tissue repair, while deficiencies or chronic excess can alter metabolism and disease risk. Some nutrients also participate in biochemical reactions involved in epigenetic regulation, but it is misleading to claim that one food simply “switches on” desirable genes. Air pollution affects the lungs, cardiovascular system, and other organs through inflammation, oxidative stress, and vascular effects. WHO’s 2026 technical brief identifies air pollution as a leading environmental health risk and summarizes evidence of effects on respiratory, cardiovascular, cerebrovascular, reproductive, and metabolic systems (World Health Organization [WHO], 2026). Environmental chemicals can also interfere with hormonal signaling or damage cells depending on hazard, dose, route, timing, and duration. Studies of prenatal exposure show why developmental timing matters: the same exposure can have different consequences when organs and regulatory systems are forming rapidly (Perera & Herbstman, 2011).

Stress, Social Conditions, and Biological Embodiment

Stress and the social environment are also biological influences. Acute stress responses can be adaptive because hormones such as adrenaline and cortisol mobilize energy and increase alertness, but repeated or prolonged stress can contribute to sleep disturbance, cardiovascular strain, metabolic change, anxiety, depression, and altered immune regulation. Social conditions shape who experiences these exposures. Income, housing quality, discrimination, neighborhood safety, occupation, family relationships, and access to healthcare can change contact with pollution, crowding, unsafe work, heat, poor nutrition, or chronic uncertainty. Hou and colleagues (2012) emphasized that environmental chemical exposures can interact with epigenetic processes, but molecular evidence should not distract from the social systems that distribute exposure unequally. A child living near heavy traffic, for example, may experience air pollution, noise, heat, limited green space, and socioeconomic stress simultaneously. These pathways demonstrate how social inequality can become biologically embodied without implying that individuals or families are responsible for the hazards imposed on them. Prevention must therefore address both molecular mechanisms and the conditions that create exposure.

Everyday Environmental Regulation of Physiology

Light, temperature, microbes, and physical activity demonstrate that the environment regulates ordinary physiology as well as disease risk. Light reaching specialized retinal cells helps synchronize circadian rhythms that influence sleep, hormone release, body temperature, appetite, and alertness. Irregular schedules, night work, or bright light late at night can disrupt these rhythms without changing a person's DNA sequence. Temperature triggers thermoregulatory responses such as sweating, skin blood-flow changes, and shivering; repeated heat exposure can produce acclimatization, while extreme temperatures can overwhelm normal compensation. Microorganisms interact continuously with humans, and the microbiome contributes to digestion and immune development, although not every difference in microbial composition is harmful or requires treatment. Physical activity likewise changes muscle, bone, metabolism, cardiovascular function, and mood, while prolonged inactivity produces deconditioning. These examples show why environmental influence should not be equated solely with toxic exposure. Human biology is inherently responsive to daily patterns of light, movement, temperature, microorganisms, food, and social experience throughout the lifespan.

Hemophilia and the Limits of Environmental Explanation

Hemophilia provides an important example of what should not be described as an environmentally transmitted condition. The Centers for Disease Control and Prevention explains that hemophilia A and B are usually inherited bleeding disorders caused by low levels of clotting factor VIII or factor IX, respectively, because of genetic variants affecting the relevant genes (Centers for Disease Control and Prevention [CDC], 2024). The genes are located on the X chromosome, which helps explain the characteristic inheritance pattern. Environmental circumstances can influence injury risk, access to specialist treatment, and health outcomes, but they do not ordinarily create the inherited variant. This distinction separates genetic inheritance from infection, exposure, and epigenetic regulation. It also shows why claims about transgenerational environmental effects require caution. Some epigenetic states can persist as cells divide, and animal studies provide evidence that certain exposure effects may extend across generations, but human inheritance is difficult to establish because descendants often share genes, environments, culture, and social conditions. Direct prenatal exposure must be distinguished from effects in generations that were never biologically exposed.

Conclusion

The environment influences human biology through many connected pathways, including nutrition, pollution, chemicals, stress, social conditions, microbes, light, temperature, physical activity, injury, and healthcare access. Epigenetic mechanisms help explain how some exposures influence gene

regulation without changing DNA sequence, but they are only one part of a much broader biological response (NHGRI, n.d.; Martin & Fry, 2018). Evidence from environmental health also shows that risks are distributed unevenly because infrastructure, work, housing, and policy determine who encounters hazardous conditions. At the same time, biological plasticity means that many environmental effects are not fixed forever. Cleaner air, better nutrition, smoking cessation, physical activity, safer workplaces, improved sleep, treatment, and stronger social conditions can reduce risk or improve function. Scientific understanding should therefore support prevention rather than fatalism. The practical goal is to create environments that reduce avoidable exposure and expand healthy development while communicating uncertainty carefully and avoiding claims that molecular findings prove destiny. Human biology is responsive, but responsiveness does not mean inevitability.

Works Cited

Bollati, V., & Baccarelli, A. (2010). Environmental epigenetics. *Heredity*, *105*, 105–112. <https://doi.org/10.1038/hdy.2010.2>

Centers for Disease Control and Prevention. (2024). *How Hemophilia Is Inherited*. <https://www.cdc.gov/hemophilia/testing/how-hemophilia-is-inherited.html>

Hou, L., Zhang, X., Wang, D., & Baccarelli, A. (2012). Environmental chemical exposures and human epigenetics. *International Journal of Epidemiology*, *41*(1), 79–105. <https://doi.org/10.1093/ije/dyr154>

Martin, E. M., & Fry, R. C. (2018). Environmental influences on the epigenome: Exposure-associated DNA methylation in human populations. *Annual Review of Public Health*, *39*, 309–333. <https://doi.org/10.1146/annurev-publhealth-040617-014629>

National Human Genome Research Institute. (n.d.). *Epigenomics Fact Sheet*. <https://www.genome.gov/about-genomics/fact-sheets/Epigenomics-Fact-Sheet>

Perera, F., & Herbstman, J. (2011). Prenatal environmental exposures, epigenetics, and disease. *Reproductive Toxicology*, *31*(3), 363–373. <https://doi.org/10.1016/j.reprotox.2010.12.055>

World Health Organization. (2026). *Health effects of air pollution: evidence and implications*. <https://www.who.int/publications/i/item/B09563>