

How the environment influences human biology

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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, stress, chemical exposure, physical activity, climate, social conditions, and developmental experience. Environmental influences can affect physiology directly, alter patterns of gene expression, shape the immune and nervous systems, and change the probability that a person will develop particular diseases. Epigenetics is one important part of this interaction. It studies molecular processes that regulate gene activity without changing the underlying DNA sequence, including DNA methylation, histone modification, chromatin organization, and regulatory RNA. These processes help cells maintain their identity and respond to changing conditions. However, environmental influence should not be reduced entirely to epigenetics. The environment also affects health through injury, infection, behavior, endocrine signaling, inflammation, and unequal access to food, clean air, housing, and healthcare.

Genes Are Not a Fixed Biological Destiny

Every person inherits genetic variants from biological parents, but the effects of those variants often depend on context. A gene may influence how an enzyme works, how a receptor responds, or how a tissue develops, yet diet, exposure, age, and other genes can modify the outcome. This is commonly described as gene-environment interaction. For example, a person may inherit a susceptibility to a disease without inevitably developing it. The risk may increase or decrease according to smoking, nutrition, medication, infection, occupational exposure, or physical activity. Conversely, people with different genetic backgrounds may respond differently to the same environmental condition. Human biology is therefore neither purely genetic nor purely environmental. It is produced by interaction across the lifespan.

What Epigenetics Means

Epigenetic regulation changes how accessible or active particular genes are without rewriting the sequence of DNA bases. DNA methylation can influence whether transcription machinery reaches a region of the genome. Histone proteins, around which DNA is organized, can be chemically modified in ways that affect chromatin structure and gene activity. Non-coding RNAs can regulate the production or stability of other RNA molecules. These mechanisms are essential to normal development. A liver cell and a neuron contain almost the same DNA, yet they express different sets

of genes because their regulatory programs differ. Environmental exposures may influence these programs, particularly during sensitive periods such as prenatal development, infancy, puberty, and aging. Nevertheless, an observed epigenetic difference does not automatically prove that an exposure caused a disease. Researchers must distinguish cause, consequence, correlation, tissue differences, and confounding factors. (Bollati & Baccarelli, 2010; Martin & Fry, 2018; National Human Genome Research Institute, n.d.)

The Exposome

The term *exposome* refers to the totality of environmental exposures experienced throughout life and the biological responses they produce. It includes external exposures such as air pollution, water quality, occupation, radiation, noise, climate, diet, housing, and social stress. It also includes internal processes such as inflammation, hormones, metabolism, and the microbiome. The exposome concept is useful because people are rarely exposed to one factor at a time. A child living near heavy traffic may experience air pollution, noise, heat, limited green space, and socioeconomic stress together. These exposures may interact rather than producing separate effects. Studying them requires epidemiology, molecular biology, geography, toxicology, and social science. The complexity makes causal conclusions difficult, but it more accurately reflects how human biology develops in real environments. (Hou et al., 2012; Perera & Herbstman, 2010)

Nutrition and Development

Nutrition supplies energy and the materials required for growth, tissue repair, hormones, immune function, and brain development. Severe undernutrition can impair physical growth and cognitive development, while excess energy intake and poor dietary quality can contribute to obesity, diabetes, cardiovascular disease, and some cancers. Nutrients also participate in biochemical pathways involved in epigenetic regulation. Folate, choline, methionine, and vitamins involved in one-carbon metabolism contribute to the availability of methyl groups used in cellular reactions. This does not mean that a particular food can simply “switch on” desirable genes or cure disease. Nutritional effects depend on dose, developmental timing, overall dietary pattern, genetics, health status, and social context. Prenatal and early-life nutrition are especially important because organs and regulatory systems are developing rapidly.

Air Pollution and Respiratory Biology

Air pollution affects human biology through the respiratory and cardiovascular systems. Fine particulate matter can reach deep areas of the lungs and contribute to oxidative stress, inflammation, vascular dysfunction, and worsening of respiratory disease. Pollutants such as nitrogen dioxide, ozone, smoke, and occupational dust can aggravate asthma or damage lung tissue. Research also

identifies associations between air pollution and changes in DNA methylation, epigenetic aging, and regulatory pathways, although mechanisms and clinical significance vary across studies. The World Health Organization identifies air pollution as a leading environmental health risk. The most effective response is not to place responsibility only on individuals but to improve energy, transport, industrial, housing, and urban policies that determine exposure. (World Health Organization, n.d.)

Chemicals and Endocrine Disruption

Humans encounter chemicals through food, water, air, consumer products, workplaces, and waste. Some substances are toxic at particular doses, while others can interfere with hormonal signaling. Endocrine-disrupting chemicals may imitate, block, or alter the production and metabolism of hormones. Because hormones regulate development, reproduction, metabolism, and behavior, exposure during sensitive periods may have lasting effects. Metals, pesticides, tobacco smoke constituents, and industrial chemicals have been studied for their influence on gene regulation and epigenetic patterns. However, evidence differs by substance and outcome. It is scientifically misleading to assume that every synthetic chemical is dangerous or every natural chemical is safe. Risk depends on hazard, dose, route, timing, duration, and susceptibility.

Stress and the Biological Stress Response

Stress is not merely a feeling; it involves coordinated activity across the nervous, endocrine, immune, and cardiovascular systems. In an immediate challenge, stress responses can be adaptive. Hormones such as adrenaline and cortisol help mobilize energy, increase alertness, and prepare the body to respond. Problems arise when stress is intense, repeated, or prolonged without adequate recovery. Chronic stress can contribute to sleep disturbance, hypertension, metabolic change, impaired immune regulation, anxiety, depression, and unhealthy coping behavior. Early adversity may influence the development of stress-regulation systems, and researchers have examined epigenetic pathways as possible mediators. Yet it would be too deterministic to claim that stress produces a fixed biological destiny or a precise reduction in life expectancy. Social support, treatment, safety, exercise, sleep, and improved material conditions can modify risk.

Social Environment and Human Biology

The environment includes social and economic conditions, not only physical surroundings. Income, discrimination, education, neighborhood safety, work, housing, family relationships, and access to healthcare influence exposure and biological response. A person living in poor housing may encounter mold, heat, crowding, and pests. An employee in insecure work may experience stress while also being exposed to hazardous substances. Structural discrimination can shape healthcare access, chronic stress, employment, and residential pollution. These social determinants become biologically

embodied through multiple pathways. Recognizing this process does not mean reducing injustice to molecular markers. Epigenetic evidence should not distract from the political and material conditions that produce unequal health.

Light, Circadian Rhythms, and Sleep

Light is an environmental signal that helps regulate circadian rhythms. Specialized cells in the retina send information to brain systems that coordinate sleep, hormone release, temperature, appetite, and daily patterns of alertness. Daylight supports synchronization with the external day, while bright light at night can delay sleep timing and suppress melatonin. Shift work, jet lag, and irregular schedules can disrupt circadian organization, affecting performance and potentially contributing to long-term health risk. The effect is regulatory rather than a rapid alteration of DNA sequence. Human biology evolved to respond to light-dark cycles, but modern artificial lighting and screen use can change the timing and intensity of exposure. Healthy sleep practices and appropriately designed workplaces can reduce disruption.

Temperature and Physiological Adaptation

Temperature affects human biology through thermoregulation. In heat, the body increases skin blood flow and sweating; in cold, it conserves heat and may increase shivering. Repeated heat exposure can produce acclimatization, including earlier sweating and improved cardiovascular stability. Extreme heat can overwhelm these mechanisms, particularly among older adults, infants, outdoor workers, and people with chronic disease. Cold exposure can contribute to hypothermia and cardiovascular strain. Across many generations, climate has also contributed to evolutionary adaptation, but evolutionary genetic change should not be confused with short-term physiological adjustment. Individual bodies acclimatize during life, while population-level genetic evolution occurs through changes in variant frequencies across generations.

Ultraviolet Radiation and Skin Biology

Sunlight illustrates both beneficial and harmful environmental influence. Ultraviolet B radiation contributes to vitamin D synthesis in the skin, while excessive ultraviolet exposure damages DNA and increases skin-cancer risk. Melanin helps protect tissue by absorbing radiation, and human variation in pigmentation reflects a long evolutionary history involving ultraviolet exposure, vitamin D, and folate protection. A person's skin may tan through increased melanin production after exposure, but this is not the same as an inherited evolutionary change. Sun protection recommendations should consider skin type, location, occupation, and medical needs. The broader lesson is that environmental factors rarely produce only beneficial or only harmful effects; dose and context matter.

Microbes, Immunity, and the Human Microbiome

Humans live in constant interaction with microorganisms. Some cause infection, while many form communities on the skin and in the mouth, intestine, and other body sites. The microbiome participates in digestion, immune development, and protection against pathogens. Diet, antibiotics, infection, birth conditions, geography, and lifestyle can alter microbial communities. Changes in the microbiome may influence inflammation and metabolism, but causal interpretation is still developing. It is inappropriate to describe all microbes as harmful or to assume that every difference in microbiome composition requires treatment. Environmental sanitation, vaccination, food safety, and responsible antibiotic use remain important, while excessive or unnecessary antimicrobial exposure can create other risks.

Physical Activity and Mechanical Environment

Muscles, bones, the cardiovascular system, and metabolism respond to use. Regular physical activity can improve insulin sensitivity, cardiovascular function, bone strength, mood, and functional ability. Conversely, prolonged inactivity can contribute to deconditioning. Bone tissue adapts partly to mechanical load, which is why weight-bearing activity is important during growth and aging. Occupational environments can also impose harmful loads through repetitive movement, vibration, heavy lifting, or poor ergonomics. These examples show that biological systems are responsive. Environmental influence is not always a chemical entering the body; it can be the pattern of movement, rest, and mechanical demand imposed by daily life.

Injury, Infection, and Direct Environmental Effects

Not every environmental effect requires altered gene expression. Trauma can damage tissue directly. Infectious organisms can invade cells or provoke harmful immune responses. Loud noise can damage sensory structures in the ear. Radiation can injure DNA. Poor water and sanitation can increase exposure to pathogens. These direct pathways are important because an excessive focus on epigenetics can make simple preventable hazards appear unnecessarily mysterious. Human biology responds through inflammation, healing, immune memory, and physiological compensation. Environmental health therefore includes reducing exposure, improving infrastructure, and providing timely medical care, not only measuring molecular changes.

Clarifying Genetic Disorders such as Hemophilia

The original discussion used hemophilia as an example of an environmentally transferred condition, but hemophilia is primarily an inherited bleeding disorder caused by variants affecting clotting factors, most commonly factor VIII or factor IX. It is not transmitted through contact, and an affected

parent does not necessarily produce a child with a more severe form. Inheritance depends on the specific type, genetic variant, and sex-chromosome pattern. Modern treatment may include replacement clotting factors, non-factor therapies, or other specialist care; continuous blood replacement is not an accurate general description. Environmental conditions can influence access to treatment, injury risk, and health outcomes, but they do not create the inherited variant in the ordinary case. This distinction illustrates why genetic inheritance, infection, exposure, and epigenetic regulation should not be conflated.

Can Environmental Effects Be Inherited?

Some epigenetic states are maintained as cells divide, which helps tissues preserve identity. Whether environmentally induced changes are inherited across human generations is a more difficult question. During reproduction and early development, much epigenetic information is reprogrammed. Animal studies provide evidence that certain exposure effects can persist across generations, but demonstrating true transgenerational inheritance in humans is challenging because descendants often share genes, environments, culture, and social conditions. Researchers therefore distinguish direct prenatal exposure from effects in generations that were not biologically exposed. Claims that trauma, diet, or pollution permanently rewrites the biology of all future descendants should be treated cautiously. The field is important, but uncertainty remains.

Reversibility and Biological Plasticity

Environmental effects are not uniformly permanent. Some injuries are irreversible, while many physiological and regulatory changes can improve when conditions change. Smoking cessation reduces future disease risk. Improved nutrition can correct deficiencies. Exercise changes metabolism and muscle function. Treatment can reduce stress symptoms and improve sleep. Some epigenetic marks are stable, but others are dynamic and tissue-specific. Biological plasticity is especially strong during development yet continues throughout life. This is an important ethical point: environmental biology should not be used to label people as permanently damaged. Understanding mechanisms should support prevention, treatment, and healthier environments rather than fatalism or stigma.

Research Limitations and Ethical Interpretation

Environmental epigenetic research faces several limitations. Human studies often measure exposures imperfectly, examine accessible tissues such as blood rather than the organ of interest, and identify associations after disease has already developed. Age, medication, diet, smoking, cell composition, and socioeconomic conditions can confound results. Large datasets also increase the risk of finding statistically significant patterns with uncertain biological importance. Researchers need replication, longitudinal designs, mechanistic studies, and careful causal methods. Ethical communication is

equally important. Molecular findings should not be used to blame parents, communities, or individuals for exposure-related illness, particularly when hazards arise from poverty, discrimination, occupation, or industrial policy.

Conclusion

The environment influences human biology through many connected pathways. Nutrition, pollution, chemicals, microbes, stress, light, temperature, physical activity, social conditions, injury, and healthcare access affect development and disease risk. Epigenetic mechanisms provide one explanation for how exposures can influence gene regulation without changing DNA sequence, but they are not the only mechanism and should not be interpreted deterministically. Human outcomes emerge from interactions among genes, timing, dose, tissue, behavior, and social structure. The most important implication is practical: healthier environments can prevent disease and expand biological opportunity. Scientific understanding should therefore guide cleaner air and water, safer workplaces, equitable social conditions, appropriate nutrition, and evidence-based healthcare while maintaining caution about claims that exceed the available evidence.

Works Cited

Bollati, Valentina, and Andrea Baccarelli. "Environmental Epigenetics." *Heredity*, vol. 105, 2010, pp. 105–112. <https://doi.org/10.1038/hdy.2010.2>.

Hou, Lifang, Xiao Zhang, Dong Wang, and Andrea Baccarelli. "Environmental Chemical Exposures and Human Epigenetics." *International Journal of Epidemiology*, vol. 41, no. 1, 2012, pp. 79–105. <https://doi.org/10.1093/ije/dyr154>.

Martin, E. M., and R. C. Fry. "Environmental Influences on the Epigenome: Exposure-Associated DNA Methylation in Human Populations." *Annual Review of Public Health*, vol. 39, 2018, pp. 309–333. <https://doi.org/10.1146/annurev-publhealth-040617-014629>.

National Human Genome Research Institute. "Epigenomics Fact Sheet." *Genome.gov*, National Institutes of Health.

Perera, Frederica, and Julie Herbstman. "Prenatal Environmental Exposures, Epigenetics, and Disease." *Reproductive Toxicology*, vol. 31, no. 3, 2011, pp. 363–373. <https://doi.org/10.1016/j.reprotox.2010.12.055>.

World Health Organization. "Environmental Health." *WHO*, www.who.int/health-topics/environmental-health.