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Review Article

Environmental Pollutants and Human Health: Mechanisms of Toxicity, Disease Burden, And Emerging Challenges in a Changing Climate

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ABSTRACT

Environmental pollution remains one of the most pressing public health concerns worldwide, contributing substantially to disease burden, premature mortality, and rising healthcare costs. Rapid industrial growth, urban expansion, fossil fuel combustion, and climate-related environmental changes have led to increased human exposure to particulate matter, toxic chemicals, persistent organic pollutants, heavy metals, and endocrine-disrupting compounds. Increasing evidence suggests that the adverse effects of these contaminants extend beyond isolated organ toxicity, affecting multiple physiological systems through interconnected mechanisms such as oxidative stress, chronic inflammation, mitochondrial impairment, endocrine disruption, immune dysfunction, epigenetic modifications, and metabolic disturbances. Among environmental contaminants, fine particulate matter (PM_{2.5}) has attracted considerable attention because of its ability to penetrate deep into the respiratory tract, enter systemic circulation, and induce pathological changes in distant organs. Similarly, exposure to heavy metals, endocrine-disrupting chemicals, and persistent organic pollutants has been linked to a wide range of chronic conditions, including diabetes, cancer, reproductive dysfunction, and developmental abnormalities. The health impacts of environmental pollution are further intensified by climate change, which influences pollutant distribution, exposure patterns, air quality, and even the stability and effectiveness of pharmaceutical products. This review provides an overview of the major environmental pollutants, the molecular mechanisms underlying their toxicity, associated health outcomes, and emerging challenges related to climate change. A better understanding of these complex interactions is essential for developing effective prevention strategies, strengthening environmental health policies, and reducing the global burden of pollution-related diseases

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INTRODUCTION

Environmental pollution has emerged as one of the most significant public health challenges of the twenty-first century. Rapid industrialization, expanding urban populations, increased transportation activities, intensive agricultural practices, and the extensive use of synthetic chemicals have substantially elevated human exposure to a wide range of environmental contaminants. Over recent decades, thousands of synthetic compounds have been released into the environment, many of which remain inadequately evaluated for their long-term effects on human health and ecosystems [1]. Findings from recent studies suggest that long-term exposure to environmental pollutants may contribute to a broad range of health problems. Certain population groups, including children, pregnant women, older adults, and individuals with pre-existing medical conditions, are especially susceptible because of physiological vulnerability, developing or weakened defense mechanisms, and greater sensitivity to environmental stressors.

There is a growing body of evidence indicating that environmental pollutants play a role in the development of various chronic illnesses, such as respiratory issues, heart disease, neurodegenerative disorders, metabolic syndrome, diabetes, reproductive problems, developmental defects, and cancer [2]. Earlier toxicological investigations largely concentrated on the harmful effects of toxic substances on particular target organs. Today, it is increasingly recognized that environmental pollutants rarely act in isolation. Instead, they can initiate a series of interconnected molecular and cellular events that disrupt the normal functioning of several organ systems simultaneously, leading to broad and long-lasting health consequences [3].

Among air pollutants, particulate matter has received considerable research attention because of its widespread occurrence and documented health effects, with PM_{2.5} attracting particular concern because of its ubiquitous exposure and strong association with adverse health outcomes [4]. Its small particle size allows it to bypass many of the body's natural defense mechanisms, reach the deepest regions of the lungs, and subsequently enter the bloodstream. This systemic distribution can trigger oxidative stress and persistent inflammation, contributing to damage in multiple organs and increasing the risk of chronic diseases [5,6].

This review examines the major classes of environmental pollutants, their mechanisms of toxicity, associated health outcomes, and emerging challenges posed by climate change.

2. Literature Search Strategy

Literature for this review was retrieved from PubMed, Scopus, Web of Science, and Google Scholar databases. Keywords including “environmental pollution”, “PM_{2.5}”, “persistent organic pollutants”, “heavy metals”, “endocrine disruptors”, “oxidative stress”, “climate change”, and “human health” were used. Relevant articles published in English between 2000 and 2026 were screened. Original research articles, systematic reviews, meta-analyses, and reports from international organizations were included based on relevance to environmental toxicology and public health.

3. Major Sources and Categories of Environmental Pollutants

Environmental contaminants originate from a wide range of both anthropogenic and natural sources. Human activities such as fossil fuel combustion, industrial production, vehicular



traffic, waste incineration, mining operations, agricultural practices, and residential heating contribute substantially to the release of pollutants into the environment. These activities introduce a complex mixture of harmful substances into the air, water, and soil, increasing the likelihood of human exposure and associated health risks [7,8]. These activities release a diverse mixture of pollutants into the environment, including particulate matter, volatile organic compounds (VOCs), heavy metals, nitrogen and sulfur oxides, ozone-forming substances, persistent organic pollutants (POPs), and endocrine-disrupting chemicals. The combined presence of these contaminants contributes to environmental degradation and increases the potential for adverse effects on human health [9,10].

3.1 Particulate Matter

Particulate matter is made up of both solid and liquid particles that are suspended in the air. These particles are categorized into PM10, PM2.5, and ultrafine particles based on their aerodynamic diameter [11,12]. Smaller particles demonstrate heightened biological activity due to their ability to penetrate more deeply into respiratory tissues and their larger surface area relative to volume, which facilitates the transport of toxic chemicals and metals [13].

3.2 Persistent Organic Pollutants

Persistent organic pollutants (POPs) encompass polychlorinated biphenyls, dioxins, furans, organochlorine pesticides, and similar compounds. These substances are resistant to environmental degradation, accumulate in tissues, and have long biological half-lives [14,15]. Exposure occurs through contaminated food and water, occupational contact, and environmental persistence.

3.3 Heavy Metals

Cadmium, arsenic, lead, mercury, and chromium are some of the most critical toxic metals. Long-term exposure to these substances is linked to neurotoxicity, disruption of the endocrine system, cancer development, cardiovascular issues, kidney dysfunction, and reproductive problems [16,17].

3.4 Endocrine-Disrupting Chemicals

Compounds that disrupt the endocrine system either imitate or obstruct natural hormonal signaling pathways. Environmental estrogens and air pollutants that disrupt estrogen have garnered significant attention due to their potential role in contributing to breast cancer and reproductive health issues [18,19].

Table 1. Major Environmental Pollutants, Sources, and Associated Health Effects

Pollutant Category	Major Sources	Principal Toxic Components	Major Health Effects
Particulate Matter (PM2.5, PM10)	Vehicle emissions, fossil fuel combustion, industrial processes, biomass burning	Carbon particles, metals, organic compounds	Asthma, COPD, cardiovascular disease, stroke, lung cancer, systemic inflammation
Persistent Organic Pollutants (POPs)	Industrial waste, pesticides, combustion processes	Dioxins, furans, PCBs, organochlorine pesticides	Endocrine disruption, diabetes, reproductive toxicity, cancer



Heavy Metals	Mining, industrial emissions, contaminated water and food	Lead, cadmium, arsenic, mercury, chromium	Neurotoxicity, nephrotoxicity, cardiovascular disease, developmental disorders, cancer
Endocrine-Disrupting Chemicals (EDCs)	Plastics, pesticides, consumer products, industrial chemicals	Bisphenol A, phthalates, parabens	Hormonal imbalance, infertility, obesity, metabolic syndrome, breast cancer
Volatile Organic Compounds (VOCs)	Solvents, paints, fuels, industrial emissions	Benzene, toluene, formaldehyde	Respiratory irritation, neurological disorders, leukemia, carcinogenesis
Nitrogen Oxides (NO _x)	Motor vehicles, power plants	Nitric oxide, nitrogen dioxide	Airway inflammation, asthma exacerbation, impaired lung function
Sulfur Oxides (SO _x)	Coal combustion, industrial activities	Sulfur dioxide	Respiratory diseases, bronchoconstriction, cardiovascular effects
Ground-Level Ozone	Photochemical reactions involving NO _x and VOCs	Ozone (O ₃)	Pulmonary inflammation, oxidative stress, decreased lung capacity

4. Molecular Mechanisms of Environmental Toxicity

4.1 Oxidative Stress

Oxidative stress is a key factor in the toxicity caused by pollutants [20]. Environmental toxins lead to an overproduction of reactive oxygen species (ROS), which surpasses the body's natural antioxidant defenses and results in harm to lipids, proteins, DNA, and cellular organelles [21,22]. PM_{2.5} components directly produce reactive oxygen species and trigger intracellular oxidative pathways that involve NADPH oxidases and mitochondrial electron transport systems [23].

Air pollutants like ozone and nitrogen dioxide cause the oxidation of lipids and proteins in cellular membranes, which triggers inflammatory reactions and leads to tissue damage. Persistent oxidative stress plays a role in the gradual decline

of lung function and the onset of systemic diseases [24].

4.2 Chronic Inflammation

Oxidative damage triggers inflammatory signaling pathways such as NF- κ B, MAPK, and responses mediated by the inflammasome. Once activated, immune cells release cytokines, chemokines, and reactive intermediates that continue to cause tissue harm [25]. Persistent inflammation contributes to vascular dysfunction, fibrosis, metabolic disturbances, and carcinogenesis [26].

4.3 Mitochondrial Dysfunction

Environmental contaminants compromise mitochondrial health and disrupt energy production processes. When mitochondria malfunction, there is a decrease in ATP synthesis, an increase in the production of reactive oxygen species (ROS), disturbances in calcium balance,



and the triggering of cell death pathways [27]. These alterations play a crucial role in the processes of cellular aging, metabolic disorders, and neurodegenerative conditions.

4.4 Endocrine Disruption

Numerous environmental pollutants possess the ability to modulate endocrine functions.

Substances such as dioxins, PCBs, pesticides, and specific metals can disrupt hormone receptor signaling, interfere with the production of steroids, and alter the regulation of endocrine feedback mechanisms [28]. These changes lead to issues with reproduction, developmental defects, weight gain, and diabetes.

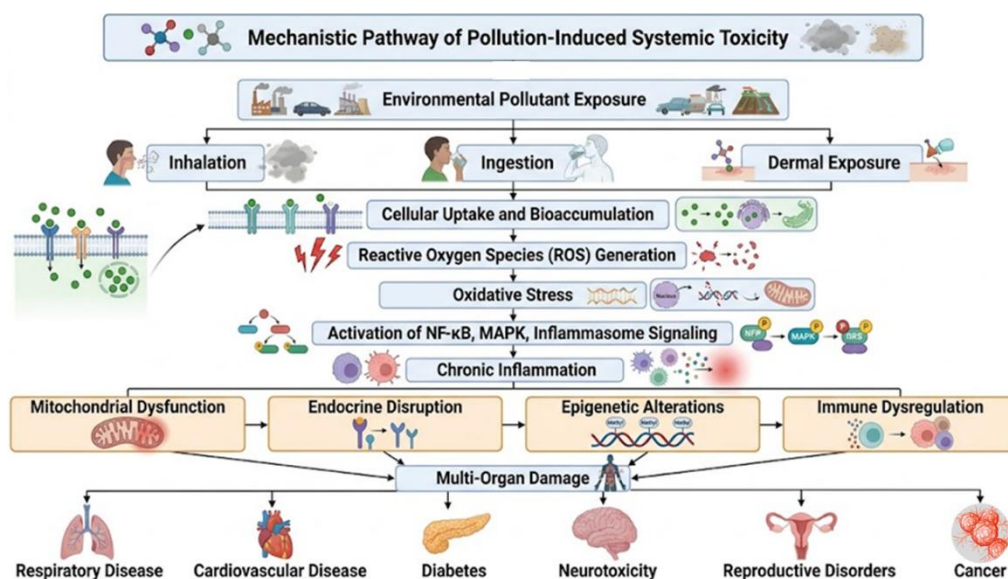


Figure 1. Mechanistic pathway of pollution-induced systemic toxicity.

Environmental pollutants enter the body through inhalation, ingestion, and dermal exposure. Following cellular uptake and bioaccumulation, pollutants stimulate excessive reactive oxygen species (ROS) generation and oxidative stress, leading to activation of inflammatory signaling pathways including NF-κB, MAPK, and

inflammasome pathways. These responses contribute to mitochondrial dysfunction, endocrine disruption, epigenetic alterations, and immune dysregulation, culminating in multi-organ damage and the development of respiratory, cardiovascular, metabolic, neurological, reproductive, and neoplastic diseases.

Mechanism	Biological Effects	Major Signalling Pathways	Associated Diseases
Oxidative Stress	Excess ROS generation, lipid peroxidation, DNA damage	NADPH oxidase, Nrf2, mitochondrial pathways	COPD, cancer, cardiovascular disease, neurodegeneration
Chronic Inflammation	Cytokine release, immune activation	NF-κB, MAPK, inflammasome signalling	Atherosclerosis, diabetes, fibrosis, cancer
Mitochondrial Dysfunction	ATP depletion, ROS amplification, apoptosis	Mitochondrial electron transport chain disruption	Neurodegenerative diseases, metabolic disorders, aging

Endocrine Disruption	Hormone receptor interference, altered steroidogenesis	Estrogen receptor, androgen receptor, AhR signalling	Infertility, obesity, diabetes, breast cancer
Epigenetic Modifications	DNA methylation, histone modification, altered gene expression	Epigenetic regulatory pathways	Cancer, developmental disorders, chronic diseases
Immune Dysregulation	Altered innate and adaptive immunity	Cytokine signalling pathways	Autoimmune disorders, infections, chronic inflammation
Metabolic Dysfunction	Insulin resistance, altered glucose metabolism	PI3K/Akt, AMPK pathways	Type 2 diabetes, obesity, metabolic syndrome

5. Systemic Health Consequences of Environmental Pollutants

5.1 Respiratory Effects

The respiratory system serves as the main entry point for airborne contaminants. Inhalation of PM_{2.5}, ozone, nitrogen dioxide, and harmful aerosols is linked to worsening asthma, chronic obstructive pulmonary disease, diminished lung capacity, lung inflammation, and a heightened risk of respiratory infections [29,30]. Tissue damage caused by oxidative stress plays a crucial role in determining these outcomes.

5.2 Cardiovascular Toxicity

Small particles can penetrate the bloodstream and have a direct impact on vascular tissues. Exposure to pollutants leads to endothelial dysfunction, vascular inflammation, thrombosis, high blood pressure, irregular heartbeats, atherosclerosis, heart attacks, and strokes [31]. Pollution-related deaths around the globe are significantly attributed to cardiovascular disease.

5.3 Metabolic Disorders and Diabetes

Recent findings indicate a strong link between environmental pollutants and metabolic disorders. Persistent organic pollutants, such as dioxins,

PCBs, and chlorinated pesticides, have been linked to a heightened risk of diabetes among individuals exposed through their occupation or environment. Numerous studies have shown that the prevalence of diabetes correlates with the level of pollutant exposure in a dose-dependent manner [32,33].

From a mechanistic perspective, environmental pollutants disrupt insulin signaling pathways, damage pancreatic β -cell function, trigger oxidative stress, and encourage persistent, low-level inflammation [34], thereby contributing to insulin resistance and metabolic dysfunction.

5.4 Cancer Development

Environmental carcinogenesis encompasses DNA damage, mutagenesis, epigenetic changes, persistent inflammation, and interference with the regulation of cell proliferation. Benzo(a)pyrene, a classic example of a polycyclic aromatic hydrocarbon, has direct toxic effects on the epithelial tissues of the respiratory system and is acknowledged as a significant environmental carcinogen [35,36].

Prolonged exposure to cadmium compounds and air pollutants containing arsenic has been linked to a higher risk of breast cancer in certain groups [37,38].

5.5 Neurodevelopmental and Neurological Effects

Due to their developmental stages and higher exposure levels relative to their body mass, children are particularly susceptible to environmental toxins. Exposure to these pollutants has been associated with impaired neurodevelopment, reduced cognitive performance, behavioral disturbances, and a range of adverse neurological outcomes [39].

6. Environmental Exposures in Vulnerable Populations

Studies have shown that exposure to environmental pollutants can interfere with normal brain development and function, leading to learning difficulties, reduced cognitive abilities, behavioral problems, and other neurological impairments [40,41]. Exposure to environmental pollutants has been increasingly recognized as a factor that can adversely affect brain development and function, contributing to learning difficulties, cognitive impairment, behavioral changes, and a variety of neurological disorders.

Similarly, older adults, pregnant women, and individuals living with chronic health conditions are particularly vulnerable to the adverse effects of environmental pollutants because of age-related

physiological changes, altered immune responses, and a reduced ability to cope with environmental stressors [42,43]. Risk assessment models are increasingly focusing on the protection of these vulnerable subgroups.

7. Climate Change as an Emerging Amplifier of Environmental Health Risks

Climate change in combination with environmental pollution increases health effects. Increased temperatures, altered rainfall patterns, wildfires, droughts, floods, and extreme weather events influence the generation, distribution, and human exposure to pollutants [44,45]. Climate change poses health threats such as increased respiratory, cardiovascular and kidney diseases, mental health problems and spread of infectious diseases.

Climate change has direct health effects, but it also impacts the stability of pharmaceuticals, the bioavailability of drugs and their therapeutic efficacy. Degradation of active pharmaceutical ingredients, changes in pharmacokinetics and compromised medication safety can result from elevated temperatures and humidity [46,47]. Bad weather can further disrupt health care systems and pharmaceutical supply chains.



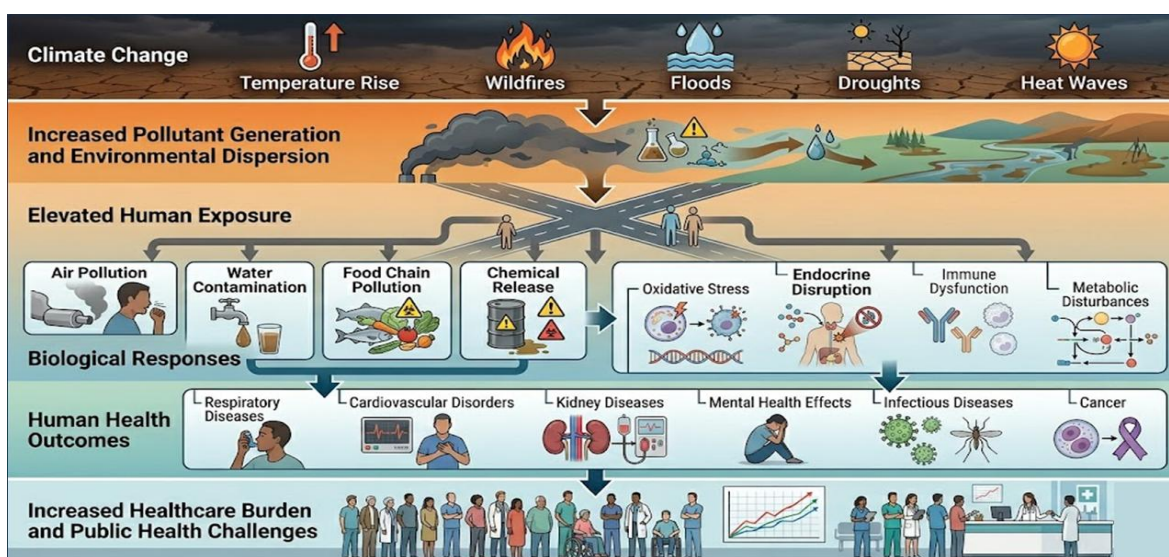


Figure 2: Climate change–driven environmental pollution and human health consequences.

Rising temperatures, wildfires, floods, droughts, and heat waves are several ways in which climate change increases pollutant creation, environmental dispersion, and human exposure. Contaminated air, water, food, and dangerous substances cause oxidative stress, endocrine disruption, immunological dysfunction, and metabolic disorders. These conditions can result in respiratory, cardiovascular, renal, neurological, infectious, and neoplastic diseases. These adverse health effects contribute to the burden of health care and public health problems worldwide.

8. Human Health Risk Assessment Approaches

Health risk assessment provides a structured framework for evaluating the potential health effects of environmental pollutants. An effective assessment requires consideration of exposure levels and duration, identification of susceptible populations, evaluation of dose–response relationships, and recognition of uncertainties associated with exposure and health outcomes. Assessing the risk of chronic diseases can be particularly challenging because these conditions often develop over long periods and are influenced by multiple interacting factors.

As environmental health challenges become more complex, researchers are increasingly integrating data from epidemiology, toxicology, biomonitoring, mechanistic studies, and computational modeling. Such multidisciplinary approaches offer deeper insight into the cumulative and interactive effects of pollutants and help guide policies aimed at reducing environmental risks and protecting public health.

9. Future Perspectives and Research Priorities

Several critical knowledge gaps remain. Future research should focus on:

- Longitudinal studies of lifetime pollutant exposure.
- Identification of early biomarkers of environmental toxicity.
- Mechanistic characterization of pollutant mixtures.
- Multi-omics approaches to understand susceptibility.
- Studies on climate–pollution interactions.
- Development of targeted preventive and therapeutic interventions.

- Improved exposure assessment technologies and personal monitoring systems.
- Translational studies linking experimental findings with clinical outcomes.

The enhanced integration of environmental science, toxicology, epidemiology, molecular biology, and public health policy is essential for reducing pollution-associated disease burdens globally.

10. CONCLUSION

The evidence reviewed in this article highlights the substantial impact of environmental pollution on human health worldwide, with its impact becoming increasingly evident across diverse populations and geographical regions. Exposure to airborne particulate matter, persistent organic pollutants, endocrine-disrupting chemicals, heavy metals, and other environmental contaminants has been linked to the development of numerous chronic diseases through complex biological processes involving oxidative stress,

inflammation, mitochondrial dysfunction, hormonal imbalance, and immune dysregulation. Growing evidence indicates that these pollutants rarely affect a single organ in isolation; instead, they exert widespread systemic effects that can compromise the function of multiple physiological systems. Certain populations, particularly children, older adults, and individuals with pre-existing health conditions, remain especially vulnerable to these adverse outcomes. The challenges posed by environmental pollution are further intensified by climate change, which influences pollutant distribution, exposure patterns, disease dynamics, and even the stability and effectiveness of therapeutic agents. Addressing these interconnected environmental and health challenges requires robust risk assessment strategies, strengthened regulatory frameworks, targeted preventive interventions, and sustained multidisciplinary research. Such efforts are essential to reduce the global burden of pollution-related diseases and promote a healthier and more sustainable future.

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