IAA Journal of Biological Sciences 13(2):17-22, 2025
©IAAJOURNALS
https://doi.org/10.59298/IAAJB/2025/1321722
www.iaajournals.org
ISSN:2636-7254
IAAJB132

Environmental Pollutants and Endocrine Disruption: Mechanistic Insights and Public Health Implications

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ABSTRACT

Environmental pollutants, particularly endocrine-disrupting chemicals (EDCs), have emerged as major public health concerns due to their ability to interfere with hormonal signaling and regulation. These agents, prevalent in industrial byproducts, pesticides, plastics, personal care products, and pharmaceuticals, mimic or antagonize natural hormones, leading to reproductive, developmental, metabolic, and neurological dysfunctions. This review explores the molecular mechanisms through which EDCs-such as bisphenol A, phthalates, dioxins, and organochlorine pesticides-disrupt endocrine homeostasis. It also examines the vulnerability of different populations, especially pregnant women, children, and wildlife, and the cumulative impact of low-dose chronic exposures. Evidence from epidemiological and experimental studies linking EDCs to obesity, diabetes, infertility, thyroid abnormalities, and hormone-sensitive cancers is discussed. Furthermore, the review addresses emerging public health strategies, regulatory challenges, and the need for stronger environmental monitoring systems. By bridging molecular toxicology with epidemiological outcomes, this article highlights the urgent need for global policy frameworks aimed at mitigating EDC exposure and protecting endocrine health.

Keywords: Endocrine Disrupting Chemicals, Hormonal Imbalance, Environmental Toxicants, Public Health, Mechanisms of Action, EDC Regulation

INTRODUCTION

The increasing incidence of endocrine-related disorders across populations has drawn global attention to the role of environmental pollutants in disrupting hormonal balance [1]. The endocrine system comprises glands and organs that produce, store, and regulate hormones-chemical messengers essential for growth, metabolism, development, reproduction, and mood regulation [2]. Endocrine-disrupting chemicals (EDCs) are exogenous substances that interfere with these hormonal processes, often by mimicking, blocking, or altering hormone levels and signaling pathways [3]. These chemicals include a broad spectrum of substances such as industrial compounds, pesticides, heavy metals, and pharmaceutical residues [4]. Their widespread use in agriculture, manufacturing, personal care products, and plastics has led to pervasive contamination of air, soil, water, and food sources [5]. Over the past two decades, advances in toxicology, molecular biology, and epidemiology have expanded our understanding of how low-dose, chronic exposure to EDCs can contribute to a range of health issues. Unlike traditional toxicants that operate through acute or high-dose toxicity, EDCs can exert their effects even at very low concentrations, particularly when exposure occurs during vulnerable developmental stages such as gestation, infancy, and puberty [6]. Furthermore, the non-monotonic dose-response curves seen with many EDCs challenge classical toxicological models that "the dose makes the poison [7]." This review aims to provide a mechanistic understanding of endocrine disruption by environmental pollutants, examine major sources and types of EDCs, explore the pathways through which they affect hormonal systems, and assess the resulting health outcomes. By doing so, the review underscores the pressing need for integrative public health and regulatory responses.

Sources and Types of Endocrine Disrupting Chemicals

Endocrine-disrupting chemicals originate from diverse sources and are found in a wide array of consumer products and industrial waste streams [8]. One of the most ubiquitous sources is the plastic industry. Compounds such as bisphenol A (BPA) and phthalates are commonly used in the production of plastic containers, food packaging,

medical devices, and personal care products [9]. These chemicals can leach into food and water supplies, especially when plastics are exposed to heat or mechanical stress. Pesticides and herbicides represent another major category. Chemicals like dichlorodiphenyltrichloroethane (DDT), atrazine, and chlorpyrifos are widely used in agriculture and can persist in the environment for decades [10]. Despite bans in some countries, these compounds continue to affect ecosystems through soil and water contamination and through bioaccumulation in food chains. Industrial pollutants such as polychlorinated biphenyls (PCBs), dioxins, and perfluoroalkyl substances (PFAS) have been widely documented as potent EDCs [11]. These are persistent organic pollutants (POPs) with high chemical stability, leading to prolonged environmental and biological half-lives. Heavy metals such as lead, arsenic, and cadmium also act as endocrine disruptors by interfering with the hypothalamic-pituitary axis and altering hormone receptor sensitivity [12]. Additionally, pharmaceutical agents, particularly synthetic estrogens and androgens used in hormone replacement therapies and contraceptives, enter water systems through human excretion and improper disposal, contributing to aquatic contamination and ecological endocrine disruption [13].

Mechanisms of Endocrine Disruption

The mechanisms by which EDCs interfere with hormonal regulation are multifaceted and operate at various levels of the endocrine system. A primary mechanism involves direct interaction with hormone receptors. Many EDCs act as agonists or antagonists to nuclear hormone receptors such as the estrogen receptor (ER), androgen receptor (AR), and thyroid hormone receptor (TR) [14]. For instance, BPA mimics the action of estradiol, binding to ERs and triggering or inhibiting estrogenic responses in target tissues [15]. Beyond receptor binding, EDCs influence the synthesis, metabolism, and elimination of hormones. Certain phthalates and pesticides alter the expression or activity of enzymes involved in steroidogenesis, such as aromatase or 5α-reductase, disrupting the biosynthesis of testosterone and estrogens [16]. Others modulate the activity of cytochrome P450 enzymes, crucial for hormone detoxification and metabolic regulation [14]. A significant emerging area is the epigenetic modulation by EDCs. These chemicals can induce DNA methylation changes, histone modifications, and alter microRNA expression, leading to persistent gene expression alterations without changing the underlying DNA sequence [17]. Such epigenetic changes are particularly concerning as they may be heritable across generations. Additionally, EDCs disrupt the transport and bioavailability of hormones by binding to hormone-binding proteins such as sex hormone-binding globulin (SHBG), altering the distribution and access of hormones to target tissues [18].

Health Outcomes Linked to EDC Exposure

The health effects of EDCs are wide-ranging and depend on the nature of the compound, the duration and timing of exposure, and the biological susceptibility of the individual. One of the most well-documented outcomes is reproductive dysfunction. In males, EDCs such as PCBs and phthalates have been associated with decreased sperm count, altered testicular development, and testosterone deficiency [19]. In females, exposure to EDCs like BPA and dioxins has been linked to irregular menstrual cycles, reduced fertility, and conditions such as endometriosis and polycystic ovary syndrome (PCOS) [20]. EDCs also pose a substantial risk to developmental health. In utero and early-life exposure to these chemicals can interfere with neurodevelopment, resulting in learning disabilities, reduced IQ, and behavioral disorders such as autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) [21]. The disruption of thyroid hormone signaling, which is critical for brain development, is a key mechanism behind these outcomes [22]. In terms of metabolic health, a class of EDCs known as "obesogens" can promote adipogenesis, insulin resistance, and lipid dysregulation, thereby contributing to the global rise in obesity and type 2 diabetes [23]. EDCs such as tributyltin (TBT), BPA, and PFAS have been implicated in disrupting glucose homeostasis and promoting chronic inflammation [24]. Endocrine disruption has also been linked to several hormone-sensitive cancers, including breast, prostate, ovarian, and testicular cancers [25]. Chronic exposure to estrogen-mimicking chemicals increases mitogenic signaling in estrogen receptor-positive cells, enhancing the risk of neoplastic transformation [26]. Finally, thyroid and adrenal disorders represent an often-overlooked outcome of EDC exposure. Chemicals that interfere with thyroid hormone production or receptor function can cause hypothyroidism, goiter, or even neuropsychiatric manifestations, particularly in children [28].

Vulnerable Populations

While EDCs pose health risks to the general population, certain groups exhibit heightened susceptibility due to physiological and developmental factors. Pregnant women and fetuses represent one of the most vulnerable populations. During pregnancy, hormonal regulation is critical for fetal development, and any disruption caused by transplacental transfer of EDCs can lead to irreversible effects [28]. Studies have demonstrated that maternal exposure to BPA, phthalates, and PCBs can result in fetal growth retardation, altered neurodevelopment, and changes in reproductive organ morphology [29]. Infants and children are also disproportionately affected due to their smaller body mass, immature detoxification systems, and developmental sensitivity [29]. Early-life exposures can set the stage for lifelong health challenges, a phenomenon referred to as "developmental origins of health and

disease" (DOHaD). For example, children exposed to lead or arsenic have shown deficits in cognitive performance and delayed puberty [30]. Similarly, early exposure to estrogenic compounds has been linked to early menarche and increased risk of hormone-dependent cancers later in life [31]. Beyond humans, wildlife species experience significant endocrine disruption due to environmental contamination. Aquatic species are particularly at risk, as wastewater effluents containing pharmaceuticals and EDCs like ethinyl estradiol have been shown to induce feminization in male fish, altered mating behavior, and decreased reproductive capacity [32]. These effects have implications for biodiversity, population dynamics, and ecosystem health.

Regulatory and Public Health Challenges

Regulating EDCs presents a complex challenge due to scientific uncertainties, diverse chemical structures, and global variability in policies. Unlike traditional toxicants, EDCs exhibit non-monotonic dose responses, where low-dose exposures may elicit stronger biological effects than high doses [33]. This complexity complicates risk assessments and often leads to underestimation of actual risks. Current regulatory frameworks, such as those by the US Environmental Protection Agency (EPA) and the European Chemicals Agency under REACH, have made progress in evaluating EDCs, yet many substances remain insufficiently tested [34]. Furthermore, chemical-by-chemical regulation is time-intensive and does not adequately address mixture effects or cumulative exposure over time [35]. There is also limited biomonitoring and surveillance infrastructure in low- and middle-income countries, where environmental contamination is often high due to poor waste management and industrial emissions [36]. Public awareness remains low, and labeling of EDC-containing products is largely absent. These gaps underscore the need for an integrated global strategy that includes standardized testing protocols, precautionary regulatory measures, and public education initiatives aimed at reducing exposure and enhancing resilience.

Policy and Mitigation Strategies

Effective mitigation of EDC-related health risks requires comprehensive public health policies, cross-sector collaboration, and technological innovation. One of the most critical strategies is the adoption of the precautionary principle in chemical regulation, whereby lack of complete scientific certainty does not delay action against potentially harmful substances [37]. Regulatory agencies must establish standardized screening and testing protocols for identifying endocrine-disrupting properties in chemicals. These should be complemented by lifecycle assessments and green chemistry initiatives that prioritize safer chemical substitutes. Governments can incentivize industries to phase out high-risk chemicals through subsidies, tax credits, or penalties. Public awareness campaigns and environmental labeling schemes can empower consumers to make informed choices. For example, labeling products as "EDC-free" can shift market demand and pressure manufacturers to adopt safer formulations [38] Community-level interventions, such as improving waste disposal systems, limiting pesticide use, and promoting sustainable agriculture, are essential in reducing environmental EDC loads. Furthermore, investments in water treatment technologies and air quality monitoring can significantly curtail population-level exposures. In the research domain, fostering interdisciplinary collaboration and funding for longitudinal epidemiological studies can generate the evidence base needed to inform dynamic policy responses.

Emerging Research and Future Directions

The evolving understanding of endocrine disruption continues to benefit from advances in molecular biology, bioinformatics, and environmental sciences. Emerging research is increasingly focused on identifying sensitive biomarkers of exposure and effect. High-throughput screening assays and omics technologies—such as transcriptomics, metabolomics, and epigenomics—offer powerful tools for uncovering the subtle yet biologically significant effects of EDCs [17]. Artificial intelligence and computational toxicology are being leveraged to predict EDC activity based on chemical structure, reducing the need for animal testing and accelerating risk assessments [39]. These approaches can also help decipher complex mixture effects and dose-response relationships. Transgenerational studies are shedding light on the heritable impacts of EDCs. Findings from rodent models have demonstrated that ancestral exposure can lead to phenotypic alterations in multiple generations, potentially via epigenetic mechanisms [40].

CONCLUSION

These revelations emphasize the long-term implications of current exposures on future generations. Looking forward, a shift toward a "one health" approach, integrating human, animal, and environmental health is essential for comprehensively tackling endocrine disruption. Collaborative efforts between governments, academia, industries, and civil society will be critical in translating scientific discoveries into effective, evidence-based policy actions. The pervasiveness of endocrine-disrupting pollutants represents a profound threat to global health. A mechanistic understanding of their actions, combined with policy enforcement, public engagement, and innovative research, is crucial in addressing this invisible epidemic. Protecting endocrine integrity should be a central aim of environmental and public health strategies in the 21st century.

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CITE AS: Ernest Nsubuga (2025). Environmental Pollutants and Endocrine Disruption: Mechanistic Insights and Public Health Implications. IAA Journal of Biological Sciences 13(2):12-22. https://doi.org/10.59298/IAAJB/2025/1321722