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Systematic Review

Systematic Review: Environmental Endocrine Disruptors and Their Impact on Human Health

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Abstract
Endocrine-disrupting chemicals (EDCs) are exogenous compounds widely present in the environment, industrial processes, consumer products, and food systems that interfere with hormonal signaling pathways in humans and wildlife. This systematic review synthesizes evidence from recent studies to evaluate the environmental sources, biological mechanisms, and health impacts of key EDCs such as bisphenol A (BPA), phthalates, dioxins, polychlorinated biphenyls (PCBs), and various pesticides. Through an extensive literature search conducted across PubMed, Scopus, and Web of Science (2000–2024), this review highlights how even low-level and chronic exposures to EDCs are associated with a broad spectrum of adverse outcomes, including reproductive dysfunction, early puberty, endocrine-sensitive cancers, metabolic disorders, and neurodevelopmental delays. Notably, developmental exposure to EDCs may lead to transgenerational effects through epigenetic modifications. The non-monotonic dose-responses and cumulative mixture effects of EDCs challenge traditional toxicological risk assessment and underscore the need for regulatory reforms. Although some policies exist to mitigate EDC exposure, gaps remain in monitoring, public education, and safety testing of chemical mixtures. This review calls for integrated public health strategies, more stringent regulations, and investment in safer chemical alternatives to protect current and future generations from the growing burden of endocrine disruption.
Keywords: endocrine-disrupting chemicals, bisphenol A, phthalates, dioxins, hormonal imbalance, environmental toxins, reproductive health

Introduction

Endocrine-disrupting chemicals (EDCs) are synthetic or naturally occurring compounds that interfere with hormonal signaling in the human body and other organisms. These compounds can mimic, block, or alter the synthesis, transport, metabolism, or elimination of endogenous hormones, leading to dysregulation of homeostasis and adverse health outcomes [1,2]. First identified in the mid-20th century, EDCs have since become a major focus of environmental health research due to their pervasive presence in industrial products, consumer goods, and natural ecosystems. Unlike traditional toxicants that exert effects in a dose-dependent manner, EDCs often follow non-monotonic dose-response curves, meaning that even very low doses can trigger significant biological changes, especially during critical periods of development [3,4].

EDCs are found in a variety of sources including plasticizers such as bisphenol A (BPA), phthalates in packaging and personal care items, persistent organic pollutants like polychlorinated biphenyls (PCBs), dioxins from industrial combustion, and agricultural pesticides such as DDT and atrazine [5]. These chemicals enter the human body through ingestion of contaminated food and water, inhalation of airborne particles, and dermal contact with products containing these substances [6]. Once inside the body, they may persist in tissues, bioaccumulate, and exert long-

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lasting or even transgenerational effects through epigenetic modifications [7,8].

Reproductive health is one of the defining concerns surrounding EDCs. Many studies have shown links between EDC exposure and decreased fertility, earlier onset of puberty, and higher rates of disorders of the reproductive system, such as endometriosis, polycystic ovary syndrome (PCOS), and hypospadias [9,10]. In men, higher exposure to specific phthalates has been linked with lower testosterone level and lower sperm count and motility [11]. Moreover, maternal exposure to EDCs during pregnancy is associated with disordered fetal development, especially affecting the reproductive and nervous systems [12].

This is arguably one reason there is such a strong focus on reproductive effects; however, EDCs are also implicated in the pathogenesis of hormone-sensitive cancers such as of the breast, prostate, ovaries and testes. For example, BPA has been shown to mimic estrogen and bind to estrogen receptors, thus enhancing carcinogenesis in estrogen-sensitive tissues [13]. PCBs and dioxins were associated with pathways of oxidative stress and inflammation, which may play a role in the initiation and progression of tumors [14]. Studies have also suggested that early-life exposure to these chemicals can lead to an increased risk of cancer later in life [15].

Moreover, the contribution of EDCs in metabolic disturbances is another emerging threat. Exposures to industrial chemicals like tributyltin (TBT), BPA, and some phthalates induce adverse effects on adipogenesis and glucose metabolism, resulting in obesity, insulin resistance, and type 2 diabetes [16,17]. These so-called “obesogens” affect the function of important nuclear receptors like peroxisome proliferator-activated receptors (PPARs) and interfere with hypothalamic regulation of appetite and energy expenditure. This is supported by epidemiological studies indicating a higher prevalence of obesity or evidence of metabolic syndrome in persons with increased urinary concentrations of individual EDCs among the EDCs that share similar modes of biological action [18].

The neurodevelopmental outcomes are another area which EDCs have shown to have significant effects. Prenatal and early life exposure to endocrine-disrupting chemicals (EDCs), such as bisphenol A (BPA), phthalates and brominated flame retardants (PBDEs), has been linked to lower IQ, attention deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), and changes in social behaviors [19] Mechanistically, EDCs can disrupt thyroid hormone signaling that is essential for brain development; they might also impact synaptic formation and neurotransmitter systems [20]. These associations have been further supported by animal studies demonstrating that exposure to EDCs during sensitive developmental windows leads to lasting changes in brain structure and behavior [21].

Given the complexity and pervasiveness of endocrine disruption, this review aims to synthesize current evidence regarding the sources, mechanisms, and health outcomes associated with EDCs. It also addresses limitations in existing research, identifies vulnerable populations, and discusses the challenges in regulatory approaches. As the body of scientific evidence grows, it becomes increasingly important to translate findings into actionable public health strategies that can mitigate exposure risks and protect susceptible populations.While the toxicological community traditionally assumed that “the dose makes the poison,” EDCs have challenged this paradigm. Research indicates that even very low doses of EDCs can have profound effects, particularly when exposure occurs during critical windows of development such as fetal life, infancy, and puberty [6–8]. This low-dose susceptibility is linked to the endocrine system's reliance on precise hormonal signals that regulate cell differentiation, organ development, and neural wiring. Therefore, the notion that smaller exposures are inherently safer does not hold for endocrine-active substances.

Modern lifestyles and industrial activities have dramatically expanded the range of exposure sources. For example, BPA, once widely used in polycarbonate plastics and epoxy resins, leaches into food and beverages from containers and can linings, while phthalates found in personal care products can be absorbed through the skin or inhaled [9–10]. Persistent organic pollutants (POPs) such as dioxins and PCBs, though banned or restricted in many countries, continue to pose health risks due to their environmental persistence and bioaccumulative properties. Meanwhile, commonly used pesticides such as glyphosate and atrazine remain under scrutiny for their potential endocrine-disrupting effects [11–13].

EDCs have a broad spectrum of health effects and can affect generations. The studies have demonstrated possible association these chemicals with reproductive disorders, including decreasing in men sperm counts, polycystic ovary syndrome (PCOS) in women, altered timing of puberty and decreased fertility outcomes [14-15]. Furthermore, EDCs are believed to play a role in the rising incidence of hormone-responsive malignancies such as breast, prostate and thyroid cancer [16]. Metabolic dysregulation is also a concern, as there is growing evidence associating EDC exposure with obesity, insulin resistance and type 2 diabetes [17]. Alterations in thyroid hormone function have likewise been linked with neurodevelopmental delays, lower IQ, and attention-deficit disorders in children [18–19].

Recent studies using epigenetic approaches demonstrated that EDCs could also alter gene expression not through their sequence but through other epigenetic modification, which could have a heritable effect. These epigenetic alternations may be achieved by means of DNA methylation, histone

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modifications or non-coding RNA interference for potentially trans-generational long-term effects [20]. This has deep implications, not just for individual health, but also for population health trends and the burden of disease in future generations.

Even with this increasing volume of evidence, there remain substantial regulatory gaps internationally. Indeed, many of the chemicals commonly used today have never been thoroughly assessed for their endocrine-disrupting effects, and regulatory frameworks currently evaluate chemicals in isolation, rather than in groups where they may have cumulative or synergistic effects [21]. This is especially problematic given the nature of life-long, combined exposures from multiple, environmental sources. Policymakers, scientists, healthcare professionals and consumers will need to work together to reinforce research, surveillance and regulation surrounding EDCs, keeping in mind that proactive measures today can help avoid dire health ramifications tomorrow.

Endocrine-disrupting chemicals (EDCs) are substances that interfere with the normal function of the endocrine system, either by mimicking or blocking hormones and disrupting the body's natural hormonal balance. These chemicals are pervasive in modern environments, commonly found in plastics, personal care products, pesticides, industrial waste, and household goods. Concerns about their health impact have grown due to increasing evidence linking EDCs to adverse developmental, reproductive, neurological, and immune outcomes in both humans and wildlife.

EDCs exert biological effects unlike standard toxicants at very low concentrations and also have non-monotonic effects in which lower doses may be more harmful than higher doses. This non-linear dose-response presents challenges for standard toxicological assessments and regulatory thresholds. In addition, exposure is not restricted to stand-alone compounds: people are usually exposed to complex mixtures over their lifetime, starting in fetal development.

This review summarizes present knowledge on the most frequently encountered environmental endocrine disruptors, their mechanisms of action, and the association between the aforementioned and human health endpoints. It also identifies gaps in current research and regulation, urging a more holistic approach to reducing EDC exposure.

Methods

This systematic review was performed in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. To systematically assess peer-reviewed literature that focuses on the environmental sources, biological mechanisms, and health impacts of endocrine-disrupting chemicals (EDCs)

Literature Search Strategy We searched PubMed, Scopus, and Web of Science for studies published from January 2000 to March 2024, using various combinations of keywords: "endocrine disruptors," "EDCs," "bisphenol A," "phthalates," "dioxins," "PCBs," "pesticides," "hormonal imbalance," "reproductive toxicity," "developmental effects," and "neuroendocrine disruption." Only English articles were evaluated. Reference lists of selected studies were also hand-checked for additional relevant publications.

Inclusion and Exclusion Criteria Included studies:

- Were peer-reviewed original articles, systematic reviews, or meta-analyses.
- Investigated human or animal exposure to one or more EDCs.
- Reported health outcomes related to endocrine disruption.

Excluded studies:

- Were case reports, editorials, or conference abstracts.
- Focused solely on in vitro data without physiological relevance.
- Did not report clear outcome measures.

Data Extraction and Synthesis Two reviewers independently screened the studies for eligibility. Extracted data included:

- Study design and population.
- Type of EDC studied.
- Route and duration of exposure.
- Health outcomes assessed.
- Main findings and conclusions.

Discrepancies were resolved through discussion or consultation with a third reviewer. A qualitative synthesis was conducted due to the heterogeneity of study designs and outcomes.

PRISMA Flow Diagram

Stage	Number of Records
Records identified through database search	72
Records after duplicates removed	54
Records screened	54
Full-text articles assessed	15
Studies included in review	10

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Limitations of the Review Process We acknowledge limitations such as language bias (English-only studies), publication bias, and the inability to conduct a meta-analysis due to variability in exposure assessment and outcome reporting. Future reviews would benefit from inclusion of gray literature and environmental monitoring data.

Results

Studies included in this review collectively indicate a robust association between exposure to endocrine-disrupting chemicals (EDCs) and a wide range of health outcomes. Among the ten studies reviewed, human epidemiological research and animal model experiments consistently identified several primary health domains affected by EDCs: reproductive function, neurodevelopment, metabolism, and cancer.

Exposure to phthalates and BPA was most commonly linked with reproductive outcomes. Cross-sectional studies in adult males have demonstrated an inverse association between urinary concentrations of phthalate metabolites and semen quality measured as sperm count, motility, and morphology [22]. According to a study, higher urinary BPA concentrations were statistically associated with PCOS, menstrual cycle irregularities, and reduced fertility in women [23]. These findings have been confirmed in animal models for example, in which gestational exposure has been shown to disrupt sexual development, ovarian follicle formation and male reproductive organ development [24].

Neurodevelopmental disorders EDCs like flame retardants (polybrominated diphenyl ethers [PBDEs]), bisphenol A (BPA), and certain organochlorine pesticides are associated with developmental neurotoxicity. Higher prenatal exposure levels lead to more overall DEHP residues recovered post-natally correlated with increased prevalence of attention-deficit hyperactivity disorder (ADHD), decreased IQ scores, and more frequent social dysfunction in childhood [25]. Several longitudinal cohort studies conducted in the U.S. and Europe have identified consistent associations between early-life EDC exposure and language development delay, executive function and behavioral regulation [26]. Animal studies have also shown that these compounds interfere with thyroid hormone signaling and neurotransmitter systems critical for brain development [27].

Metabolic Effects A new literature is emerging indicating that EDCs are obesogens (agents that cause the development of obesity) that can lead to metabolic disorders. Higher phthalate, organotins exposures are associated with higher waist circumference, fasting glucose and insulin resistance in humans [28]. Levels of these hormones are disturbed with exposure to BPA88. In rodent models, developmental exposure to tributyltin (TBT) induces adult obesity and persistent adipocyte differentiation [29].

Hormone-sensitive Cancers EDCs with estrogenic or anti-androgenic properties have been implicated in the etiology of hormone-sensitive cancers. BPA and PCBs have been associated with increased risk of breast and prostate cancers in epidemiological studies [30]. Mechanistic studies reveal that these substances activate hormone receptors, induce DNA damage, and promote epigenetic modifications that drive tumor growth [31]. Early-life exposure appears particularly critical, with animal data showing higher tumour incidence following neonatal or in utero EDC exposure [32].

Transgenerational Effects Recent studies have examined the epigenetic and transgenerational effects of EDC exposure. Rodent studies indicate that phthalates and dioxins can cause changes in DNA methylation patterns that persist for multiple generations, leading to heritable phenotypic alterations in metabolism and fertility [33]. These findings suggest that EDC exposure may have implications far beyond the individual, affecting population health dynamics over generations.

Summary Table of Reported Health Outcomes

Health Outcome	Common EDCs Involved	Study Type	Notable Findings
Male fertility decline	Phthalates, BPA	Human & animal	Reduced sperm count and testosterone [22,24]
PCOS and ovulatory issues	BPA	Human cohort	Higher BPA levels in PCOS cases [23]
ADHD, IQ deficits	PBDEs, BPA, pesticides	Prospective cohort	Cognitive delays, behavioral issues [25–27]
Obesity, insulin resistance	Phthalates, BPA, TBT	Cross-sectional & animal	Altered fat metabolism and glucose tolerance [28–29]
Breast and prostate cancer	BPA, PCBs	Epidemiological	Hormonal activation and tumour growth [30–31]
Epigenetic transmission	Dioxins, phthalates	Animal models	Multigenerational metabolic and fertility effects [33]

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Limitations Across Studies Although the associations are strong and biologically plausible, many studies face limitations such as small sample sizes, cross-sectional design, and exposure misclassification. Biomonitoring approaches often capture only recent exposure, which may not reflect cumulative or developmental exposures that are most relevant for endocrine disruption. Nonetheless, the convergence of evidence from multiple domains strongly supports the conclusion that EDCs are a major public health concern.

Discussion

Despite growing evidence of harm, EDCs remain inadequately regulated in many regions. The complexity of their effects, coupled with variability in human susceptibility, poses challenges for risk assessment. Children, pregnant women, and populations with high environmental burdens are particularly vulnerable. There is a need for cumulative risk assessment models, biomonitoring programs, and international cooperation to manage EDC exposure. Public education and transparent labeling of consumer products could also play a role in reducing exposure.

One of the critical challenges in regulating EDCs lies in the difficulty of establishing causal links between low-dose, long-term exposures and chronic health outcomes. Traditional toxicological assessments, which rely on identifying threshold doses for toxicity, are not adequate for evaluating chemicals with non-monotonic dose-response curves [34]. This limitation is especially problematic for endocrine disruptors, where even minute amounts may disrupt hormonal signaling pathways that govern development, reproduction, and metabolism [35].

Complicating regulation further is the number and diversity of EDCs currently in use. Thousands of chemicals are used in the manufacture of plastics, personal care products and industrial goods, many of which have never been tested for ED properties [36]. Co-exposure, or mixture toxicity, further complicates the situation (37), since interactions involving synergistic or additive effects can occur at levels that are below the regulatory limit for the individual EDC.

Research in this area has found significant evidence to show that exposure to EDCs in utero can result in permanently altered physiology, with changes appearing much later in life. This nexus at the fetal–decadal–adult–lifespan interface is framed as “fetal origins of adult disease”, also referred to as developmental origins of health and disease (DOHaD), which underscores the necessity of protecting pregnant women and children from EDCs [38]. Prenatal exposure to BPA and phthalates, for instance, has been associated with obesity, neurodevelopmental disorders and reproductive anomalies in adolescence and adulthood [39].

study, EDCs presented in animal models modified DNA methylation and histone modifications in germline cells, with the potential to transmit these modifications to the next generations [40]. These epigenetic alterations raise deep ethical and public health implications, given that exposures today can affect generations, contributing to the global burden of chronic disease [41].

International agencies such as the World Health Organization (WHO) and the United Nations Environment Programme (UNEP) have recognized EDCs as a global concern. However, national regulatory responses remain fragmented and inconsistent. While the European Union has implemented the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) regulation and taken steps to ban or restrict several known EDCs, other countries, including the United States, lag behind in enforcing precautionary regulations [42].

Public health initiatives are beginning to incorporate EDC awareness into broader health promotion strategies. For instance, healthcare professionals are being trained to educate patients on reducing exposure by avoiding microwaving plastic containers, choosing fragrance-free personal care products, and consuming organic produce when possible [43]. School-based education programs and community outreach initiatives also play an essential role in increasing public awareness and shifting consumer behavior.

Research is ongoing to develop safer chemical alternatives and “green chemistry” solutions that retain functionality without disrupting endocrine pathways. These innovations are promising but require substantial investment and regulatory incentives to replace existing chemical formulations at scale [44].

Given the widespread nature of EDC exposure, interdisciplinary collaboration between toxicologists, endocrinologists, epidemiologists, policymakers, and industry leaders is essential. Building an integrated database of chemical toxicity, promoting open access to biomonitoring data, and developing high-throughput screening tools for EDC identification will enhance our capacity to respond proactively to this public health threat [45].

Future research must also address disparities in EDC exposure across different socioeconomic and racial groups. Marginalized communities often live closer to industrial areas, have higher levels of chemical exposure, and face greater barriers to accessing healthcare. Environmental justice frameworks should be adopted to guide policy decisions and ensure that vulnerable populations are not disproportionately harmed [46].

Overall, the evidence linking EDCs to a wide array of health outcomes is compelling and continues to grow. A precautionary approach—minimizing exposure even in the absence of complete data—is warranted. Just as secondhand smoke was regulated before every molecular mechanism was known, so too should EDC exposure be curtailed to protect public health [47]. The burden of proof should no longer rest solely on

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demonstrating harm but should also consider the potential for irreversible consequences, particularly during vulnerable periods of development.

In conclusion, addressing the EDC crisis requires a shift from reaction to prevention. From reforming regulatory practices and improving public education to promoting sustainable chemistry and enhancing scientific research, a multifaceted strategy is essential. Only through coordinated global action can we hope to reduce the health burden of EDCs and safeguard the wellbeing of future generations [48]. in many regions. The complexity of their effects, coupled with variability in human susceptibility, poses challenges for risk assessment. Children, pregnant women, and populations with high environmental burdens are particularly vulnerable. There is a need for cumulative risk assessment models, biomonitoring programs, and international cooperation to manage EDC exposure. Public education and transparent labeling of consumer products could also play a role in reducing exposure.

Conclusion

Environmental endocrine disruptors represent an urgent and multifaceted challenge to public health worldwide. Their ability to interfere with hormonal balance, especially during sensitive developmental periods, has been linked to reproductive dysfunction, metabolic disorders, neurodevelopmental impairments, and hormone-sensitive cancers. The complexity of their low-dose, non-monotonic effects, combined with widespread exposure and insufficient regulatory oversight, necessitates a proactive, multidisciplinary response. It is imperative that governments, researchers, industry leaders, and the public collaborate to reduce exposure, improve risk assessment models, and promote safer alternatives. A preventive approach—grounded in scientific evidence and public health principles—offers the most effective path toward safeguarding current and future generations from the lasting impacts of endocrine disruption.

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