

Review Article

## Environmental pollutants as endocrine disruptors

Seema Srivastava<sup>1</sup>, Sharey Balyan<sup>1</sup>

<sup>1</sup>Department of Zoology, University of Rajasthan, Jaipur, Rajasthan, India.



**\*Corresponding author:**

Seema Srivastava,  
Department of Zoology,  
University of Rajasthan, Jaipur,  
Rajasthan, India.

[drseema07@gmail.com](mailto:drseema07@gmail.com)

Received: 16 June 2025

Accepted: 16 August 2025

Published: 15 September 2025

DOI

10.25259/JRHM\_22\_2025

Quick Response Code:



### ABSTRACT

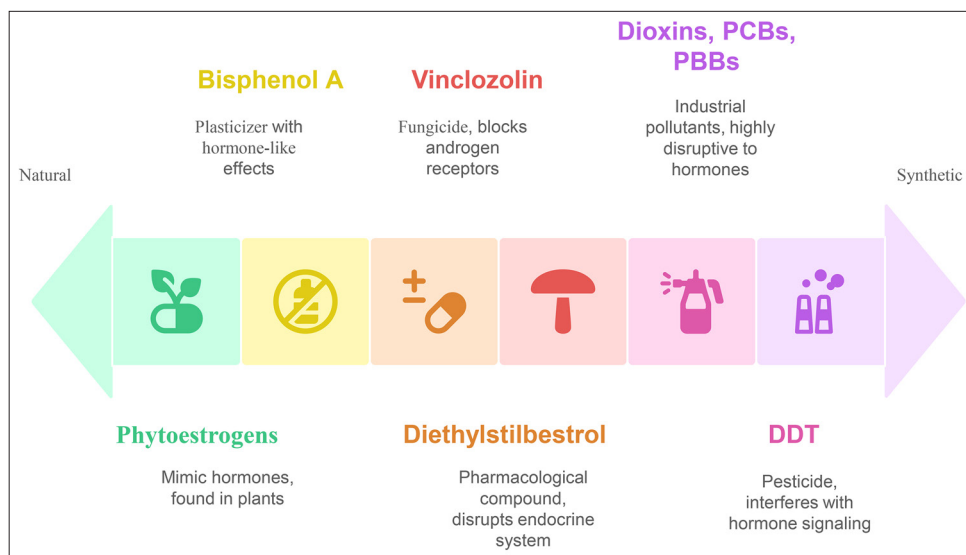
This review examines environmental pollutants that act as endocrine-disrupting chemicals (EDCs), focusing on their harmful effects on human and wildlife health. EDCs interfere with the endocrine system, which regulates hormones essential for growth, development, metabolism, and reproduction. Exposure to these pollutants has been linked to adverse health outcomes such as reproductive abnormalities, developmental delays, immune dysfunction, and metabolic disorders such as obesity and diabetes. By disrupting hormonal balance during critical developmental stages, EDCs threaten both individual health and ecosystem stability. The paper emphasizes the need for international regulatory frameworks, continuous scientific research, and increased public awareness to address the risks posed by these chemicals. EDCs often operate at low doses through complex biochemical pathways, making detection and regulation difficult. The review categorizes major EDCs, including industrial chemicals, pesticides, plastics, and pharmaceuticals and highlights their significant impacts on reproductive health in both humans and animals. Ultimately, the study advocates for informed policy decisions and preventive strategies to protect both environmental and public health.

**Keywords:** Bisphenol A, Endocrine disruptors, Environment pollutants, Heavy metals, Polychlorinated biphenyls

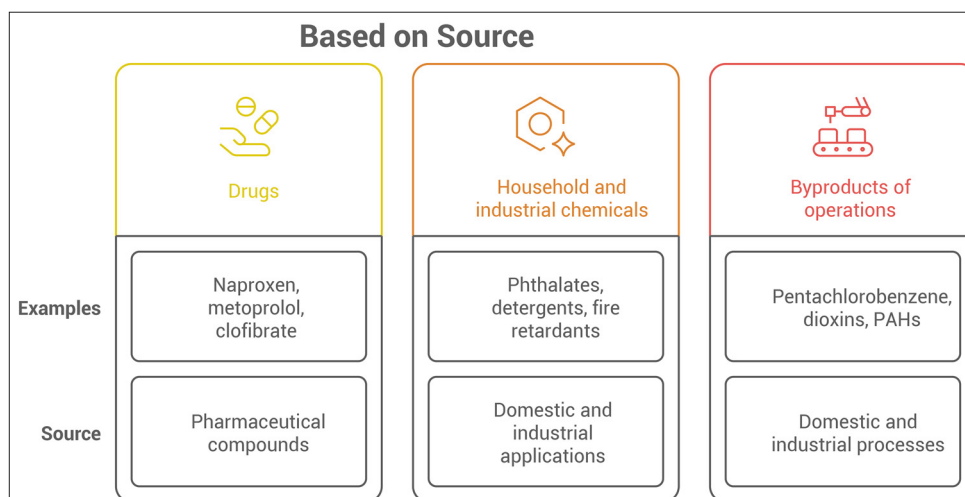
### INTRODUCTION

The endocrine system is crucial for regulating hormones, which are involved in numerous physiological processes such as growth, metabolism, reproduction, and mood regulation. Environmental pollutants, particularly those released from industrial, agricultural, and urban sources, have been identified as endocrine disruptors. These substances mimic, block, or alter the normal functioning of hormones, leading to adverse health effects in both humans and wildlife. This review aims to summarize the current understanding of how environmental pollutants affect the endocrine system and the resulting health implications.

“Any agent that interferes with the synthesis, secretion, transport, binding, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, reproduction, development, and/or behavior” is what the U.S. Environmental Protection Agency defines as an endocrine-disrupting compound.<sup>[1]</sup> To put it simply, endocrine disruptors are substances or combinations of substances that interfere with the normal action of hormones. Because of the high toxicity and carcinogenic nature of endocrine-disrupting chemicals (EDCs), prolonged exposure to these can lead to long-term health impacts. Owing to the traits of being persistent and bioaccumulative, both humans and the environment are currently confronted with a catastrophe that has not yet materialized.<sup>[2]</sup> Figures 1 and 2 categorize EDCs based on their type and source, respectively. Figure 1 specifies EDCs by type, distinguishing between naturally occurring and synthetic compounds, with examples<sup>[3]</sup>, and Figure 2 highlights their typical



**Figure 1:** Classification of endocrine-disrupting chemicals by type. PCB: Polychlorinated biphenyl, PBB: Polybrominated biphenyl, DDT: Dichlorodiphenyltrichloroethane



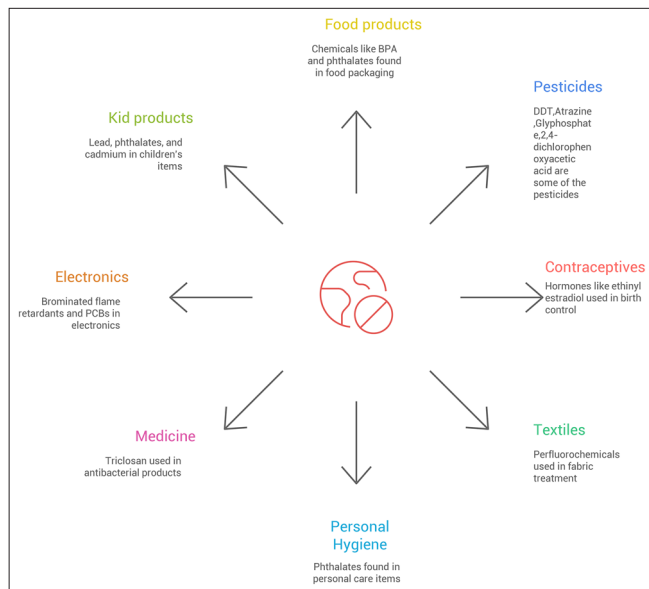
**Figure 2:** Classifications of endocrine-disrupting chemicals by source. PAH: Polycyclic aromatic hydrocarbons.

applications or sources.<sup>[4]</sup> Figure 3 highlights various EDCs – including bisphenol A (BPA), phthalates, lead, and atrazine – commonly found in everyday items such as food products, personal care items, electronics, textiles, medicines, and pesticides.<sup>[5,6]</sup>

### BRIEF INTRODUCTION TO THE ENDOCRINE SYSTEM AND ITS ROLE

The endocrine system, a network of glands and organs distributed throughout the body, plays a crucial role in regulating various physiological processes through the production and secretion of hormones directly into the bloodstream. These hormones, acting as chemical

messengers, travel through the circulatory system to reach specific target organs and tissues, where they influence a wide array of bodily functions. While endocrine signaling is slower than the nervous system's rapid electrical communication, its effects are far-reaching and enduring, significantly impacting development, metabolism, mood, reproduction, and the maintenance of homeostasis. Central to this system are glands, which serve as the primary producers and secretors of hormones, releasing them directly into the bloodstream to orchestrate vital activities such as immune regulation, growth, metabolism, reproduction, and electrolyte balance. Key glands include the pituitary gland, often called the “master gland” for its regulatory control over other endocrine glands and critical processes; the thyroid



**Figure 3:** Common endocrine-disrupting chemicals and their uses. DDT: Dichlorodiphenyltrichloroethane, BPA: Bisphenol A. PCB: Polychlorinated biphenyl.

gland, which governs metabolic rate and energy balance; and the adrenal glands, responsible for hormone production that aids stress response and maintains electrolyte and fluid homeostasis. Alongside other important glands such as the pancreas, ovaries, and testes, these organs form a complex hormonal network essential for sustaining the body's internal equilibrium and overall health. Disruption of this finely tuned hormonal network by external compounds, such as EDCs, can lead to significant physiological imbalances, and understanding how these chemicals interfere with hormone function at the molecular level is essential to assessing their impact on human and environmental health.

### HORMONAL FEEDBACK LOOPS

To maintain hormonal balance, the endocrine system primarily relies on feedback mechanisms, with negative feedback being the most common. In this process, elevated hormone levels trigger a response that suppresses further secretion. For example, when blood glucose rises, the pancreas releases insulin to promote glucose uptake; as glucose levels return to normal, insulin release is reduced. In contrast, positive feedback is less common and involves the amplification of hormone release. A classic example occurs during childbirth: Oxytocin-induced uterine contractions stimulate further oxytocin release, continuing until delivery.

While these feedback systems are vital for physiological stability, growing scientific concern has emerged over environmental pollutants that act as EDCs – substances capable of mimicking or interfering with natural hormones.

Recent research has highlighted the broad impact of EDCs on hormonal regulation and development. A 2024 study by the National Institute of Environmental Health Sciences identified musk ambrette, a fragrance in personal care products, as an EDC that may prematurely activate the reproductive axis in children by stimulating receptors such as gonadotropin-releasing hormone receptor and KISS1R, potentially contributing to early puberty in girls.<sup>[7]</sup> Similarly, a 2024 study from the Barcelona Institute for Global Health found that prenatal exposure to EDC mixtures was associated with poorer metabolic health in children, increasing the risk of metabolic syndrome later in life. These findings emphasize the urgent need for stronger public health measures to reduce EDC exposure, especially during critical developmental periods. To understand the mechanisms behind these effects, it is essential to examine how EDCs disrupt hormone signaling at the molecular level.

### WHY IT'S A GROWING CONCERN

The growing concern surrounding EDCs is amplified by their ubiquity, as these substances are not confined but rather are widespread, permeating various products we use daily and existing pervasively within the environment itself. Adding to this concern is their persistence; many EDCs exhibit a resistance to natural degradation processes, leading to their accumulation not only within diverse ecosystems but also within the bodies of living organisms over extended periods. Furthermore, research has highlighted the potential for low-dose effects, indicating that even exposures to relatively small amounts of these chemicals, particularly during critical and sensitive developmental windows such as the *in utero* period, can precipitate long-term and potentially adverse health consequences. The environmental ramifications of EDCs extend to the contamination of soil and waterways, subsequently impacting wildlife populations. Within aquatic environments, fish and amphibian species may exhibit noticeable signs of feminization or various reproductive abnormalities as a consequence of EDC exposure. Similarly, terrestrial wildlife, including birds and mammals, can experience disruptions to their natural breeding cycles and exhibit altered behavioral patterns due to the presence of these endocrine-interfering substances in their environment.

Endocrine disruptors act through various mechanisms that interfere with the normal functioning of hormones and their receptors. These mechanisms can be divided into several key categories. Some pollutants resemble naturally occurring hormones, such as estrogen, and can bind to hormone receptors, triggering similar biological responses.

For example, the synthetic estrogen-like compound BPA can bind to estrogen receptors and disrupt the endocrine system. Certain pollutants have structures similar to natural hormones like estradiol, allowing them to bind to hormone

receptors and trigger (or block) hormonal responses like BPA mimics estrogen.<sup>[8-10]</sup> BPA can bind to estrogen receptors  $\alpha$  and  $\beta$ , causing similar gene activation as estradiol.<sup>[11]</sup> Some pollutants can activate hormone receptor (agonists) and also can block receptor and prevent natural hormones from binding (antagonists) like dichlorodiphenyltrichloroethane (DDT) and its metabolite dichlorodiphenyldichloroethylene (DDE). DDE is an androgen receptor antagonist, affecting male reproductive development.<sup>[12]</sup> Some EDCs disrupt enzymes responsible for hormone production or degradation, altering hormonal balance. For example, polychlorinated biphenyls (PCBs) affect thyroid hormone levels by increasing their clearance from the blood.<sup>[13]</sup>

A few pollutants bind to hormone-binding proteins like thyroxine-binding globulin, reducing the availability of active hormones. EDCs can alter deoxyribonucleic acid (DNA) methylation and histone modification, leading to long-term or trans generational effect.<sup>[14]</sup> Vinclozolin, a fungicide, causes epigenetic changes in sperm, affecting several generations.<sup>[15]</sup> Natural hormones cannot bind and have their desired effects when some contaminants block hormone receptors. Dioxins, for instance, have the ability to bind to the aryl hydrocarbon receptor and interfere with regular signaling pathways.<sup>[16]</sup> Environmental pollutants can also interfere with the synthesis, metabolism, or elimination of hormones, leading to imbalances. Numerous pathways expose humans to a wide variety of substances. In the course of daily life, environmental contaminants are typically transferred to the human body unconsciously. Ingestion is the primary way that humans are exposed to EDCs, with inhalation and skin absorption also playing a role.

## ENVIRONMENT POLLUTANTS: ENDOCRINE DISRUPTORS

### Industrial chemicals

**BPA:** The plastic industry widely uses BPA ( $(\text{CH}_3)_2\text{C}(\text{C}_6\text{H}_4\text{OH})_2$ ), a synthetic organic chemical with two hydroxyphenyl groups, in the manufacturing of bottles, pipes, sports gear, compact discs, digital versatile discs, and numerous other consumer goods. Because of its structural resemblance to the estrogen hormone estradiol, BPA has been shown to have detrimental effects on both human and animal health in numerous studies. In both humans and animals, exposure to BPA is linked to serious endocrine-related disorders.<sup>[8]</sup> BPA is a major toxicant that interferes with the normal estrogen/androgen pathways leading to infertility in both sexes through many ways, including DNA damage in spermatozooids, altered methylation pattern, histone modifications, and miRNA expression.<sup>[17]</sup> There is increasing evidence that BPA has an impact on human fertility and is responsible for the reproductive pathologies,

*e.g.*, testicular dysgenesis syndrome, cryptorchidism, cancers, and decreased fertility in male and follicle loss in female.<sup>[18]</sup>

### Phthalates

Phthalates, a group of chemical compounds commonly used as plasticizers to increase the flexibility and durability of plastics, are found in a wide range of consumer products, including toys, food packaging, medical devices, and personal care items. Scientific studies have demonstrated that exposure to certain phthalates can interfere with the body's endocrine system, particularly by disrupting androgen signaling – a hormone pathway crucial for male reproductive development. This disruption has been linked to a variety of adverse health outcomes, including reduced testosterone levels, decreased sperm quality, and developmental abnormalities in the reproductive system, especially when exposure occurs during critical windows such as prenatal or early childhood development.

### PCBs

PCBs are a class of man-made chemicals that fall under the category of persistent organic pollutants (POPs), meaning they resist environmental degradation and can accumulate in the food chain over time. Although their production has been banned or severely restricted in many countries due to health concerns, PCBs continue to pose environmental and health risks because of their long-lasting presence in soil, water, and air. Research has shown that PCBs can interfere with the normal functioning of thyroid hormones, which are critical regulators of metabolism, growth, and brain development. Disruption of thyroid hormone signaling by PCBs has been particularly concerning in the context of neurodevelopment, as these hormones play a vital role during prenatal and early childhood brain development. Consequently, exposure to PCBs – especially during pregnancy and early life – has been associated with cognitive impairments, behavioral problems, and delays in neurological development.

### Pesticides and herbicides

#### DDT

DDT is a synthetic pesticide that was widely used in agriculture and for vector control, particularly to combat malaria-carrying mosquitoes. Although its use has been banned or restricted in many countries due to environmental and health concerns, DDT remains persistent in the environment and continues to be detected in soil, water, and even in human and animal tissues. One of the primary concerns associated with DDT is its estrogenic activity – it can mimic the hormone estrogen by binding to estrogen receptors, thereby disrupting the body's endocrine system.

This hormonal interference can lead to a range of adverse health effects, particularly in reproductive and neurological development. Studies have linked DDT exposure to altered sexual development, reduced fertility, and increased risk of reproductive disorders. Furthermore, prenatal or early-life exposure has been associated with neurodevelopmental impairments, including cognitive deficits, attention problems, and behavioral abnormalities, highlighting the long-term impact of EDCs like DDT on human health.

### ***Atrazine***

Atrazine is one of the most widely used herbicides in the world, commonly applied to control broadleaf and grassy weeds in crops such as corn and sugarcane. Despite its agricultural benefits, atrazine has raised significant environmental and public health concerns due to its persistence in soil and water and its potential to act as an EDC. Research has shown that atrazine can interfere with the endocrine system by altering estrogenic signaling pathways, even at low environmental concentrations. This disruption can lead to abnormal hormonal regulation, including the inappropriate activation of estrogen receptors, which may result in feminization of male organisms, reproductive abnormalities, and altered sexual development. Such effects have been observed in a range of species, particularly amphibians, where atrazine exposure has been linked to the development of hermaphroditic traits and impaired reproductive function. These findings have prompted ongoing debates about the safety and regulatory oversight of Atrazine use, particularly regarding its potential risks to wildlife and human health.

### ***Heavy metals***

Various heavy metals have been identified as EDCs, capable of interfering with hormonal signaling in both humans and wildlife. Heavy metals such as lead and mercury are well-documented for their harmful effects on the endocrine system. Lead, primarily known for its neurotoxic properties, can also disrupt thyroid hormone function, thereby impairing normal growth and developmental processes. Similarly, mercury exposure has been associated with disturbances in both thyroid and reproductive hormone activity, particularly in aquatic organisms, where it can alter reproductive cycles and developmental outcomes. Pharmaceuticals and personal care products are another major source of EDCs. For instance, synthetic estrogens found in oral contraceptives can enter aquatic ecosystems through wastewater effluents and mimic natural estrogens, disrupting the hormonal balance of fish and other aquatic life. Triclosan, an antimicrobial agent widely used in soaps, toothpaste, and other hygiene products, has also been shown to interfere with thyroid hormone regulation. These examples highlight the pervasive presence

of endocrine disruptors across various environmental sources and underscore the importance of monitoring and regulating their impact on both ecological and human health.

## **HEALTH IMPACTS OF ENDOCRINE DISRUPTION**

The effects of endocrine disruption are varied and influenced by factors such as the type of chemical involved, the dose, duration, and, importantly, the timing of exposure. Health outcomes associated with EDCs span multiple systems, including the neurodevelopmental, metabolic, and reproductive domains. Exposure during sensitive developmental windows – particularly prenatal and early postnatal periods – has raised concerns about links to cognitive and behavioral disorders. Although conditions such as attention-deficit hyperactivity disorder and autism spectrum disorders (ASD) are complex and multifactorial, involving genetic, environmental, and developmental influences, some studies suggest that certain EDCs may contribute to neurodevelopmental risk by disrupting hormone-regulated brain development.<sup>[19,20]</sup> EDCs are also strongly implicated in metabolic disturbances, contributing to obesity and type 2 diabetes by interfering with insulin signaling and lipid metabolism.<sup>[21,22]</sup> In the reproductive system, these chemicals can cause developmental and functional abnormalities, including altered sexual differentiation, reduced fertility, and hormonal imbalances.<sup>[3,23]</sup> Substances such as BPA, phthalates, and specific pesticides have been closely linked to such reproductive outcomes. Given the breadth of potential health impacts and the ongoing exposure to these pollutants, further research and stronger regulatory frameworks are essential to better understand risks and reduce harm, particularly during vulnerable stages of human development.

## **REPRODUCTIVE HEALTH**

EDCs, such as BPA, phthalates, and perfluoroalkyl substances (PFASs), have been extensively studied for their adverse effects on human reproductive health. A growing body of evidence has linked exposure to these compounds with impaired fertility in both males and females. In males, BPA exposure has been associated with reduced sperm count, abnormal sperm morphology, decreased motility, increased DNA fragmentation, hormonal dysregulation, and testicular atrophy. Similarly, phthalate exposure has been correlated with compromised semen quality and disrupted androgen synthesis.<sup>[24]</sup> PFASs have also been shown to impair male reproductive function by disrupting hormonal balance, damaging seminiferous tubules, and reducing sperm quality and count.<sup>[25]</sup> These effects are primarily attributed to the interference of EDCs with the hypothalamic–pituitary–gonadal (HPG) axis and the modulation of key reproductive hormones, including testosterone and follicle-stimulating

hormone.

In females, BPA and phthalates disrupt hormonal homeostasis essential for ovulatory function, leading to menstrual irregularities, reduced ovarian reserve, and increased risks of infertility. BPA exposure, in particular, has been implicated in the etiology of polycystic ovary syndrome and endometriosis. Phthalates may also impair ovarian folliculogenesis and alter uterine receptivity, contributing to reproductive disorders.<sup>[26]</sup> Furthermore, both BPA and phthalates are capable of crossing the placental barrier, raising concerns regarding their impact during critical windows of fetal development. Prenatal exposure to these chemicals has been linked to structural malformations of the reproductive tract, as well as long-term neurodevelopmental impairments in offspring. Another study highlighted EDCs, oxidative stress, inflammation, and altered intra-ovarian signaling collectively impair ovarian function and female reproductive health.<sup>[27]</sup>

Collectively, these findings underscore the need for robust regulatory frameworks aimed at limiting human exposure to EDCs, particularly during sensitive developmental stages.<sup>[28]</sup> Continued research is essential to further elucidate the mechanisms of EDC action and to inform public health interventions that mitigate their reproductive and developmental consequences.

### The male reproductive system

The male reproductive system consists of the testes, epididymis, vas deferens, prostate, and seminal vesicles. These organs work in close coordination to carry out spermatogenesis, maturation, storage, and ejaculation of

sperm [Table 1]. In addition to these internal organs, two external structures – the scrotum and the penis – also play important roles. The scrotum provides a protective sac and maintains the optimal temperature for sperm production, while the penis is responsible for delivering sperm during ejaculation.

Androgens are a major group of hormones that are required for male development. The main androgen is testosterone, which is produced in Leydig cells, other hormones include inhibin B and Müllerian inhibiting substance produced in Sertoli cells. HPG axis modulates and regulates the developmental and functional attributes of the male reproductive system.<sup>[29]</sup> Owing to the complex process of spermatogenesis, testes are inherently vulnerable to disrupting chemicals and environmental factors. Previous studies has noted that due to year an increase in adverse environmental and lifestyle conditions, sperm count in humans has declined over the past 50 years.<sup>[30]</sup> POPs, chemical exposure, and lifestyle significantly affect testicular functions and spermatogenesis.<sup>[31]</sup> Therefore, in a control setup, the conditions resulted following exposure could not accurately represent the adversity caused by a single compound.

Importantly, the adverse effects of EDCs are not limited to humans but extend across species, impacting wildlife and entire ecosystems. Many wildlife species, particularly aquatic organisms, are sensitive to low levels of pollutants, which can disrupt reproductive cycles, cause intersex conditions, and reduce biodiversity. The effects on ecosystems can have cascading impacts on food webs and ecosystems. Recent scientific studies have further elucidated the detrimental effects of EDCs on wildlife and ecosystems, particularly aquatic organisms. For instance, research has shown that exposure to EDCs can lead to reproductive abnormalities in fish species, such as intersex conditions where individuals exhibit both male and female characteristics. These disruptions not only affect individual species but can also lead to population declines and altered community structures within aquatic ecosystems. Moreover, the presence of EDCs in water bodies has been linked to changes in reproductive behaviors and success rates among various aquatic species, potentially leading to long-term ecological consequences. These findings underscore the critical need for monitoring and regulating EDCs to protect wildlife and maintain ecosystem integrity.<sup>[32]</sup>

Recent scientific investigations have further elucidated the detrimental effects of EDCs such as BPA and phthalates on reproductive health and fetal development. A notable 2025 study conducted in Italy revealed the presence of microplastics in the ovarian follicular fluid of women undergoing fertility treatments.<sup>[33]</sup> These microplastics are significant because they can act as vectors for EDCs like BPA

**Table 1:** Participants of the reproductive system and their main functions.

Organs	Functions
Penis	Protects the urethra, contains corpora cavernosum and prepuce protects the glans penis
Scrotum	Protective loose sac provides compartments for organs such as testis, epididymis, and vas deferens. Regulates temperature
Testes	Production of sperm and testosterone
Epididymis	Storage and transportation, sperm maturation
Vas deferens	Transportation of sperm from the epididymis to the urethra
Spermatic cord	Protects the ductus deferens or vas, internal and external spermatic arteries, etc.
Prostate gland	Production traces of seminal fluid and prostatic fluid that increases sperm viability
Seminal vesicle	Production of seminal fluid.

and phthalates, thereby introducing these harmful chemicals directly into the ovarian environment. This contamination is concerning, as it may impair oocyte (egg) development by disrupting the delicate hormonal balance necessary for folliculogenesis and ovulation, potentially leading to reduced fertility or compromised outcomes in assisted reproductive technologies.<sup>[34]</sup>

Moreover, the impact of EDCs extends beyond reproductive organs to influence fetal brain development. Prenatal exposure to BPA has been increasingly associated with adverse neurodevelopmental outcomes. A 2024 epidemiological study linked elevated maternal BPA levels during pregnancy to a higher incidence of ASD in male offspring. The proposed mechanism involves BPA's disruption of aromatase activity, an enzyme critical for converting androgens to estrogens in the developing brain. This disruption may alter the delicate hormonal milieu that guides neural differentiation and synapse formation, resulting in neurological and behavioral changes observed in ASD.<sup>[35]</sup> These findings add to a growing body of evidence suggesting that even low-dose exposure to EDCs during sensitive periods of prenatal development can have profound and lasting effects on offspring health.

Collectively, these studies underscore the urgent need for stringent regulatory policies aimed at reducing human exposure to EDCs, particularly during critical windows such as preconception, pregnancy, and early childhood. Public health initiatives should focus on minimizing contact with known sources of BPA, phthalates, and microplastics – ranging from consumer products to environmental contamination – to protect reproductive function and neurodevelopment. Continued research is essential to further clarify the mechanisms by which these chemicals exert their effects and to identify safer alternatives that mitigate risk.

## REGULATORY AND MITIGATION EFFORTS

In response to growing concerns over the health and environmental impacts of EDCs, many countries have implemented regulations aimed at limiting the use of these harmful substances. These regulations seek to reduce human and environmental exposure to pollutants, especially those that are persistent and bioaccumulative. Key international frameworks, such as the Stockholm Convention, address POPs such as PCBs and DDT,<sup>[36]</sup> while the European Union's Registration, Evaluation, Authorization, and Restriction of Chemicals (REACH) regulation focuses on managing chemical risks and ensuring safer use of chemicals.<sup>[37]</sup>

Despite these efforts, significant challenges remain. Many EDCs persist in the environment for extended periods, making remediation difficult. In addition, the current regulatory testing frameworks often lack comprehensive

methods to fully assess endocrine-disrupting potential. Specifically, there is a critical need for more advanced *in vitro* assays, such as receptor-binding and cell-based reporter gene assays, as well as high-throughput screening techniques that can evaluate multiple hormonal pathways simultaneously. These methods would complement traditional *in vivo* animal testing, providing faster and more mechanistic insights into how EDCs affect endocrine function.

Moreover, a notable gap exists between scientific knowledge and regulatory implementation, particularly in developing countries. For example, in parts of Southeast Asia, where pesticide use remains high and regulatory enforcement is limited, exposure to banned or unregulated EDCs continues unabated, contributing to adverse health outcomes without adequate policy response.<sup>[38]</sup> This illustrates the urgent need for capacity building, improved infrastructure, and international collaboration to support these regions in adopting and enforcing effective regulations.

Addressing these challenges will require strengthened global cooperation, the integration of cutting-edge testing methods into regulatory frameworks, and targeted efforts to close policy gaps, especially in vulnerable and resource-limited regions. Only through such comprehensive action can the widespread risks posed by EDCs to human health and ecosystems be effectively mitigated.

## CONCLUSION

Endocrine-disrupting environmental pollutants present considerable risks to human health and ecosystems by interfering with hormonal regulation and impacting reproductive, metabolic, and developmental processes. While significant advances have been made in elucidating their mechanisms of action, substantial challenges persist in effectively regulating and mitigating exposure. Ongoing research is imperative to refine detection techniques – particularly for low-dose exposures and complex biochemical pathways – and to understand the combined and synergistic effects of multiple chemicals. Moreover, closing the gap between scientific evidence and regulatory implementation, especially in resource-limited regions, remains essential. Strengthening international collaboration through frameworks such as the Stockholm Convention and the European Union's REACH regulation, alongside evidence-based policy-making and targeted preventive measures, is critical to safeguarding public health, preserving ecosystems, and protecting future generations from the adverse effects of endocrine disruptors.

**Ethical approval:** The Institutional Review Board approval is not required.

**Declaration of patient consent:** Patient's consent is not required as there are no patients in this study.

**Financial support and sponsorship:** Nil.

**Conflicts of interest:** There are no conflicts of interest.

**Use of artificial intelligence (AI)-assisted technology for manuscript preparation:** The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

## REFERENCES

- Kavlock RJ, Daston GP, DeRosa C, Fenner-Crisp P, Gray LE, Kaattari S, *et al.* Research needs for the risk assessment of health and environmental effects of endocrine disruptors: A report of the U.S. EPA-sponsored workshop. *Environ Health Perspect* 1996;104 Suppl 4:715-40.
- UNEP and WHO United Nations Environment Programme and the World Health Organization. State of the science of endocrine disrupting chemicals-2012. Switzerland: WHO; 2013. p. 1.
- Diamanti-Kandarakis E, Bourguignon JP, Giudice LC, Hauser R, Prins GS, Soto AM, *et al.* Endocrine-disrupting chemicals: An endocrine society scientific statement. *Endocr Rev* 2009;30:293-342.
- Caliman FA, Gavrilescu M. Pharmaceuticals, personal care products and endocrine disrupting agents in the environment - a review. *CLEAN Soil Air Water* 2009;37:277-303.
- Gore AC, Crews D, Doan LL, La Merrill M, Patisaul H, Zota A. Introduction to endocrine disrupting chemicals (EDCs): A guide for public interest organizations and policy-makers. Washington, DC: Endocrine Society; 2014. p. 21-2.
- Dodson RE, Nishioka M, Standley LJ, Perovich LJ, Brody JG, Rudel RA. Endocrine disruptors and asthma-associated chemicals in consumer products. *Environ Health Perspect* 2012;120:935-43.
- Demir A, Aydin A, Büyükgebiz A. Thematic review of endocrine disruptors and their role in shaping pubertal timing. *Children (Basel)* 2025;12:93.
- Rubin BS. Bisphenol A. An endocrine disruptor with widespread exposure and multiple effects. *J Steroid Biochem Mol Biol* 2011;127:27-34.
- Molina-Molina JM, Jiménez-Díaz I, Fernández MF, Rodríguez-Carrillo A, Peinado FM, Mustieles V, *et al.* Determination of bisphenol A and bisphenol S concentrations and assessment of estrogen- and anti-androgen-like activities in thermal paper receipts from Brazil, France, and Spain. *Environ Res* 2019;170:406-15.
- Yuan M, Chen S, Zeng C, Fan Y, Ge W, Chen W. Estrogenic and non-estrogenic effects of bisphenol A and its action mechanism in the zebrafish model: An overview of the past two decades of work. *Environ Int* 2023;176:107976.
- Matthews JB, Twomey K, Zacharewski TR. *In vitro* and *in vivo* interactions of bisphenol A and its metabolite, bisphenol A glucuronide, with estrogen receptors  $\alpha$  and  $\beta$ . *Chem Res Toxicol* 2001;14:149-57.
- Kelce WR, Stone CR, Laws SC, Gray LE, Kemppainen JA, Wilson EM. Persistent DDT metabolite p,p'-DDE is a potent androgen receptor antagonist. *Nature* 1995;375:581-5.
- Zoeller RT. Environmental chemicals impacting the thyroid: Targets and consequences. *Thyroid* 2007;17:811-7.
- Boas M, Feldt-Rasmussen U, Skakkebaek NE, Main KM. Environmental chemicals and thyroid function. *Eur J Endocrinol* 2006;154:599-611.
- Anway MD, Cupp AS, Uzumcu M, Skinner MK. Epigenetic transgenerational actions of endocrine disruptors and male fertility. *Science* 2005;308:1466-9.
- Mandal PK. Dioxin: A review of its environmental effects and its aryl hydrocarbon receptor biology. *J Comp Physiol B* 2005;175:221-30.
- Amir S, Shah ST, Mamoulakis C, Docea AO, Kalantzi OI, Zachariou A, *et al.* Endocrine disruptors acting on estrogen and androgen pathways cause reproductive disorders through multiple mechanisms: A review. *Int J Environ Res Public Health* 2021;18:1464.
- Rochester JR. Bisphenol A and human health: A review of the literature. *Reprod Toxicol* 2013;42:132-55.
- Mustieles V, Fernández MF. Bisphenol A shapes children's brain and behavior: Towards an integrated neurotoxicity assessment including human data. *Environ Health* 2020;19:66.
- La Merrill MA, Vandenberg LN, Smith MT, Goodson W, Browne P, Patisaul HB, *et al.* Consensus on the key characteristics of endocrine-disrupting chemicals as a basis for hazard identification. *Nat Rev Endocrinol* 2020;16:45-57.
- Janesick AS, Blumberg B. Obesogens: An emerging threat to public health. *Am J Obstet Gynecol* 2016;214:559-65.
- Heindel JJ, Blumberg B, Cave M, Machtinger R, Mantovani A, Mendez MA, *et al.* Metabolism disrupting chemicals and metabolic disorders. *Reprod Toxicol* 2017;68:3-33.
- Bergman Å, Heindel JJ, Kasten T, Kidd KA, Jobling S, Neira M, *et al.* The impact of endocrine disruption: A consensus statement on the state of the science. *Environ Health Perspect* 2013;121:A104-6.
- Salami EA, Rotimi OA. The impact of Bisphenol-A on human reproductive health. *Toxicol Rep* 2024;13:101773.
- Singh S, Singh SK. Male reproductive health at risk due to exposure to perfluoroalkyl substances: Recent research highlights. *J Reprod Healthc Med* 2021;2:13.
- Basso CG, De Araujo-Ramos AT, Martino-Andrade AJ. Exposure to phthalates and female reproductive health: A literature review. *Reprod Toxicol* 2022;109:61-79.
- Biswas S, Mukherjee U, Maitra S. Endocrine disruption and female reproductive health: Implications on cross-talk between endocrine and autocrine/paracrine axes in the ovary. *J Reprod Healthc Med* 2020;1:2.
- Hoffman SS, Tang Z, Dunlop A, Brennan PA, Huynh T, Eick SM, *et al.* Impact of prenatal phthalate exposure on newborn metabolome and infant neurodevelopment. *Nat Commun* 2025;16:2539.
- Silva MS, Giacobini P. New insights into anti-Müllerian hormone role in the hypothalamic-pituitary-gonadal axis and neuroendocrine development. *Cell Mol Life Sci* 2021;78:1-16.
- Swan SH, Elkin EP, Fenster L. The question of declining sperm density revisited: An analysis of 101 studies published 1934-1996. *Environ Health Perspect* 2000;108:961-6.
- Sharpe RM. Environmental/lifestyle effects on spermatogenesis. *Philos Trans R Soc B* 2010;365:1697-712.
- Carnevali O, Santangeli S, Forner-Piquer I, Basili D, Maradonna F. Endocrine-disrupting chemicals in aquatic

- environment: What are the risks for fish gametes? *Fish Physiol Biochem* 2018;44:1561-76.
33. Grechi N, Franko R, Rajaraman R, Stöckl JB, Trapphoff T, Dieterle S, *et al.* Microplastics are present in women's and cows' follicular fluid and polystyrene microplastics compromise bovine oocyte function *in vitro*. *eLife* 2023;12:86791.
  34. Zurub RE, Cariaco Y, Wade MG, Bainbridge SA. Microplastics exposure: Implications for human fertility, pregnancy and child health. *Front Endocrinol (Lausanne)* 2024;14:1330396.
  35. Thongkorn S, Kanlayaprasit S, Kasitipradit K, Lertpeerapan P, Panjabud P, Hu VW, *et al.* Investigation of autism-related transcription factors underlying sex differences in the effects of bisphenol A on transcriptome profiles and synaptogenesis in the offspring hippocampus. *Biol Sex Differ* 2023;14:8.
  36. United Nations Environment Programme (UNEP). Stockholm convention on persistent organic pollutants (POPs); 2021. Available from: <https://www.pops.int> [Last accessed on 2025 Jul 31].
  37. European Chemicals Agency (ECHA). REACH regulation (EC); 2020. Available from: <https://echa.europa.eu/regulations/reach> [Last accessed on 2025 Jul 31].
  38. United Nations Environment Programme (UNEP). Chemicals in Asia: Emerging threats and opportunities; 2023. Available from: <https://www.unep.org> [Last accessed on 2025 Jul 31].

**How to cite this article:** Srivastava S, Balyan S. Environmental pollutants as endocrine disruptors. *J Reprod Healthc Med.* 2025;6:17. doi: 10.25259/JRHM\_22\_2025