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THE ROLE OF ENDOCRINE DISRUPTORS IN EARLY PUBERTY AND HORMONAL IMBALANCES IN WOMEN

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ABSTRACT

In recent decades, puberty has been occurring at younger ages, particularly among girls. While genetics and nutrition are well-known influences, increasing evidence suggests that endocrine-disrupting chemicals (EDCs) also play a role. EDCs are compounds that interfere with hormones by mimicking, blocking, or altering their activity. Found in plastics, cosmetics, pesticides, and processed foods, they represent widespread, unavoidable exposures in modern life.

Early puberty carries significant health risks beyond physical changes. Girls who mature earlier are more likely to develop hormonal imbalances, menstrual irregularities, metabolic disorders, and reproductive conditions such as polycystic ovary syndrome, endometriosis, and infertility. Prolonged estrogen exposure is also linked to hormone-sensitive cancers. Metabolic disruptions may further increase risks of obesity, insulin resistance, and cardiovascular disease.

Evidence on EDCs remains mixed: some studies associate higher levels of bisphenol A, phthalates, and parabens with early puberty, while others report inconsistent effects. Variability may stem from genetic susceptibility, metabolism, or cumulative exposure to environmental factors.

This review explores how EDCs may influence pubertal timing and long-term hormonal health, emphasizing the need to recognize environmental contributors to female reproductive development and to strengthen the regulation of endocrine-disrupting chemicals.

KEYWORDS

Endocrine Disruptors, Puberty, Bisphenol A, Phthalates, Parabens, Dioxins, Pesticides, Polycystic Ovary Syndrome, Endometriosis

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Introduction.

Over the past several decades, the age at which puberty begins has steadily declined, raising concerns among healthcare professionals, researchers, and the public alike. This trend is particularly pronounced among girls and has sparked intense investigation into the underlying causes. The phenomenon of earlier pubertal onset poses not only a medical and scientific challenge, but also a societal one, as it profoundly affects physical health, mental well-being, and social development (Kaplowitz, 2025). While improvements in nutrition and lifestyle changes are often cited as primary factors, mounting scientific evidence suggests that the pervasive presence of endocrine-disrupting chemicals (EDCs) is a significant and often overlooked contributor to the issue (Endocrine Society, 2025).

EDCs are found in a wide array of everyday items, including plastics, personal care products, household cleaners, and pesticides, making exposure nearly unavoidable in modern life. These chemicals have the ability to interfere with the body's intricate hormonal systems, either by mimicking, blocking, or altering the activity of hormones (Wróbel, 2014). The repercussions of such disruption are far-reaching, extending beyond early puberty to impact long-term reproductive health, metabolic processes, and even the risk of hormone-dependent cancers. As our understanding of the complex interplay between environmental exposures and biological development grows, so does the urgency to identify and mitigate these risks (Mayo Clinic Staff, 2025).

Understanding the role of EDCs is therefore crucial for protecting the health and well-being of current and future generations. This article examines the impact of EDCs on pubertal timing and hormonal balance in women, highlighting current scientific knowledge, research gaps, and the urgent need for regulatory and public health interventions (Wróbel, 2014).

This study aims to investigate the impact of endocrine-disrupting chemicals (EDCs) on the onset of early puberty, particularly in girls, and their potential long-term effects on hormonal balance and reproductive health. The primary objective is to explore the mechanisms by which EDCs, such as bisphenol A (BPA), phthalates, parabens, dioxins, and pesticides, interfere with hormonal regulation, leading to alterations in pubertal timing and subsequent reproductive health complications. This research will also assess the cumulative effects of these chemicals on metabolic health and investigate their contribution to conditions like polycystic ovary syndrome (PCOS), endometriosis, and hormone-sensitive cancers (Endocrine Society, 2025; Sharma, 2019).

Methodology

This review paper employs a comprehensive and systematic approach to gather, evaluate, and synthesize current scientific knowledge regarding the role of endocrine-disrupting chemicals (EDCs) in early puberty and hormonal imbalances in women. The methodology is structured as follows:

A literature search strategy identified relevant studies through electronic database searches using PubMed, Google Scholar, and Scopus. The search included peer-reviewed articles, systematic reviews, meta-analyses, clinical trials, and guidelines published in English and Polish up to 2025. Search terms included combinations of "endocrine disruptors," "puberty," "hormonal imbalance," "women's health," "bisphenol A," "phthalates," "parabens," "dioxins," "pesticides," "PCOS," and "early menarche."

Inclusion and exclusion criteria included studies that addressed the effects of EDCs on pubertal timing, hormonal regulation, reproductive health, or metabolic outcomes in females. Animal studies, human epidemiological studies, and mechanistic research were considered. Articles focusing solely on male health, unrelated environmental exposures, or non-peer-reviewed sources were excluded.

Key findings from the selected literature were extracted, including study design, sample size, exposure assessment methods, main outcomes, and limitations. The collected data were synthesized to provide a balanced overview of the current evidence, with particular attention paid to areas of consensus and ongoing controversy.

The methodological quality of studies was assessed based on criteria such as sample representativeness, exposure measurement accuracy, and control for confounding factors. Discrepancies and research gaps were highlighted to inform recommendations for future investigation.

A literature review was conducted using PubMed and Google Scholar. The study encompasses international and Polish guidelines, scientific articles, and clinical data related to endocrine disruptors and their impact on hormonal imbalances in women, as well as during puberty.

Results

Puberty and Precocious Puberty in Women

Puberty is a pivotal developmental stage during which children undergo significant physical and hormonal changes, transitioning into sexual maturity. Typically, girls begin puberty between the ages of 8 and 13, while boys start between 9 and 14. However, over the past 150 years, there has been a notable decline in the average age of puberty onset. For instance, in 1860, the average age for girls to begin puberty was approximately 16.6 years; by 1980, it had decreased to around 12.5 years (NHS, 2025; Boseley, 2012).

This trend toward earlier puberty, especially among girls, has been observed across various populations. Factors contributing to this shift include improved nutrition, resulting in an increased body mass index (BMI), and exposure to endocrine-disrupting chemicals present in the environment. Additionally, genetic predispositions and certain lifestyle factors, such as reduced physical activity, have been implicated (Carel et al., 2023)

Precocious puberty, defined as the onset of puberty before age 8 in girls and before age 9 in boys, has become an area of growing concern. This condition can lead to various complications, including reduced adult height due to early closure of growth plates, psychological challenges stemming from the mismatch between physical development and emotional maturity, and potential long-term health risks like increased susceptibility to certain cancers (Mayo Clinic Staff, 2025).

The etiology of precocious puberty can be categorized into central and peripheral causes. Central precocious puberty (CPP) results from the premature activation of the hypothalamic-pituitary-gonadal axis. While many cases are idiopathic, especially in girls, potential causes include central nervous system

abnormalities such as tumors, infections, or injuries (Cleveland Clinic, 2025). Peripheral precocious puberty arises from the secretion of sex hormones independent of gonadotropin stimulation, often due to conditions like congenital adrenal hyperplasia, ovarian cysts or tumors, and exposure to external sources of estrogen or testosterone (Sadeghi et al., 2014; Kaplowitz, 2025)

Early diagnosis and management are crucial to mitigate the adverse effects associated with precocious puberty. Treatment strategies typically involve addressing the underlying cause and may include the use of gonadotropin-releasing hormone (GnRH) analogs to halt the progression of puberty until a more appropriate age. Regular monitoring and a multidisciplinary approach are essential to ensure optimal outcomes for affected children (Kaplowitz, 2025).

In summary, while puberty traditionally occurs within a specific age range, recent trends indicate an earlier onset in both girls and boys. Understanding the factors contributing to this shift and recognizing the signs of precocious puberty are vital for timely intervention and support.

Mechanism of Endocrine Disruptors in Endocrine Dysregulation

Endocrine-disrupting chemicals (EDCs) are exogenous substances that interfere with the body's hormonal systems, leading to adverse health outcomes. These chemicals disrupt endocrine function through several primary mechanisms (Endocrine Society, 2025).

According to the Endocrine Society, there are over 85000 man-made substances, and nearly 1000 of them can be linked to hormonal imbalances and other potentially harmful effects. Most of these chemicals have never been thoroughly tested, making it challenging to estimate their far-reaching consequences (Endocrine Society, 2025).

There are multiple ways in which EDCs can interfere with the hormonal system, one of which is mimicking natural hormones. EDCs can imitate endogenous hormones by binding to specific hormone receptors, thereby triggering similar physiological responses. For example, bisphenol A (BPA), commonly found in plastics, acts as a xenoestrogen by binding to estrogen receptors. This mimicry can lead to altered reproductive development and function (Endocrine Society, 2025).

Another way EDCs can interfere with hormonal balance is by blocking hormone receptors. Some EDCs function as antagonists, binding to hormone receptors without activating them, thus preventing natural hormones from exerting their effects. Phthalates, used as plasticizers, have been shown to interfere with testosterone production by inhibiting androgen receptors, leading to reproductive anomalies in males and females (Tiwari et al, 2024).

It can also alter hormone metabolism by modifying the synthesis, breakdown, or clearance of hormones, disrupting hormonal balance. Parabens, commonly used as preservatives in cosmetics, can affect the metabolism of estrogens, potentially leading to hormonal imbalances (Gore et al., 2010).

Understanding these mechanisms is crucial for assessing the risks associated with EDC exposure and implementing strategies to mitigate their impact on human health (Rochester, 2013).

Major Endocrine Disruptors

Bisphenol A (BPA)

Bisphenol A has become widely recognized as an environmental endocrine disruptor with significant implications for women's health, particularly in terms of reproductive function. BPA is a man-made chemical often used as a plasticizer in the manufacturing of polycarbonate plastics and epoxy resins. People can be exposed to BPA when it leaches from food and drink containers into their contents, especially when heated, at a specific pH level, or during regular use (Tiwari et al, 2024). BPA acts as an endocrine disruptor by weakly binding to estrogen receptors ERα and ERβ. Additionally, BPA can affect the body by activating the GPER (GPR30) membrane receptor and other receptors, such as estrogen-related receptors (ERRs (Rochester, 2013).

BPA's potential to interfere with hormone regulation is due to its ability to mimic estrogen, a hormone crucial for many physiological processes, including reproduction. Studies indicate that BPA exposure can lead to hormonal imbalances in women, affecting menstrual cycles, ovulation, and ovarian reserve, the total number of viable eggs in the ovaries (Vandenberg et al., 2012). This disruption can result in a reduced ability to conceive and may contribute to conditions like polycystic ovarian syndrome (PCOS), which is characterized by hormonal irregularities, cysts on the ovaries, and infertility (Sugiura-Ogasawara et al., 2014). Furthermore, BPA exposure has been linked to the development of fibroids, non-cancerous growths in the uterus, and endometriosis, a condition where uterine-like tissue grows outside the uterus, causing chronic pain and infertility (Gore et al., 2015). These conditions are not only associated with reduced fertility but also with

significant long-term health complications. Additionally, BPA exposure may increase the risk of metabolic disorders, including obesity and diabetes, which have been linked to hormone disturbances (Zhang et al., 2021). The widespread use of BPA in plastics and food containers raises concerns about cumulative exposure to this chemical, especially given its ability to leach into food and beverages, particularly when containers are heated (Martínez-Pacheco et al., 2021). The potential toxic effects of BPA on women's hormonal health highlight the importance of regulating its presence in consumer products, especially those that come into direct contact with food. Research continues to uncover the complex ways in which BPA impacts female reproductive health, suggesting the need for precautionary measures to protect women from its potential harm

Phthalates

Pthalates exert their endocrine-disrupting effects by interfering with the body's hormonal signaling pathways, particularly those regulated by the hypothalamic-pituitary-gonadal (HPG) axis. These chemicals can act as anti-androgens, reducing testosterone levels, and they may also interfere with estrogen synthesis and signaling (Gore et al., 2015). One key mechanism involves the inhibition of steroidogenesis, the process by which hormones like estrogen and progesterone are produced. For example, di-(2-ethylhexyl) phthalate (DEHP) and its metabolites have been shown to suppress the activity of aromatase, an enzyme responsible for converting androgens into estrogens, leading to hormonal imbalances that can affect puberty timing and menstrual cycle regularity (Zhang et al., 2021).

Additionally, phthalates can alter the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus, which in turn affects the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the pituitary gland. Disruptions in LH and FSH levels can impair ovarian function, leading to anovulation or irregular cycles, conditions commonly seen in disorders such as polycystic ovary syndrome (PCOS) (Martínez-Pacheco et al., 2021). Some phthalates may also bind to estrogen receptors (ERα and ERβ), either blocking or mimicking natural estrogen activity, which can result in inappropriate hormonal signaling, early breast development, or delayed reproductive maturation (Kay et al., 2022).

The cumulative effect of these disruptions can have long-term consequences for female reproductive health, increasing the risk of infertility, hormone-related cancers, and metabolic disorders. Given their widespread presence in everyday products, limiting phthalate exposure, especially during critical developmental windows, may be essential in preventing these adverse effects Martínez-Pacheco et al., 2021).

Parabens

Parabens are a class of preservatives widely used in cosmetics, pharmaceuticals, and personal care products. They have been implicated in endocrine disruption, particularly in relation to female reproductive health, puberty, and hormonal regulation. These chemicals can mimic estrogen by binding to estrogen receptors (ERα and ERβ), potentially leading to altered hormonal signaling and reproductive dysfunction (Darbre & Harvey, 2014). Research suggests that exposure to parabens, particularly during critical developmental windows, may contribute to early puberty, menstrual irregularities, and fertility issues by disrupting the hypothalamic-pituitary-gonadal (HPG) axis (Boberg et al., 2016).

One of the primary mechanisms by which parabens affect hormonal balance is through their weak estrogenic activity. Although parabens are less potent than natural estrogen, their cumulative effect, especially with prolonged exposure, can disrupt normal endocrine function. Studies have shown that parabens, such as butylparaben and propylparaben, can increase the expression of estrogen-responsive genes and stimulate breast cell proliferation, raising concerns about their potential role in hormone-dependent conditions like breast cancer and endometriosis (Wróbel, 2014). Additionally, parabens can interfere with gonadotropin-releasing hormone (GnRH) secretion, which regulates the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), potentially leading to ovulatory dysfunction and decreased fertility (Vo et al., 2010).

Paraben exposure has also been linked to earlier onset of puberty in girls, with studies indicating that higher urinary concentrations of parabens correlate with increased breast development at a younger age (Harley et al., 2019). Given their widespread presence in everyday products, concerns about their long-term impact on female reproductive health have led to increasing regulatory scrutiny and consumer demand for paraben-free alternatives. Further research is necessary to fully understand the extent of their endocrine-disrupting effects and establish safe exposure limits (Wróbel, 2014).

Dioxins

Dioxins are a group of highly toxic environmental pollutants that have been linked to significant disruptions in female reproductive health, puberty, and hormonal regulation. These persistent organic pollutants (POPs) are primarily byproducts of industrial processes and can accumulate in fatty tissues, leading to long-term endocrine disruption. Dioxins exert their effects primarily by binding to the aryl hydrocarbon receptor (AhR), a transcription factor that regulates genes involved in hormone metabolism and reproductive function (Mimura, 2003). This interaction can disrupt the normal function of the hypothalamic-pituitary-gonadal (HPG) axis, resulting in menstrual irregularities, delayed puberty, and reduced fertility (Eskenazi et al., 2018).

One of the key mechanisms by which dioxins disrupt hormonal balance is through their impact on estrogen signaling. By activating AhR, dioxins can modulate the expression of cytochrome P450 enzymes, such as CYP1A1 and CYP1B1, which are involved in estrogen metabolism. This can lead to increased estrogen breakdown, reducing its bioavailability and potentially causing conditions like oligomenorrhea and early menopause (Mahoney, 2021). Additionally, dioxins can interfere with progesterone receptor signaling, impairing ovulation and increasing the risk of reproductive disorders like polycystic ovary syndrome (PCOS) and endometriosis (Rier, 2002).

Dioxins also affect puberty timing by disrupting gonadotropin-releasing hormone (GnRH) secretion from the hypothalamus, which in turn alters the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the pituitary gland. Studies have shown that prenatal exposure to dioxins can lead to delayed breast development and altered pubertal timing, likely due to changes in estrogen and androgen levels (Warner et al., 2017). Furthermore, dioxins have been associated with increased risks of hormone-dependent cancers, such as breast and ovarian cancer, due to their ability to promote oxidative stress and inflammation (La Merrill et al., 2020).

Given their persistence in the environment and ability to bioaccumulate, dioxins pose a long-term risk to female reproductive health. Reducing exposure through dietary changes, such as limiting consumption of animal fats where dioxins accumulate, and stricter industrial regulations are essential strategies for minimizing their endocrine-disrupting effects (Mahoney, 2021).

Pesticides

Pesticides such as DDT (dichlorodiphenyltrichloroethane) and atrazine have been widely studied for their endocrine-disrupting effects, particularly in relation to female reproductive health, puberty, and hormonal regulation. These chemicals can interfere with the hypothalamic-pituitary-gonadal (HPG) axis, disrupting normal hormonal signaling and increasing the risk of menstrual irregularities, infertility, and hormone-dependent diseases. Both DDT and atrazine act through different mechanisms but ultimately lead to estrogenic and anti-androgenic effects that can alter reproductive development and function (Gore et al., 2015).

DDT, however, was banned in 1976 in Poland, as well as in other places, and its metabolite, DDE (dichlorodiphenyldichloroethylene), is a xenoestrogen, meaning it can bind to estrogen receptors and mimic or disrupt natural estrogen activity. This can lead to early puberty, irregular menstrual cycles, and increased risks of breast cancer and endometriosis (Cohn et al., 2015). Additionally, DDT exposure has been linked to altered gonadotropin levels, reducing luteinizing hormone (LH) and follicle-stimulating hormone (FSH) secretion, which can impair ovulation and fertility (Teng et al., 2022). Prenatal exposure to DDT has also been associated with early menarche and altered mammary gland development, suggesting long-term reproductive consequences (La Merrill et al., 2020).

Atrazine

Atrazine, a widely used herbicide, was banned in Poland in 2007. It is an anti-androgenic and estrogenic endocrine disruptor. It does not bind directly to estrogen receptors but instead increases aromatase activity, an enzyme responsible for converting androgens into estrogens. This leads to excess estrogen production, which can disrupt ovarian function, cause early puberty, and increase the risk of estrogen-dependent disorders such as polycystic ovary syndrome (PCOS) and breast cancer (Hayes et al., 2010).

Studies in animal models have shown that atrazine exposure can lead to ovarian dysfunction, reduced fertility, and changes in hormone receptor expression, suggesting similar risks in humans (Gely-Pernot et al., 2017).

Both DDT and atrazine have been associated with altered puberty timing. Epidemiological studies have found that girls exposed to high levels of DDT in utero tend to experience earlier menarche. At the same time, atrazine exposure has been linked to delayed puberty due to disruptions in gonadotropin-releasing hormone (GnRH) secretion (Rosenberg et al., 2008). Additionally, these pesticides have been linked to increased risks

of hormone-sensitive cancers, such as breast and ovarian cancer, due to their ability to promote cell proliferation and disrupt normal estrogen signaling (Kassotis, 2020).

Given the persistence of DDT and atrazine in the environment, despite the ban, these chemicals remain a significant concern for female reproductive health. Reducing exposure through dietary choices, regulatory measures, and alternative pest control methods may help mitigate their endocrine-disrupting effects.

Effects of EDCs on Female Hormonal Health

Endocrine-disrupting chemicals (EDCs) play a significant role in altering the timing of puberty, particularly by advancing the age of menarche and accelerating breast development in girls. Over the past few decades, the average age of puberty onset has declined, with numerous studies linking this trend to increased exposure to estrogenic EDCs such as bisphenol A (BPA), phthalates, parabens, dioxins, and pesticides (Watkins et al., 2017). BPA, a widely used plasticizer, has been shown to mimic estrogen by binding to estrogen receptors (ERα and ERβ), leading to early thelarche (breast development) and altered pubertal timing (Harley et al., 2019). Research suggests that girls with higher urinary BPA levels tend to experience earlier breast development and menarche, likely due to BPA's ability to stimulate estrogen-responsive genes and interfere with the hypothalamic-pituitary-gonadal (HPG) axis (Elsen, 2022). Furthermore, exposure to phthalates and parabens, common in cosmetics and personal care products, has been associated with premature adrenarche and irregular menstrual cycles, which may set the stage for long-term reproductive dysfunction (Teng et al., 2022).

Beyond direct estrogenic effects, obesity and insulin resistance (IR) are key metabolic factors accelerating puberty, particularly in girls. Adipose tissue serves as an endocrine organ, producing leptin and adipokines that influence GnRH secretion and drive early pubertal onset (Ahmed et al., 2009). Insulin resistance, a condition that is increasingly common due to rising childhood obesity rates, enhances ovarian androgen production while simultaneously promoting the conversion of excess estrogen in adipose tissue, creating a hormonal environment that favors early puberty (Biro et al., 2018). Additionally, EDCs such as BPA and dioxins are known to act as obesogens, disrupting metabolic pathways and contributing to both weight gain and altered puberty timing (La Merrill et al., 2020). This interplay between EDC exposure and metabolic dysfunction increases the likelihood of developing polycystic ovary syndrome (PCOS), a leading cause of anovulation and infertility. PCOS is characterized by hyperandrogenism, irregular cycles, and insulin resistance, and studies suggest that early-life exposure to EDCs like DDT and phthalates may contribute to its pathogenesis by disrupting ovarian follicular development and androgen regulation (Teng et al., 2022).

The long-term implications of early estrogen exposure due to EDCs extend beyond puberty, increasing the risk of hormone-dependent gynecological cancers, such as breast, ovarian, and endometrial cancer. Estrogenic EDCs, particularly BPA, dioxins, and pesticides (such as DDT and atrazine), have been shown to induce epigenetic modifications and DNA damage, promoting carcinogenesis in estrogen-sensitive tissues (Kim et al., 2020). Early menarche and prolonged estrogen exposure are well-established risk factors for breast cancer, and studies indicate that girls exposed to high levels of EDCs in utero have a higher lifetime risk of developing estrogen-dependent malignancies (Cohn et al., 2019). Moreover, the estrogenic and inflammatory properties of these chemicals can exacerbate endometriosis, a condition associated with chronic pelvic pain and infertility (Rier & Foster, 2002). Given these significant health risks, there is an urgent need for stricter regulations on EDCs, public health interventions to limit exposure, and further research to fully understand the mechanisms by which these chemicals shape reproductive health across the lifespan.

Thyroid

Thyroid health is particularly vulnerable to endocrine-disrupting chemicals (EDCs), as many of these compounds interfere with thyroid hormone synthesis, metabolism, and receptor signaling. Chemicals such as polychlorinated biphenyls (PCBs), dioxins, perfluoroalkyl substances (PFAS), and polybrominated diphenyl ethers (PBDEs) have been shown to suppress thyroid hormone production, disrupt iodine uptake, and alter deiodinase enzyme activity, leading to hypothyroidism, goiter, and neurodevelopmental impairments (Boas et al., 2006). In pregnant women, maternal thyroid dysfunction due to EDC exposure can have severe consequences for fetal brain development, increasing the risk of cognitive deficits, reduced IQ, and attention disorders in offspring (Zoeller et al., 2012). Additionally, exposure to pesticides such as glyphosate and organochlorines (e.g., DDT) has been linked to autoimmune thyroid diseases like Hashimoto's thyroiditis and Graves' disease, as these chemicals induce immune dysregulation and oxidative stress in thyroid tissue (Turunen et al., 2021). Moreover, PFAS, widely found in water-resistant and non-stick products, are known to

bind to thyroid transport proteins, leading to disrupted thyroid hormone distribution in the body (Jain, 2018). Given the critical role of thyroid hormones in growth, metabolism, and neurological function, addressing environmental thyroid disruptors should be a public health priority. Strategies such as monitoring drinking water contamination, banning hazardous flame retardants, and reducing PFAS exposure through regulatory action could help prevent rising rates of thyroid disorders linked to chemical pollution (Cohn et al., 2019).

Polycystic ovary syndrome (PCOS)

PCOS is one of the most common endocrine disorders in women of reproductive age, characterized by hyperandrogenism, menstrual irregularities, polycystic ovarian morphology, and metabolic dysfunction. While the exact etiology of PCOS remains unclear, increasing evidence suggests that endocrine-disrupting chemicals (EDCs) such as bisphenol A (BPA), phthalates, dioxins, and pesticides (DDT, atrazine) play a significant role in its development by interfering with ovarian folliculogenesis, insulin signaling, and androgen production (Wróbel, 2014). BPA, a known xenoestrogen, disrupts the hypothalamic-pituitary-ovarian (HPO) axis by binding to estrogen receptors and altering gonadotropin secretion, resulting in increased luteinizing hormone (LH) and suppressed follicle-stimulating hormone (FSH), a hormonal pattern commonly observed in PCOS (Rattan et al., 2021). Additionally, phthalates and pesticides have been linked to ovarian dysfunction, oxidative stress, and chronic inflammation, further exacerbating PCOS symptoms (Patisaul, 2009). Another key factor in PCOS is insulin resistance (IR), which is present in up to 70% of affected women and contributes to excess androgen production from the ovaries and adrenal glands (Goodarzi et al., 2011). Studies indicate that EDC exposure during critical windows of development, such as fetal life or early puberty, may reprogram metabolic and reproductive pathways, increasing the risk of PCOS later in life Rattan et al., 2021). Given the growing body of evidence linking EDCs, obesity, and metabolic dysfunction to PCOS, efforts to reduce exposure to these chemicals, particularly during gestation, infancy, and puberty, may be crucial in preventing and managing this condition (Kahn, 2020).

Fertility issues in women have been on the rise, with environmental factors and endocrine-disrupting chemicals (EDCs) playing a major role in impairing ovarian function, oocyte quality, and implantation success. EDCs such as bisphenol A (BPA), phthalates, dioxins, and pesticides disrupt the hypothalamic-pituitary-gonadal (HPG) axis, leading to ovulatory dysfunction, anovulation, and reduced ovarian reserve (Caserta et al., 2021). BPA, for example, has been shown to alter folliculogenesis by increasing oxidative stress in ovarian cells, leading to poor oocyte quality and decreased fertilization rates (Machtinger et al., 2018). Additionally, phthalates and persistent organic pollutants (POPs), such as dioxins and PCBs, can accumulate in the body over time, causing hormonal imbalances that interfere with corpus luteum function and progesterone production, which are essential for successful implantation and maintaining pregnancy (Toft et al., 2020). Furthermore, exposure to pesticides such as DDT and atrazine has been linked to higher risks of miscarriage, preterm birth, and endometriosis, a condition that contributes to infertility by affecting uterine receptivity and embryo implantation (Teng et al., 2022). EDCs may also contribute to premature ovarian insufficiency (POI) by inducing epigenetic modifications in ovarian cells, accelerating follicular atresia, and depleting the ovarian reserve at a younger age (Gore et al., 2015). Given the significant impact of EDCs on reproductive health, reducing exposure through lifestyle changes such as choosing organic foods, avoiding plastic containers, and using natural personal care products may help preserve fertility and improve reproductive outcomes for women.

Avoiding Exposure to Endocrine-Disrupting Chemicals (EDCs)

Reducing exposure to endocrine-disrupting chemicals (EDCs) requires a combination of personal lifestyle changes and systemic policy interventions. One of the most effective ways to minimize EDC exposure is through dietary modifications, as many of these chemicals, such as bisphenol A (BPA), phthalates, dioxins, and pesticides, accumulate in food and packaging materials (Kahn et al., 2020). Choosing organic produce can reduce ingestion of pesticides like atrazine and DDT, while avoiding high-fat animal products lowers exposure to lipophilic chemicals such as dioxins and polychlorinated biphenyls (PCBs), which accumulate in animal fat (Kim et al., 2020). Additionally, reducing plastic use, especially plastic containers marked with recycling codes 3 (phthalates), 6 (styrene), and 7 (BPA or BPS), can significantly lower exposure to hormone-disrupting compounds that leach into food and beverages (Sugiura-Ogasawara et al., 2014). Instead, opting for glass, stainless steel, or BPA-free materials when storing or microwaving food helps prevent EDC contamination. Personal care products are another major source of EDC exposure, with parabens, phthalates, and triclosan frequently found in cosmetics, lotions, and shampoos. Switching to fragrance-free, natural, or certified organic personal care items can reduce dermal absorption of these chemicals (Dodson et al., 2012). Household products, such as flame retardants in

furniture and perfluorinated compounds (PFCs) in non-stick cookware, also contribute to long-term EDC exposure, making it important to choose chemical-free furniture, cookware, and cleaning supplies (Kahn et al., 2020). Awareness of indoor air quality is also crucial, as EDCs like phthalates and PBDEs (polybrominated diphenyl ethers) are commonly found in household dust. Regular vacuuming with HEPA filters and ventilating indoor spaces can help minimize inhalation exposure. While individuals can take many steps to reduce EDC exposure, systemic regulatory measures are necessary to ensure widespread protection.

Policies and Public Health Interventions to Reduce EDC Exposure

Government regulations and public health interventions play a critical role in reducing human exposure to endocrine-disrupting chemicals (EDCs). Some nations have already banned or restricted the use of certain EDCs, such as the European Union's ban on BPA in baby bottles and restrictions on phthalates in children's toys (European Chemicals Agency, 2021). In contrast, the United States has lagged behind in comprehensively regulating EDCs, with policies such as the Toxic Substances Control Act (TSCA) failing to adequately address the full scope of chemical exposure risks (Rudel et al., 2011). Stronger chemical safety laws such as requiring pre-market testing of chemicals for their potential to disrupt endocrine stems could significantly reduce public health risks. Public health campaigns aimed at educating consumers about EDCs have also been effective; for example, studies show that informed individuals actively reduce their exposure to BPA and phthalates by altering their purchasing habits (Rudel et al., 2011). Additionally, expanding environmental monitoring programs to track EDC contamination in water, food, and air is essential for identifying exposure hotspots and protecting vulnerable populations, such as pregnant women and children (Kahn et al., 2020). International agreements, such as the Stockholm Convention on Persistent Organic Pollutants (POPs), have played a crucial role in phasing out hazardous chemicals like DDT and PCBs, demonstrating that coordinated global efforts can successfully mitigate EDC risks (UNEP, 2021). However, further policy efforts are needed to expand bans on known EDCs, promote safer chemical alternatives, and hold manufacturers accountable for producing hazardous substances. Ultimately, a precautionary approach where chemicals must be proven safe before widespread use could prevent further public health crises caused by endocrine disruptors.

Limitations and Research Gaps in EDC Science

Despite growing evidence linking endocrine-disrupting chemicals (EDCs) to reproductive and metabolic disorders, significant gaps remain in research, regulation, and risk assessment. One major limitation is the lack of long-term human studies that track EDC exposure across multiple generations; most current research relies on animal models or short-term epidemiological studies, making it difficult to fully assess lifetime risks (Kahn et al., 2020). Additionally, many studies focus on individual chemicals, while real-world exposure involves complex mixtures of EDCs that may have additive or synergistic effects (Kortenkamp, 2017). This "cocktail effect" is not well understood, and standard risk assessment models often fail to account for the way multiple EDCs interact in the body (Birnbaum, 2012). Another challenge is the non-monotonic dose-response relationship of many EDCs, meaning that low doses may have more significant endocrine effects than high doses, contradicting traditional toxicological assumptions (Martínez-Pacheco et al., 2021). This makes it difficult to establish "safe" exposure thresholds and has led to regulatory agencies underestimating the risks of widespread low-dose EDC exposure. Furthermore, most toxicology tests fail to consider critical windows of vulnerability, such as fetal development, puberty, and pregnancy, where hormonal disruptions can have long-lasting effects (Diamanti-Kandarakis et al., 2009). There is also an urgent need for biomonitoring studies to identify populations with high EDC body burdens and to determine the long-term health consequences of exposure (Meeker et al., 2009). Lastly, the lack of safer chemical alternatives remains a barrier to phasing out hazardous EDCs; while some BPA-free plastics have been introduced, replacements like BPS and BPF have shown similar endocrine-disrupting properties, highlighting the need for rigorous testing of chemical substitutes before market approval (Rochester, 2015) Addressing these research gaps will require interdisciplinary collaboration, increased funding for long-term studies, and more stringent regulatory frameworks to ensure public health protection from EDC exposure.

Future Directions in Navigating Endocrine-Disrupting Chemicals (EDCs)

The future of mitigating endocrine-disrupting chemical (EDC) exposure lies in advancing early detection technologies, epigenetic research, and the development of safer alternatives. One promising avenue is the use of biosensors and chemical detection binaries that can identify low-dose EDC exposure in real time. Emerging tools such as hormone receptor-based biosensors and wearable EDC monitors could revolutionize

public health by providing early warnings about environmental contamination and individual exposure levels (Kumar et al., 2021). Additionally, research into epigenetic modifications induced by EDCs, such as DNA methylation, histone modifications, and changes in microRNA expression, is shedding light on how early-life exposure can lead to transgenerational effects on reproductive and metabolic health (Walker et al., 2020). Understanding these epigenetic markers could lead to the development of novel biomarkers for predicting EDC-related diseases and the creation of personalized intervention strategies. At the same time, the push for EDC-free product development is gaining momentum, with researchers exploring green chemistry approaches to create safer plastic alternatives, non-toxic flame retardants, and paraben-free cosmetics (Vogel et al., 2019). Investment in high-throughput screening methods, such as computational toxicology and machine learning models, will be critical in identifying hazardous chemicals before they reach the market (ToxCast Program, 2021). Moving forward, interdisciplinary collaboration between toxicologists, chemists, public health officials, and regulatory agencies will be essential to develop science-based policies, enhance consumer awareness, and reduce the global burden of EDC-related diseases.

Conclusions

This paper provides a comprehensive and up-to-date analysis of how endocrine-disrupting chemicals (EDCs) impact hormonal health, reproductive function, and metabolic diseases, filling critical gaps in current research by integrating mechanistic insights, epidemiological evidence, and policy implications. Unlike many existing reviews that focus solely on one class of EDCs or a single health outcome, this paper takes a holistic approach, examining the broad spectrum of hormonal imbalances induced by EDC exposure, including early puberty, PCOS, infertility, thyroid disorders, and cancer risks (Teng et al., 2022). Additionally, it highlights emerging research areas such as epigenetic modifications, real-time biosensor detection technologies, and safer chemical alternatives, positioning itself at the forefront of future directions in EDC mitigation (Wróbel, 2014). The discussion of obesity, insulin resistance, and metabolic dysfunction as accelerators of EDC-induced endocrine disruption provides a novel perspective on how environmental and metabolic factors interact to exacerbate health risks (Zhang et al., 2021). Furthermore, this paper highlights the urgent need for policy interventions, underscoring the regulatory gaps that permit hazardous chemicals to persist in consumer products despite mounting scientific evidence of harm. By synthesizing findings from toxicology, endocrinology, public health, and regulatory science, this paper serves as a valuable resource for researchers, healthcare professionals, and policymakers, offering actionable insights to reduce exposure, enhance regulation, and protect future generations from the long-term consequences of EDC exposure (Kahn, 2020).

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