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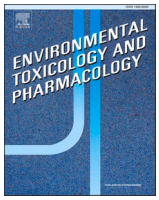

Effects of endocrine disruptive chemicals (EDCs) and therapeutic approaches to the polycystic ovary syndrome (PCOS): A current state-of-the-art

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ABSTRACT

Polycystic ovary syndrome (PCOS) is an endocrine condition affecting women of reproductive age, characterized by hyperandrogenism, ovulatory dysfunction, and some metabolic disturbances. Emerging evidence has been suggesting a link between certain endocrine-disrupting chemicals (EDCs), such as bisphenol A (BPA) and phthalates, and the pathogenesis and/or exacerbation of PCOS. These exogenous compounds interfere with hormonal function, potentially inducing epigenetic modifications and disrupting insulin sensitivity, primarily when exposure occurs during specific periods. This review explores the role of EDCs in the onset and progression of the condition, and it also outlines current therapeutic strategies, including hormonal contraceptives, insulin sensitizers, ovulation induction agents, and anti-androgenic treatments. Novel approaches targeting gut microbiota, cytokine modulation, and antioxidant therapies are discussed, alongside preventive measures to reduce EDC exposure. Thus, this review highlights the need for a personalized, multimodal management of PCOS, integrating pharmacological, lifestyle, and behavioural intervention. Nevertheless, for some of these therapies, it also emphasizes some lack of causality, as well as limited clinical trial data to support their efficacy.

1. Introduction

Polycystic Ovary Syndrome (PCOS) is an endocrine disorder affecting approximately 6–13 % of women of reproductive age (World Health Organization [WHO], 2025). PCOS is primarily characterized by hyperandrogenism, ovulatory dysfunction, morphological alterations of the ovaries, and dysregulation of the hypothalamic-pituitary-ovarian axis. Furthermore, it is frequently associated with insulin resistance and an elevated risk of type 2 diabetes. PCOS is usually diagnosed during adolescence, although symptoms may evolve and become more apparent later in life (Dong and Rees, 2023).

Hyperandrogenism, a hallmark of PCOS, results from excessive androgen production by the ovaries, primarily testosterone. Insulin resistance contributes to elevated circulating insulin levels, which further stimulate androgen synthesis. Additionally, dysfunction of the HPO axis leads to an imbalance in luteinizing hormone (LH) and follicle-stimulating hormone (FSH), characterized by elevated LH levels and reduced FSH levels. This hormonal imbalance disrupts normal follicular development, leading to anovulation and the accumulation of immature ovarian follicles (WHO, 2025; Silva et al., 2023). Clinical signs of hyperandrogenism include hirsutism, acne, and androgenetic alopecia. These symptoms are driven by increased testosterone and its conversion to dihydrotestosterone (DHT) via 5-alpha reductase. DHT promotes excessive hair growth, sebum production, and miniaturization of scalp hair follicles (Azziz et al., 2006; Dumesic et al., 2015). Menstrual irregularities, such as oligomenorrhea and amenorrhea, are relatively common and reflect the underlying ovulatory dysfunction. Approximately 85 % of women with PCOS experience ovulatory disturbances, although some may have regular cycles despite anovulation (AE-PCOS Society, 2024; Dumesic et al., 2015).

Chronic low-grade inflammation is another characteristic of PCOS, with elevated cytokine levels that may contribute to metabolic and hormonal disturbances. Oxidative stress, driven by an excess of free radicals and insufficient antioxidant defences, is also implicated in the

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pathogenesis of PCOS and its associated complications, including insulin resistance and cardiovascular disease. Vitamin D deficiency is common among women with PCOS and may exacerbate these effects due to its role in modulating inflammation and insulin sensitivity (Han et al., 2024). Dyslipidemia is also frequently observed, characterized by elevated triglycerides and low-density lipoprotein (LDL) cholesterol, along with reduced high-density lipoprotein (HDL) cholesterol. These lipid abnormalities are responsible for the increased risk of cardiovascular disease in women with PCOS.

PCOS is associated with a significantly increased prevalence of psychological disorders, including anxiety, depression, and body image dissatisfaction. These disturbances are thought to result from a complex interplay between hormonal imbalances, metabolic alterations, infertility, and phenotypic features such as hirsutism and obesity, all of which may affect neurochemical pathways involved in mood regulation (Alur-Gupta et al., 2019; Li et al., 2024; WHO, 2025). Emerging evidence also links PCOS to gut microbiota dysbiosis. Elevated levels of *Bacteroides vulgatus* have been associated with insulin resistance, bile acid dysregulation, and ovarian dysfunction in animal models, suggesting a potential role of the gut microbiome in PCOS pathophysiology and management (Dong and Rees, 2023; Qi et al., 2019).

Endocrine-disrupting chemicals (EDCs) are exogenous substances that interfere with the normal functioning of the endocrine system, potentially leading to adverse health effects in exposed individuals as they alter endocrine function and cause adverse outcomes in intact organisms or populations (Srnovršnik et al., 2023). EDCs disrupt the synthesis, secretion, transport, metabolism, or elimination of natural hormones responsible for maintaining homeostasis, reproduction, and development. Besides PCOS, they have been implicated in a wide range of reproductive disorders, including infertility, hormone-dependent cancers (e.g., breast and prostate), and metabolic diseases such as diabetes and obesity (Jozkowiak et al., 2023), although a definitive causal relationship between exposure to most EDCs and the development of PCOS has not yet been established and remains under investigation.

EDCs such as bisphenol A (BPA), parabens, triclosan, and per- and polyfluoroalkyl substances (PFAS) are widely used in consumer products and industrial applications, contributing to widespread human exposure and environmental contamination. The estimated global volume of BPA utilization was 7.69 million metric tons in 2015 (Manzoor et al., 2022). Parabens, are used as preservatives in cosmetics, pharmaceuticals, and food, with methylparaben (MeP), ethylparaben (EtP), and propylparaben (PrP) being the most common (Cashman and Warshaw, 2005). Triclosan is an antimicrobial agent found in soaps and toothpaste so frequently used that 75 % of the U.S.A. population is likely exposed to this compound via consumer goods and personal care products (Weatherly et al., 2017). PFAS, known for their persistence and bioaccumulation, are used in non-stick cookware, waterproof textiles, and firefighting foams. The global PFAS waste management market value stood at almost 13.98 billion U.S. dollars in 2025 (Taylor et al., 2024).

Currently, there is no definitive cure for PCOS; current therapeutic strategies focus on the management of clinical manifestations. Management of the syndrome often necessitates a multifaceted approach, combining pharmacological treatments, nutritional supplementation, and lifestyle modifications. Moreover, PCOS is a chronic condition that requires sustained follow-up. Thus, the ideal treatment for PCOS remains unknown and requires an individualized approach. Therefore, this review aims to provide a summary of the current knowledge about the involvement of exposure to EDC as a risk factor in the development of PCOS and to summarise the multiple therapeutic options available, including underdeveloped approaches.

2. EDCs and PCOS

A growing amount of evidence suggests that both genetic and environmental factors influence the pathogenesis of PCOS. Epigenetic modifications, including DNA methylation, alter gene expression without altering the DNA sequence and can be inherited across generations. They are influenced by intrauterine conditions, such as maternal hormone levels and lifestyle during pregnancy. This phenomenon, known as fetal programming, may explain the high heritability of PCOS, with studies indicating that 60–70 % of daughters born to women with PCOS develop symptoms themselves later in life. Several genes have also been identified as possible causes, including those involved in reproductive function and metabolic regulation. These genes interact with one another and with environmental exposures, contributing to the etiology of PCOS (Mimouni and Giacobini, 2024). These changes may be passed on to future generations, potentially increasing susceptibility to PCOS through disrupted hormone secretion and insulin sensitivity. Prenatal or early-life exposure to EDCs may therefore play an important role in the manifestation of PCOS in adulthood (Jozkowiak et al., 2023).

EDCs can interfere with CYP17A1, a critical enzyme in androgen biosynthesis, by altering its expression and activity, thereby contributing to the hyperandrogenism characteristic of PCOS. Additionally, these chemicals activate oxidative stress-related signaling pathways such as MAPK/JNK and NF-κB, which promote inflammation and disrupt ovarian follicular development. EDCs also influence the expression of microRNAs (miRNAs), which regulate gene networks involved in steroidogenesis and apoptosis. For instance, BPA exposure has been linked to altered miRNA profiles that affect granulosa cell function and ovarian morphology (Guarnotta et al., 2022). Lipophilic EDCs, which accumulate in adipose tissue, may exacerbate PCOS symptoms over time. These substances are not only stored in fat but may also promote fat accumulation by disrupting insulin and lipid metabolism. Therefore, chronic exposure may lead to a vicious cycle: increased adiposity enhances EDC storage, which in turn worsens metabolic and hormonal imbalances (Darbre, 2017; Islam et al., 2022). Furthermore, EDCs can be retained in biological fluids such as amniotic fluid, breast milk, serum, and urine, amplifying their long-term effects. This accumulation is particularly concerning in individuals with obesity, as greater fat mass correlates with higher EDC retention and more severe PCOS symptoms (Islam et al., 2022).

Animal studies have demonstrated that prenatal exposure to high levels of anti-Müllerian hormone can disrupt fetal development, leading to PCOS symptoms in offspring, including hyperandrogenism, oligoanovulation, insulin resistance, and altered body composition. Such findings underscore the importance of a balanced hormonal environment during pregnancy to prevent long-term reproductive and metabolic consequences (Mimouni and Giacobini, 2024). Zebrafish exposed to specific EDCs exhibited ovarian dysfunction, altered steroidogenesis, and disrupted hypothalamic-pituitary-gonadal axis signaling—hallmarks of PCOS (Ramya Ranjan Nayak et al., 2023).

EDCs such as BPA, phthalates, and pesticides are pervasive in the environment and can enter the human body through ingestion, inhalation, or dermal absorption (Silva et al., 2023; Darbre, 2017). BPA has been shown to disrupt kisspeptin-mediated signalling, which plays a critical role in the regulation of gonadotropin-releasing hormone (GnRH) secretion. Disruption of this pathway impairs the release of FSH and LH, leading to downstream effects on sex hormone production and ovarian function. Moreover, elevated urinary phthalate levels in women have been associated with reduced fertility, increased miscarriage rates, and diminished ovarian reserve (Silva et al., 2023). Women working in agriculture may be especially vulnerable to EDC exposure due to contact with pesticides such as DDT and glyphosate. These compounds accumulate in adipose tissue and may also act as endocrine disruptors (Darbre, 2017).

Moreover, PFAS, parabens, triclosan, and flame retardants are also reported as relevant EDCs that pose significant risks to reproductive health. PFAS persist in the environment and bioaccumulate in human tissues, interfering with hormone transport and signalling, particularly during pregnancy and early development. Parabens, widely used as preservatives in personal care products, mimic estrogen and disrupt reproductive hormone balance, leading to altered sperm quality, follicle-

stimulating hormone levels, and potential infertility. Triclosan been shown to interfere with thyroid hormone metabolism and estrogenic activity, affecting reproductive outcomes. Flame retardants, as polybrominated diphenyl ethers (PBDEs), disrupt sex hormone synthesis and are linked to delayed puberty, reduced fertility, and adverse pregnancy outcomes. These chemicals act through various mechanisms including receptor binding, epigenetic modifications, and oxidative stress (Gore et al., 2024; Isaac et al., 2025).

2.1. Bisphenol A (BPA) and PCOS

Among multiple EDCs, BPA is one of the most extensively studied in relation to PCOS. BPA is a synthetic compound commonly found in plastics and food packaging, and it has been shown to mimic estrogen by binding to estrogen receptors, thereby disrupting hormonal regulation. BPA has been implicated in the dysregulation of steroid hormone production, particularly by increasing levels of 17β-estradiol. This exacerbates the already existing imbalance between estrogens and androgens in PCOS, potentially worsening menstrual irregularities and ovarian dysfunction. Animal studies have demonstrated that BPA's estrogenic activity can lead to excessive endometrial proliferation, further supporting its role in reproductive disruption (Rochester and Bolden, 2015).

Eslami et al. (2017) investigated urinary BPA concentrations in 51 women diagnosed with PCOS and 51 control participants. The results revealed significantly higher BPA levels in the PCOS group (3.34 ± 2.63 ng/mL) compared to the control group (1.43 ± 1.57 ng/mL, $p < 0.001$). Logistic regression analysis confirmed a strong association between BPA exposure and PCOS, with an odds ratio of 1.53, indicating a 53 % increased likelihood of PCOS per unit increase in urinary BPA, after adjusting for confounding factors such as age, BMI, and reproductive history.

Additional studies have corroborated these findings. Srnovršnik et al. (2023) reported elevated BPA levels in plasma, urine, and follicular fluid of women with PCOS. Kandaraki et al. (2011) found higher mean serum BPA concentrations in PCOS patients (1.05 ± 0.56 ng/mL) compared to controls (0.72 ± 0.37 ng/mL). Similarly, Eslami et al. (2017) observed increased urinary BPA levels in PCOS women (2.19 ± 1.88 ng/mL) versus controls (1.04 ± 0.98 ng/mL), and Wang et al. (2017) reported higher BPA concentrations in follicular fluid of PCOS patients (2.09 ± 0.12 ng/mL) compared to controls (1.04 ± 0.10 ng/mL).

These studies also identified positive correlations between BPA levels and markers of hyperandrogenism, including serum androstenedione and testosterone, as well as insulin resistance, hepatic steatosis, and chronic inflammation. Conversely, negative correlations were found between BPA and markers of ovarian reserve (Zhou et al., 2016), sex hormone-binding globulin (SHBG), and vitamin D-binding protein (Jedrzejuk et al., 2019).

Moreover, a recent case-control study found significantly elevated BPA levels in women diagnosed with PCOS compared to healthy controls, with a notable positive correlation between BPA and LH levels, suggesting a potential role in disrupting the hypothalamic-pituitary-ovarian axis. Additionally, consistent associations between high BPA exposure and PCOS-related hormonal imbalances have been reported, although the directionality of causation remains under investigation. Another clinical study demonstrated that BPA exposure was linked to altered hormonal profiles in PCOS patients, including elevated testosterone and disrupted insulin sensitivity, reinforcing its role as a contributing environmental factor (Kechagias et al., 2020, Patel et al., 2024).

Although BPA exhibits anti-androgenic activity, this does not necessarily translate into symptom improvement in PCOS. On the contrary, BPA may further disrupt the endocrine feedback mechanisms that regulate LH secretion. In PCOS, reduced hypothalamic sensitivity to estradiol and progesterone leads to persistently elevated LH levels, which in turn stimulate excessive androgen production. This feedback disruption may be exacerbated by BPA exposure (Moore, 2022).

Efforts to define safe exposure thresholds for BPA have evolved. While the previously accepted limit in Europe and the United States was 50 μg/kg/day, recent European guidelines have drastically reduced this to 0.0002 μg/kg/day, reflecting growing concern over BPA's health risks (EFSA, 2023). Animal studies have shown adverse effects at exposure levels close to the former threshold (Fernández et al., 2010), and BPA has been detected in 80 % of urine samples from the general population, indicating widespread daily exposure (Patel et al., 2024).

Given the absence of a clearly defined safe exposure level in humans, and the consistent evidence linking BPA to hormonal and metabolic disturbances in PCOS, it is essential to consider the cumulative effects of chronic, low-dose exposure in both clinical management and public health policy (Patel et al., 2024; Rutkowska and Rachoń, 2014).

3. Multimodal therapeutic approach to PCOS

As mentioned, PCOS does not have a specific treatment or cure, requiring an individual approach to the different symptoms, depending on the clinical case, reproductive status and personal intentions of the patient. Table 1 summarises the therapeutic options available for PCOS management.

3.1. Hormonal Regulation and Contraceptives

Combined oral contraceptives (COCs) are widely used as first-line therapy for managing hormonal dysregulation and hyperandrogenic symptoms such as acne, hirsutism, and menstrual irregularities. These formulations normally contain estrogen and a progestin, which work synergistically to suppress ovarian androgen production and increase sex hormone-binding globulin, thereby reducing free testosterone levels (Teede et al., 2019). COCs containing drospirenone or cyproterone acetate are particularly effective due to their anti-androgenic properties (Bhathena, 2005; Feng et al., 2016).

Progestin-only contraceptives (POCs) offer an alternative for women who cannot tolerate estrogen or have cardiovascular risk factors. While less effective in treating hyperandrogenic symptoms, POCs are safer in specific populations and can help regulate menstrual cycles (Oguz and Yildiz, 2021).

Intrauterine devices (IUDs), both hormonal and copper-based, provide long-term contraception. Hormonal IUDs releasing levonorgestrel may offer additional benefits by reducing endometrial hyperplasia risk, particularly in women with obesity or insulin resistance (Hardeman and Weiss, 2014). Some clinical references have suggested that progestin IUDs may be considered in PCOS patients who are at risk of endometrial hyperplasia or who have contraindications to systemic hormonal therapies. However, their use remains limited to symptom management rather than addressing the broader metabolic and endocrine dysfunctions of PCOS (Huddleston and Dokras, 2022; Oguz and Yildiz, 2021). In fact, a recent network meta-analysis of pharmacological interventions for PCOS did not include hormonal IUDs among the most effective treatments for metabolic or androgen-related outcomes (Bo et al., 2025). Thus, while IUDs may be beneficial in select cases, their role in comprehensive PCOS management requires further investigation through robust studies.

3.2. Insulin sensitizers and metabolic modulators

Metformin, a biguanide antidiabetic agent, is commonly prescribed to improve insulin sensitivity and reduce hyperinsulinemia, which in turn lowers ovarian androgen production. It is particularly beneficial in overweight or obese women and may enhance ovulatory function and fertility outcomes (Attia et al., 2023). Myo-inositol, a naturally occurring insulin sensitizer, has also shown efficacy in improving metabolic and reproductive parameters, especially when combined with vitamin D (Kiani et al., 2022; Mohan et al., 2023). In zebrafish, isatin-linked pyrazole K1 derivatives have been shown to modulate insulin sensitivity by

Table 1 –
Summary of the different therapeutic strategies for PCOS, according to the goal.

Goal	Therapy	Benefit	Contraindications or Disadvantages	Ref
Manage hyperandrogenism	COCs	Reduces androgen levels, regulates cycles, improves acne/hirsutism	Risk of thromboembolism, not suitable for women with cardiovascular risk	Teede et al., (2019); Bhathena, (2005)
	POCs	Safer for women with cardiovascular/ metabolic risks	Less effective on acne/hirsutism	Oguz and Yildiz, (2021)
	IUDs	Contraception; reducing endometrial hyperplasia risk	Copper based may represent a risk	Hardeman and Weiss, (2014)
Treat insulin resistance	Metformin	Reduces insulin resistance and androgen levels, improves ovulation	Gastrointestinal side effects, off-label use	Attia et al., (2023)
	Myo-inositol + Vitamin D	Enhances insulin sensitivity, improves ovulation and fertility	Mild GI discomfort requires consistent intake	Kiani et al., (2022)
	Isatin-linked pyrazole K1 derivatives	Also helps reducing ovarian fibrosis	Limited human data. Requires further clinical validation	Ramamurthy et al., (2025b))
Induce ovulation	Clomiphene citrate	Stimulates ovulation	Risk of multiple pregnancy, hot flashes	Cunha and Póvoa, (2021)
	Letrozole	Higher ovulation and live birth rates than clomiphene	Headaches, fatigue, and off-label use	Legro et al., (2014)
Treat hirsutism, acne and/ or alopecia	Spironolactone	Reduces androgen effects, improves hair and skin symptoms	Risk of hyperkalemia, requires monitoring	Reiser et al., (2023)
	Minoxidil (topical)	Stimulates hair growth	Requires continuous use	Herskovitz and Tosti, (2013)
Weight management	Lifestyle modification (diet/ exercise)	Improves insulin sensitivity, ovulation, and lipid profile	Requires long-term adherence	Barrea et al., (2019), (2023)
	GLP−1 receptor agonists	Promotes weight loss, improves insulin sensitivity and menstrual cycles	GI side effects, cost	Erguc et al., (2021)
	SGLT2 inhibitors	Reduces glucose and weight, improves insulin sensitivity	Risk of urinary infections	Artasensi et al., (2023)
Reduce EDC impact	Antioxidants (Vitamins A, C, D, etc.)	Neutralise oxidative stress, support detoxification	Limited human data, mostly preclinical evidence	Ogunlade et al., (2022)
	Curcumin, NAC, alpha-lipoic acid, omega 3 fatty acids…	Anti-inflammatory, supports liver detox pathways	Requires further clinical validation	Geng et al., (2017)

reducing the blood glucose level, lowering lipid levels, and significantly reducing collagen deposition in ovarian tissues, indicating that it may reduce fibrosis associated with PCOS (Ramamurthy et al., 2025b).

3.3. Ovulation induction agents

Clomiphene citrate and letrozole are first-line agents for inducing ovulation in women with PCOS-related infertility. Letrozole, an aromatase inhibitor, has demonstrated superior efficacy and safety compared to clomiphene in several trials (Legro et al., 2014). In resistant cases, combination therapy may be employed to enhance follicular response (Mejia et al., 2019).

3.4. Aldosterone antagonists and dermatological treatments

Spironolactone, an aldosterone antagonist with anti-androgenic effects, is effective in treating hirsutism, acne, and androgenetic alopecia. It increases SHBG and accelerates testosterone clearance, although it requires monitoring for hyperkalemia (Reiser et al., 2023). Topical minoxidil and laser-assisted hair removal are also additional options for managing androgenetic alopecia and hirsutism, respectively (Herskovitz and Tosti, 2013; Hosseini et al., 2022).

3.5. GLP-1 receptor agonists, SGLT2 inhibitors and cyproterone acetate (CPA)

GLP-1 receptor agonists, such as liraglutide, promote weight loss, improve insulin sensitivity, and reduce androgen levels. These agents are beneficial in women with obesity or fatty liver disease (Erguc et al., 2021). Furthermore, they have been pointed out as having a “superior efficacy” in improving metabolic and hormonal outcomes in women with PCOS, comparing to other treatment approaches (Bo et al., 2025).

Moreover, SGLT2 inhibitors enhance glucose excretion and promote weight loss, offering metabolic benefits in PCOS patients with insulin resistance (Artasensi et al., 2023).

Cyproterone acetate (CPA), when combined with ethinyl estradiol, remains a potent anti-androgenic option for managing acne and hirsutism, although regulatory restrictions in some countries may limit its use (Bitzer et al., 2017).

3.6. Microbiota and cytokine-based therapies

Recent research has highlighted the role of the gut microbiota in the pathophysiology of PCOS. The elevation of *Bacteroides vulgatus* in the gut microbiota of PCOS patients, which has been associated with reduced levels of interleukin-22 (IL-22), a cytokine involved in maintaining intestinal barrier integrity and regulating inflammation. In experimental models, transplantation of microbiota from PCOS patients—especially those enriched in *B. vulgatus* — was sufficient to induce PCOS-like symptoms in mice, including ovarian dysfunction and metabolic disturbances. Supplementation with IL-22 was able to reverse these effects, improving insulin sensitivity, reducing systemic inflammation, and restoring gut barrier function. These findings suggest that IL-22 therapy may offer a promising strategy to counteract PCOS symptoms linked to gut dysbiosis and immune-metabolic imbalance (Qi et al., 2019, Geng et al., 2023, Sudhakaran et al., 2025).

3.7. EDC exposure therapies and preventive measures

Given the role of EDCs in PCOS, there are also therapeutic approaches to neutralize or eliminate these compounds, namely micronutrients and bioactive compounds. Antioxidants such as vitamins A, C, D, alpha-lipoic acid, and curcumin have shown potential in mitigating EDC-induced oxidative stress and inflammation. These compounds may act by neutralizing free radicals and restoring redox balance, which is often disrupted by EDC exposure. Vitamin C scavenges reactive oxygen species and enhances overall cellular defense. Vitamin D plays a role in modulating immune responses and reducing inflammatory cytokine production. Vitamin A supports epithelial integrity and immune regulation. Alpha-lipoic acid also has na antioxidant effect and an ability to amplify other antioxidant effects. Curcumin has been shown to suppress inflammatory pathways such as NF-κB and reduce oxidative damage at

both cellular and systemic levels. Together, these antioxidants form a synergistic defense system that can counteract the harmful effects of EDCs (Ogunlade et al., 2022; Geng et al., 2017). Moreover, compounds such as omega-3 fatty acids and polyphenols can modulate insulin signaling, reduce oxidative stress, and restore steroidogenic enzyme balance. These findings support the role of nutritional interventions in counteracting metabolic and reproductive disturbances in PCOS, especially those exacerbated by EDC exposure (Nayak et al., 2025). Another study, using the zebrafish model, reported that zinc coumarin derivatives effectively mitigate metabolic and neurobehavioral disturbances associated with PCOS, also suggesting their potential as comprehensive therapeutic agents (Ramamurthy et al., 2025a).

Pharmacological modulation of detoxification enzymes, including cytochrome P450 and glutathione S-transferase, is currently under investigation. Gene therapy, epigenetic interventions, and nanotechnology-based drug delivery systems are also being explored as future avenues for targeted treatment (He et al., 2024; Stener-Victorin and Deng, 2021).

Preventive strategies aimed at reducing exposure are also recognized as essential public health measures. These strategies are particularly critical during vulnerable life stages, such as pregnancy and early childhood, when hormonal systems are most susceptible to disruption (WHO, 2013). Raising public awareness is a cornerstone of effective prevention. Educational initiatives can empower individuals to make informed choices about the products they use and the environments they inhabit. Campaigns should focus on common sources of EDC exposure, including food packaging, personal care products, and household items (El Ouazzani et al., 2021).

Consumers can significantly reduce their exposure to EDCs by opting for safer alternatives in everyday products. Key recommendations include (Martin et al., 2022):

- Avoid using plastic containers for food and beverages, especially when reheating, and opt for glass, stainless steel, or ceramic alternatives instead.
- Reducing consumption of canned foods, as metal can linings often contain BPA.
- Choosing organic and home-cooked meals over processed or packaged foods, which are more likely to contain phthalates and bisphenols.
- Using natural cleaning products, such as vinegar, baking soda, or black soap, instead of conventional chemical cleaners.
- Selecting cosmetics and personal care products labelled “paraben-free,” “phthalate-free,” and “triclosan-free,” and favouring certified labels like COSMOS Organic® or ECOCERT®.
- Ventilating indoor spaces regularly to reduce the accumulation of airborne EDCs from furniture, paints, and household dust.
- Avoid using non-stick cookware containing PFAS and opt for alternatives like cast iron or ceramic instead.
- Using glass or metal water bottles instead of plastic ones, which may release microplastics and EDCs.
- Applying perfumes to clothing rather than directly to the skin, and preferring natural or certified EDC-free fragrances.

Pregnant women, infants, and children are particularly vulnerable to the effects of EDCs. Special attention should be paid to baby products and toys, ensuring they are free from BPA, phthalates, and flame retardants. Additionally, it is essential to drink good-quality water, as it may contain pesticide residues. Activated carbon or reverse osmosis filters are recommended for home use (Schreiber et al., 2024).

3.8. Weight management and lifestyle change

Lifestyle modification remains a foundational component of PCOS management. A modest weight loss of 5–10 % can significantly improve insulin sensitivity, ovulatory function, and lipid profiles. Dietary strategies such as the Mediterranean diet, low-glycemic index diet, and very low-calorie ketogenic diet (VLCKD) have shown benefits in clinical studies (Barrea et al., 2019, 2023). These interventions should be personalized and sustainable to ensure long-term adherence and efficacy.

4. Conclusions

PCOS is a multifactorial condition with significant reproductive, metabolic, and psychological implications. A deeper understanding of the interplay between genetic, hormonal, and environmental contributors to PCOS (including the role of different EDCs) is crucial for prevention and development of new therapeutic strategies.

Given the heterogeneity of PCOS, a personalized and multimodal therapeutic approach is essential to effectively address its diverse clinical manifestations. For instance, a patient presenting with hirsutism, acne, and irregular menstrual cycles may benefit from COCs, which suppress ovarian androgen production and regulate menstrual function. However, if laboratory findings reveal insulin resistance, the addition of metformin or myo-inositol plus vitamin D may be more appropriate to improve metabolic parameters and restore ovulatory cycles. In cases of infertility, clomiphene citrate or letrozole are first-line agents for ovulation induction, with letrozole often preferred due to its superior efficacy in inducing mono-ovulatory cycles. For patients with severe hyperandrogenism, spironolactone may be added to reduce androgenic symptoms, while topical minoxidil can be used to manage androgenic alopecia. Obese patients or those with glucose intolerance may benefit from GLP-1 receptor agonists or SGLT2 inhibitors, which offer weight loss and glycemic control advantages. Additionally, vitamins A, C, and D, along with curcumin, alpha-lipoic acid, and omega-3 fatty acids, may help mitigate oxidative stress and inflammation. Finally, lifestyle modifications, including tailored diet and exercise plans, remain foundational and should complement pharmacological interventions. A multimodal treatment and preventive strategies—particularly those addressing environmental factors such as EDCs exposure—will certainly provide a more personalized and holistic approach to the disease.

Future studies should prioritize longitudinal exposure assessments to better understand the cumulative and developmental impacts of EDCs such as BPA and phthalates, particularly in reproductive-age women. Integrating multi-omics approaches—including genomics, transcriptomics, metabolomics, and microbiomics—will be essential to unravel the complex molecular interactions driving PCOS and to identify novel biomarkers for early detection and intervention. More studies evaluating antioxidant therapies, microbiota-targeted interventions, and nutritional strategies are also critical to translating mechanistic insights into effective treatments. Finally, regulatory policies must evolve to restrict the use of high-risk EDCs in consumer products, in order to prevent the development or progression of reproductive disorders, namely in reproductive-age women.

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CRediT authorship contribution statement

Adèle Lemogne Robert: Writing – original draft. **Catarina Jota Baptista:** Writing – review & editing, Supervision. **Edite Oliveira**

Torres: Writing – review & editing, Supervision.

Consent to participate

This is not applicable

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This is not applicable

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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