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ENDOCRINE DISRUPTORS IN FEMALE FERTILITY

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ABSTRACT

Introduction: Nowadays, the world is facing a global issue of infertility. Endocrine-disrupting chemicals (EDCs) influence homeostasis and contribute to the development of various diseases. The endocrine system plays a crucial role in maintaining proper body functions, including metabolism regulation, fluid and electrolyte balance, and, importantly, the regulation of reproductive functions. The EDCs analyzed in this study (pesticides, BPA, phthalates, parabens and phenols, and dioxins) are commonly found in everyday items such as food containers, personal care products, and even children's articles. Recent studies show that their impact on the endocrine system contributes to female infertility.
Purpose of the study: This study aims to investigate the relationship between EDCs found in everyday-use products and the female reproductive system, with an emphasis on their mode of action, substance-specific effects, and duration of exposure.
Materials and methods: An overview of 31 articles sourced from PubMed and open-access databases was conducted. The reviewed literature focused on the disruption of follicular development, prolonged time to pregnancy, and reduced ovarian function. Most of the studies were conducted on women of reproductive age.
Conclusions: EDCs have a negative impact on the female reproductive system on many levels, including hormonal regulation, cellular function, and gene expression.

KEYWORDS

Endocrine-Disrupting Chemicals, EDCs, Endocrine System, Female Reproductive System, Female Fertility

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1. Introduction

World Health Organisation (WHO) and The International Programme on Chemical Safety (IPCS) defined endocrine disrupting compounds (EDCs) as exogenous substances or mixtures that alter function of the endocrine system and consequently cause adverse health effects in an intact organism or its progeny or populations [1]. Not every molecule that alters endocrine function can be classified as EDC, it must cause measurable harm through a plausible endocrine mode of action [2]. The best characterised compounds are bisphenols, phthalates, parabens, polychlorinated biphenyls, dioxins, pesticides and herbicides [3]. They are found in various sources including food, water, air, dust and soil, as well as in personal care products, building materials, packaging and toys. Exposure can occur through ingestion, inhalation or skin absorption [4]. As far as we know EDCs are associated with disruption of steroidogenesis, along with cross interaction with various cellular receptors including estrogen receptors (ERα and ERβ), androgen receptors (ARs) and G protein-coupled estrogen receptor (GPER). This results in a modulatory effect on signalling pathways, potentially leading to altered gene expression and impaired cellular function [5, 6]. Literature review revealed EDCs association with women reproductive health, especially its negative impact on menstrual cycle, longer conception time and disruption of the hypothalamic-pituitary-gonadal axis, resulting in reduced fertility and potentially contributing to polycystic ovary syndrome (PCOS) and endometriosis development.

Infertility manifests by failure to establish a clinical pregnancy after 12 months of regular and unprotected sexual intercourse [7]. Global prevalence of infertility is estimated at 17.5%, which implies that one in six individuals worldwide is impacted. The peak prevalence of primary infertility occurs in women aged 20-24, while secondary infertility peaks at 35-44. In 2021, 124.7 million cases of infertlilty and associated 71.6 thousand years lived with disability (YLDs) were reported [8]. Female infertility may result from hypogonadotropic hypogonadism, hyperprolactinemia, premature ovarian insufficiency, polycystic ovary syndrome, endometriosis, uterine fibroids and endometrial polyps, along with disorders of ciliary function, cystic fibrosis, infections, systemic diseases and lifestyle related factors [7]. Global infertility epidemics arouse interest in association between chemical exposure and reproductive impairments. Contributing to continuously growing attention given to EDCs [9].

Polycystic ovary syndrome is characterised by signs and symptoms of androgen excess and ovarian dysfunction. It is strongly influenced by epigenetic and environmental factors, such as diet and lifestyle [10]. The pathophysiology of PCOS is complex and yet not fully uncovered. Nevertheless studies present that key elements in PCOS development are insulin resistance and hyperandrogenism, resulting in metabolic and reproductive disturbance. Intinaly the core abnormality is elevated ratio of luteinizing hormone (LH) to follicle stimulating hormone (FSH), induced by increased frequency of gonadotropin-releasing hormone (GnRH) pulses. As a result theca cells produce more androgens, responsible for hirsutism, hair loss and acne. Symptoms can additionally involve irregular cycles and infertility [11, 12]. The occurrence of PCOS fluctuates between 6% and 20%. Moreover the incidence and the prevalence of PCOS are consistently increasing [8, 13].

Endometriosis is a chronic estrogen-dependent condition in which endometrial glands and stroma implants outside the uterine cavity. Clinically, it is primarily characterized by chronic pelvic pain and reduced fertility. The most typical localizations of ectopic endometrium are the ovaries, the posterior broad ligament, the anterior cul-de-sac, pouch of Douglas and the uterosacral ligament. Additionally, ectopic endometrial tissue can infiltrate the uterine myometrium, exacerbating symptoms and further impair fertility [14]. The underlying mechanisms are complex and not entirely elucidated, however multiple theories were proposed, such as retrograde menstruation, metaplasia, genetic susceptibility, extrauterine-sourced stem cells, hematogenous or lymphatic spread and Müllerian rest induction [15, 16]. Approximately 10% of the general female population

suffer from endometriosis, the prevalence rises to 30-40% of women with dysmenorrhea and up to 50-60% of women with infertility [17]. The social burden connected with endometriosis is significant and impacts various aspects of a woman's life. Studies suggest that roughly 40% of women with endometriosis experience impaired career growth due to the condition and nearly 50% report a decreased ability to work because of chronic pain symptoms [18].

Research indicates that economical costs associated with EDCs in the European Union are approximately €1.5 billion annually, considering only DDE-attributable fibroids and phthalate-attributable endometriosis. These figures reflect a significant public health burden implicating in urgent need of regulatory action on EDCs prevalence [19].

2. State of Knowledge

Pesticides are widely used in households and agriculture, so they can easily pollute water, soil, air and food. Studies have been conducted showing reduced fertility in women associated with exposure to pesticides. Elevated levels of such pesticides as dichlorodiphenyldichloroethylene (DDE), β-hexachlorocyclohexane (β-HCH) and hexachlorobenzene (HCB) in umbilical cord blood were associated with a longer time of getting pregnant [4]. Animal studies have shown that organochlorine pesticides negatively affect the ovaries by reducing their mass, follicle growth, and oocyte viability. Additionally, methoxychlor increased the incidence of ovarian cysts. In studies on rats, the carbamate pesticide molinate inhibited the frequency of LH pulses, leading to delayed ovulation, indicating a potential impact of pesticide exposure on pituitary gland function [21]. Organochlorines (DDE) disrupt female reproduction by altering the length of the menstrual cycle. Their serum concentrations are associated with reduced levels of progesterone and estrogen. Moreover, some studies have shown a link between these substances and lower ovarian reserve as well as an earlier age of menopause [22].

Bisphenol A [2, 2-bis(4-hydroxyphenyl)propane] (BPA) is a substance primarily found in polycarbonate resin, which is mainly used in the production of plastic bottles and packaging, particularly for food and beverages. It is also present in containers intended for microwave use. Unfortunately, when these containers are exposed to heat, they can release small amounts of BPA into the food. In Italy, a prospective cross-sectional study was conducted involving 48 women aged 18–40 affected by infertility, aiming to investigate the role of BPA in reproductive health. The number of individuals with detectable BPA levels – at the detection limit of 0, 5 ng/ml – was higher among infertile women compared to fertile ones [23]. Bisphenol A can disrupt the hypothalamic-pituitary-gonadal (HPG) axis and its feedback circuit. It has been shown that this substance can alter levels of gonadotropin-releasing hormone (GnRH) by regulating kisspeptin expression, which in turn affects the release of follicle-stimulating hormone (FSH), luteinizing hormone (LH), and sex hormones. This can lead to dysregulation of the HPG axis [24]. Additionally, it is postulated that bisphenol A may contribute to the development of polycystic ovary syndrome (PCOS), which also disrupts women's reproductive health [25].

Phthalates, or phthalic acid esters (PAE), are compounds that increase the plasticity of polyvinyl chloride (PVC). They are mainly used in cosmetics, as well as in the production of packaging, including beverages and food [26]. Diethylhexyl phthalate (DEHP) can affect all stages of follicle development up to ovulation in rodents. Therefore, exposure to this substance during fetal life may lead to a significant reduction in the number of oocytes, dysregulation of meiosis, and depletion of the follicle pool. The study based on data from the National Health and Nutrition Examination Surveys (NHANES), which included a total of 857 women of reproductive age (18–45 years), indicates that phthalate metabolites are associated with infertility in women of this age [27].

Parabens and phenols are also widespread environmental contaminants. Parabens are compounds which are widely used in products such as plastics and personal care items. Due to their antimicrobial properties, their presence appears to be unavoidable. Benzophenone-3 is an UV filter used in sunscreens, and although few studies have been conducted so far, it is suggested that it may exhibit weak estrogenic and anti-androgenic activity. Triclosan, which has antibacterial and antifungal properties, is added to products such as toothpaste and soaps. However, there is evidence from animal studies indicating that this substance can affect reproductive hormone levels. Parabens show relatively low affinity for estrogen receptors, yet it has been documented that with prolonged exposure, they can exhibit certain agonistic properties, which are associated with adverse health effects and alterations in reproductive hormone levels [28].

Dioxins are a group of chemicals released into the environment as by-products of municipal waste and fuel combustion, or resulting from chemical production, such as pesticide production. They pollute the environment and can be a source of water and food contamination. Studies on the effects of dioxins on women's health have shown that exposure to these substances is associated with adverse reproductive outcomes, as well as reduced fetal growth, even at intake levels below the tolerable weekly intake of 14 picograms of toxic equivalents per kilogram of body weight per week [29].

3. Results

Growing amount of evidence confirms the earlier existing hypothesis of strong connection between environmental exposure and female reproductive system dysfunction. Cumulative data shows that EDCs (endocrine disrupting compounds) interfere with hormone signalling, reduce fertility, disrupt follicular development and are linked to the pathogenesis of female reproductive system disorders such as polycystic ovary syndrome (PCOS) and endometriosis. Pesticides, especially organochlorine compounds, were consistently linked to prolonged time to pregnancy and reduced ovarian function. Elevated blood concentrations of DDE, β-HCH, HCB were associated with altered ovulatory cycles, lower progesterone and estradiol levels and reduced ovarian reserve [4, 22]. Animal studies show morphological changes in ovaries and pituitary gland dysfunction through disrupted LH pulses and cyst formation [21]. BPA is one of the most intensively studied EDCs today. Several studies suggest that BPA contributes to PCOS pathogenesis through hyperandrogenism and metabolic disturbance mechanisms [25]. BPA altering FSH, LH secretion through interfering with hypothalamic-pituitary-gonadal axis and interference with kisspeptin-mediated GnRH regulation [23, 24]. Phtalates were linked to significant disruption of folliculogenesis. Animal studies show impaired meiosis, decreasing of an ovarian reserve and follicular atresia [25]. Human supported epidemiological research supports these findings [27]. Parabens and related phenols displayed lower estrogen receptor affinity, but still induced endocrine activity, in particular changes in estrogen and androgen levels, upon prolonged exposure [28]. Dioxins were shown to affect the reproductive system even at low exposure levels, highlighting their significance as environmental toxins [29]. In conclusion all presented data confirm that EDCs contribute significantly to female reproductive system pathogenesis. They alter hormonal signalling, interfere with gametogenesis, ovulation and contribute to chronic gynecological illnesses.

4. Discussion

Present-day research strongly confirms that exposure to endocrine disrupting chemicals (EDCs) is a significant risk factor for female reproductive health, affecting hormonal homeostasis, folliculogenesis, and overall fertility. The substances examined in our review—including pesticides, bisphenol A (BPA), phthalates, parabens, phenols, and dioxins—interfere with various endocrine pathways. These disruptions are particularly concerning in women of reproductive age, where hormonal balance is critical for ovulation and successful conception [2, 23].

Pesticides, especially organochlorine compounds such as DDE, β-HCH, and HCB, have been consistently associated with prolonged time to pregnancy and decreased ovarian function [2, 21]. Both epidemiological and animal studies highlight their capacity to disrupt luteinizing hormone (LH) pulsatility, lower estrogen and progesterone levels, and cause structural changes in ovarian tissue [21, 22]. Although some of the data are based on animal models, these findings support the hypothesis that environmental pesticide exposure may directly impact pituitary-ovarian axis functionality.

Bisphenol A (BPA), a ubiquitous compound found in food packaging and plastic containers, has been shown to dysregulate the hypothalamic-pituitary-gonadal (HPG) axis. It alters gonadotropin secretion and interferes with kisspeptin-mediated regulation of GnRH, which is crucial for maintaining regular ovulatory cycles [23, 24]. Several studies support its role in the pathogenesis of polycystic ovary syndrome (PCOS) through mechanisms involving hyperandrogenism, insulin resistance, and chronic anovulation [25]. Importantly, BPA exhibits endocrine-disrupting effects even at low doses, which raises questions about current safety thresholds and regulatory standards [23, 26].

Phthalates, commonly used in plastics and cosmetics, are also associated with adverse reproductive outcomes. Data suggest that prenatal or prolonged exposure can impair meiosis, deplete ovarian follicle reserve, and increase follicular atresia [25]. These effects may compromise fertility even before clinical symptoms become apparent. Human studies, such as those based on NHANES data, further support a correlation between phthalate metabolites and infertility in women of reproductive age [27].

Parabens and phenols, although characterized by lower estrogen receptor affinity, can still exert estrogenic and anti-androgenic activity, especially upon chronic exposure. Their widespread use in personal care products, combined with accumulating evidence of hormonal modulation, calls for more comprehensive toxicological evaluation, particularly in adolescent and reproductive-age women [28].

Dioxins, known for their environmental persistence and high toxicity, can negatively impact female reproductive health even at exposure levels below current safety thresholds. These compounds accumulate in adipose tissue and can be transmitted transgenerationally, further amplifying their potential long-term

consequences. Some of their effects include reduced fetal growth, altered menstrual cycles, and increased risk of endometriosis [20, 29].

Despite the increasing body of evidence, several limitations must be acknowledged. First, results are sometimes inconsistent across studies—especially between animal models and human cohorts—which may stem from differences in exposure windows, individual susceptibility, and co-exposure to other environmental toxins such as heavy metals [30]. Second, many studies fail to adequately assess the effects of EDC mixtures, which may act synergistically or antagonistically. Third, the long latency period between exposure and clinical manifestation complicates risk assessment [22, 23, 31].

From a clinical and public health standpoint, our findings underscore the need for stricter regulatory policies, especially regarding substances present in consumer goods targeted at women of reproductive age [20]. Biomonitoring strategies, including routine testing for EDC biomarkers (e.g., BPA in urine), could help identify high-risk individuals. Furthermore, educational initiatives promoting awareness of EDC sources and safer alternatives are essential, especially for adolescents, women planning pregnancy, and healthcare providers [26].

Finally, there is a pressing need for long-term, mechanistic and transgenerational studies. Many molecular pathways involved in EDC-related reproductive dysfunction remain unclear, particularly their epigenetic effects [23, 24, 26]. Addressing these gaps requires interdisciplinary collaboration between clinicians, toxicologists, epidemiologists and policymakers.

In conclusion, this review highlights the multifactorial impact of EDCs on female fertility and reproductive health. While scientific understanding has advanced significantly, actionable progress depends on translating this knowledge into both clinical practice and regulatory frameworks.

5. Conclusions

Growing amounts of evidence, including findings discussed in our review, points to the potential risks EDCs pose to female fertility. EDCs analysed in the study (pesticides, BPA, phthalates, parabens and phenols, dioxins) interfere with hormone signaling and disrupt ovarian function through multiple mechanisms – inluencing steroidogenesis, folliculogenesis, and the regulation of the hypothalamic-pituitary-gonadal axis. As a result, they are involved in pathogenesis of disorders such as PCOS and endometriosis.

Stricter regulations are absolutely necessary to curb exposure to EDCs, especially in products aimed at women of reproductive age. By incorporating biomonitoring and early detection methods, we can better identify individuals and groups who are at risk. Ongoing investment in long-term and mechanistic research is vital to fully understand how EDCs work and guide the creation of safer alternatives. Additionally, it is crucial to raise public awareness about the reproductive dangers linked to EDCs. Without a broad understanding and informed choices from individuals, efforts to minimize exposure – particularly for vulnerable populations like girls and women of childbearing age – will fall short. Safeguarding female fertility in a world filled with EDCs demands not just scientific and regulatory advancements, but also empowered and well-informed communities.

In conclusion, the presented data confirms the contribution of EDCs to the pathogenesis of female infertility, which happens in mechanisms such as altering hormonal signaling, interfering with gametogenesis and ovulation. These pathologies lead to chronic gynecological ilnesses, and as a result – impairment of the female reproductive system.

Conflict of Interest Statement: No conflicts of interest to declare.

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