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Endocrine disruptors in male fertility – a review

Abstract

Introduction. Endocrine-Disrupting Chemicals (EDCs) have an impact on health and disease. We are exposed to EDCs from the earliest years of life, from children's products, personal care products, food containers, pesticides, and herbicides. These chemicals often bind to endogenous receptors (e.g., estrogen receptor, steroid receptor) and disrupt normal brain function, reproductive organs, development, immune system, and many other organs. Recent studies indicate a potential link between exposure to EDCs and male infertility. In this review we also provide practical public health implications, prevention strategies and policy recommendations aligned with the WHO and the EU frameworks for reducing EDCs exposure.

Aim. This review aims to explore the relationship between exposure to Endocrine-Disrupting Chemicals, we can find in items used on a daily basis and male reproductive health focusing on mechanisms of action, exposure timing and substance specific consequences.

Materials and methods. A narrative review of 62 articles retrieved from PubMed and open access sources (published up to May 2025) was conducted. The included literature covered molecular mechanism, function of Leydig and Sertoli cells, testosterone synthesis, semen analysis. Emphasis was placed on studies investigating adolescent and adult populations as well as animal models.

Conclusions. EDCs interfere with hormonal homeostasis and spermatogenesis through numerous often very subtle mechanisms. Alarming is high sensitivity of the male reproductive system during prenatal development and puberty, while even low-dose exposures can lead to lasting or even transgenerational effects.

Keywords: Endocrine Disrupting Chemicals; EDCs, hormonal system, male reproductive system, male fertility.

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INTRODUCTION

Endocrine-disrupting chemicals (EDCs) are a diverse class of natural and synthetic compounds that can interfere with the body's hormonal system. They occur in everyday items, such as plastic containers, cosmetics, cleaning products and even food packaging,so their potential biological effects are a growing public-health concern. According to the World Health Organization (WHO) and the United Nations Environment Programme (UNEP), EDCs are substances that can alter one or more endocrine functions, leading to adverse health outcomes in individuals, their offspring or entire populations [1]. More recent expert statements reaffirm that EDCs "mimic, block or otherwise interfere" with hormone action, often at doses well below traditional safety thresholds [2].

Human exposure to EDCs is widespread and largely unavoidable, occurring through ingestion, inhalation and dermal contact. Such exposure now extends far beyond occupational settings, because EDC-containing consumer products dominate modern life [3]. Prominent examples include phthalates, bisphenol A (BPA), polychlorinated biphenyls (PCBs), dioxins, parabens and numerous pesticides, all of which are routinely detected in food, water, air and household dust [4,5].

Experimental and epidemiological data show that EDCs can act as hormone mimics or antagonists, disrupt hormone synthesis and metabolism, and interfere with intracellular signaling - often at very low concentrations. Notably, many EDCs exhibit nonmonotonic dose–response curves, whereby small doses during sensitive life stages (e.g., in utero development or puberty) can result in disproportionately strong biological effects [6,7]. Emerging work also demonstrates that EDC exposure can remodel the epigenome, promoting heritable changes in gene expression that persist across generations [8].

Male fertility appears to be particularly vulnerable. A regional and global meta-analysis covering data up to 2021 reported an ≈50 % decline in sperm concentration and total sperm count since the 1970s [9]. Parallel increases in disorders such as cryptorchidism, hypospadias and testicular dysgenesis are increasingly attributed to environmental EDC exposures. The male reproductive system, especially during its develop-

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mental stages, is highly vulnerable to hormonal disturbances, and mounting evidence shows that in-utero or early-life exposure to EDCs can permanently impair spermatogenesis and hormone regulation [10,11].

Given the ubiquity of EDCs, the complexity of their molecular actions and the inconsistencies among regulatory guidelines, an up-to-date synthesis of the current evidence is urgently needed. This narrative review summarises findings on the impact of endocrine disruptors on male fertility, highlights key mechanistic insights and identifies critical research gaps. By clarifying these issues, we aim to support a coordinated scientific, clinical and policy response to this escalating public-health challenge.

State of knowledge

Widely spread antiandrogens, xenoestrogens and dioxins are found to be best characterised endocrine disruptors of male reproductive system. Especially early exposure to EDCs in utero or during breast-feeding, impacts future fertility [2]. In the year 1976 a chemical factory explosion, which was located near Seveso in Italy led to enormous contamination of the environment with 2,3,7,8-tetracholorodibenzo-p-dioxin (TCDD or dioxin). Dioxin is not only an EDCs but also a well known human carcinogen. It has the ability to accumulate in fat, hence its long half-life. The intensity of adverse events was associated with distance from the accident, therefore researchers divided polluted areas into zones. Observational studies proved that sons from zones A and B exposed in utero or breast-fed showcased lower sperm concentration, progressive motility and total motile count compared to unexposed males. Additionally in utero-exposed groups higher follicle stimulating hormone and lower inhibin B was found, that strongly supports dioxin impact on Sertoli cell proliferation along with permanently reduced sperm quality [12,13]. Another study investigated the long-term effect of dioxin after Seveso incident in three age groups 1-9, 10-17 and 18-26 years old. Conversely, displayed results suggested that puberty exposure (10-17 years old) resulted in a stimulatory effect on semen parameters. In this group researchers found increased total sperm count and FSH, also reduced estradiol. During adulthood (18-26 years old) no statistically significant differences in sperm variables were detected [14]. Generally considered, dioxins are by-products of industrial processes, volcanic eruptions and forest fires. Humans come across them in food, especially dairy products, meat, fish and shellfish [15].

Climbazole (CBZ) is another EDC. It has an antifungal effect that is widely used in personal care products for example shampoos. Studies suggest that CBZ disrupts hormonal balance in male by acting as agonist of estrogen receptor alfa (ER alfa) and androgen receptor antagonist. Moreover, CBZ induces premature capacitation, which leads to reduced sperm efficiency within the female reproductive tract. The CBZ upregulates hormone receptors: ESR1, ESR2 and FSHR in non-capacitated sperm. In capacitated sperm CBZ downregulates ESR1 and ESR2, but maintains elevated FSHR expression. Another specific mechanism is that CBZ alters tyrosine phosphorylation by changing localisation of phosphorylated residues in the acrosome, midpiece and tail regions, disrupting the combinations critical for motility regulation and acrosomal exocytosis. Long term exposure causes compromised sperm function, hormonal imbalance, increased DNA damage, as well as transgenerational consequences [16,17].

Attention must also be paid to phthalates (phthalic acid diesters), chemicals produced mainly for the manufacture of polyvinyl chloride (PVC) plastics. Phthalates are present everywhere, in food casing, bottles, wrappers, microwave food, dress materials, dwelling houses and scent retainers in personal care products. They are mostly lipophilic, easily absorbed to human blood and quickly metabolised, some of these metabolites act as EDCs [18]. According to present knowledge, phthalates especially affect Leydig cell function, by increased LH levels coupled with largely unaffected testosterone. This implies that a more powerful LH signal is needed to produce the same amount of testosterone. Studies show that larger or sustained LH stimul ation may stress Leydig cells and lead to wearing them out of function in adulthood. Over time these changes can potentially reduce male fertility. The most vulnerable window of exposure detected was early life years [19]. Phthalates present differences in potency or activity. Diethylhexyl phthalate (DEHP) and dibutyl adipate (DBP) are strongly associated with anogenital distance alterations, semen parameters and testosterone levels. Only slight evidence links diisobutyl phthalate (DIBP) and diethyl phthalate (DEP) with male reproductive outcomes, due to fewer studies conducted and lower exposure levels. Furthermore, animal studies on DEP exposure have shown reduced anti-androgenic effect compared to other phthalates [20]. EDCs are found to be responsible for both multigenerational and transgenerational effects on reproductive health. Research indicates that ancestral exposure to DBP alters anogenital distance, causes early puberty and testicular disease, decreases fertility and sperm count, alters sperm morphology and changes sex steroid hormone levels [21].

Another EDC to consider is bisphenol A (BPA), a substance used to manufacture polycarbonate plastic, also a transparent and rigid type of plastic, applied in water dispensers, food containers and reusable beverage bottles. In addition it is adapted to create resins, which are found in protective coating and lining for food and beverage cans and vats [22]. Animal study conducted on mice revealed that prenatal BPA exposure leads to decreased serum testosterone levels and testis weight, reduced sperm count, motility and acrosome integrity, arrested meiotic transition from zygotene to pachytene in spermatocytes, reduced sperm-egg binding capacity and abnormal embryonic cleavage [23]. Besides, outcomes of BPA exposure depend on the dosage, low BPA levels (0,0001 μM) reduces mitochondrial membrane potential and ATP levels, while high levels (100 μM) increase ROS rates, inducing mitochondrial mediated apoptosis in Leydig and Sertoli cells. It was also revealed that BPA impairs stereogenic enzyme mRNA expression and testosterone synthesis in Leydig cells and reduces anchoring junction proteins in Sertoli cells [24]. Moreover, BPA disrupts the hypothalamic-pituitary-gonadal axis negative feedback loop, thus decreasing male fertility [25]. Studies conducted on humans highlight the negative impact of BPA on male reproductive system, as well as the fact that all participants tolerable daily intake of BPA (set by the European Food Safety Authority) was exceeded, which raises a concern about population exposure levels [26,27].

It cannot be overlooked that pesticides, widely used in agriculture, also disrupt male reproductive health by rising global infertility rates. The most widespread, organochlorines (OCs) were successfully used in the control of malaria and typhus, but many advanced countries banned their usage [28].

OCs are linked with massive influx of calcium ions into germ cells, which reduces mitochondrial membrane potential, together with increased sperm DNA condensation [29]. Farmers daily exposed to pesticide presented significantly different reproductive hormone levels between growing periods, when pesticides are actively used and non-growing periods. A ten-year retrospective study of men in Cameroon found that farmers over the age of 50 were the most susceptible to poorer semen quality, an effect interpreted as the combined effect of pesticide exposure and high temperatures. A comparison of semen from farmers in Myanmar who were occupationally exposed to various types of pesticides on a daily basisshowed reduced sperm counts during crop-growing periods compared with non-growing periods, and lower counts than those observed in men from the general population [30]. A study conducted in Costa Rica, revealed that pesticides, especially mancozeb, dieldrin or chlorothalonil, cause the decreased semen quality and testicular dysfunction. Additionally, pesticides are found to alter synthesis, activity and concentration of thyroid hormone, crucial to reproductive health [31].

Differences in male fertility can also be observed in terms of geographic distribution also within the same continet. A threefold higher incidence of testicular cancer, which may be linked to prenatal exposure to EDCs,has been reported in Denmark and Norway compared with Estonia and Finland. This led researchers to investigate semen quality, which in turn revealed an east–west gradient in semen quality across the Nordic-Baltic region [32].

the impact of EDCs on male reproductive health has not yet been fully explored. Further research is crucial to establish the full role of EDCs. The research highlights that the development stage of male reproductive organs is the most vulnerable to environmental toxins and implicates the importance of minimising infancy and puberty exposure. Underlying mechanisms of EDCs action involve receptor binding interference, hormone production and secretion alterations, oxidative stress and epigenetic alteration [33].

There is also the concept of synergism. It refers to situations where the combined effect is greater than the sum of activities of the individual components at the same level of exposure. EDCs, particularly pesticides and heavy metals, may interact with each other and enhance their toxicity. On the other hand, the mechanisms underlying their mutual synergistic interactions have not yet been adequately studied [34]. It has also been shown that many antiandrogenic chemicals act in combination, eliciting effects at doses that, on their own, are not associated with any observable responses. Thus, mixed exposures to EDCs may have a pivotal impact on male fertility [35].

METHODS

The literature selection process was based on clearly defined inclusion and exclusion criteria. Studies were included if they addressed the effects of endocrine-disrupting chemicals on male fertility, covering aspects such as semen quality, hormone levels, testicular function, reproductive outcomes, or molecular mechanisms. Eligible sources consisted of peer-reviewed original research articles (including human studies, animal models, and in vitro experiments), systematic reviews, meta-analyses, and high-quality review papers providing mechanistic or epidemiological insights. The target population included human males from the general population, occupationally exposed groups, and infertile men, as well as animal studies with translational relevance to human fertility. Only articles published within the last 20 years (2005-2025) were considered to ensure inclusion of recent evidence and current understanding. Additionally, only English-language publications with full-text availability, either through institutional access or open access, were selected.

Exclusion criteria encompassed articles not directly related to endocrine disruptors, non-scientific sources, studies with unclear methodology, insufficient data or lack of outcomes related to male fertility, as well as duplicate publications or preliminary reports later updated by the same authors. Literature searches were conducted in the following databases: PubMed, ScienceDirect, and Web of Science. The quality appraisal favored studies with robust designs, including human epidemiological research with adequate sample size and appropriate control for confounding factors, as well as animal studies employing validated exposure models and relevant reproductive endpoints.

Selection stages:

1. Records identified in databases (n=2713).
2. Records after removing duplicates (n=2202).
3. Records after screening titles and abstracts (n=530).
4. Full texts assessed for eligibility (n=350).
5. Studies included in the review (n=60).

RESULTS

This overview identifies and explores research findings on the impact of EDCs on male fertility, with a focus on mechanisms of action, exposure timing and substance specific consequences.

Studies after the Seveso disaster showed that in utero and early exposure to TCDD significantly reduced sperm quality, motility and count, with elevated FSH and decreased inhibin B indicating Sertoli cell dysfunction.

Exposure during puberty showed paradoxical stimulatory effects on semen parameters, while no significant effect was observed in adult males [12-15].

Phthalates, such as diethylhexyl phthalate (DEHP) and dibutyl phthalate (DBP) were shown to disrupt Leydig cell function by increasing LH levels without corresponding increase in the level of testosterone, suggesting a compensatory overstimulation. Early exposure was most strongly associated with reduced anogenital distance, reduced semen quality and decreased fertility. Animal studies indicated transgenerational consequences, including testicular abnormalities and hormonal disruption [18-20].

Animal models showed that prenatal BPA exposure reduced testosterone levels, testis weight, sperm motility and acrosomal integrity and also impaired meiotic progression. BPA triggered oxidative stress, apoptosis in Leydig and Sertoli cells, and disrupted hypothalamic-pituitary-gonadal axis. Human studies revealed widespread BPA exposure exceeding tolerable daily intake limits, with correlation to altered hormone ratios and reduced sperm quality [23,24].

Climbazole, an antifungal agent in cosmetics, disrupts hormonal balance by modulating receptors. Acting as an androgen antagonist and estrogen agonist, it alters sperm receptor expression, leading to premature capacitation and reduced motility [25,26].

Pesticides are difficult to avoid, with farmers being at greatest risk. Organochlorines disrupt germ cells by altering calcium influx and DNA integrity, while mancozeb, chlorothalonil and dieldrin reduce semen quality and cause testicular dysfunction. Studies of sex hormones in farmers during the pesticide use season, compared with the season without their use, additionally showed their negative impact on reproductive health.

Dioxins, climbazole, phthalates, bisphenol A (BPA) and pesticides significantly impair male reproductive health, particularly during developmental stages, when children are most vulnerable to toxic exposures. Exposure to dioxin lowers sperm quality and causes hormonal imbalance in exposed men, which was established by Sevenso et al. in 1976. Another substance, climbazole, impacts hormonal balance and function of sperm through receptor modulation. Phthalates, present in food containers, toys, medical devices, cosmetics and paints, are widespread in the environment, contaminating soil, water and air during production, use and disposal [36]. Phthalates negatively influence Leydig cells leading to decreased testosterone production. As another dangerous substance is BPA, the exposure to BPA has many proven adverse effects, including reduction of sperm count and quality, and of testosterone levels. BPA, used in epoxy resins and food packaging, alters estrogen pathways, increasing estradiol and the levels of sex hormone-binding globulin (SHBG) and disrupting the androgen-to-estrogen ratio [6]. Pesticides, another major group of EDCs, reduce semen quality and contribute to rising infertility rates.

In order to enhance clarity, a summary of the main endocrine-disrupting chemicals and their effects is presented in Table 1.

TABLE 1. Summary of the main endocrine-disrupting chemicals and their effects.

EDC	Exposure source	Primary target(s)	Mechanism of action	Observed effect
Dioxins (TCDD)	Industrial by-products, contaminated food (fish, diary, meat)	Sertoli cells	Binds Ahr receptor, disrupts Sertoli cells proliferation,	↓ Sperm concentration, motility, total motile count, ↑ FSH, ↓ inhibin B
Climbazole (CBZ)	Personal care products	Sperm cells, hormone receptors	ERα agonist, AR antagonist, alters tyrosine phosphorylation	↓ Sperm efficiency, motility, ↑ DNA damage, hormonal imbalance, transgenerational risk
Phthalates (DEHP, DBP and others)	Plastics,food containers, personal care products	Leydig cells	Anti-androgenic effect, ↑ LH without ↑ testosterone, disrupts steroidogenesis	Altered anogenital distance, ↓ semen quality, Leydig cell dysfunction
Bisphenol A (BPA)	Plastics, epoxy resins, food packaging, thermal paper	Leydig & Sertoli cells, HPG axis	ER modulator, oxidative stress inducer, mitochondrial disruption, epigenetic changes	↓ Testosterone, ↓ sperm count/motility, impaired spermatogenesis, altered hormone ratios
Pesticides (OCs, mankozeb, diedrin, chlorothalonil)	Agriculture, food residues	Germ cells, thyroid function alteration	Mitochondrial dysfunction, oxidative stress, thyroid disruption	↓ Sperm count and quality, DNA damage, testicular dysfunction, altered hormone levels

DISCUSSION

The findings of recent research strongly confirms that exposure to endocrine disruptors is a significant risk factor for male infertility, as it affects not only semen quality but also hormonal homeostasis.

Human biomonitoring is a tool necessary for measuring the presence and concentration of chemicals in the human body by examining blood, urine, or hair samples. The European Human Biomonitoring Initiative, HBM4EU, conducted a study in which urine samples were taken from 2,756 adults from 11 different countries (Croatia, the Czech Republic, Denmark, France, Finland, Germany, Iceland, Luxembourg, Poland, Portugal, and Switzerland) to measure the concentration of Bisphenol A and two other bisphenols used as substitutes for BPA, bisphenol S and bisphenol F. BPA was detected in 92% of the adult participants from 11 European countries. BPA levels in European urine were assessed with reference to the HBM-GV, the threshold below which no adverse effects are expected [37,38]. The tolerable daily intake (TDI) threshold was recently updated by the European Food Safety Authority (EFSA) based on evidence that BPA affects the immune system. The new TDI of 0.2 ng/kg bw/day translates to a HBM-GV value of 11.5 ng/L total BPA in urine in adults [39]. In 11 countries participating in the biomonitoring initiative, the level of BPA exceeded 71% to 100%. Exposure to BPA in Europe is too high and poses a potential health risk [37].

Swan et al. carried out a study, in which metabolites such as mono-(2-ethylhexyl) phthalate (MEHP) and mono-n-butyl phthalate (MnBP) have been extensively measured in urine to evaluate dose-response relationships with sperm motility, densityand DNA integrity [40]. The study investigated prenatal phthalate exposure and its impacts on male infants, using anogenital distance (AGD) as a developmental marker. Data from 134 boys aged 2-36 months indicated AGD was strongly related to penile volume and to incomplete testicular descent. Elevated prenatal monobutyl phtalate (MBP) levels were linked to a 10.2 odds ratio for decreased anogenital index [anogenital index=AGD/weight (mm/kg)] (95% CI, 2.5 to 42.2). These findings suggest that environmental levels of phthalates are damaging to the development of male reproduction, with implications for phthalate-related reproductive syndrome knowledge.

Advanced analytical techniques like high-performance liquid chromatography coupled with tandem mass spectrometry (HPLC-MS/MS) allow for sensitive quantification and identification of such biomarkers. Silva et al. in their study compared urinary levels of seven monoester metabolites of phthalates in 2,540 NHANES 1999-2000 samples and reported ubiquitous exposure across the United S tates. [41]. Over 75% of samples had detectable levels of monoethyl phthalate (MEP), MBP, monobenzyl phthalate (MBzP), and MEHP. Interestingly, non-Hispanic blacks had significantly higher levels of MEP than others, and children had significantly higher levels of MBP, MBzP, and MEHP than adolescents and adults.

Despite the growing amount of evidence, there are some significant inconsistencies in the interpretation of the impact of EDCs on reproductive functions. In a cohort study of 266 fertile men from the Faroe Islands, who had been previously exposed to high levels of polychlorinated biphenyls (PCBs), researchers found no direct association between exposure and semen parameters, despite the disruption of testosterone-to-estradiol ratio [42]. This apparent contradiction may be the

result of individual sensitivity, timing of exposure (prenatal versus adult), or interactions with other environmental factors, such as heavy metals or pesticides [6]. Additionally, results of studies on animal models indicate that the effects of EDC exposure may not appear until several generations later, which makes risk assessment from a transgenerational perspective even more difficult [36].

There are multipronged clinical implications of these findings. First of all there is a strong need to implement rigoristic regulations concerning the use of endocrine disrupting substances in medical and cosmetic products. There are alternative plasticisers, such as DINCH (diisononyl cyclohexane-1,2-dicarboxylate), which exhibit less toxicity [20]. Secondly, implementation of exposure biomarker tests (e.g., phthalate metabolites in urine) should be included in the routine diagnostic process for male infertility, which would enable better identification of high-risk subpopulations [43]. Finally, long-term cohort studies are essential to evaluate the cumulative effects of EDC mixtures, considering epigenetic mechanisms of damage inheritance [6]. An interdisciplinary approach combining exposure assessment, molecular research and health policy is essential to address male reproductive health risks.

The current scope of research does not cover the impacts of EDCs on male reproductive health, hence more scientific research is needed. Still many disrupting substances or their mechanisms of action remain unknown. Another pressing issue is the necessity of informing the general public about endocrine disruptors, as this topic is still not a very widely known problem. It is crucial to minimise exposure of males during infancy and puberty. The lack of knowledge in this case may cause lifelong consequences not only for men's reproductive health, but also for their overall well-being and that of their partners.

In most countries, infertility is not covered by public funds, but by the private sector, which is driven by profit. Individuals and families undergoing these procedures may experience not only a huge financial burden, but also physical and psychological strain. The desire to have a child can be extremely strong, and infertility can have a huge impact on mental health, leading to anxiety, depression, and relationship problems. Stigma associated with infertility and IVF can also cause feelings of shame and isolation [44].The growing problem of infertility and the decline in birth rates coincides with the increase in life expectancy. For many individuals, a longer life may seem like good news, but combined with low fertility rates, it will lead to significantly ageing populations in the future, as well as a decline in the proportion of the population of working age. An aging population will increase fiscal pressure, as government spending (including pensions, health services, and services for the elderly) will increase, while tax revenues may decrease as the working-age population shrinks [45]. As the working-age population decreases, there is more room for migration, which entails the need to compensate for labor shortages. However, it is also an opportunity for education among young people, as well as greater participation of women in the workforce [46]. Furthermore, extending the period of professional activity,for example, by raising the retirement age, as well as future increases in life expectancy ,can reduce the pressure on pension systems [47,48].

Public health implication, prevention strategies and policy context (WHO, EU)

Given the overwhelming presence of EDCs in consumer products and food contact materials primary prevention is necessary. International public health organisations emphasise reduction of exposure to EDCs, improving testing and identification methods, as well as human biomonitoring to guide policy[1,49-52].

The European Food Safety Authority re-evaluated BPA with a dramatically reduced the tolerable daily intake [39].

WHO/UNEP assessments highlight EDCs risks and recommend exposure reduction, as well as research and health sector engagement. In the EU the Chemicals Strategy for Sustainability under the Green Deal and Zero Pollution programs promotes measures on EDCs identification. BPA is currently banned in plastic feeding bottles for infants and restricted in thermal paper. Pesticides regulation includes scientific criteria to identify EDCs [51,53-55].

Practical recommendations for different groups:

1. Clinicians (primary care, andrology, fertility medicine, obstetrics):
   - Question patients about their environmental exposure history (workplace, fragranced products, food-contact plastic,pesticides).
   - Advise patients to avoid heating food in plastic, choosing fresh uncanned food where possible and switching to glass or stainless steel food storage [56,57].
   - Discuss personal care and cosmetic choices, advise to use products that are fragrance-free and without phthalates/parabens where possible [58].
   - Advise to limit the use of thermal receipts and preferring digital receipts if possible, especially for pregnant patients [53,59].
   - In occupational high-risk cases consider biomonitoring (e.g., urinary phthalates metabolites, bisphenols).
2. General population:
   - Prioritise non-chemical pest control for gardening, follow label instructions strictly if pesticides are used.
   - Ventilate often and reduce dust accumulation.
   - Store food in glass or stainless steel containers.
   - Avoid heating plastics in the microwave.
   - Decline or minimise handling of thermal receipts. Wash your hands after handling.
   - Use glass baby bottles if possible. If needed choose bottles that specifically labelled 'bisphenol-free', while BPA in plastic infant bottles is banned in the EU broader bisphenol substitutes may still be present [37,60,61].
3. Public health care systems:
   - Extending restrictions in food-contact materials.
   - Improve consumer information labeling for materials containing food.
   - Invest in HBM4EU/PARC aligned biomonitoring to evaluate exposure trends and track the impact of interventions [52,62].

CONCLUSION

There is a growing evidence base, including the study findings, which highlights serious threats of EDCs to male reproduction. These compounds occur not only in industrial processes but also in everyday consumer products.

They interfere with hormonal homeostasis and spermatogenesis through numerous often very subtle mechanisms. Alarming is high sensitivity of the male reproductive system during prenatal development and puberty, while even low-dose exposures can lead to lasting or even transgenerational effects. Despite progress in identifying EDCs and their effects, key challenges remain: variable susceptibility, inconsistent findings, and the complexity of multiple environmental exposures.

In addition, gaps remain in our understanding of dose-response relationships, epigenetic transmission and long-term cumulative effect. This highlights the importance of addressing this public health concern and necessity of a multidisciplinary research. Stricter regulatory frameworks for EDC use in cosmetics and medicine are needed. The integration of biomonitoring could enable early detection of at-risk individuals. Further investment in longitudinal research will be necessary to fully understand EDCs biological effect and pathways, that will lead to development of safer alternatives. Finally, public awareness about the potential consequences must be elevated. Without widespread public understanding of EDCs negative effects, attempts to reduce exposure, especially among sensitive groups like infants, children and adolescents, have a lower chance to succeed. Male reproductive health protection requires not only scientific and medical advancement, but also educated individual decisions and strong public health policy.

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