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Xenoestrogens and Microplastics as Environmental Endocrine Disruptors: Mechanisms, Health Effects, and Relevance to Physical Activity

Marcin Wieleba
ORCID https://orcid.org/0009-0006-9815-4340
E-mail marcin.wieleba09@gmail.com
Medical University of Lublin, Poland

Barbara Izabela Krupska
ORCID https://orcid.org/0009-0002-5755-9377
E-mail bkrupska07@wp.pl
Medical University of Lublin, Poland

Franciszek Włodarczyk
ORCID https://orcid.org/0009-0005-0754-4558
E-mail fwlodarczyk576@gmail.com
University Clinical Hospital No. 1 in Lublin, Poland

Magdalena Zapalska
ORCID https://orcid.org/0009-0007-5777-2959
E-mail magda.zapalska1@gmail.com
Medical University of Lublin, Poland

Weronika Maria Woźniak
ORCID https://orcid.org/0009-0007-4906-7125
E-mail weronika7801@gmail.com
Medical University of Lublin, Poland

Zuzanna Winiarska
ORCID https://orcid.org/0009-0002-3177-4271
E-mail zuzawiniarska22@gmail.com
Medical University of Lublin, Poland

Eliza Wiercioch
ORCID https://orcid.org/0009-0004-0044-301X
E-mail eliza.wiercioch2001@gmail.com
Medical University of Lublin, Poland

Wiktoria Katarzyna Woroszyło
ORCID https://orcid.org/0009-0009-5356-4911
E-mail wiktoria.woroszylo2001@gmail.com
Medical University of Lublin, Poland

Michał Krzysztof Woszczek
ORCID https://orcid.org/0009-0002-1013-6297
E-mail michal.woszczek.00@gmail.com
Medical University of Lublin, Poland

Bartosz Jakub Minik
ORCID https://orcid.org/0009-0009-8315-9087
E-mail bartoszminik2000@gmail.com
1 Military Clinical Hospital with Outpatient Clinic: Lublin, Poland

Corresponding Author:
Marcin Wieleba
marcin.wieleba09@gmail.com

Abstract

Background. Xenoestrogens and microplastics are widespread environmental contaminants with potential endocrine-disrupting effects. Their exposure may interfere with hormonal signaling and contribute to disturbances in reproductive, metabolic, thyroid, and developmental processes.

Aim. The aim of this review was to summarize current evidence on the sources, mechanisms, and health effects of xenoestrogens and microplastics, with particular emphasis on their relevance to physical activity.

Materials and methods. A narrative literature review was conducted based on available human, animal, and in vitro studies addressing exposure to xenoestrogens, endocrine-disrupting chemicals, microplastics, and nanoplastics, as well as their potential endocrine and health effects.

Results. Xenoestrogens and plastic-associated chemicals are encountered through food, drinking water, cosmetics, household products, indoor environments, and other consumer materials, mainly via ingestion, inhalation, and dermal contact. Current evidence suggests potential associations with impaired male and female reproductive health, altered thyroid function, metabolic disturbances, and adverse pregnancy-related outcomes. In physically active populations, limited evidence indicates possible associations between selected EDCs and anaerobic performance, while sports supplements may represent an additional source of estrogenically active compounds. However, the available evidence is heterogeneous and largely observational or derived from animal and in vitro studies.

Conclusions. Xenoestrogens and microplastics may contribute to endocrine disruption and have potential implications for reproductive, metabolic, thyroid, and exercise-related health. However, causal relationships remain insufficiently established. Further longitudinal human studies are needed to determine the clinical relevance of these exposures, particularly in physically active populations.

Keywords: xenoestrogens, endocrine-disrupting chemicals, microplastics, nanoplastics, endocrine disruption, reproductive health, metabolism, physical activity

1. Introduction

Increasing attention has recently been paid to the impact of environmental factors on human hormonal health. Among these factors, endocrine-disrupting chemicals (EDCs) have emerged as an important public health concern due to their widespread presence in the environment and their potential to interfere with endocrine function.

EDCs are defined as exogenous chemicals or mixtures of chemicals that interfere with hormone action. The United States Environmental Protection Agency (EPA) originally defined EDCs as exogenous agents that interfere with specific aspects of hormone action. To account for the continuously expanding knowledge regarding endocrine mechanisms, Gore et al. proposed a simplified definition, describing an endocrine disruptor as "an exogenous chemical, or mixture of chemicals, that interferes with any aspect of hormone action" (Zoeller et al., 2012). Because hormone signaling is receptor-mediated and frequently characterized by nonlinear dose-response relationships, the biological effects of EDCs may also exhibit nonlinear responses, making their toxicological assessment particularly challenging (Zoeller et al., 2012).

Xenoestrogens constitute an important subgroup of EDCs. As exogenous compounds capable of interfering with hormone action, they may contribute to disturbances in endocrine homeostasis through interactions with hormone signaling pathways (Zoeller et al., 2012).

Growing evidence suggests that EDCs may adversely affect multiple physiological systems. Numerous studies have demonstrated associations between EDC exposure and impaired reproductive function, with female fertility being particularly susceptible across a wide range of species, including humans (Rattan et al., 2017). Furthermore, EDCs have been implicated in disturbances of thyroid hormone homeostasis and other endocrine pathways (Zoeller et al., 2012). Poorly defined or inadequately enforced environmental policies may further increase global human exposure to EDCs, contributing to a substantial burden of disease, disability, and economic costs (Duh-Leong et al., 2023).

In recent decades, the increasing prevalence of metabolic disorders has paralleled profound changes in the chemical environment. Besides traditional risk factors, exposure to EDCs originating from plastics, plasticizers, pesticides, electronic waste, and food additives has been proposed as a contributing factor to the worldwide rise in diabetes and other metabolic diseases (Kumar et al., 2020).

Plastics represent one of the major sources of human exposure to EDCs, including xenoestrogens. Exposure of plastic materials to high temperatures can promote the release of microplastics, nanoplastics, and plastic-associated chemicals into the environment. These particles may migrate from packaging materials into food and beverages and subsequently accumulate throughout the food chain, resulting in continuous human exposure through ingestion, inhalation, and dermal contact (Kumar et al., 2020). Biomonitoring studies have demonstrated the presence of microplastics and plastic-associated additives in humans, indicating that exposure is widespread and may pose potential health risks (Kumar et al., 2020). Everyday activities, including the consumption of bottled water and contact with thermal paper receipts, may further contribute to chronic exposure to plastic-derived chemicals (Kumar et al., 2020).

The aim of this review is to summarize current evidence regarding the endocrine effects of xenoestrogens and microplastics, with particular emphasis on their mechanisms of action, major sources and routes of exposure, potential health consequences, and possible relevance to physically active populations.

2. Research material and methods

A narrative literature review was conducted using the PubMed and Scopus databases to identify relevant publications on xenoestrogens, endocrine-disrupting chemicals (EDCs), microplastics, and nanoplastics and their potential effects on endocrine and human health. The search included studies published from January 2012 to July 2026. The following keywords and their combinations were used: “xenoestrogens,” “endocrine-disrupting chemicals,” “EDCs,” “microplastics,” “nanoplastics,” “endocrine disruption,” “reproductive health,” “thyroid function,” “metabolism,” “physical activity,” and “sports supplements.”

The inclusion criteria comprised original research articles, systematic reviews, narrative reviews, and meta-analyses published in English that addressed the sources, mechanisms of action, exposure, or potential health effects of xenoestrogens, EDCs, microplastics, and nanoplastics. Human, animal, and in vitro studies were considered. Articles not written in English, conference abstracts, opinion pieces, and studies with insufficient methodological information or without available full text were excluded from the analysis.

3. Research results

3.1. Sources and Routes of Exposure

Xenoestrogens and other EDCs are widely distributed in the environment and may be encountered through numerous everyday sources. Particular attention has been given to food and drinking water, as plastic packaging and processing materials may facilitate the migration of endocrine-active chemicals into food products. In a study conducted in India, bisphenol A

(BPA) and 13 phthalate esters were detected across a wide range of food products as well as supply and packaged drinking water. Higher levels of phthalates were observed in high-fat foods such as cottage cheese and fish, while packaging materials, food processing, and environmental contamination were identified as plausible sources of exposure. BPA and phthalates were also detected in drinking water, highlighting the importance of dietary and waterborne exposure pathways (Chakraborty et al., 2022).

Beyond food and water, potential sources of exposure include cosmetics, household products, indoor dust, synthetic textiles, and plastic-based materials used in everyday environments. Cosmetics may contain compounds such as phthalates and parabens with potential xenoestrogenic activity, while synthetic textiles and household materials can contribute to the accumulation of microplastics and associated chemicals in indoor environments (Dewika et al., 2023; Gachowska et al., 2024; Kek et al., 2024).

Human exposure may occur through several routes, with oral ingestion, inhalation, and dermal contact representing the major pathways. Ingestion may result from contaminated food, drinking water, and food packaging, whereas inhalation may occur through airborne particles and indoor dust, particularly those originating from synthetic textiles and other plastic-containing materials. Dermal exposure may occur through direct contact with cosmetics, personal-care products, clothing, and plastic-based surfaces (Akanyange et al., 2021; Dewika et al., 2023).

3.2. Mechanisms of Hormonal Disruption

Xenoestrogens may disrupt endocrine homeostasis through several mechanisms that interfere with normal hormone signaling. Their effects can involve both genomic and non-genomic pathways, resulting in alterations in cellular signaling, gene expression, proliferation, and differentiation. Importantly, xenoestrogens may exert biological effects even at relatively low concentrations, while their dose-response relationships can be nonlinear, complicating the assessment of their endocrine activity (Wang et al., 2021; Watson et al., 2014). Furthermore, endocrine disruption may depend on the duration and timing of exposure, with developmental and reproductive periods being particularly sensitive to hormonal disturbances (Wang et al., 2021).

A major mechanism of xenoestrogen activity involves interactions with nuclear and membrane-associated hormone receptors. Due to structural similarities with endogenous estrogens, some xenoestrogens can bind to estrogen receptor alpha (ERα) and estrogen receptor beta (ERβ) and either mimic or antagonize physiological estrogen signaling. These interactions may activate downstream intracellular signaling pathways, thereby altering cellular responses (Watson et al., 2014). In addition to estrogen receptors, EDCs may interact with androgen receptors and other nuclear receptors, potentially contributing to disturbances in reproductive, metabolic, and developmental processes (Wang et al., 2021). Xenoestrogens may also interfere with endocrine function by altering hormone synthesis, metabolism, transport, and elimination. Such effects may involve disruption of steroidogenic pathways and changes in the activity of enzymes responsible for the synthesis and metabolism of sex hormones. Consequently, exposure to EDCs may modify circulating hormone concentrations and disturb physiological endocrine feedback mechanisms (Wang et al., 2021). These effects may extend beyond estrogen signaling, as endocrine-active compounds can also interfere with androgen and thyroid hormone pathways, potentially contributing to broader disturbances in endocrine homeostasis (Wang et al., 2021).

Microplastics and nanoplastics represent an additional concern because they may contain or carry EDCs, including BPA and phthalate-based additives. Due to their physicochemical properties, these particles can act as carriers of associated chemicals and facilitate their exposure to biological systems (Tyc et al., 2025; Ullah et al., 2022). Combined exposure to microplastics and their associated chemicals may contribute to endocrine disruption through several mechanisms, including interference with hormone receptors, oxidative stress, mitochondrial dysfunction, and altered intracellular signaling (Kovacs et al., 2025). Therefore, microplastics may represent not only a source of particle exposure but also a potential vector of endocrine-active compounds with possible implications for reproductive and metabolic health.

3.3. Potential Health Effects

3.3.1. Male Reproductive Health

Exposure to EDCs has been suggested as one of the environmental factors potentially contributing to the decline in male reproductive health. EDCs may interfere with male reproductive function at multiple levels, including the hypothalamic-pituitary axis, testicular cells, spermatogenesis, semen quality, DNA integrity, and epigenetic processes. Reported alterations in semen quality include changes in sperm count, motility, and morphology, which may potentially contribute to male hypofertility and some cases of in vitro fertilization failure (Lahimer et al., 2023).

Among EDCs, BPA has received particular attention. A recent systematic review and meta-analysis of epidemiological studies found that BPA exposure was associated with increased sex hormone-binding globulin (SHBG) and estradiol (E2) levels, as well as reduced sperm concentration and total sperm count, while no significant effect on sperm motility was observed (Lü et al., 2024). However, the overall evidence should be interpreted cautiously. An earlier review of epidemiological studies concluded that evidence linking male BPA exposure with semen quality, reproductive hormones, and reproductive outcomes remained limited and inconclusive. Important methodological limitations included differences between study populations and exposure levels, reliance on single urinary BPA measurements despite its short half-life, predominantly cross-sectional study designs, and uncertainty regarding the biologically relevant exposure window. Furthermore, urinary BPA concentrations were not associated with more clinically relevant reproductive outcomes such as time to pregnancy, implantation, or live birth, although the available studies had limited statistical power to detect small or moderate effects (Mínguez-Alarcón et al., 2016). Overall, current evidence suggests a potential adverse association between BPA and selected markers of male reproductive health, particularly sperm concentration, sperm count, and hormonal parameters, but further prospective studies are required to clarify causality and determine the clinical relevance of these associations.

3.3.2. Female Reproductive Health

Xenoestrogens and other EDCs may interfere with female reproductive function through multiple mechanisms. Xenoestrogens can mimic naturally occurring estrogens and interfere with hormonal homeostasis, while some compounds may also exert their effects through pathways independent of classical estrogen receptor signaling. Their biological effects appear to be tissue-, dose-, and time-dependent, which may contribute to considerable variability in observed reproductive outcomes (Wang et al., 2021).

Exposure to EDCs, including BPA, phthalates, parabens, pesticides, triclosan, polychlorinated biphenyls, and heavy metals, has been associated with a range of female reproductive disorders, including endometriosis, uterine fibroids, polycystic ovary syndrome, premature ovarian failure, menstrual irregularities, altered menarche, and infertility (Hassan et al., 2024). More specifically, a recent meta-analysis including seven studies and 2,488 participants found that BPA exposure was associated with a significantly higher risk of endometriosis (odds ratio [OR] = 1.36, 95% confidence interval [CI] = 1.13–1.63, $p = 0.008$). Sensitivity analyses indicated that the results were relatively robust, although the limited number of studies and their observational design prevent causal conclusions (Chen et al., 2026).

Nevertheless, the overall evidence should be interpreted cautiously. A substantial proportion of research on xenoestrogens is observational or based on animal models, randomized controlled trials are scarce, and numerous confounding factors make direct causal relationships difficult to establish. Furthermore, the potentially dose- and time-dependent nature of xenoestrogen effects may require longer follow-up periods to adequately characterize their consequences for female reproductive health (Wang et al., 2021).

3.3.3. Thyroid Function

Growing evidence suggests that microplastic exposure may interfere with thyroid homeostasis. In a cohort of 1,250 mother–child pairs, higher concentrations of microplastics, including polyvinyl chloride (PVC), polypropylene (PP), and polybutylene succinate (PBS), detected in placental tissue were associated with lower cord-blood thyroxine (T4) and thyroid-stimulating hormone (TSH) concentrations and a reduced T4/triiodothyronine (T3) ratio, with dose-response relationships observed for overall microplastic exposure (Chen et al., 2026). Recent evidence has also demonstrated the presence of microplastics in human thyroid tissue, with higher concentrations observed in patients with autoimmune thyroiditis, particularly for PVC, although the cross-sectional nature of the study does not establish causality (Yu et al., 2026). Mechanistic studies further suggest that micro- and nanoplastics may interfere with thyroglobulin synthesis and thyroid hormone regulation through alterations in PAX8 and CREB expression and modulation of TSH signaling (J. Zhang et al., 2024).

3.3.4. Metabolism, Insulin Sensitivity, and Body Composition

Microplastics have also been investigated as potential contributors to metabolic dysfunction and impaired insulin sensitivity. In vitro exposure of human skeletal muscle cells to polystyrene micro- and nanoplastics reduced glucose uptake and induced mitochondrial dysfunction and oxidative stress, suggesting a potential mechanism linking microplastic exposure with skeletal muscle insulin resistance (Tang et al., 2025). Similarly, animal studies have demonstrated that oral exposure to polystyrene microplastics may disrupt the gut–liver axis, alter intestinal microbiota and metabolic processes, and increase fasting glucose and insulin concentrations (Shi et al., 2022). A recent systematic review integrating epidemiological, experimental, and mechanistic evidence further reported associations between microplastic exposure and metabolic disturbances, including insulin resistance, obesity, and dyslipidemia; however, the available human evidence remains insufficient to establish a causal relationship (Albukhari, 2025).

3.3.5. Possible Developmental and Pregnancy-Related Effects

Pregnancy may represent a particularly sensitive period for the potential effects of microplastic exposure, as the placenta may serve as an important site of microplastic accumulation and fetal exposure. Microplastics have been detected in human placental tissue and fetal meconium, raising concerns about their potential effects on fetal development and pregnancy outcomes (Hunt et al., 2024). In a cohort of 1,250 mother–child pairs, higher concentrations of placental microplastics were associated with alterations in thyroid hormone profiles in cord blood, including lower T4 concentrations and a reduced T4/T3 ratio, suggesting a potential influence on fetal endocrine regulation (X. Zhang et al., 2025). A recent systematic review of human and animal studies further reported potential associations between micro- and nanoplastic exposure and adverse obstetric outcomes, including preterm birth, low birth weight, intrauterine growth restriction, and miscarriage (Novillo-Del Álamo et al., 2026). However, the available evidence remains limited and heterogeneous, and the observational nature of most human studies does not allow causal relationships to be established.

3.4. Relevance to Sport and Physical Activity

Although research directly examining the effects of EDCs on physical performance remains limited, emerging evidence suggests that exposure to certain EDCs may be associated with parameters relevant to athletic performance. In a study involving 54 physically active young adults, dos Santos et al. investigated the relationship between exposure to BPA, bisphenol S (BPS), and 2-naphthol and peak anaerobic power. Higher urinary BPA concentrations were negatively associated with peak anaerobic power, with BPA remaining a significant predictor after adjustment for sex, height, and age. Sex-specific differences were also observed, as both BPA and 2-naphthol were negatively associated with peak anaerobic power in women (Dos Santos et al., 2025). These findings suggest a potential relationship between exposure to selected EDCs and anaerobic performance; however, the small sample size and observational design preclude conclusions regarding causality.

Sports supplements may represent an additional and relatively understudied source of exposure to compounds with estrogenic activity. In an analysis of 50 sports supplements using a validated estrogen-responsive reporter gene assay, estrogenic activity expressed as 17β-estradiol equivalents exceeded levels reported for a typical omnivorous diet in 33 products, while 13 exceeded the acceptable daily intake considered in the study (Plotan et al., 2014). These findings indicate that sports supplements may constitute a potential source of exposure to estrogenically active compounds among physically active individuals. However, because the results were based on in vitro biological activity and exposure modelling, they do not demonstrate that consumption of these products results in clinically relevant endocrine disruption in athletes (Plotan et al., 2014).

More broadly, athletes may experience specific patterns of environmental exposure associated with diet, supplement use, and the training environment, supporting the concept of an athlete-specific exposome encompassing multiple exposure pathways (Wu et al., 2026). Nevertheless, evidence directly linking these exposures to clinically relevant endocrine disturbances or impaired athletic performance remains limited, highlighting an important area for future research (Wu et al., 2026).

4. Discussion

Current evidence regarding the endocrine effects of xenoestrogens and microplastics is limited by the predominance of in vitro and animal studies, which may not fully reflect human exposure and physiological responses. In addition, substantial methodological heterogeneity exists between studies, including differences in particle characteristics, exposure concentrations, experimental models, and analytical methods, making direct comparisons difficult. Another important limitation is the difficulty of distinguishing the biological effects of plastic particles themselves from those of associated chemical additives and other endocrine-disrupting compounds. Finally, the scarcity of long-term human studies, particularly in physically active populations, limits the assessment of chronic exposure and its potential effects on hormonal regulation, metabolism, reproductive health, and exercise adaptation.

5. Conclusions

Xenoestrogens may interfere with multiple hormonal pathways through interactions with hormone receptors and alterations in hormone synthesis, metabolism, and signaling. Microplastics may further contribute to endocrine disruption through biological effects and by acting as carriers of endocrine-disrupting chemicals. Current evidence indicates associations with reproductive, thyroid, metabolic, and developmental outcomes, although the strength of evidence differs substantially between outcomes and much of the available literature does not establish causality. Evidence in physically active populations is particularly limited, despite preliminary findings suggesting potential associations with performance-related parameters and sport-specific sources of exposure. Further well-designed longitudinal and epidemiological studies are therefore needed to clarify the relationships between xenoestrogen and microplastic exposure, hormonal function, metabolism, reproductive health, and exercise-related outcomes.

Disclosure

Author Contributions

Conceptualization: Marcin Wieleba, Barbara Izabela Krupska, Franciszek Włodarczyk, Magdalena Zapalska, Weronika Maria Woźniak

Methodology: Marcin Wieleba, Zuzanna Winiarska, Wiktoria Katarzyna Woroszyło

Software: Marcin Wieleba, Magdalena Zapalska, Michał Krzysztof Woszczek

Check: Barbara Izabela Krupska, Eliza Wiercioch, Bartosz Jakub Minik

Formal analysis: Franciszek Włodarczyk, Weronika Maria Woźniak, Barbara Izabela Krupska

Investigation: Marcin Wieleba, Weronika Maria Woźniak, Zuzanna Winiarska

Resources: Barbara Izabela Krupska, Wiktoria Katarzyna Woroszyło, Bartosz Jakub Minik

Data curation: Franciszek Włodarczyk, Magdalena Zapalska, Michał Krzysztof Woszczek

Writing-rough preparation: Marcin Wieleba, Barbara Izabela Krupska, Franciszek Włodarczyk

Writing-review and editing: Magdalena Zapalska, Weronika Maria Woźniak, Eliza Wiercioch

Project administration: Marcin Wieleba

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The authors deny any conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

In preparing this work, the author(s) used ChatGPT for language improvement and grammatical correction. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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