REVIEW ARTICLE

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Endocrine and metabolic effects of microplastic exposure – current evidence and biological mechanisms

Mikroplastik jako środowiskowy czynnik zaburzający gospodarkę hormonalną i metaboliczną człowieka

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Abstract

Introduction and Objective. Microplastics are widespread environmental contaminants, and their potential impact on human health has attracted increasing scientific attention. Due to their small size and physicochemical properties, they may enter the human body mainly through ingestion and inhalation, and have been detected in several biological matrices, including placenta, blood and lung tissue. The aim of this review is to summarise current knowledge on the endocrine-disrupting effects of microplastics and their potential metabolic consequences.

Brief description of the state of knowledge. Available evidence suggests that microplastics may interfere with endocrine function through oxidative stress, inflammatory signalling, interactions with endocrine-disrupting chemicals, and disruption of intracellular pathways involved in hormone synthesis. Human bio-monitoring studies confirm internal exposure, whereas experimental studies provide mechanistic data on activation of inflammatory pathways, impairment of steroidogenesis, altered insulin signalling, gut microbiota dysbiosis, and adipose tissue dysfunction. These mechanisms may affect major hormonal axes, including the HPA, HPT, and HPG axes, and contribute to disturbances in glucose and lipid metabolism, insulin resistance, and obesity-related processes.

Summary. Current data suggest that microplastics may act as potential modulators of endocrine and metabolic processes. However, most mechanistic evidence comes from *in vitro* and animal studies, and direct causal relationships between microplastic exposure and endocrine or metabolic diseases in humans remain unconfirmed. Further studies using standardized analytical methods, reliable contamination control, and clinically relevant endocrine and metabolic endpoints are needed.

Key words

inflammation, microplastics, oxidative stress, obesity, insulin resistance, endocrine disruptors

Streszczenie

Wprowadzenie i cel pracy. Mikroplastik stanowi powszechnie występujące zanieczyszczenie środowiskowe, którego potencjalny wpływ na zdrowie człowieka jest przedmiotem rosnącego zainteresowania. Ze względu na niewielki rozmiar i właściwości fizykochemiczne cząstki mikroplastiku mogą przedostawać się do organizmu głównie drogą pokarmową i inhalacyjną, przy czym zostały wykryte w kilku matrycach biologicznych człowieka. Celem pracy było podsumowanie aktualnej wiedzy dotyczącej indukowana przez mikroplastik zaburzeń układu endokrynnego i ich potencjalnych konsekwencji metabolicznych.

Opis stanu wiedzy. Dostępne dane wskazują, że mikroplastik może wpływać na funkcję układu endokrynnego poprzez stres oksydacyjny, sygnalizację zapalną, interakcje ze związkami endokrynnie czynnymi oraz zaburzenie wewnątrzkomórkowych szlaków regulujących syntezę hormonów. Badania eksperymentalne ukazują jego wpływ na steroidogenezę, sygnalizację insulinową, skład mikrobioty jelitowej i funkcję tkanki tłuszczowej. Mechanizmy te mogą oddziaływać na główne osie hormonalne, w tym HPA, HPT i HPG, oraz sprzyjać zaburzeniom metabolizmu glukozy i lipidów.

Podsumowanie. Aktualne dane sugerują, że mikroplastik może modulować procesy endokrynne i metaboliczne. Bezpośredni związek przyczynowy między ekspozycją na mikroplastik a chorobami endokrynnymi lub metabolicznymi

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u ludzi nie został jednak potwierdzony. Konieczne są dalsze badania z użyciem wystandaryzowanych metod analitycznych i klinicznie istotnych punktów końcowych.

Słowa kluczowe
insulinooporność, stres oksydacyjny, otyłość, mikroplastik, stan zapalny, związki endokrynnie czynne

INTRODUCTION AND OBJECTIVE

In recent years, the use of plastics – a material, initially perceived as easy and inexpensive to produce – has significantly increased and has now become a subject of global environmental concern. Microplastics (MPs) are defined as plastic particles ranging from 1 μm to 5 mm, whereas nanoplastics (NPs) are generally defined as plastic particles smaller than 1 μm. MPs may be produced intentionally or formed as a result of the degradation of larger plastic materials. These ubiquitous particles are distributed through water, soil, food, and air, entering living organisms and leading to continuous human exposure. Estimated intake varies widely depending on exposure source, diet, drinking water consumption, and analytical methodology, and the potential health consequences remain uncertain [1].

An increasing number of studies have demonstrated the presence of microplastic particles in human tissues and body fluids, suggesting their potential for bioaccumulation. However, despite growing evidence, the biological consequences of this exposure remain incompletely understood [2].

An important aspect of debate regarding the safety of microplastics is their potential impact on the human endocrine system, through endocrine-disrupting chemicals (EDCs) such as bisphenols and phthalates, as well as their direct biological activity [3]. Once inside the body, these particles may act through multiple pathways, including modulation of endocrine receptors, alteration of hormone synthesis, and disruption of endocrine gland function, ultimately affecting major hormonal axes, including the hypothalamic-pituitary axis [4].

Microplastics may also exert deleterious cellular effects through the induction of oxidative stress and chronic inflammation. In experimental models, these processes have been associated with reproductive, metabolic, neurotoxic, and immunotoxic effects [5].

Despite often groundbreaking discoveries, significant research gaps remain, particularly regarding the long-term effects of human exposure [6].

MATERIALS AND METHOD

This narrative review was prepared based on a structured literature search focused on human exposure to microplastics, their detection in human biological matrices, and experimental evidence concerning endocrine and metabolic effects. The literature search was performed using PubMed and Google Scholar and was last updated on 12 August 2026. The final PubMed search was conducted across three predefined thematic areas: human exposure and biomonitoring, endocrine effects, and metabolic effects. The searches identified 602, 441, and 188 records, respectively (1,231 records in total). After removal of 92 duplicate records based on PMID, 1,139 unique records were considered during title and abstract screening for relevance to the predefined scope of the review. Google Scholar was used as a supplementary source to identify additional relevant publications and to trace primary studies cited in review articles. Articles published between 2015–2026 were considered.

The search strategy included combinations of terms related to microplastic exposure, human biomonitoring, endocrine disruption, and metabolic effects. The main search terms were: 'microplastics', 'nanoplastics', 'human exposure, 'human blood', 'placenta', 'lung tissue', 'endocrine disruption', 'oxidative stress', 'inflammation', 'steroidogenesis', 'thyroid hormones', 'insulin resistance', 'glucose metabolism', 'obesity', 'adipogenesis', bisphenol A', and 'phthalates'.

Original studies were prioritized, particularly human biomonitoring studies, and experimental studies describing molecular mechanisms of endocrine or metabolic disruption. Review articles were used to provide background information, identify relevant primary studies, and summarise broader toxicological concepts. Studies were selected according to their relevance to the predefined scope of the review, with priority given to original human biomonitoring studies and experimental studies providing direct mechanistic evidence on endocrine or metabolic effects. Publications were included if they addressed at least one of the following areas: sources of human exposure, detection or quantification of microplastics in human tissues or biological fluids, endocrine-disrupting mechanisms, oxidative or inflammatory signaling pathways, or metabolic outcomes.

Studies were excluded if they were not directly related to microplastic exposure, endocrine disruption, or metabolic effects, or if they focused exclusively on environmental contamination without relevance to human exposure or biological mechanisms. Due to substantial heterogeneity in analytical methods, biological matrices, particle-size detection limits, exposure models, and outcome measures, the findings were summarized narratively rather than pooled quantitatively. Particular attention was paid to distinguishing human biomonitoring data from evidence derived from *in vitro* and animal studies. No formal risk-of-bias assessment tool was applied because of the narrative design of the review. However, studies were critically interpreted with consideration of study design, sample size, analytical methodology, contamination control, particle-size detection limits, exposure model, and the relevance of experimental exposure conditions to human exposure.

DESCRIPTION OF THE STATE OF KNOWLEDGE

Sources and pathways of human exposure to microplastics. Plastic particles, once considered mainly an environmental issue, are now increasingly recognized as a potential factor of human exposure. The main concern regarding microplastics is associated with their ubiquitous presence in water, food, air, and soil, which results in continuous human exposure [7, 8]. Humans are exposed to microplastics primarily through ingestion and inhalation, while dermal contact is generally considered a less significant route of exposure [9].

Table 1. Data on selected sources of human exposure to microplastics

Exposure source	Study material / sample size	Quantitative findings	Reference
Tap water	159 tap water samples	Anthropogenic particles detected in 81% of samples; 0–61 particles/L; mean 5.45 particles/L	Kosuth et al., 2018 [13]
Commercial sea salt	12 brands	Particles detected in all tested brands; mean 212 particles/kg; range 46.7–806 particles/kg	Kosuth et al., 2018 [13]
Bottled water	259 bottles: 11 brands, 19 locations, 9 countries.	93% of samples contaminated; mean 10.4 particles/L >100 µm and 325 particles/L including particles 6.5–100 µm; concentrations ranged from 0 – >10,000 particles/L	Mason et al., 2018 [14]
Fish and seafood	Edible tissues of 4 shellfish species: 2 two shrimp species, 1 crab species, 1 squid species.	Mean 2.7 ± 12 microplastic particles/kg wet weight of edible tissue; substantial variation between species.	Daniel et al., 2021 [10]

Table 2. Quantitative data on microplastics detected in human tissues and biological fluids

Biological matrix	Sample size	Detection frequency	Quantitative findings	Analytical method	Reference
Placenta	6 placentas	4/6 (66.7%)	12 fragments, 5–10 µm	Raman microspectroscopy	Ragusa et al., 2021 [18]
Placenta	62 placentas	100%	6.5–685 µg/g tissue	Pyrolysis–gas chromatography–mass spectrometry (Py-GC/MS)	Garcia et al., 2024 [19]
Blood	22 donors	17/22 (77.3%)	Mean concentration approximately 1.6 µg/mL; PET, PE, styrene polymers and PMMA	Double-shot pyrolysis–gas chromatography/ mass spectrometry (Py-GC/MS)	Leslie et al., 2022 [20]
Lung tissue	13 samples	11/13 (84.6%)	39 particles; 1.42 ± 1.50 MP/g tissue; 0.69 ± 0.84 MP/g after blank subtraction	µFTIR spectroscopy	Jenner et al., 2022 [21]

The data summarized in Table 1 indicate that human exposure to microplastics is not limited to a single source, but results from the simultaneous contribution of dietary, waterborne, and airborne pathways.

Microplastics in the environment can be classified as primary microplastics, intentionally manufactured in microscopic size, and secondary microplastics, formed through the fragmentation or abrasion of larger plastic materials. Primary MPs are used as ingredients in many everyday products, for instance, as abrasive particles in toothpaste and exfoliating products or as formulation bases in creams and aerosols. However, secondary microplastics are considered the major source of environmental contamination. Important sources of secondary microplastics include degradation of plastic waste, abrasion of synthetic textiles, and tire wear particles [10, 11].

Regarding human ingestion, studies indicate that fish and seafood constitute a major source of microplastic intake; however, this largely depends on dietary habits of the studied population [10, 12]. Other dietary and waterborne sources of exposure include table salt, tap water and bottled water [13, 14]. Representative quantitative data describing contamination of major exposure sources are summarized in Table 1. These findings demonstrate considerable variability in reported concentrations depending on the analyzed matrix and applied analytical methodology. Inhalation exposure primarily involves airborne microplastic particles and fibers, originating from both indoor and outdoor sources. Their concentration may be influenced by urbanization, traffic-related emissions, textile fibers and indoor dust [15].

The ubiquitous presence of microplastics in the environment is undeniable and indicates that humans are exposed to these particles simultaneously through multiple pathways. The widespread exposure raises important questions regarding their potential impact on human health, particularly on endocrine system function [16].

Microplastics in human tissues and fluids. The widespread presence of microplastics in the natural environment has raised scientific concern regarding their ability to enter the human body and cross biological barriers. In recent years, human biomonitoring studies have reported the detection of microplastics in several biological matrices, including placenta, blood and lung tissue. These findings support the concept of internal exposure; however, the extent of accumulation, clearance mechanisms and clinical significance remain uncertain [17].

Representative human biomonitoring studies reporting the occurrence and concentrations of microplastics in different biological matrices are summarized in Table 2.

Exposure may begin even before birth. Ragusa et al. provided the first evidence of microplastic contamination in human placental tissue, detecting 12 pigmented microplastic fragments, 5–10 µm in size, in 4 of 6 placentas analyzed [18]. A more recent quantitative study by Garcia et al., using pyrolysis-gas chromatography-mass spectrometry, detected micro- and nanoplastics in all 62 examined placental samples, with concentrations ranging from 6.5–685 µg/g of placental tissue [19]. In human blood, Leslie et al. detected plastic particles in 17 of 22 healthy donors, corresponding to a detection frequency of approximately 77%. The reported mean concentration was approximately 1.6 µg/mL, and the identified polymers included PET, PE, styrene polymers and PMMA [20]. In lung tissue, Jenner et al. identified microplastics in 11 of 13 samples analyzed. A total of 39 particles were detected, with a mean concentration of 1.42 ± 1.50 MP/g tissue, or 0.69 ± 0.84 MP/g after blank subtraction. The most abundant polymer types were polypropylene, polyethylene terephthalate and resin [21]. Microplastic particles have also been identified in the liver, kidneys, and central nervous system; however, the evidence for these matrices is still more limited and less standardized than for placenta, blood and lung tissue [9].

Collectively, available human biomonitoring studies demonstrate that microplastics can be detected in multiple biological matrices, including placenta, blood and lung tissue. Detection frequencies reported in these studies range from approximately 67% in the first placental study to 100% in a recent quantitative placental analysis. These findings support the concept of internal exposure to microplastics, but they should not yet be interpreted as evidence of clinically significant bio-accumulation or toxicity. Although reported concentrations differ substantially between studies, mainly because of differences in analytical techniques, contamination control procedures, particle-size detection limits and reporting units, direct quantitative comparisons remain difficult. Spectroscopic methods, such as FTIR and Raman microscopy, provide information on particle number, size and morphology, but are constrained by method-dependent size detection limits, whereas pyrolysis-GC/MS quantifies polymer mass without preserving information on individual particles. Differences in sample preparation and the risk of procedural contamination further contribute to uncertainty in reported concentrations. Nevertheless, these findings indicate that internal exposure to microplastics is a recurrent observation rather than an isolated finding.

ENDOCRINE-DISRUPTING EFFECTS OF MICROPLASTICS

The widespread presence of microplastics in the human body raises questions regarding their potential biological effects. Particular attention has focused on their potential impact on endocrine function through multiple toxicological mechanisms.

Mechanisms of endocrine action of microplastics. The impact of microplastics on the endocrine system results from multiple interacting biological mechanisms, related both to their physicochemical properties and their ability to bind endocrine-disrupting chemicals (EDCs), such as bisphenols and phthalates. As a consequence, microplastics may affect hormonal regulation at various levels of biological organization, ranging from cellular receptors to intracellular processes, including the induction of oxidative stress and inflammatory responses, which play a key role in cellular damage and disruption of hormonal signaling.

Increased production of reactive oxygen species (ROS) leads to mitochondrial dysfunction, enhanced lipid peroxidation, and DNA damage. Activation of redox-sensitive signalling pathways, such as NF-κB and JNK, contributes to the disruption of cellular homeostasis and the maintenance of a pro-oxidative state, which is considered one of the key pathophysiological mechanisms associated with microplastic exposure [22].

Experimental studies further indicate that oxidative stress is not only a consequence of microplastic exposure, but also an upstream trigger of inflammatory signalling. In an *in vitro* model using Caco-2 cells, exposure to polystyrene microplastics increased intracellular ROS production, resulting in activation of the NF-κB/NLRP3/IL-1β signalling pathway and myosin light chain kinase (MLCK). These molecular alterations were accompanied by reduced expression of the tight junction proteins ZO-1, occludin, and claudin-1. Importantly, pretreatment with the antioxidant N-acetylcysteine markedly attenuated ROS generation and inhibited activation of this pathway, supporting oxidative stress as an initiating mechanism rather than a secondary consequence of cellular injury [5]. These mechanisms may act synergistically, leading to disturbances in endocrine homeostasis at multiple levels of biological organization [3].

Particular importance in the context of endocrine disruption is also attributed to alterations in major hormonal axes, including the hypothalamic–pituitary–adrenal (HPA), hypothalamic–pituitary–thyroid (HPT), and hypothalamic–pituitary–gonadal (HPG) axes [23]. Importantly, the endocrine effects of microplastics are unlikely to result from a single molecular pathway. Experimental evidence suggests that oxidative stress, chronic inflammation, endocrine-disrupting chemicals adsorbed onto microplastic particles, and direct alterations of intracellular signaling pathways may act simultaneously, leading to cumulative impairment of hormone synthesis, secretion, receptor activation, and target tissue responsiveness. The biological response is further influenced by particle characteristics, including polymer type, particle size, surface charge, concentration, and duration of exposure, which may explain the considerable variability observed among experimental studies [3, 22, 23].

The mechanisms described above are summarized in Figure 1.

Disruption of major endocrine axes. Current evidence on microplastic-related disruption of major endocrine axes is derived predominantly from experimental studies, while direct evidence in humans remains limited [23]. Alterations of the HPA axis may affect stress-hormone signalling and downstream metabolic processes, including glucose metabolism and adipose tissue regulation. Accumulation of microplastic particles may also be associated with disruption of thyroid hormone synthesis and function, thereby affecting the entire HPT axis. These hormones play a crucial role in regulating metabolic homeostasis. Disruption of the HPG axis may lead to dysregulation of sex hormones, affecting estrogen- and androgen-dependent metabolic processes leading to problems with reproductive health [3, 23].

Experimental studies provide further mechanistic evidence for these endocrine effects. In a chronic exposure study, Jin et al. demonstrated that mice receiving polystyrene microplastics in drinking water for 180 days exhibited significantly reduced serum testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) concentrations, accompanied by impaired spermatogenesis and histopathological alterations of the testes. Mechanistically, these effects were associated with downregulation of the LH-mediated LHR/cAMP/PKA/StAR signalling pathway, resulting in decreased expression of key steroidogenic enzymes, including P450scc, CYP17A1, 3β-HSD, and 17β-HSD, ultimately reducing testosterone biosynthesis. Furthermore, restoration of LHR expression partially reversed these alterations, supporting a direct role of this signalling pathway in microplastic-induced endocrine dysfunction [24].

Together, these alterations highlight the complex role of microplastics in disrupting endocrine regulation across multiple hormonal axes.

Role of endocrine-disrupting chemicals (EDCs) associated with microplastics. Microplastics can act as carriers of endocrine-disrupting chemicals (EDCs), such as

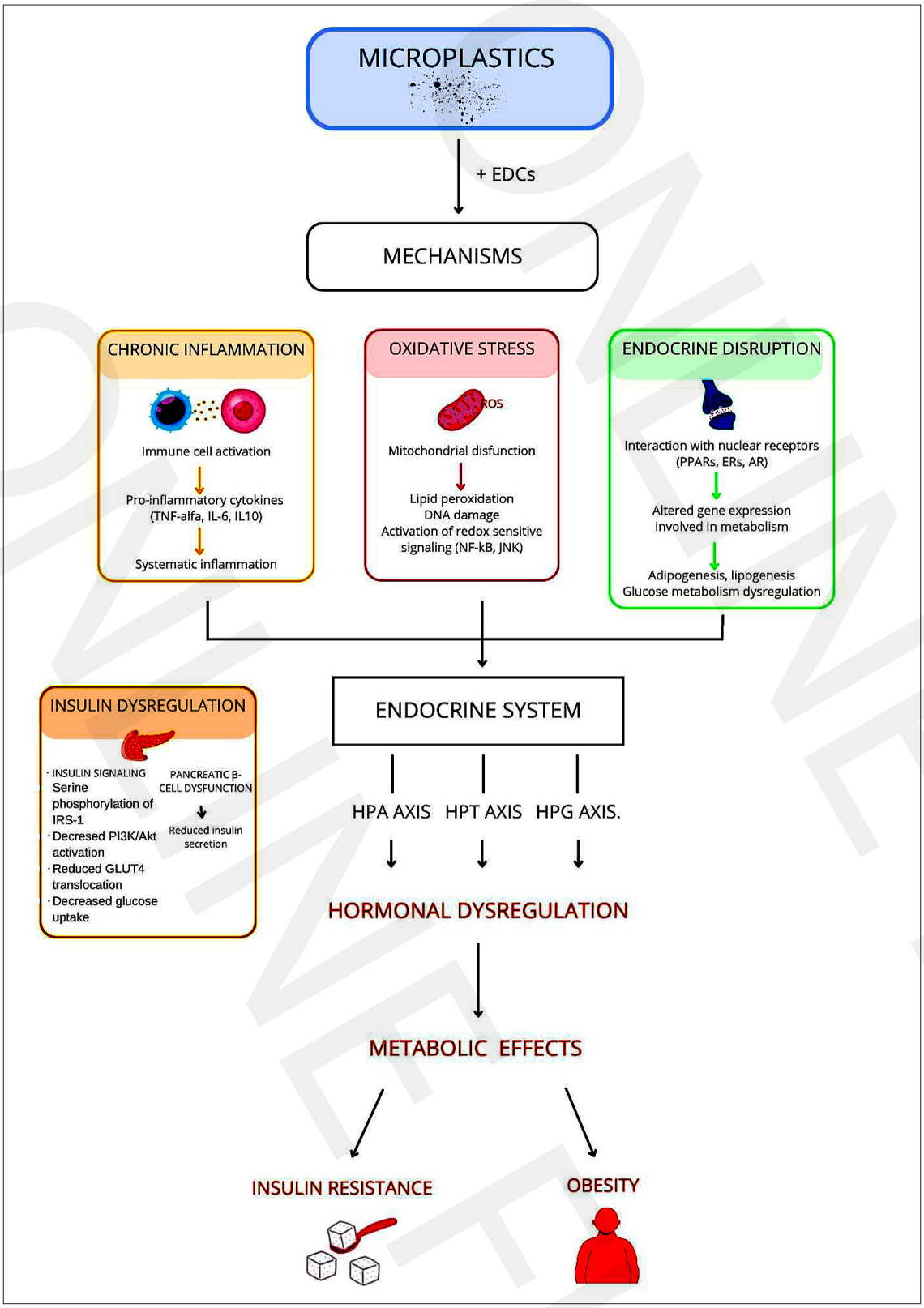

Figure 1. Mechanisms of microplastic-induced endocrine disruption and associated metabolic effects. Microplastics and associated endocrine-disrupting chemicals (EDCs) may cause oxidative stress, chronic inflammation, and receptor-mediated effects, leading to alterations in major hormonal axes (HPA, HPT, HPG) and contributing to metabolic disturbances. The mechanisms described in the text include activation of NF-κB/NLRP3/IL-1β signalling, disruption of LHR/cAMP/PKA/StAR-dependent steroidogenesis, impairment of IRS1/PI3K-mediated insulin signaling, gut microbiota dysbiosis, and adipose tissue dysfunction. The pathways presented are based predominantly on findings from *in vitro* and animal studies and require confirmation in humans

bisphenols, phthalates, and other additives added during plastic production. These substances may interact with hormone receptors, modulate gene expression, and influence the synthesis and metabolism of hormones. This carrier function has been supported by original experimental data. Chen et al. analyzed field-collected marine microplastics and mesoplastics and demonstrated that estrogenic compounds were the dominant group of EDCs associated with plastic particles. Bisphenol A showed the highest detection frequency, being identified in 75% of samples, with an average concentration of 475 ± 882 μg/kg. The authors also observed a particle-size effect, as smaller microplastics released greater quantities of EDCs than larger plastic fragments, suggesting that particle fragmentation may increase the availability of plastic-associated endocrine-active compounds [25]. As a result, EDCs may function as agonists or antagonists of hormonal receptors, leading to abnormal activation or inhibition of signaling pathways. Receptor-mediated endocrine activity of chemicals released from plastic materials has been demonstrated experimentally. Zimmermann et al. showed that chemicals migrating from plastic products under realistic use conditions induced anti-androgenic activity in 13 of 24 samples tested, while estrogenic activity was detected in one sample. These findings indicate that plastic-derived chemical mixtures may interfere with nuclear receptor signalling, particularly androgen receptor inhibition, although the magnitude and direction of the effect depend on polymer type and chemical composition of the leachate [1]. Attention is given to their impact on steroid hormones and thyroid hormones, which play a crucial role in regulating metabolic, reproductive, and developmental processes. Moreover, long-term exposure to EDCs may result in cumulative effects, with potential consequences emerging later in life; however, the contribution of microplastic-associated EDC exposure to chronic endocrine disorders in humans remains uncertain [3, 4].

Disruption of endocrine homeostasis induced by microplastics may translate into significant alterations in metabolic regulation, given the close interplay between hormonal signaling and energy balance in the human body. These mechanisms collectively create a biological basis for the metabolic disturbances described in the following section.

METABOLIC CONSEQUENCES OF MICROPLASTICS EXPOSURE

Current experimental evidence suggests that microplastic exposure may contribute to alterations in metabolic regulation. In particular, these effects may involve alterations in glucose and lipid metabolism and energy homeostasis.

Insulin resistance and disturbance in glucose homeostasis. Microplastic exposure may contribute to disturbances in carbohydrate metabolism, including impaired insulin signalling and insulin resistance, as suggested by experimental studies. In a mouse model, Huang et al. demonstrated that exposure to polystyrene microplastics induced insulin resistance in animals fed both a normal chow diet and a high-fat diet. These effects were accompanied by increased plasma lipopolysaccharide (LPS), elevated pro-inflammatory cytokines, including TNF-α and IL-1β, reduced gut microbiota richness and diversity, and increased relative abundance of Gram-negative bacteria. Moreover, 5 μm polystyrene particles accumulated in the liver, kidneys, and blood vessels, while hepatic insulin signalling was impaired through inhibition of IRS1 and decreased expression of PI3K. These findings suggest that microplastic-induced insulin resistance may result from the interaction between gut microbiota dysbiosis, low-grade inflammation, tissue accumulation of particles, and direct inhibition of insulin signaling pathways [26]. Microplastic-associated endocrine-disrupting chemicals may further contribute to metabolic dysregulation through receptor-mediated mechanisms. Plastic-derived chemical mixtures have been shown to exert antiandrogenic and, less frequently, estrogenic activity, suggesting that associated EDCs may influence nuclear receptor signaling involved in energy balance, adipogenesis and glucose metabolism [1, 3].

Hormonal imbalances, particularly within the HPA and HPT axes, may contribute to dysregulation of blood glucose levels through their effects on cortisol and thyroid hormone secretion, both of which play essential roles in energy metabolism. Therefore, the metabolic effects observed in experimental models of microplastic exposure are likely to reflect overlapping mechanisms, including inflammatory signalling, disruption of insulin transduction pathways and endocrine modulation, rather than as a consequence of a single isolated pathway [3, 26].

Obesity-related processes and adipose tissue dysfunction. In addition to disturbances in glucose metabolism, microplastic exposure has been associated with alterations in adipose tissue function and adipogenesis. In an *in vitro* study using murine 3T3-L1 cells, Molonia et al. investigated the effects of 5 μm polystyrene microplastics in 2 exposure scenarios: fully differentiated mature adipocytes and preadipocytes undergoing differentiation. In mature adipocytes, exposure did not significantly alter selected markers of adipogenesis, endoplasmic reticulum stress, or inflammation, including PPARγ, FASN, p-eIF2α, GRP78, IL-6, and MCP-1. However, exposure during adipocyte differentiation promoted adipocyte hypertrophy and was associated with upregulation of adipogenic markers, increased ROS production, activation of endoplasmic reticulum stress responses, and apoptosis. These findings suggest that the metabolic effects of microplastics may depend on the developmental stage and functional state of adipose tissue cells, with differentiating adipocytes appearing more susceptible than mature adipocytes [27].

Limitations of the study. Current evidence on MPs and their impact on human health remains limited, particularly in the context of long-term exposure and direct clinical outcomes. Human bio-monitoring studies have confirmed the presence of microplastics in several biological matrices, including placenta, blood and lung tissue; however, these studies are usually based on relatively small sample sizes and differ substantially in sample preparation, contamination control, analytical techniques, particle-size detection limits, and units used to report results [18–21].

These methodological differences make direct comparison between studies difficult and limit the possibility of defining exposure thresholds that could be considered clinically relevant. For example, some studies report particle number per gram of tissue, whereas others quantify polymer mass per volume or mass of biological material. In addition, particles

below the detection limit of commonly used spectroscopic methods may be missed, while insufficient control of procedural contamination may lead to overestimation of exposure. Therefore, currently available quantitative data should be interpreted as evidence of internal exposure rather than as a precise estimate of toxic dose.

Another important limitation is that most mechanistic evidence comes from *in vitro* experiments and animal models [3, 22, 23]. Although these studies provide valuable information on oxidative stress, inflammatory pathways, steroidogenesis, insulin signallling, and adipocyte dysfunction, they often use particle concentrations, exposure durations, and administration routes that may not reflect real-world human exposure. This limits direct dose extrapolation from experimental models and precludes the definition of biologically relevant exposure thresholds in humans. Consequently, causal relationships between microplastic exposure and endocrine or metabolic diseases in humans cannot yet be established.

Future research should therefore focus on standardized analytical protocols, rigorous contamination control, harmonized reporting of particle size and concentration, and well-designed human studies integrating biomonitoring with endocrine and metabolic endpoints [4, 16]. Particular attention should also be paid to dose-response relationships, particle characteristics, co-exposure to plastic-associated EDCs, and the identification of molecular biomarkers that could link internal microplastic burden with clinically meaningful endocrine or metabolic outcomes [22, 23, 25].

SUMMARY

Microplastics have become ubiquitous contaminants with potential relevance for human health. Their widespread presence in the environment, combined with their detection in human tissues and biological fluids, highlights the relevance of continuous human exposure.

Current evidence suggests that MPs may interfere with endocrine function through multiple mechanisms, including oxidative stress, inflammatory signalling, and interactions with endocrine-disrupting chemicals. These mechanisms may also disrupt intracellular pathways regulating hormone synthesis and affect major hormonal axes, potentially contributing to metabolic dysregulation. However, these effects have been demonstrated predominantly in experimental models and remain insufficiently characterized in humans.

In particular, experimental data indicate a potential link between microplastic exposure and metabolic disturbances, such as insulin resistance and obesity-related processes. Proposed mechanisms include impaired insulin signalling, low-grade inflammation, gut microbiota dysbiosis, and adipose tissue dysfunction, although direct causal relationships in humans remain to be established.

Despite increasing scientific interest, significant gaps remain in understanding the long-term health consequences of exposure to MPs. Further research is required, especially well-designed human studies using standardized analytical methods and clinically relevant endocrine and metabolic endpoints, to clarify their role in the development of endocrine and metabolic disorders.

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