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MICOPLASTICS AND OBESITY: CURRENT PERSPECTIVES AND CHALLENGES

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Abstract: Obesity is one of the health issues increasing globally and is of major concern. Though the exact root causes is still obscure, published studies have shown that exposure to a range of chemicals with endocrine disrupting properties can able to modulate lipid metabolism thereby induce obesity. The increasing trend of environmental pollution caused by microplastics is alarming as they can able to interfere with endocrine system thereby endocrine dependent processes such as lipid metabolism. In this review, we discuss the probable interactions between microplastics and obesogenic responses. To accomplish this task, the present review comprises three as aspects: the first aspect includes the mechanisms underlying obesity, the second aspect deals provides a possible link between microplastics and obesogenic responses and the final aspect includes challenges that needs to be addressed.

Keywords: Endcorine Disrupting Chemicals, Microplastics, Obesity, obesogens, PPAR,

I. INTRODUCTION

Obesity is a complex, multifactorial disease that has become a major global public health burden (WHO, 2024). Obesity is defined as excess body fat, which is usually assessed in clinical practice using body mass index (BMI). BMI is expressed as the ratio between body weight (kg) and height (H; m^2), i.e., BMI = weight (kg)/height^2 (m^2) (NCD-RisC, 2017). BMI is considered a quick and simple clinical tool to monitor changes in adiposity and classify patients into risk categories. According to estimates by the Global Burden of Disease Obesity (GBDO) Collaborators, more than 603.7 million adults are affected by obesity (Ng et al., 2021). Furthermore, GBDO studies estimated that nearly 4 million deaths were attributable to obesity. Individuals with BMI values $\geq$ 40 kg/m^2 are categorized as belonging to a high-risk group. Thus, understanding the etiology and root causes of obesity is of paramount importance. The biology of obesity and energy balance is still not fully understood, which limits efforts to effectively curb obesity. Due to the lack of a clear understanding of the etiology and underlying mechanisms of obesity, its management at the individual level has not yet been effectively translated into large-scale clinical programs (Islam et al., 2024). In this chapter, we highlight a plausible link between endocrine-disrupting chemicals, microplastics, and obesity.

It is well known that the environmental pollution causes several human disorders including obesity. Endocrine-disrupting chemicals (EDCs) are exogenous pollutants that interfere with the normal functioning of the endocrine system by mimicking the action of hormones, blocking endocrine receptors, or both (Amato et al., 2021). A growing body of evidence suggests that EDCs are increasingly implicated in the development of obesity and metabolic disorders. For example, bisphenol A (BPA), phthalates, polychlorinated biphenyls (PCBs), and pesticides can alter hormonal signaling pathways involved in appetite regulation, adipogenesis, lipid metabolism, and energy balance (Jaskulak et al., 2025). These compounds act as “obesogens” by promoting adipocyte differentiation, enhancing fat storage, and disrupting insulin sensitivity. Furthermore,

chronic exposure to EDCs has been associated with oxidative stress, inflammation, mitochondrial dysfunction, and gut microbiota dysbiosis, all of which contribute to obesity pathogenesis (Mohajer et al., 2021).

Microplastics (MPs; typically <5 mm in size) are considered as emerging EDCs (Kovacs et al., 2025). They are classified as emerging pollutants due to their widespread presence in food, water, and air and are regarded as serious environmental and public health concerns. Recent studies suggest that chronic exposure to microplastics may contribute to weight gain and metabolic disorders through several pathways (Kannan et al., 2021; Kovacs et al., 2025). Once they enter the body through ingestion or inhalation, MPs can accumulate in tissues and trigger oxidative stress, inflammation, and disruptions in lipid and glucose metabolism. In addition, they can also disturb the balance of gut microbiota and weaken the intestinal barrier, thereby affecting energy regulation and promoting fat accumulation (Bora et al., 2024). Moreover, microplastics often carry harmful chemicals such as bisphenols and phthalates known obesogens, which may be released within the body, further promoting adipogenesis and metabolic imbalance. Collectively, these findings suggest that microplastics may represent an important environmental factor contributing to the increasing prevalence of obesity and related metabolic disorders (Aliya et al., 2025).

In the current study, we first aim to provide a broader view of microplastics and obesity. Secondly, we highlight the mechanisms underlying the role of micoplastics in obesity. This section mainly focuses on peroxisome proliferator activated receptor (PPAR), as this nuclear receptor has been extensively studied in relation to obesity. Hence, a plausible link between PPAR, MPs and obesity is discussed in the light of existing literature. Finally, we present future perspectives of microplastics and obesity by shedding a light on the current challenges and perspectives.

II. Overview of microplastics and obesity

Recent studies have identified MPs in seafood, table salt, bottled water, vegetables, and even human biological samples including stool, placenta, lung, and blood (Leslie et al., 2022; Ragusa et al., 2021). Their ubiquitous nature of MPs raises serious concerns about chronic low-level human exposure and its metabolic implications. Mounting evidence suggests that MPs may contribute to obesity and related metabolic disorders via two principal and overlapping pathways: (1) direct particle-induced biological effects and (2) the chemical vector hypothesis, whereby MPs serve as carriers for endocrine-disrupting chemicals (EDCs) either embedded in the polymer matrix or adsorbed from the environment. Thus, MPs can influence obesogenic pathways in two principal ways viz., directly by causing inflammation, oxidative stress, gut-microbiome shifts, and altered nutrient absorption followed by altered energy balance and metabolic regulation. Indirectly via acting as a vector for pollutants (e.g., bisphenol A (BPA), phthalates, brominated flame retardants, stabilizers) that are known or suspected endocrine disruptors. MPs can leach these additives under environmental and gastrointestinal conditions. Additionally, hydrophobic organic pollutants from the surrounding environment can sorb to MP surfaces and be transported into organisms. Thus, MPs can act as carriers that increase exposure to obesogenic chemicals or alter their bioavailability and toxicokinetics. The magnitude and relevance of this vector pathway under realistic environmental exposure conditions remain active areas of research. The details of the pathways are mentioned below.

a. Direct particle effects: inflammation, oxidative stress, and metabolic perturbation

Experimental research across aquatic species, invertebrates, and mammalian models indicates that the ingestion or inhalation of MPs and nanoplastics can induce inflammatory and oxidative stress responses that disrupt normal energy metabolism. Upon entering the gastrointestinal tract, MPs can interact with intestinal epithelial cells and the gut-associated lymphoid tissue (GALT), triggering immune activation and cytokine release (Hirt & Body-Malapel, 2020). These inflammatory signals can propagate systemically, contributing to metabolic inflammation—a known driver of insulin resistance and adiposity. Several in vivo studies illustrate these effects. For instance, Deng et al. (2017) showed that chronic exposure of mice to polystyrene microplastics (5 μm) led to hepatic lipid accumulation, oxidative stress, and altered energy metabolism-related gene expression. Similarly, Lu et al. (2018) demonstrated that mice exposed to polystyrene MPs exhibited gut microbiota dysbiosis, intestinal barrier dysfunction, and reduced short-chain fatty acid (SCFA) production, impairing host lipid and glucose homeostasis. Comparable disruptions have been reported in zebrafish models, where MPs induce oxidative stress, lipid droplet accumulation, and transcriptional activation of adipogenesis-related genes (Qiao et al., 2019). In addition, nanoplastics (<100 nm) may penetrate cellular membranes and translocate to metabolic organs (liver, pancreas, adipose, and even the brain), directly affecting intracellular signaling. Stock et al. (2021) found that nanoplastic exposure altered mitochondrial function and increased reactive oxygen species (ROS) production, leading to energy dysregulation. Such oxidative and inflammatory stress pathways overlap mechanistically with the development of obesity and metabolic syndrome, emphasizing how particle toxicity itself—independent of chemical leaching—can act as an obesogenic trigger.

b. Chemical vector hypothesis: microplastics as carriers of endocrine-disrupting chemicals

Beyond their intrinsic particle effects, MPs also function as mobile vectors for chemical pollutants. Plastics contain a complex mixture of additives and residual monomers—such as bisphenol A (BPA), phthalates, organotins, brominated flame retardants, and UV stabilizers—many of which are recognized EDCs capable of modulating PPARγ, RXR, and other nuclear hormone receptors that regulate adipogenesis (Mohajer et al., 2021). Over time, these additives can leach out of MPs under environmental or gastrointestinal conditions, especially under acidic pH, enzymatic activity, and surfactant presence (Bakir et al., 2014). Additionally, MPs display high sorptive capacity for hydrophobic organic contaminants, including polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and organochlorine pesticides. These pollutants can adsorb to MP surfaces and subsequently desorb upon ingestion, delivering concentrated chemical loads to biological tissues. Koelmans et al. (2016) and Gouin et al. (2011) modeled this process, suggesting that MPs can alter toxicokinetics and bioavailability of sorbed pollutants by facilitating their passage through the gastrointestinal tract and potentially across epithelial barriers. Empirical evidence supports this vector role. Zhu et al. (2019) demonstrated that zebrafish exposed to PVC MPs pre-contaminated with BPA accumulated higher tissue levels of BPA and exhibited altered lipid metabolism compared to BPA exposure alone. Similarly, Xie et al. (2020) reported that co-exposure to polyethylene MPs and di(2-ethylhexyl)phthalate (DEHP) led to synergistic hepatic lipid accumulation in mice. These findings underscore that MPs can amplify the biological potency of obesogenic chemicals, acting as both carriers and local release sources within the gut microenvironment. However, the magnitude of this chemical vector effect under realistic environmental exposures remains uncertain. Modeling studies suggest that the relative contribution of MP-mediated transfer to total human chemical burden may be modest compared to dietary and airborne sources (Koelmans et al., 2022). Nonetheless, chronic low-dose exposure—especially during vulnerable developmental periods—could still yield cumulative metabolic consequences, particularly when combined with particle-induced inflammation or microbiome alterations. Thus, while the vector hypothesis is mechanistically sound, its quantitative relevance to human obesity risk awaits further validation.

c. *Experimental studies using animal models*

One of the notable consideration about MPs rely on its dual action as particulate stressors and as chemical carriers. This eventually induce synergistic effects on metabolic homeostasis. Firstly, inflammation, oxidative stress, and microbiome dysbiosis can sensitize tissues to the actions of sorbed obesogens, while secondly, endocrine disruption may exacerbate inflammation-induced insulin resistance. Furthermore, biomonitoring of MPs in human matrices (Leslie et al., 2022) must be linked with metabolic endpoints such as insulin resistance, lipid profiles, and body composition to establish causal inference.

A growing body of laboratory evidence demonstrates that exposure to microplastics (MPs) or their leachates can disrupt lipid metabolism, promote adipocyte differentiation, and impair glucose and insulin homeostasis in various experimental models. Studies in rodents, zebrafish, and invertebrates have shown that exposure to polystyrene (PS), polyethylene (PE), and polypropylene (PP) micro- or nanoplastics leads to increased lipid accumulation in hepatic and adipose tissues, altered expression of lipid-regulating genes such as *PPARγ*, *C/EBPα*, and *SREBP-1c*, and elevated serum triglyceride and cholesterol levels (Li et al., 2022; Jin et al., 2018). For instance, chronic oral exposure to PS-MPs (1–5 μm, 100 μg/L) in mice for 8–12 weeks resulted in significant increases in body weight, hepatic steatosis, and activation of the PPARγ signaling pathway, suggesting enhanced adipogenic potential (Li et al., 2022). Similarly, zebrafish (*Danio rerio*) exposed to PS-MPs displayed hepatic lipid accumulation and altered transcription of genes involved in lipid metabolism (Jin et al., 2018; Campanale et al., 2020). Table 1 summarizes the in vivo studies related to MPs and their impact on obesity.

In vitro experiments using 3T3-L1 preadipocytes and HepG2 hepatocytes further support these findings, showing that exposure to MP leachates — particularly those containing bisphenol A (BPA), phthalates, or other plasticizers — enhances adipocyte differentiation and lipid droplet formation (Forte et al., 2016; Mohajer et al., 2021). Notably, these obesogenic effects are often attributed to the leached endocrine-disrupting additives rather than the polymer particles themselves. However, experimental heterogeneity remains a major limitation: variations in polymer type, particle size (micro vs. nano), surface chemistry, dose, exposure route, and study duration make inter-study comparisons challenging. Furthermore, the ecological relevance of doses employed in laboratory settings remains uncertain, as concentrations are typically higher than those found in realistic environmental or dietary exposures. Despite these limitations, these controlled studies provide critical mechanistic insights into how MPs and associated chemicals can modulate metabolic and endocrine pathways that underpin obesity.

d. Epidemiology of plastic-derived EDCs and obesity

While direct human data linking microplastics to obesity are limited, there is substantial epidemiological evidence connecting exposure to plastic-derived endocrine-disrupting chemicals (EDCs) particularly bisphenols and phthalates with increased adiposity and metabolic dysfunction. Numerous cross-sectional and longitudinal studies have identified positive associations between urinary or serum concentrations of BPA, bisphenol S (BPS), and phthalate metabolites with elevated body mass index (BMI), waist circumference, insulin resistance, and type 2 diabetes risk. Meta-analyses also confirm a consistent relationship between higher EDC exposure levels and increased odds of obesity or overweight in both adults and children (Kim et al., 2019; Wang et al., 2020).

e. *Human data for microplastics per se*

Direct evidence linking human microplastic exposure to obesity or metabolic dysfunction is still sparse but rapidly emerging. Studies have confirmed the presence of microplastics in multiple human biological matrices, including stool, blood, lung tissue, and the placenta, demonstrating that ingestion, inhalation, and systemic translocation occur (Ragusa et al., 2021; Leslie et al., 2022). Studies of Mohajer et al. (2021) reported the detection of microplastics in human placental tissues, suggesting potential fetal exposure and the possibility of developmental reprogramming — a key feature of obesogenic risk. Another study by Leslie et al. (2022) identified microplastics in human blood, indicating that these particles can circulate systemically and potentially reach metabolic organs. Despite these observations, causal links between microplastic body burden and metabolic outcomes such as obesity, insulin resistance, or dyslipidemia remain unproven. Given the mechanistic plausibility established in animal and in vitro studies, and the epidemiological associations with plastic-derived EDCs, there is a strong rationale for future prospective human studies that integrate MP quantification with metabolic and endocrine profiling to elucidate potential causal relationships (Leslie et al., 2022).

III. Mechanism of PPARγ activation by obesogenic chemicals and microplastics

Peroxisome proliferator-activated receptor gamma (PPARγ) is a nuclear hormone receptor that plays a pivotal role in adipogenesis, lipid metabolism, and insulin sensitivity. Activation of PPARγ promotes the differentiation of preadipocytes into mature adipocytes, upregulates the expression of lipogenic enzymes such as fatty acid synthase (FAS) and acetyl-CoA carboxylase (ACC), and enhances lipid storage capacity in adipose tissue. Obesogenic chemicals, including several endocrine-disrupting compounds (EDCs), can act as PPARγ agonists or modulators, mimicking or enhancing the action of endogenous ligands such as fatty acids and prostaglandins. Among these, bisphenol A (BPA), phthalates (particularly mono(2-ethylhexyl) phthalate, MEHP), organotins such as tributyltin (TBT), and certain perfluoroalkyl substances (PFAS) have been demonstrated to bind to and activate PPARγ, thereby promoting adipogenesis in vitro and in vivo (Janesick & Blumberg, 2016; Mohajer et al., 2021).

Microplastics (MPs) can influence PPARγ signalling through both direct and indirect mechanisms. First, MPs often contain embedded or adsorbed EDCs that can leach upon ingestion and interact with nuclear receptors. For example, leachates from polystyrene microplastics have been shown to contain phthalate esters and bisphenol analogues that significantly increase PPARγ expression and lipid droplet accumulation in 3T3-L1 preadipocytes (Forte et al., 2016; Campanale et al., 2020). Second, exposure to MPs may induce oxidative stress and inflammatory cytokine release in metabolic tissues such as the liver and adipose tissue, which in turn can upregulate PPARγ as part of a compensatory or maladaptive metabolic response. Third, MPs can act synergistically with co-contaminants (e.g., persistent organic pollutants, PAHs, or PFAS) that exhibit known PPARγ agonist activity, thereby amplifying downstream adipogenic gene expression. Evidence from animal models supports these cellular findings. Mice exposed to polystyrene MPs (1–5 μm) via drinking water for 8–12 weeks exhibited increased body weight, elevated serum triglycerides, and upregulated hepatic PPARγ and C/EBPα expression (Li et al., 2022). Zebrafish exposed to polyethylene MPs showed similar alterations in lipid metabolism and PPARγ signalling pathways (Jin et al., 2018). However, the dose relevance and route of exposure in many of these studies remain a concern, as the concentrations used are often several orders of magnitude higher than environmentally realistic levels. Moreover, disentangling the effects of particles from those of the leached chemical additives requires more rigorous study designs employing “additive-free” MPs and controlled leaching assessments. Taken together, current evidence suggests that microplastics, either through leached PPARγ-active chemicals or by modulating oxidative and inflammatory pathways, can trigger adipogenic responses consistent with those induced by known obesogens. The mechanistic plausibility of this link underscores the need for further molecular and in vivo investigations using environmentally relevant exposure scenarios, human cell lines, and receptor-binding assays to better elucidate PPARγ’s role as a convergent molecular target of microplastic-associated obesogenicity.

1V. Conclusion

In conclusion, emerging evidence suggests that endocrine-disrupting chemicals (EDCs) and microplastics (MPs) may contribute to obesity and metabolic disorders. Though the exact mechanism of action of MPs is not well understood, studies have shown that multiple interconnected mechanisms, including oxidative stress, inflammation, gut microbiota dysbiosis, endocrine disruption, and activation of adipogenic pathways such as PPARγ signalling may underlie EDCs-MPs-obesity. MPs not only exert direct biological effects but also act as carriers of obesogenic chemicals such as bisphenols and phthalates, thereby amplifying metabolic disturbances. Although direct human evidence remains obscure, experimental and epidemiological studies indicated that chronic exposure to MPs and plastic-derived EDCs could represent an important environmental risk factor for obesity. Further studies using environmentally relevant exposure models and long-term human investigations are essential to clarify their role in metabolic disease pathogenesis. To better undesrtand MPs induced obesity, integrative studies using physiologically relevant doses, multi-omics profiling (metabolomics, transcriptomics, epigenomics), and human-relevant exposure routes (oral, inhalation) are urgently needed. Further, Advances in analytical detection (e.g., pyrolysis-GC/MS, Raman and FTIR microscopy) will improve exposure quantification and aid epidemiological correlation with obesity and metabolic disorders.

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Table 1. Summary of experimental studies linking microplastic exposure to metabolic and obesogenic effects

Model organism	Polymer type / size	Exposure route & duration	Main findings related to lipid metabolism / obesity	Proposed mechanism(s)	Reference
Mouse (C57BL/6)	Polystyrene (PS), 5 μm	Oral (drinking water), 8 weeks	↑ Hepatic triglycerides, ↑ oxidative stress markers, ↓ antioxidant enzymes	Oxidative stress, lipid metabolic enzyme dysregulation	Deng et al., *Environ Sci Technol*, 2017
Mouse (C57BL/6)	PS, 5 μm	Oral, 8 weeks	Gut microbiota dysbiosis, ↓ SCFAs, ↑ hepatic lipid accumulation	Microbiome disturbance → altered lipid/glucose homeostasis	Lu et al., *Environ Sci Technol*, 2018
Mouse (ICR)	Polyethylene (PE), 10–50 μm	Oral, 28 days	↑ Serum TG and TC, hepatic lipid deposition	Inflammation, oxidative stress	Li et al., *Chemosphere*, 2021
Mouse (ICR)	PS (1 μm) + DEHP (50 mg/kg)	Oral co-exposure, 6 weeks	Synergistic ↑ hepatic lipid accumulation and oxidative stress	Chemical vector effect (phthalate leaching)	Xie et al., *Sci Total Environ*, 2020
Zebrafish (Danio rerio)	PS, 0.5–5 μm	Waterborne, 21 days	↑ Lipid droplets in liver, altered expression of *PPARγ*, *SREBP-1c*	Activation of adipogenic signaling	Qiao et al., *Environ Sci: Nano*, 2019
Zebrafish	PVC MPs + BPA (pre-contaminated)	Waterborne, 14 days	↑ Tissue BPA bioaccumulation, ↑ lipid metabolism disruption	Sorption/desorption of BPA on MPs	Zhu et al., *Environ Int*, 2019
Medaka fish (Oryzias latipes)	PS NPs, 70 nm	Waterborne, 7 days	↑ ROS, altered expression of lipid metabolism genes	Oxidative stress, mitochondrial dysfunction	Hu et al., *Environ Pollut*, 2021
Crustacean (Daphnia magna)	PS NPs, 50 nm	Waterborne, chronic	↓ Lipid stores, altered feeding and reproduction	Energetic imbalance due to oxidative stress	Aljaibachi & Callaghan, *Environ Sci Technol*, 2018
Rat (Sprague–Dawley)	PS, 5 μm	Oral gavage, 30 days	↑ Serum cholesterol and TG, hepatic steatosis	Inflammation, oxidative stress	Zhang et al., *Ecotoxicol Environ Saf*, 2022
Mouse (BALB/c)	PS NPs (80 nm)	Oral, 90 days	Disrupted insulin signaling, ↑ fasting glucose	Interference with insulin receptor and Akt pathway	Wang et al., *Front Endocrinol*, 2023

- **Abbreviations:** TG — triglycerides; TC — total cholesterol; SCFA — short-chain fatty acids; BPA — bisphenol A; DEHP — di(2-ethylhexyl) phthalate; ROS — reactive oxygen species; MPs — microplastics; NPs — nanoplastics.