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Microplastics Effect on Gut Microbiota

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BACKGROUND

Microplastics (MPs) are plastic particles smaller than 5 mm. Classifications are primary where they are specifically produced for a product such as microbeads in face care, or secondary which are larger pieces such as tires and water bottles that break down into MPs. Due to their small size and persistence within the environment, MPs are distributed in the environment, including food, water, and air.

Figure 1. Pathways to human exposure to microplastics(according to Li et al., 2023).

Exposure

As a result, human exposure is unavoidable and occurs via ingestion, inhalation, and dermal contact (Li et al., 2023; Refosco et al., 2025). Due to their small size, MPs cross intestinal epithelial barriers and can bind with heavy metals and other contaminants to increase their toxicity and transport within the body (Charters et al., 2024). They create a biomolecular corona camouflage by binding with proteins, lipids, polysaccharides and heavy metals by chemical changes involving hydroxyl and carboxyl groups. Figure 2 shows the MP corona camouflage using the surface receptors to enter the cell.

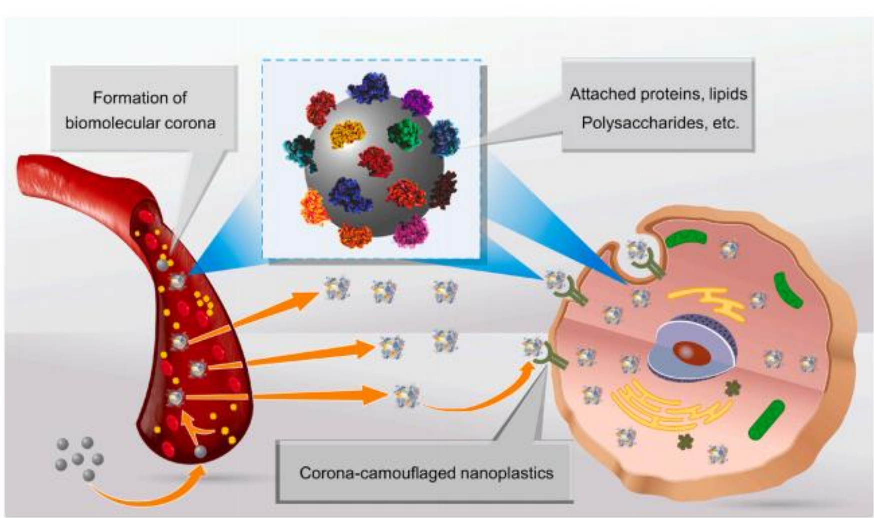

Figure 2. The formation of biomolecular corona on NPs. The NP surface interacts with various molecules and is rapidly coated by the proteins (protein-corona), as well as other biomolecules, including proteins, polysaccharides, lipids, *etc.*, to form a biomolecule corona. The corona critically affects the interactions between NPs and living systems, such as camouflage the foreign particles to enter or cross cells. These mixtures of adsorbed molecules further alter the persistence, bioavailability, and ecotoxicity of NPs (according to Wu et al., 2022).

Gut Microbiome

The gut microbiome plays a critical role in maintaining metabolism, immune regulation, and intestinal homeostasis (Gao et al., 2025). Due to the MP accumulation within the gastrointestinal tract, they can alter the microbial composition and gut function. Dysbiosis, the imbalance of the microbiome, impairs the host-microbe relationship which has been linked to inflammation, metabolic disorders, and disease.

Mechanisms

1. Reactive oxidative species (ROS)
2. Short chain fatty acids (SCFAs)
3. Mitochondrial Dysfunction
4. Immune Dysfunction

MECHANISMS

(M1) ROS

MP exposure has been shown to increase the production of ROS, which leads to oxidative stress within the microbes and gut (Kovacs et al., 2025). As seen in Figure 3, excess ROS damages lipids, proteins, and DNA, causing an impair in normal cellular function. The oxidative damage weakens the intestinal epithelial barrier, increases permeability, and promotes inflammation (Bhagat et al., 2021). In addition, MPs may carry pollutants into the body which increases the uptake of the pollutants (Holmes et al., 2012). Additionally, the oxidative environment disrupts microbial balance leading to dysbiosis.

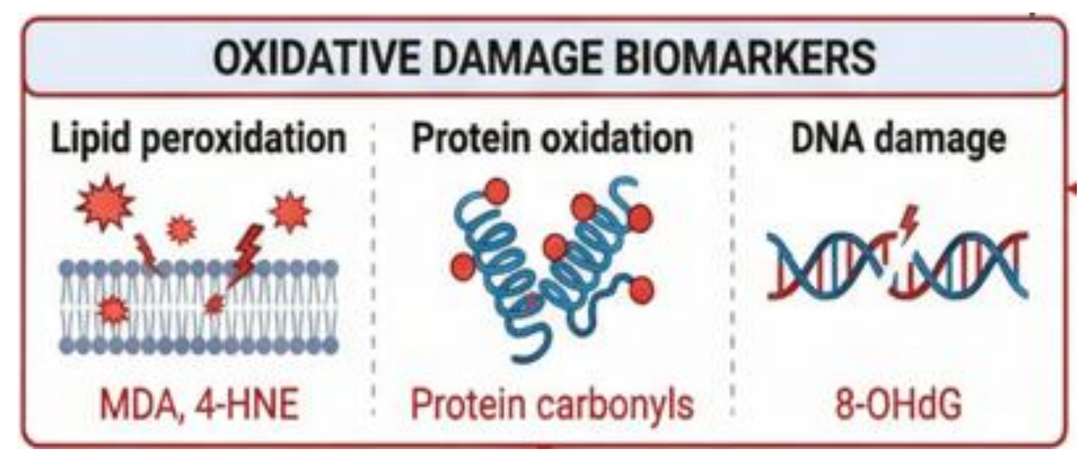

Figure 3. Mechanistic pathways linking microplastic/nanoplastic exposure to oxidative damage markers (according to Kovacs et al., 2025)

(M2) SCFAs

MP exposure alters the composition and diversity of the gut microbiota which reduces the beneficial bacteria that produce SCFAs such as butyrate (Gao et al., 2025). Butyrate fuels colonocytes, maintains the integrity of the gut and regulates inflammation. As a result, the decrease in SCFA weakens the barrier which decreases epithelial defenses and increasing intestinal permeability (Ren et al., 2025). This reduction of SCFAs is links metabolic disruption in the gut and an increase in inflammatory responses (Thin et al., 2025). Thus, linking microbial dysbiosis to impaired host health.

(M3) Mitochondrial Dysfunction

MPs disrupt mitochondrial structure and function, impairing cellular energy metabolism. Damage to the mitochondria reduces ATP production while increasing ROS generation (Figure 4) (Wu et al., 2022). This cycle of oxidative stress causes a dysfunction which can trigger apoptosis and weaken the gut epithelial barrier. Additionally, damaging the signaling pathways within the host-microbe relationship which leads to dysbiosis and promotes the growth of pro-inflammatory bacteria which increasing metabolic imbalance and cellular stress (Yang et al., 2026).

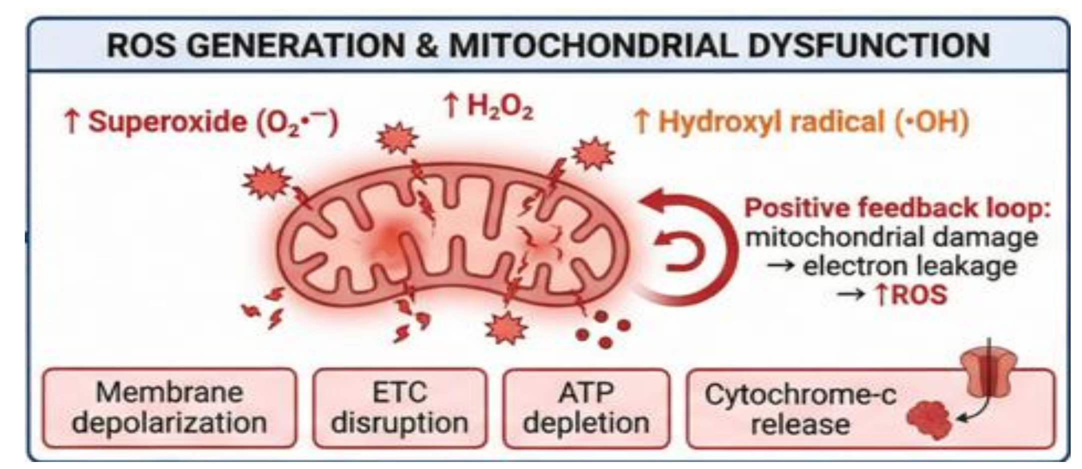

Figure 4. Mechanistic pathways linking microplastic/nanoplastic exposure to ROS generation and Mitochondria dysfunction (according to Kovacs et al., 2025)

(M4) Immune Dysregulation

MPs can activate immune responses within the gut leading to chronic low-grade inflammation (Chartres et al., 2024). This stimulates immune pathways and increases pro inflammatory cytokines which disrupts immune signaling causing microbial imbalance and damage to intestinal tissues (Wu et al., 2022). Studies have linked the presence of MPs within gut to inflammatory disease states such as Inflammatory Bowel Disease (Yan et al., 2021). This state of dysregulation leads to chronic illnesses such as Inflammatory Bowel Dysfunction (IBD) .

EVIDENCE

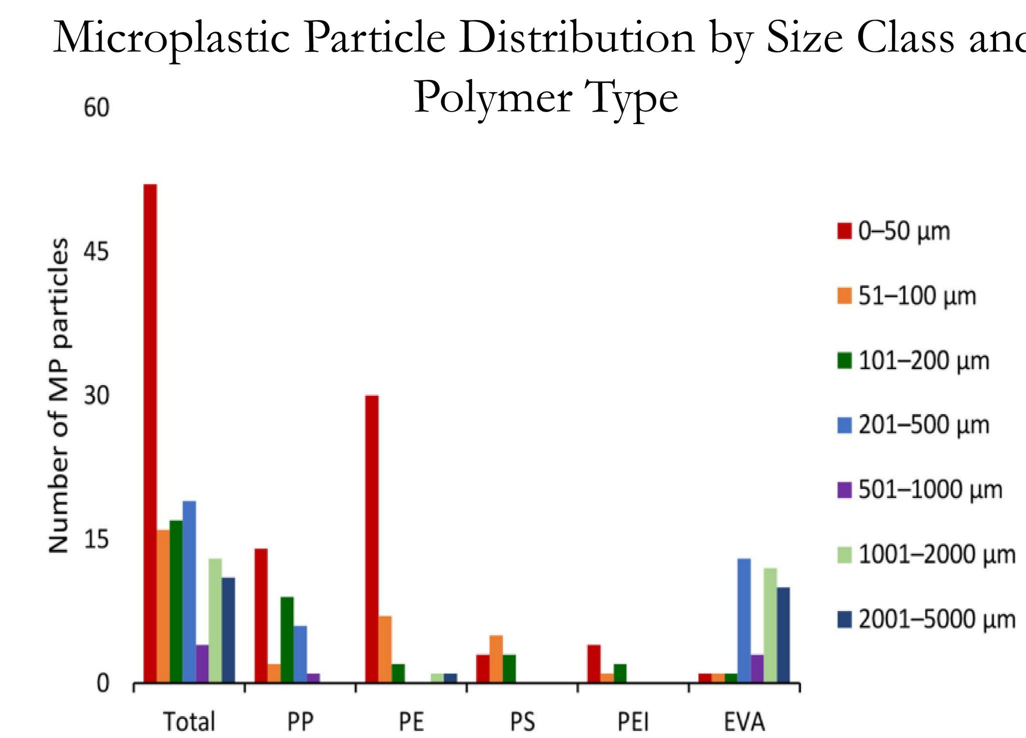

Figure 5. Size composition of MPs in fecal samples presented for the total number of particles and for each polymer identified through µ-FTIR analysis. Size classification is based on the major dimension of the particles. EVA: ethylene-vinyl acetate; PEI: polyethyleneimine; PS: polystyrene; PE: polyethylene; PP: polypropylene (according to Refosco et al., 2025).

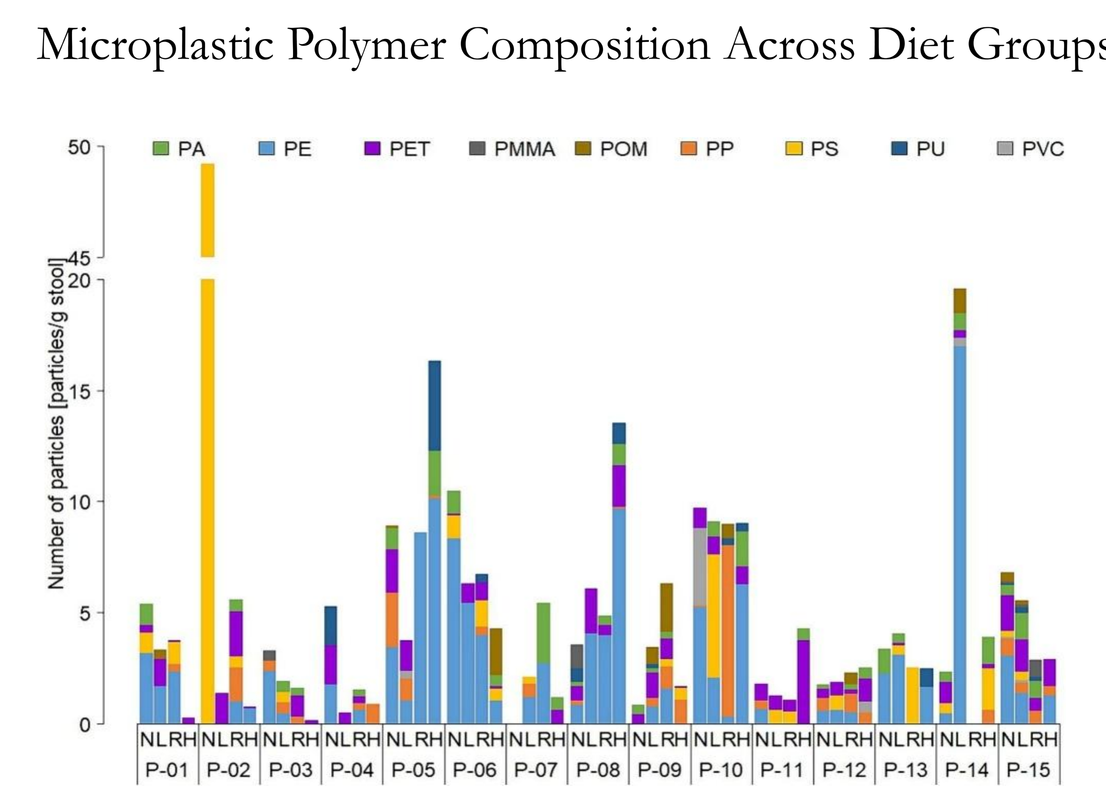

Figure 6. Number of particles of different plastic types detected per g stool (sum of size fractions 50 500 + 500–5000 µm) [particles/g] in the four plastic use and food consumption scenarios (normal diet (here: N), low plastic use and food consumption scenario (L), reset phase (R), and high plastic use and food consumption scenario (H)) per participant (according to Hui-Anderson, 2023).

CONCLUSION

Microplastics are a significant threat to human health, with evidence showing how these small particles disrupt cellular and molecular processes within the gut microbiome. By altering reactive oxygen species, short-chain fatty acid production, and mitochondrial homeostasis, MPs compromise the function and communication of microbes with the host, leading to dysbiosis. This imbalance impairs nutrient metabolism, energy homeostasis, and immune signaling within the intestinal epithelial barrier and activates pro-inflammatory pathways.

In addition, MPs act as vectors for harmful chemicals and heavy metals to enter the body, further weakening gut barrier integrity, increasing intestinal permeability, and promoting inflammation. Altogether, these disturbances contribute to chronic low-grade inflammation and increase the risk of diseases such as inflammatory bowel disease, highlighting the importance of understanding MP exposure for human health.

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