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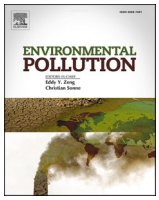

Review

The microplastic-crisis: Role of bacteria in fighting microplastic-effects in the digestive system☆

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ABSTRACT

Plastic particles smaller than 5 mm, referred to as Microplastics, pose health risks, like metabolic, immunological, neurological, reproductive, and carcinogenic effects, after being ingested. Smaller plastic particles are more likely to be absorbed by human cells, with nanoplastics showing higher potential for cellular damage, including DNA fragmentation and altered protein functions. Micro- and nanoplastics (MNPs) affect the gastrointestinal tract by altering the microbial composition, they could influence digestive enzymes, and possibly disrupt mucus layers. In the stomach, they potentially interfere with digestion and barrier functions, while in the intestines, they could increase permeability via inflammation and tissue disruption. MNPs can lead to microbial dysbiosis, leading to gastrointestinal symptoms. By activating inflammatory pathways, altering T cell functions and affecting dendritic cells and macrophages, immune system homeostasis could possibly be disrupted. Probiotics offer potential strategies to alleviate plastic effects, by either degrading plastic particles or directly countering health effects. We compared genetic sequences of probiotics to the genome of known plastic degraders and concluded that no probiotic bacteria could serve the role of plastic degradation. However, probiotics could directly mitigate MNP-health effects. They can restore microbial diversity, enhance the gut barrier, regulate bile acid metabolism, reduce inflammation, regulate insulin balance, and counteract metabolic disruptions. Antioxidative properties protect against lipid peroxidation and MNP-related reproductive system damage. Probiotics can also bind and degrade toxins, like heavy metals and bisphenol A. Additionally, bacteria could be used to aggregate MNPs and reduce their impact. Therefore, probiotics offer a variety of strategies to counter MNP-induced health effects.

1. The Plasticene

The term plastic primarily refers to synthetically produced polymers, composed by polymerization of monomers. These monomers derive from oil or gas, and various chemical additives are usually introduced to achieve the desired properties (Cole et al., 2011; Derraik, 2002; Rios et al., 2007; Thompson et al., 2009).

Leo Baekeland developed the first plastic in 1907, known as Bakelite, made from synthetic components (Baekeland, 1909; Crespy et al., 2008). Since the 1940s, plastic polymers have evolved to be an important part of our lives (Thompson et al., 2009), since they show a plethora of useful characteristics, like being mouldable, extremely durable, resistant against corrosion, and having excellent insulating properties, making them versatile for a variety of applications while being extremely lightweight at the same time (Imhof et al., 2012). In 1950, annual production of plastics was already estimated at 2 million tons, which quickly increased to 380 million tons per year in 2015, including resin and fibre products (Geyer et al., 2017). In 2022, 400.3 million tons of plastics were produced, including 2.3 million tons of bio-based and bio-attributed plastics and 35.5 million tons of recycled

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post-consumer plastic products (Plastics Europe, 2023). The largest demand for plastics is in the packaging industry, which consumes 39% of the plastics produced in Europe, followed by building and construction with 23% (Plastics Europe, 2023). The cumulative waste generation over the 65 years from 1950 to 2015 is 6.3 billion tons, of which only 9% was recycled and 12% incinerated. 79% of the waste produced was landfilled or ended up elsewhere in the environment (Geyer et al., 2017). The environmental impacts even sparked a new name for the geological period called "Plasticene" (Haram et al., 2020).

2. Omnipresence of microplastics

The continuous fragmentation of plastics leads to an exponential rise in particles smaller than 5 mm (Kaberi et al., 2013), considered microplastics (MPs) according to regularly used definitions (Arthur et al., 2009; Hanke et al., 2013). The lower border of MPs is more disputed, with many publications considering particles smaller than 1 μm as nanoplastics (NPs) (Gigault et al., 2018; Materic et al., 2021; Shi et al., 2024; Yong et al., 2020), others only considering particles smaller than 100 nm to be NPs (Fournier et al., 2020; Prust et al., 2020). In order to unify the terminology, we use the terms MP for particles >1 μm, NP for particles <1 μm. If both, micro- and nanoplastics are to be considered, we are referring to them as MNPs.

A distinction is normally made between primary and secondary MPs. Primary MPs are produced in microscopic dimensions and are used, for example, as abrasive agent. Secondary MPs, on the other hand, are produced when large plastic is broken down by various environmental influences. Those influences include sunlight, temperature fluctuations or biological degradation (Muthukumar and Veerappapillai, 2015).

The small size of MNPs leads to human uptake and accumulations via the food web. It can be absorbed through the digestive tract as well as the lungs. Uptake in the human body can occur orally, respiratory for airborne particles and by dermal absorption. Once in the body, MNPs are distributed according to size, whereas smaller particles are more largely distributed. Particles smaller than 10 μm can be internalized by cells, allowing them to cross intestinal barriers (Li and Liu, 2024; Wu et al., 2022) and even a crossing of the blood-brain-barrier by MNPs is possible (Kopatz et al., 2023; Li and Liu, 2024). Literature suggests that even particles up to 130 μm could migrate through the intestinal barrier by paracellular transport, via kneading of solid particles through loose junctions in the epithelium (Li and Liu, 2024; Wright and Kelly, 2017). Plastic particles were also measured in human blood with a mean concentration of 1.6 μg/ml, suggesting active or passive crossing of the intestinal barrier or other barriers like the lung epithelial barrier (Leslie et al., 2022). Another study discovered particles up to 3000 μm length and 800 μm width inside human blood (Leonard et al., 2024), posing the question of how these particles pass into the blood. Once the MP enters the blood, it may spread throughout the body and be deposited in a variety of tissues (Dong et al., 2020; Fournier et al., 2020; Hua et al., 2022; Leslie et al., 2022; Lu et al., 2018; Prust et al., 2020; Volkheimer, 1975; Wright and Kelly, 2017). Besides blood (Leslie et al., 2022), plastic particles have already been found in human stool (Schwabl et al., 2019) and various other tissues like liver (Horvatits et al., 2022) and placenta (Braun et al., 2021), with 0.5 μm particles showing stronger accumulation than for example 5 μm particles (Zhang et al., 2024d). Interestingly, women have a significantly higher MP load in their body than men (Zhu et al., 2024) but in general, humans are estimated to ingest approximately 39000–52000 plastic particles per year (Cox et al., 2019). Other estimations based on particles found in different types of food range from 0.1 to 5g of plastic intake per week (Senathirajah et al., 2021), highlighting the great uncertainty regarding human MP consumption.

The majority of polymers found in MPs are Polyethylene (PE), Polypropylene (PP), Polyethylene terephthalate (PET), Polyvinyl chloride (PVC), Polystyrene (PS), Polyurethane (PU), Nylon and Acrylic (Demarquoy, 2024; Thakur et al., 2023).

3. Potential impact of MNPs on human health

Micro- and nanoplastics pose multiple potential health risks for humans including alterations of the metabolism, disruptions of the immune system, neurotoxicity, reproductive toxicity and carcinogenity, but also effects on the cell level like oxidative stress, cytotoxicity and inflammation (Rahman et al., 2021; Sini Francis et al., 2021; Wu et al., 2022; Yong et al., 2020). Potential health effects may differ between the different plastic types, according to the material itself but also associated additives. Lots of information is still missing and further research into the dangers of MNPs is necessary (Brachner et al., 2020). Polyethylene terephthalate for example may include endocrine disruptors (Sax, 2010) and polyethylene seems to be leading to reproductive difficulties in mice (Park et al., 2020). PP is associated with respiratory disorders in humans, as observed in PP flocking workers (Atis et al., 2005), and polyvinylchloride proofs to be carcinogenic and toxic to major human organ systems, by inducing oxidative stress and showing endocrine activity, as tested in human cell lines (Sini Francis et al., 2021; Zimmermann et al., 2019). Chemicals used in polytetrafluoroethylene-production even seem to be associated with tumor development and neonatal death in rodents (Sini Francis et al., 2021; Steenland et al., 2010). Cell uptake into human cells has also been described in recent publications (Akhatova et al., 2022; Liu et al., 2022; Xu et al., 2021), with new research demonstrating the potential of PS beads to affect cell migration and having pro-metastatic effects in tumour cells (Brynzak-Schreiber et al., 2024).

Since the size of the particles is a crucial factor in regards of the toxicity, a rising number of publications is paying attention to NPs, after years of being neglected due to limitations in methods (Allen et al., 2022; Cunningham et al., 2023). Cell uptake of smaller particles is more likely and has already been proven in multiple publications including human cell lines (Brynzak-Schreiber et al., 2024; Lim et al., 2019; Prietl et al., 2014; Wu et al., 2019; Xu et al., 2019). A further reduction in the size of MPs by environmental degradation, including physical degradation by mechanical forces or temperature differences, photodegradation, chemical degradation and biodegradation (Bozkurt et al., 2022) to NPs leads to an exponential increase in the number of NPs. This is raising concerns regarding their potential environmental and health impact, since the effects on humans are largely unknown.

Nanoplastics can lead to damage on the level of the most fundamental building blocks of life, including DNA fragmentation and damaged cell membranes as shown in a type of microalgae (Sendra et al., 2019). Protein modifications are also predicted, according to simulated models (Holloczki and Gehrke, 2019) and alterations in gene expression in nematodes (Qu et al., 2019a; Qu et al., 2019b) or transcription factors in *Daphnia* (Zhang et al., 2020) have been shown (Kihara et al., 2021). The toxic effects might be based on the high surface area of the particles, generating oxidative stress, inflammation, physical damage or apoptosis, but no relevant direct cytotoxic effects were found in experiments with human colonic cells (Cortés et al., 2019; Li and Liu, 2024).

In the following chapters, we discuss the potential plethora of health effects of MNPs on different parts of the gastrointestinal tract of mammals, as the particles pass through the body and potential strategies to alleviate damages of MNP on the gastrointestinal tract.

3.1. Impact on the gastrointestinal tract

Micro- and nanoplastics enter the gastrointestinal tract via the mouth, where they can already change the oral microbiota, shown in a study including the impact of take-away containers on humans (Zha et al., 2023). Furthermore, they inhibit alpha amylase activity in human sputum (Azhagesan et al., 2024; Huang et al., 2022) and directly interact with oral epithelium and the mucus layer. Increased viscosity of saliva and decreased viscosity of mucus is the consequence, as shown in experiments with artificial mucus and saliva (Przekop et al., 2023). Both are associated with pathogen overgrowth: less viscous mucus enables

pathogens to reach the epithelial surface, while viscous saliva is linked to periodontal disease and caries. Increased viscosity leads to decreased bacterial co-aggregation, impaired oral clearance and, consequently, pathogen growth and oral microbiome dysbiosis (Biesbrock et al., 1992; Yas, 2013).

From the oral cavity, swallowing brings MNPs to the oesophagus, where they might exert toxic effects by accumulation of ROS species and inflammation leading to cell death in human oesophageal cell lines (Guanglin and Shuqin, 2024).

Reaching the stomach, MNPs exert a broad variety of actions depending on type of plastic, particle size, surface characteristics, electric charge, ageing and many more. Micro- and nanoplastics are able to inhibit gastric juice secretion, mucus production, disrupt gastric barrier function and suppress antioxidant ability, as examined in mouse experiments and human gastric cell lines (Sun et al., 2024b). Furthermore in vitro simulated human digestion showed, they influence digestion processes, since they can bind and reduce the function of Pepsin as well as bind food proteins, which can then escape digestion or at least elongate their retention time in the stomach (Krishna de Guzman et al., 2023). To further increase their possible spectrum of action and their properties MNPs can generate surface coronas around them by binding lipids, proteins, or a combination of both by directly bonding with different amino acids according to plastic type (Vismara and Gautieri, 2024). The corona composition is influenced by different food matrices (Li et al., 2023b) and the presence of endogenous proteins. Even though coronas enhance the particle size, they also allow MNPs cellular uptake by changing uptake kinetics and properties (Dorsch et al., 2023). This effect is also dependent on particle size, since particle coronas containing coagulation factors, apolipoproteins and vitronectin, elevate especially the uptake of small MNPs in a human lung cell line (Brouwer et al., 2024; Wang et al., 2023b). Micro- and nanoplastics can then act on the cellular level in the gut. Gastric epithelial cells can internalize MNPs, which leads to increased oxidative stress, DNA damage and mitochondrial dysfunction and finally apoptosis or necrosis dependent on particle size and concentration (Han et al., 2024; Sun et al., 2024b).

Helicobacter pylori, a common human pathogen associated with chronic gastritis, seems likely to be interacting with MNPs. In artificial gastric juice experiments *H. pylori* adheres to MNPs to form a biofilm. Furthermore, in vivo interaction between MNPs and *H. pylori* contributes to the rapid bacterial colonization of gastric mucosal epithelial cells, accelerates entry into tissues, and promotes gastric injury and inflammation (Tong et al., 2022). Additional investigations are needed to determine if and how much MNPs contribute to *H. pylori* induced gastritis and risk of gastric cancer development.

Dietary habits could also influence the effects of MNPs in the gut and in the whole body, making it challenging to distinguish the effects of MNPs and diet. Insufficient food intake worsens oxidative stress in the liver and bile acid disorder due to increased bioaccumulation of MNPs, reduced water intake or elevated pathogenic bacteria abundance in mice (Lv et al., 2023), indicating a potential higher risk for people living in parts of the world with insecure food and water supply or people with diseases impairing food and water intake. Furthermore, all effects seen with high fat diet like inflammation, higher blood glucose and serum lipids were worsened by MNPs in a mouse study (Okamura et al., 2023). Micro- and nanoplastics can also influence the provision of the body with nutrients, since they reduce the bioavailability of dietary calcium, copper, zinc, manganese and magnesium in the small intestine, while leading to higher iron bioavailability in the intestine, liver and kidney, as observed in a mouse model (Chen et al., 2023b). In mice, the uptake of PS beads was associated with the development of obesity and cardiometabolic diseases, including disturbed glucose and lipid metabolism (Zhao et al., 2024; Zhao et al., 2022). However, not only accessibility to nutrients but also to toxic compounds like arsenic are regulated by MNPs according to data from mouse models (Chen et al., 2023c). Via the gut-liver-axis MNPs increase hepatic inflammation, promote fibrosis, alter the hepatic expression profile (Zhang et al., 2023a), and trigger changes in hepatic receptors and signalling molecules as shown in mice (Zhao et al., 2024).

3.2. Impact on the gut barrier

Moving forward in the gastrointestinal system, MNPs change the intestinal tissue structures by villus erosion, decrease of numbers of crypts and large infiltrations of inflammatory cells in the gut, as shown in mice (Li et al., 2024). Furthermore, MNPs increase intestinal permeability by various mechanisms as demonstrated by mouse experiments. Directly, they lead to reduced expression of tight junction proteins, compromise gut barrier integrity and function and decrease mucus secretion. Indirectly, they increase local inflammation, further increasing the leaky-gut syndrome (Jia et al., 2023; Kim et al., 2024; Li et al., 2024; Xu et al., 2023; Zeng et al., 2024). On the cellular level, intestinal uptake of MNPs occurs directed via microfold cells as sensors, capturers and transporters of larger particles and undirected in epithelial cells internalizing the particles in a size-, concentration-, and time-dependent manner by diffusion (Chen et al., 2023e) or endocytosis (Hou et al., 2022), based on findings from human intestinal organoids. Once taken up, MNPs agglomerates seem to move freely within epithelial cells and are even found within nuclei (DeLoid et al., 2021). Micro- and nanoplastics were shown in cell culture experiments to be able to permeate into lipid bilayers, decreasing their density, changing fluidity and thickening the membrane, whereas larger MP aggregates are likely to cause pore formation (Wang et al., 2022). Internalization of MNPs induces metabolic changes by inducing oxidative stress, increasing glycolysis via lactate to maintain energy metabolism and glutamine metabolism to sustain anabolic processes in human intestinal cells. This is quite similar to a typical mechanism of cancer cells to optimize nutrients utilization and adapt to stress, indicating that chronic MNPs exposure might be a risk factor for colorectal cancer (Bonanomi et al., 2022; Li et al., 2023a). Micro- and nanoplastics that enter the blood are able to form complexes with hemoglobin, albumin and fibrinogen, changing their physiological functions (Ghosal et al., 2023; Leslie et al., 2022; Tran et al., 2022; Wang et al., 2023c; Wang et al., 2024b).

3.3. Impact on the immune system/inflammation

After crossing the gut barrier MNPs encounter different cells of the immune system and research has shown diverse immunotoxic effects on different types of immune cells by MNPs, affecting immune responses at various levels. NF-kB and other proinflammatory pathways in the intestine are activated (Jia et al., 2023; Kim et al., 2024; Li et al., 2024; Zeng et al., 2024), intestinal secretory immunoglobulin A levels as well as the differentiation of $CD4^+$ and $CD8^+$ T cells are decreased (Zhang et al., 2023b) and the transcriptome and metabolism of gut immune cells are disturbed in mice (Harusato et al., 2023). Understanding these effects requires a closer examination of how MPs interact with specific immune cell populations, including T cells, dendritic cells (DCs), macrophages, neutrophils, and other immune cell types.

3.3.1. Impact on T cells

T cells, the central orchestrators of adaptive immunity, play a crucial role in mounting specific immune responses against pathogens (Sun et al., 2023a). However, the complex modulation of T cell function by MNPs poses significant disruptions to immune homeostasis. Emerging evidence from mouse experiments indicates that MNPs interfere with various T cell signalling pathways, leading to disruptions in cytokine balance, an important aspect of T cell communication and function. These disruptions ultimately lead to tissue damage as a consequence of aberrant immune responses. A key mechanism through which MPs exert their effects on T cells is the generation of reactive oxygen species (ROS). While ROS are essential for cell signalling and homeostasis, the

excessive production induced by MPs can result in oxidative stress, leading to cellular and tissue damage (Wu et al., 2023). Moreover, the elevated ROS levels may trigger the release of danger-associated molecular patterns, which in turn activate toll-like receptors (TLRs) on T cells, further fuelling inflammatory responses. This cascade of events not only directly affects T cell function but also contributes to broader inflammatory processes mediated by these cells (Gong et al., 2020).

In studies investigating the impact of MNPs on human T cells, it has been observed that exposure to MNPs, such as PS and poly(methyl methacrylate) (PMMA), results in altered cytokine profiles and increased T cell activation. Specifically, analysis of peripheral blood mononuclear cells exposed to MNPs revealed downregulation of certain cytokines like IL-1β and IFN-γ, alongside upregulation of others such as CCL2, IL-17A, and IL-10. While T cells may not be directly destroyed by MNPs, their functional state is significantly altered, potentially leading to dysregulated immune responses (Wolff et al., 2023).

Furthermore, investigations into the effects of different-sized NPs on murine splenocytes have shown that larger PS-NPs are more readily internalized and induce decreased cell viability and increased apoptosis. This process obstructs T cell differentiation by inhibiting key signalling pathways such as PKCθ/NFκB and IL-2R/STAT5, thereby potentially increasing susceptibility to infections and cancer (Li et al., 2022).

In animal models, such as C57BL/6 mice, exposure to high concentrations of polyethylene MPs has been associated with increased gut inflammation, alterations in T cell subsets, and upregulation of TLR4 and IRF5 expression. These findings underscore the urgent need for interventions aimed at preventing and treating MP-related diseases, as the dysregulation of immune responses induced by MPs poses significant health risks (Li et al., 2020).

3.3.2. Impact on dendritic cells

DCs serve as vital mediators between innate and adaptive immunity, crucial for initiating specific immune responses by presenting antigens to T cells (Steinman, 2007). Upon exposure to MPs, experiments showed that human DCs may undergo dysregulated intracellular signalling, compromising their ability to effectively activate T cells and initiate immune responses. This disruption in DC function poses a significant risk to immune surveillance and the body's ability to combat pathogens efficiently.

Micro- and nanoplastics can interact with DC membranes and intracellular proteins, forming a protein corona that alters DC behaviour and function, intensifying the immunotoxic effects of MNPs on these cells. Research indicates that while T cell viability remains largely unaffected by MNP exposure, DCs exhibit decreased viability, particularly at higher concentrations of MNPs. Moreover, DCs derived from monocytes show a shift towards anti-inflammatory phenotypes, characterized by decreased expression levels of CD40 and CD80, potentially impairing their capacity to initiate robust immune responses (Wolff et al., 2023).

In an in vivo study using a mouse model and PE-MPs, exposure significantly inhibited the maturation of dendritic cells (CD11b−/CD11c+) in a dose-dependent manner. This inhibition of mature DCs could compromise immune responses, rendering individuals more susceptible to infections and impairing immune surveillance (Park et al., 2020).

3.3.3. Impact on macrophages

Macrophages, pivotal components of the innate immune system, swiftly respond to particle exposure by phagocytosing these foreign particles, initiating the initial defence mechanism (Ross et al., 2021). However, prolonged exposure to MNPs can provoke dysregulated immune responses characterized by excessive inflammation and tissue damage. Notably, human macrophages exhibit heightened sensitivity to MNPs, with their phagocytic activity significantly altered by these particles. Similar to DCs, macrophages display heightened sensitivity to higher concentrations of MNP polymers, resulting in downregulation of markers associated with pro-inflammatory M1 macrophages, such as MHC-II and CD80/86. This compromised phenotype may diminish the host's capacity for innate immune defence (Wolff et al., 2023).

Moreover, an investigation into the impact of sulfate-modified polystyrene NPs on murine macrophages unveiled adverse effects, including cytoplasmic accumulation of lipid droplets, leading to foam cell formation. Additionally, these NPs induced heightened levels of ROS production, impaired lysosomal function, and perturbed lipid metabolism in macrophages (Florance et al., 2021).

3.3.4. Impact on neutrophils

As integral part of the innate immune system, Neutrophils serve as the body's rapid responders, crucial for preventing immunotoxicity by swiftly forming phagosomes that target foreign pathogens (Rosales, 2018). Neutrophil extracellular traps (NETs) can capture nanoparticles, forming macroaggregates that facilitate their phagocytosis by neutrophils or macrophages, illustrating the intricate interplay between MNPs and immune cells (Bilyy et al., 2020).

MPs derived from marine sources, such as PE, PP, and PS, are ingested by fish and induce the release of NETs leading to alterations in fish behaviour, physiology, and metabolism, potentially impacting disease resistance (Tanaka and Takada, 2016). Activation of neutrophils has also been observed in zebrafish models following exposure to MPs, highlighting their susceptibility to MP-induced responses (Limonta et al., 2019).

In vivo studies with mice revealed that the interaction between neutrophils and hydrophobic nanocrystals from NPs results in the formation of occlusive aggregated NETs, obstructing vital tubular structures like capillaries and bile ducts, thus posing severe health risks (Bilyy et al., 2020). While serving as a defence mechanism, neutrophils and NETs can also contribute to pathological conditions when interacting with MNPs and other nanoparticles.

3.3.5. Impact on immune homeostasis

Multiple studies on human epithelial cell lines and on mice have demonstrated that MNPs can activate the immune system, triggering an inflammatory cascade characterized by the release of cytokines such as IL-1β, IL-6, IL-8 and the activation of NF-κB, leading to cell membrane damage and consequential structural and functional impairments across various tissues (Dong et al., 2020; Hu and Palic, 2020; Wang et al., 2023a; Xu et al., 2019). Transcriptomic analyses have further highlighted the involvement of inflammation pathways, including the alternative complement pathway, mitogen-activated protein kinase and Ras signalling pathways upon MNP stimulation in mice and zebrafish (Chen et al., 2023a; Umamaheswari et al., 2021; Wu et al., 2020).

Moreover, the gastrointestinal tract serves as a primary site of MNP exposure due to ingestion, where MNPs can accumulate and potentially disrupt immune homeostasis, leading to cytotoxicity and immunotoxic effects. A recent study on PET MPs using mouse models found that chronic low-dose exposure to PET MP did not significantly alter intestinal pathology or the numbers of key immune cells. However, RNA-seq analysis revealed substantial impacts on the transcriptome of gut immune cells and their metabolisms, suggesting an unexpected role in influencing gut immune homeostasis without detectable inflammation (Harusato et al., 2023). Additionally, as discussed before, MNPs can interact with gut microbiota, potentially disrupting the delicate balance of the intestinal ecosystem, leading to altered immune responses and increased susceptibility to infections and inflammatory diseases.

3.4. Impact on the intestine and its microbiome

3.4.1. In vitro data

First insights in the effects of MNPs on human gut microbiota have been provided by artificial or simulated digestion models: Static digestion of MPs showed that PE (30–140 μm size) increases *Clostridum, Bacteroides and Escherichia* in the gut microbiota, with even more drastic changes, if the plastic additive tetrabrombisphenol A was present

(Huang et al., 2021). In a standardized in vitro model combined with a dynamic gastrointestinal model, PET particles underwent biotransformation during digestion and altered gut microbiota, increasing *Escherichia/Shigella* and *Bilophila* and other opportunistic pathogens like *Phascolarctobacterium*, *Lachnoclostridium* and *Megasphaera*. Biofilm formation was promoted by microbial binding in a multi-compartmental model (Tamargo et al., 2022). Repeated exposure to PE in the Mucosal Artificial Colon model increased *Desulfovibrionaceae* and *Enterobacteriaceae* while decreasing *Christensenellaceae* and *Akkermansiaceae* in adult stool samples, whereas in infant stool, alpha diversity, *Dethiosulfovibrionaceae* and *Enterobacteriaceae* increased (Fournier et al., 2023a; Fournier et al., 2023b). PS and PE MPs promote overgrowth of opportunistic bacteria (*Enterobacteriaceae, Desulfovibrio* spp., *Clostridium group I, and Atopobium-Collinsella*) while reducing most beneficial bacteria (Nissen et al., 2024). In the mucosal simulator of the human intestinal microbial ecosystem, PET particles affected both intestinal luminal and mucosal microbiota, with more pronounced changes in the lumen due to the stabilizing effect of mucus. Gut microbes like *Acidaminococcus* and *Morganella* can release phthalates from MPs, strongly impacting luminal and mucosal microbiota (Yan et al., 2022b).

How much the gut microbiota contributes to release harmful plastic compounds is currently topic of further research. Various chemical additives are regularly used to gain specific properties or enhance the performance of the polymers, including flame-retardants, colorants, thermal stabilizers, UV stabilizers, antioxidants, lubricants or biocides. Hazardous additives can additionally contain heavy metal substances like cadmium, chromium, cobalt, lead, mercury, tin, and zinc (Groh et al., 2019). MNPs have additionally been shown to be able to bind a plethora of substances from the environment, which might enter the human body with their help. Micro- and nanoplastic bound toxins, like heavy metals (Hu et al., 2024), polycyclic aromatic hydrocarbons, pharmaceuticals (McDougall et al., 2022), microcystins (Pestana et al., 2021), natural and artificial radionuclides (El Zrelli et al., 2023), plastic additives (Lopez-Vazquez et al., 2022), endocrine disruptors like Bisphenol A and benzophenone (Oliveira et al., 2024) can leech from MNPs in the human gut, due to changing pH- and salt-concentrations (Luo et al., 2019). Those substances could then possibly provoke gastrointestinal symptoms, including nausea and vomiting (Abbasi et al., 2021) or lead to endocrinal disruption and allergies (Iftikhar et al., 2024). In addition, synergistic effects are likely, like seen with Bisphenol A and MNPs together, which change the gut microbiota dramatically: Enterococcus and Lactobacillus show decreased abundance while Escherichia, Clostridium and Bacteroides increased (Huang et al., 2021).

Simulated digestion and fermentation models inoculated with human samples show that some biodegradable plastics, namely Poly (ε-caprolactone) (PCL) and Poly(lactic acid) (PLA) can also be degraded in the small intestine and degraded as well as oligomerized by fermentation in the colon. The resulting oligomers reduce the alpha diversity of the gut microbiota and can inhibit *Bifidobacteria, Lactobacilli, Faecalibaterium, Limosilactobacilli, Blautia, Romboutsia* and *Ruminococcus* (Peng et al., 2024). PLA shows changed morphology after simulated gastric digestion and the ability to form a microbial biofilm after the intestinal and colonic phase, possibly by interaction with the human microbial colonic community but it did not change overall microbiota composition directly, although Bifidobacterium levels were increased in a different study (Jimenez-Arroyo et al., 2023). Artificial digestion experiments also showed that gastric juice and the interaction with organic matter influenced the surface properties and agglomeration behaviour of MNPs, changing their interaction with intestinal barriers (Paul et al., 2024).

By changing the gut microbiota, MNPs can influence the production of microbial metabolites in the gut. Via this pathway they might reduce the synthesis of neurotransmitters (Alipour Nosrani et al., 2021; Moser et al., 2019; Wen et al., 2024) and short chain fatty acids (SCFAs) (Moser et al., 2019; Yan et al., 2022b) adding to neuro- and hepatotoxic effects of MNPs. Their influence on bile acid metabolism is multifaceted. By changes in the intestinal microbiota, they alter the ratio of primary to secondary bile acids in mice and by inducing liver injury, they can cause cholestasis (Wen et al., 2024). Furthermore, MNPs are present in human gallstones and may aggravate cholelithiasis by forming large heteroaggregates with cholesterol (Zhang et al., 2024a).

3.4.2. *In vivo data*

Multiple studies on the microbiome of animals, like fish (Huang et al., 2020; Qiao et al., 2019; Tamura et al., 2024) and mice (Chen et al., 2023d; Chen et al., 2022; Jin et al., 2019; Lu et al., 2018) showed that MP intake could lead to dysbiosis, with unknown follow-up problems. Data from animal experiments are still inconclusive since plastic particle effects depend largely on the polymer properties, including their type, size and aging state. In vivo data on plastic types and their effects on the human gut microbiota are very scarce. Microplastics are present in infant and adult human faeces (Ho et al., 2022; Luqman et al., 2021; Schwabl et al., 2019; Wibowo et al., 2021; Yan et al., 2022a; Zhang et al., 2021b) including stool of pregnant women (Amqam et al., 2022) and meconium (Liu et al., 2023; Zhang et al., 2021a), reviewed in (Barcelo et al., 2023). A comparison of gut microbiota of Indonesian costal and highland dwellers revealed differences in the MP contamination of both groups. People living near the coast mainly had HDPE and people living in the highlands mainly had PP in their faeces. Microplastic contamination did not affect overall gut microbial composition, although, negative and positive correlations between specific types of MP with certain bacterial taxa, especially from the genera *Roseburia, Clostridium* and *Prevotella* were detected (Nugrahapraja et al., 2022). In stool of Chinese preschool children PVC, PET, PE and polyamide 6 was found. The primary source seems to be the use of feeding bottles, with higher PVC levels being associated with longer feeding times per meal. Children with high MP content, especially PVC, showed lower alpha diversity levels. MP exposure possibly impacted probiotic taxa like *Parabacteroides* and *Alistipes*. The changes on genus level were dependent on the main plastic type ingested. PVC showed a positive correlation with the abundance of the genus *Faecalibacterium* and a negative correlation with *Alistipes* and *[Eubacterium]_coprostanoligenes_group*, while PE was negatively correlated with the abundances of *[Eubacterium]_coprostanoligenes_group*, *Parabacteroides*, and *Lachnospiraceae_NK4A136_group* and PET was positively correlated with *Agathobacter*. (Ke et al., 2023). Furthermore, the frequency of plastic bottle use seems to be important, since infants who were plastic bottle fed at every meal had lower faecal microbiota alpha diversity than infants with less feeding bottles use, after adjustment for potential confounders (Tilves et al., 2023).

Already in the placenta sixteen types of MPs, mostly PA and PU, can be found and they seem to be transferred to the foetus, since they are also detectable in the meconium, affecting beta diversity and bacterial abundance based on the predominant MP type (Liu et al., 2023). In adults consuming hot food served in plastic containers, stool samples showed higher MP concentrations with significant changes in *Actinobacteria, Proteobacteria, Firmicutes* and *Bacteroidota* and an increase in *Collinsella* (Zha et al., 2023; Zhang et al., 2024b). A separate study focusing on take-away food linked high MP exposure with increased *Veillonella*, *Alistipes*, and *Dothideomycetes*, potentially contributing to obesity (Hong et al., 2024).Plastic factory workers, reasonably classified as high MP exposure group, showed elevated levels of PU, silicone resin, ethylene-vinyl acetate copolymer and PE. They had an increased abundance of *Bifidobacterium, Streptococcus* and *Shingomonas* and a decreased abundance of *Ruminococcus Torquesgroup, Dorea, Fusobacterium* and *Coprococcus* in the gut (Zhang et al., 2022).

In the clinical setting, a comparison of faeces of healthy and IBD patients revealed higher MP concentrations in patients. Fifteen types of MPs were detectable in both groups with PET and PA being the dominant ones. A positive correlation exists between faecal MP concentration and severity of IBD, strongly indicating an association with microbiota dysbiosis (Yan et al., 2022a). Micro- and nanoplastic induced microbial disequilibrium results in symptoms like abdominal pain, bloating, and

changes in bowel habits (Jin et al., 2019).

Due to these far-reaching contaminations, most of which are still unexplored in humans, as well as the potential negative impacts on human health, methods need to be developed to protect us from the potential health problems. As more and more MNP pollution is found, the pressure to develop solutions quickly is increasing. Reduction of plastic consumption, development of biodegradable plastic, better waste management, including for bio-based plastics, standardized labelling and sorting instructions, but also strategies for plastic mineralization are needed (Prieto, 2016). Multiple research gaps have been identified to be able to develop meaningful policies (Courtene-Jones et al., 2022). This review focuses on methods to prevent MNPs from causing damage in the body.

4. Modulating the microbiome to tackle MNP effects on the human body

The reduction of plastic uptake in humans must be promoted by global endeavors, focusing on the reduction of consumption, improved waste management and removal of plastic debris (Zhao and You, 2024), but may be currently difficult on a personal level (C.P. manuscript in preparation), with plastic particles being present in a wide variety of food and beverages (Cox et al., 2019; Karami et al., 2017; Kosuth et al., 2018; Senathirajah et al., 2021; Sobhani et al., 2020). Therefore, alternative methods to reduce the influence of MNPs on the gut, the microbiome and the immune system need attention. This review focuses on the two possibilities of either degrading synthetic polymers inside the human gastrointestinal system using the microbiome or modulating the microbiome in a way to directly tackle the health effects posed by plastic particles.

4.1. Enzymatic degradation of MNPs

Microplastic particles form a novel ecological niche for bacteria by providing a growth support structure (Yuan et al., 2020). Due to their small size, microorganisms can have close contact with the surface area of plastics and some of them can produce various enzymes with plastic degrading properties, possibly even using plastics as nutrient and energy source (Chapelle, 2000; Sini Francis et al., 2021). Multiple reviews about bacterial degradation of plastic particles have already been published (Amobonye et al., 2021; Matjasic et al., 2021; Pathak and Navneet, 2017; Sun et al., 2023b), so this publication intends to focus on the question of whether enzymatic degradation of MP could happen within the human body. Therefore, enzymes derived from bacteria that have been associated with the human gut, possibly enabling a future application in humans, will be the focus of this review.

Various studies were able to show that bacteria working synergistically in consortia can do plastic degradation (Amobonye et al., 2021; Huerta Lwanga et al., 2018; Shah et al., 2008). Due to the high bacterial density and variety in the human gut (Arumugam et al., 2011; Cresci and Bawden, 2015; Gomaa, 2020; Guarner and Malagelada, 2003), consortia-formation seems to be likely, but for the sake of possible human applications each bacterium, and its ability to degrade plastics, will be examined individually.

Plastic polymers have a simple basic chemical structure. They mostly derive from chemical products obtained from refined petroleum and are synthesized to produce long-chained hydrocarbon polymers (Gan and Zhang, 2019; Hees et al., 2019; Shimao, 2001). Despite being relatively new materials, synthetic polymers can be degraded by certain enzymes due to structural similarities with natural compounds. Polymers found in nature include lipids, carbohydrates, and proteins (Pathak and Navneet, 2017). For instance, polyethylene exhibits a structure similar to certain components found in beeswax. Consequently, enzymes produced by organisms like the wax moth, which typically feed on beeswax, can facilitate the degradation of PE (Bombelli et al., 2017). In recent years, an increasing number of organisms capable of, at least partly, degrading plastics have been identified. This trend suggests a potential evolutionary response to the growing abundance of persistent particles like plastics in the environment (Jang and Kikuchi, 2020; Sini Francis et al., 2021).

Plastic degradation usually follows a specific pattern, starting with bio-deterioration, a superficial degradation driven by biotic and abiotic factors, changing the physicochemical properties of the plastics. The next step is called bio-fragmentation and includes the cleavage of polymers into oligo- and monomers, usually by extracellular enzymatic activity of microorganisms and the release of oxidative agents. This is followed by the assimilation of oligo- and monomers into microorganisms by active and passive transportation, where they can be used as carbon source. The last possible step is mineralization, which is the oxidation of oligo- and monomers to H_2O, CO_2, N_2, and CH_4 (Amobonye et al., 2021; Atanasova et al., 2021; Claire Dussud, 2014; Pathak and Navneet, 2017).

The MP-degrading enzymes can roughly be separated into extracellular and intracellular enzymes, with the extracellular ones performing the bio-fragmentation step of cleaving the long polymer chains into short chains, enabling the assimilation into the cells. Examples for extracellular enzymes, that are able to degrade the backbone of plastic polymers mostly by hydrolysis are cutinases, which can degrade PET, or different depolymerases, lipases, hydrolases and proteases, but also styrene oxide isomerase, degrading PS, can be produced (Carniel et al., 2017; Lin et al., 2022b; Oelschlagel et al., 2012; Skariyachan et al., 2022; Tokiwa et al., 2009). Inside the cells, among others, esterases and lipases, degrading for example PE, PET, PVC and PU, are active and convert shortened chains into carbon sources (Akutsu et al., 1998; Howard, 2002; Lin et al., 2022b). The challenge to be faced is the high variability of plastics. Already small variations of the chemical structure can lead to vastly different properties and can change the rate of biodegradability immensely (Gu, 2003).

When polymer particles do not follow the complete mineralization process, intermediates may be directed into diverse chemical pathways (Amobonye et al., 2021). For instance, the degradation of PE has been proposed to involve the formation of acetic acid, entering the TCA cycle or lipid synthesis (Amobonye et al., 2021; Wilkes and Aristilde, 2017). Styrene, the monomeric basic building block of PS is mainly oxidized to phenylacetate, then also metabolized in the TCA cycle (Ho et al., 2018). Not much is known about plastic degradation products and further interference with human health is possible. Degradation without complete mineralization might prove dangerous, since smaller particles are more likely to pass biological barriers (Xu et al., 2019). The release of additives during the degradation process is also likely and might disrupt biological processes (Yu et al., 2024b).

The removal of environmental pollutants via the use of biological systems is also called bioremediation and can happen aerobically or anaerobically. For the purpose of this review and possible applications inside the human gut, anaerobic systems are necessary, so fungi, which are typically dependent on the presence of oxygen (Bardaji et al., 2020), can be excluded. Bacteria on the other hand can possibly degrade plastics under aerobic and anaerobic conditions. Anaerobic biodegradation produces organic acids, H_2O, CO_2 and CH_4 as end products (Gu, 2003). According to literature, it was shown that single bacterial strains could also produce toxic metabolites during the later stages of polymer degradation, which could inhibit bacterial growth. Bacterial consortia, like the gut microbiome, on the other hand could counteract this effect, by eliminating these toxic end products (Dobretsov et al., 2013; Lin et al., 2022b; Yuan et al., 2020).

The transit time in the human gut can vary strongly between individuals (Minnebo et al., 2023), leaving a highly variable time span for possible plastic degradation. Plastic degradation rates also vary significantly depending on the type of plastic and environmental conditions. Lin et al. (2022b) showed in an overview about degradation efficiency and time, that weeks, months or even years are sometimes necessary to only partly degrade different polymers. In some instances, complete

degradation may take several hundred years (Lithner et al., 2011), rendering it impossible for it to occur within the timeframe of gut passage. For possible applications in humans, this needs to be considered.

Bacteria, that could be applied within the human gut, need to be tested for their safety and compatibility with the human microbiome. These requirements are met by probiotic strains, that are either already used in different available products, or possibly in the future, by including next-generation probiotic strains. According to an expert conference of the World Health Organization and the Food and Agriculture Organization of the United Nations probiotics were defined as "live microorganisms which when administered in adequate amounts confer a health benefit on the host" in 2001 (as cited in (Hill et al., 2014)), becoming one of the most used and accepted definitions for probiotics. The bacterial strains, that fall under this definition and are fit for human application, have already been tested for their safety and are generally QPS (Qualified Presumption of Safety)-certified by the European Food Safety Authority (EFSA), while also having the GRAS (Generally Recognized As Safe)-status in the USA, as provided by the United States Food and Drug Administration (FDA).

4.2. Sources of MP-degrading bacteria/enzymes

Bacteria capable of degrading plastics have already been identified in various ecological habitats, including dumpsites and landfills (Gaytan et al., 2019; Muhonja et al., 2018), recycling facilities (Yoshida et al., 2016), cold marine surroundings (Urbanek et al., 2018), and the gastrointestinal tracts of insects (Ren et al., 2019).

Several databases (Buchholz et al., 2022; Gambarini et al., 2022; Gan and Zhang, 2019), as well as an extensive literature research that was carried out between January 2023 and June 2024, were used to collect a comprehensive list of plastic degrading bacteria. By comparing found bacteria with the data repository for human gut microbiota (GMrepo – accessed via https://gmrepo.humangut.info/taxon), bacteria also found in the human gut were identified and included in Supplementary Table 1. Only bacteria with a known species name and/or with the known plastic degrading enzyme were included.

4.3. Scanning databases for genetic sequences of plastic degrading enzymes in probiotic bacteria

With the aim of scanning the genome of commonly used species in probiotic products for plastic degrading enzymes, publicly available genomes from relevant species were downloaded from the NCBI database (https://www.ncbi.nlm.nih.gov/) using NCBI Datasets command-line tools. From Supplementary Table 1, the GenBank ID and UniProt ID's were used to download the corresponding nucleotide and amino acid sequences. These sequences were used as a query for alignment to the downloaded genomes with BLAST (Camacho et al., 2009). The hit with the highest bitscore is shown in Tables 1 and 2 for nucleotides and amino acids respectively. We use the guidelines from EFSA of a minimum identity match of 80% and a minimum coverage of 70% (Authority, 2021), to consider the presence of the gene.

No known plastic degrading enzymes were found on the publicly available genomes of conventional probiotic species and next-generation probiotic species based on the applied criteria, as visible in Tables 1 and 2 This indicates that probiotics do not contribute to plastic degradation in the human gut. Given that the degradation products of plastics have not been thoroughly studied and may pose significant health risks to humans, the absence of such degradation and also the absence of a risk of additive-release by microbial degradation (Yan et al., 2022b; Yu et al., 2024b), could be considered a benefit and safety criterion of a probiotic.

Since degradation of plastics does not seem to be the solution for the MNP health risks, due to lack of degradation and unknown dangers, other ways to prevent MNP related effects in the human gut via modulating the gut microbiome are needed (see Fig. 1).

Table 1
Nucleotide comparison of plastic degrading enzymes in probiotic species. The best hit is provided via its GenBank ID. Percentage of identity and coverage of the gene are shown, if a best hit was identified.

	best hit	Nucleotide analysis		
		Enzyme	% identity	% coverage
Bacillus coagulans	x		x	x
Bifidobacterium animalis	x		x	x
Bifidobacterium bifidum	x		x	x
Bifidobacterium breve	MW659701.1	Rubber oxygenase	76.7	63.9
Bifidobacterium longum	AF095748.1	Phthalate dioxygenase reductase	73.0	4.5
Lacticaseibacillus casei	x		x	x
Lacticaseibacillus paracasei	x		x	x
Lacticaseibacillus rhamnosus	AF095748.1	Phthalate dioxygenase reductase	82.4	0.9
Lactiplantibacillus plantarum	AF095748.1	Phthalate dioxygenase reductase	75.5	7.2
Lactiplantibacillus pentosus	x		x	x
Lactobacillus acidophilus	x		x	x
Lactobacillus delbrueckii	x		x	x
Lactobacillus gasseri	x		x	x
Lactobacillus helveticus	x		x	x
Lactococcus lactis	x		x	x
Latilactobacillus sakei	x		x	x
Levilactobacillus brevis	x		x	x
Limosilactobacillus reuteri	x		x	x
Ligilactobacillus salivarius	x		x	x
Propionibacterium freudenreichii	AF095748.1	Phthalate dioxygenase reductase	85.0	2.0
Streptococcus thermophilus	x		x	x
Next Generation Probiotics:				
Akkermansia muciniphila	AF095748.1	Phthalate dioxygenase reductase	82.7	5.1
Bacteroides fragilis	AB264778.2	Nylon-oligomer-degrading enzymes	97.8	0.6
Clostridium butyricum	x		x	x
Eubacterium hallii	AF095748.1	Phthalate dioxygenase reductase	76.7	5.0
Faecalibacterium prausnitzii	AF095748.1	Phthalate dioxygenase reductase	73.2	4.8
Prevotella copri	AF095748.1	Phthalate dioxygenase reductase	77.0	4.9

5. Modulating the microbiome to indirectly fight microplastic effects

Modulating the human gut microbiome could prove to be very useful in the prevention of MNP-related effects, that could be countered with

Table 2
Amino acid comparison of plastic degrading enzymes in probiotic species. The best hit is provided via its GenBank or UniProt ID. Percentage of identity and coverage of the gene are shown, if a best hit was identified.

	best hit	Amino acid analysis		
		Enzyme	% identity	%coverage
Bacillus coagulans	WP_041847557.1	carboxylesterase/lipase family protein	61.2	98.6
Bifidobacterium animalis	ALS54749.1	Esterase	40.9	95.5
Bifidobacterium bifidum	L0EF70	Carboxylic ester hydrolase	38.8	58.5
Bifidobacterium breve	AXY54091.1	Alkane 1-monooxygenase	64.0	96.3
Bifidobacterium longum	AKZ20829.1	Esterase	50.6	101.3
Lacticaseibacillus casei	ARF18137.1	Glutamyl-tRNA amidotransferase	39.6	86.7
Lacticaseibacillus paracasei	EMR44439.1	Alpha/beta hydrolase	45.2	82.9
Lacticaseibacillus rhamnosus	ARF18137.1	Glutamyl-tRNA amidotransferase	36.5	101.7
Lactiplantibacillus plantarum	EMR44439.1	Alpha/beta hydrolase	45.2	83.5
Lactiplantibacillus pentosus	ARF18137.1	Glutamyl-tRNA amidotransferase	38.4	88.8
Lactobacillus acidophilus	ARF18137.1	Glutamyl-tRNA amidotransferase	35.0	103.0
Lactobacillus delbrueckii	ARF18137.1	Glutamyl-tRNA amidotransferase	33.3	104.2
Lactobacillus gasseri	ARF18137.1	Glutamyl-tRNA amidotransferase	35.3	104.4
Lactobacillus helveticus	ARF18137.1	Glutamyl-tRNA amidotransferase	34.8	103.0
Lactococcus lactis	EMR44439.1	Alpha/beta hydrolase	49.0	72.3
Latilactobacillus sakei	EMR44439.1	Alpha/beta hydrolase	46.2	82.9
Levilactobacillus brevis	EMR44439.1	Alpha/beta hydrolase	47.3	82.9
Limosilactobacillus reuteri	AQX53942.1	Esterase	95.6	59.6
Ligilactobacillus salivarius	BAF98449.1	Polyethylene glycol-aldehyde dehydrogenase	35.8	101.7
Propionibacterium freudenreichii	AXY54091.1	Alkane 1-monooxygenase	75.5	48.2
Streptococcus thermophilus	ALS54749.1	Esterase	40.9	95.5
Next Generation Probiotics:				
Akkermansia municiphila	Q0KCI0	Intracellular poly(3-hydroxybutyrate) depolymerase	65.4	46.7
Bacteroides fragilis	ALS54749.1	Esterase	56.6	98.7
Clostridium butyricum	BAF98449.1	Polyethylene glycol-aldehyde dehydrogenase	39.4	102.1
Eubacterium hallii	L0EF70	Carboxylic ester hydrolase	62.7	57.2
Faecalibacterium prausnitzii	ALS54749.1	Esterase	48.9	98.3
Prevotella copri	ALS54749.1	Esterase	54.8	99.6

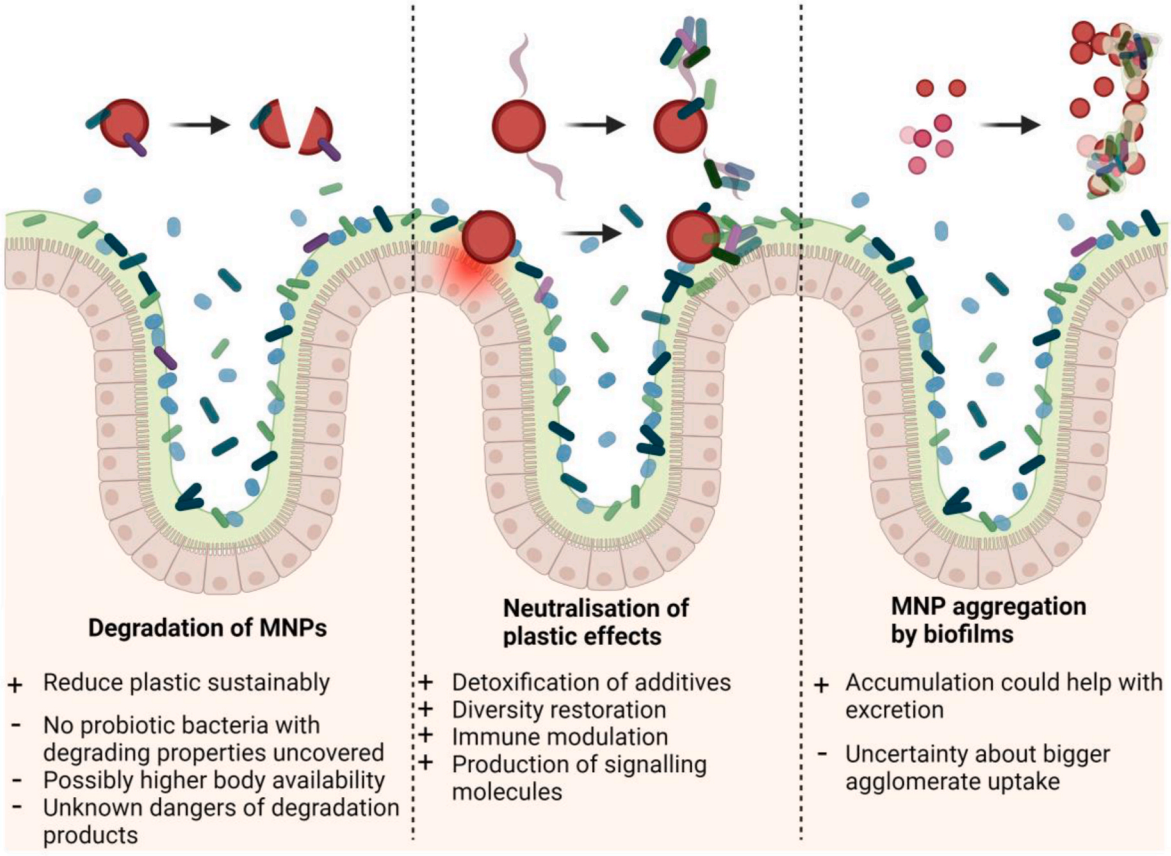

Fig. 1. Bacterial mechanisms that could be used to tackle MNP pollution inside the human gut.

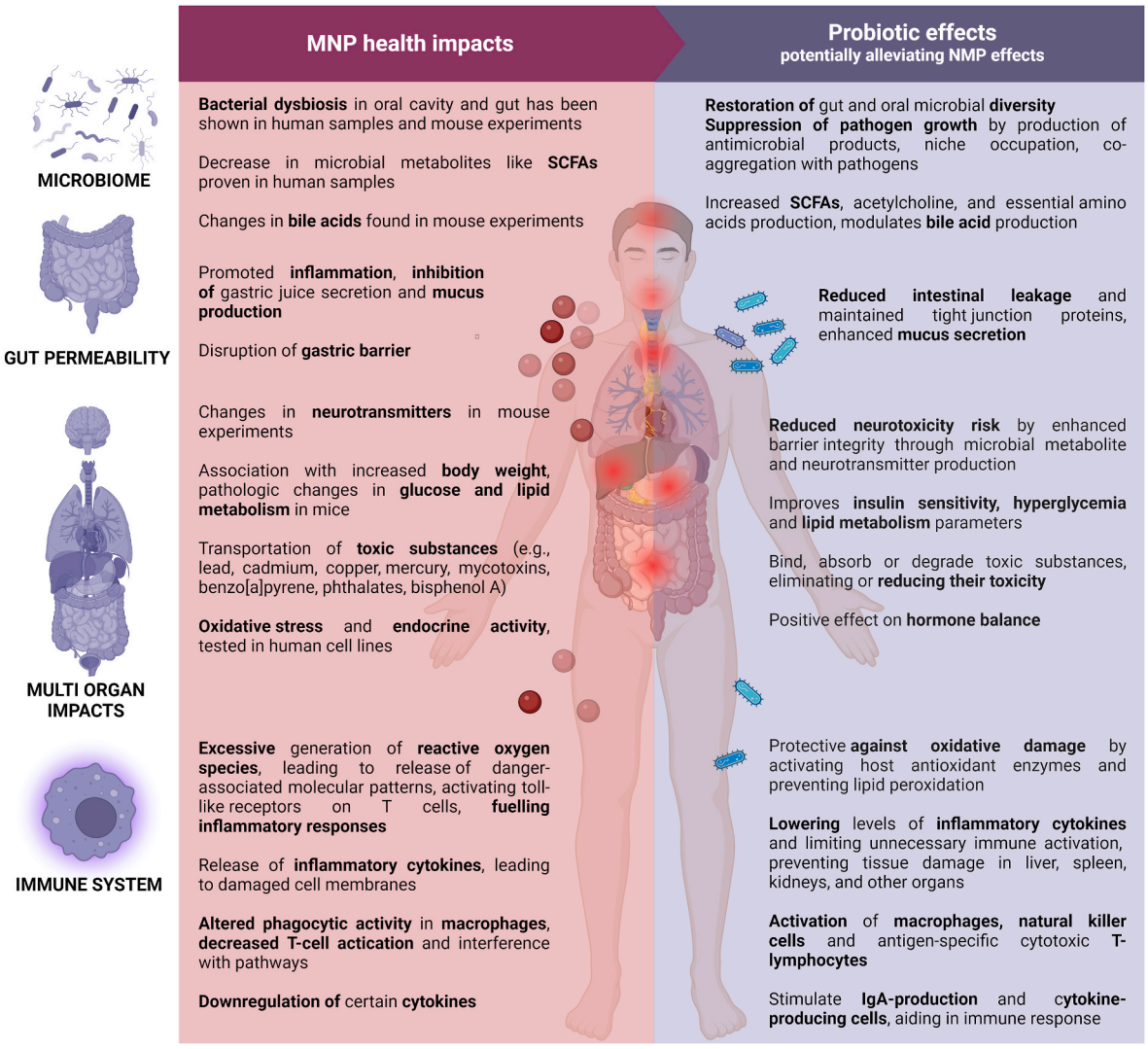

Fig. 2. Probiotic effects directly countering effects triggered by the presence of MNPs.

the use of probiotics (Hung et al., 2021; Lin et al., 2022a; Wasser et al., 2023). Fig. 2 summarizes the MP effects that can be countered with probiotic effects, as explained in more detail in Table 3. As shown in many clinical studies, probiotics can restore the gut and oral microbial diversity (Lin et al., 2022a; Stadlbauer et al., 2019) and improve the gut microbial population (Kienesberger et al., 2022; Labenz et al., 2022), directly counteracting the diversity changes caused by the plastic particles. By occupying ecological niches and co-aggregating with pathogens, probiotics prevent the proliferation of harmful bacteria, thereby reducing the risk of MNP-induced dysbiosis. In the case of toxins produced by pathogens, multispecies probiotics possess various mechanisms to counteract. They can produce antimicrobial substances or acids to reduce pathogen growth, displace pathogens by colonization resistance, reduce the adherence of pathogens to the mucosa, co-aggregate with pathogens and modulate the host's immune system (Campana et al., 2017; Müller, 2016).

By improving the gut barrier, increasing epithelial mucus secretion and decreasing intestinal leakage (Haidmayer et al., 2020; Leblhuber et al., 2018; Moser et al., 2019; Yu et al., 2024a) as well as reducing the destruction of tight junction proteins by decreasing the level of lipopolysaccharides (Horvath et al., 2020a; Szulinska et al., 2018b) probiotics may have a huge potential in protecting the host and his tissue from MNP invasion, which is facilitated by the compromised gastric and gut barrier.

Recent research shows that MNPs can breach the blood-brain barrier (BBB), posing neurotoxic risks as shown in mouse experiments, in silico studies, but also human derived models (Antunes et al., 2023; Chen et al., 2023d; Jeong et al., 2024; Kang et al., 2023; Kopatz et al., 2023; Vojnits et al., 2024; Wang et al., 2024a; Yang et al., 2023). Probiotics can strengthen the BBB by promoting the production of microbial products and neurotransmitters, which may reduce these neurotoxic effects (Loh et al., 2024; Parker et al., 2020).

Additionally, probiotics help lower inflammatory molecule secretion, preventing unnecessary immune activation—a crucial factor in reducing tissue damage in the liver, spleen, kidneys, and other organs (Cristofori et al., 2021; Daniel et al., 2021; Niers et al., 2009; Phillippi et al., 2022; Singer et al., 2018; Strauss et al., 2023).

Given that MNPs have been linked to obesity and metabolic dysfunction (Zhao et al., 2024; Zhao et al., 2022), probiotics could mitigate these effects by improving insulin sensitivity and regulating glucose and lipid metabolism (Alokail et al., 2013; Horvath et al., 2020b; Kobyliak et al., 2016; Mazruei Arani et al., 2019; Sabico et al., 2019; Sabico et al., 2017; Skrypnik et al., 2018; Szulinska et al., 2018a; Szulinska et al., 2018b; Zhang et al., 2016).

Antioxidant properties of probiotics may counteract MNP-induced oxidative stress by preventing lipid peroxidation and activating host antioxidant enzymes (Baralic et al., 2021; Lamprecht et al., 2012; Oumeddour et al., 2019; Tong et al., 2020; Yu et al., 2017), potentially

Table 3
Effects of probiotics onto human health.

Probiotic Effect	Details	References
Microbial diversity restoration	Restores gut and oral microbial diversity and improves gut microbial population.	(Kienesberger et al., 2022; Labenz et al., 2022; Lin et al., 2022a; Stadlbauer et al., 2019)
Enhanced mucus secretion & bile acid metabolism	Stimulates epithelial mucus secretion and modulates bile acid metabolism in mice.	Yu et al. (2024a)
Microbial metabolite & neurotransmitter production	Increases SCFAs, acetylcholine, and essential amino acids production, potentially aiding neuroprotection.	(Alipour Nosrani et al., 2021; Kreuzer et al., 2022; Moser et al., 2019; Wang et al., 2024a)
Hormonal balance	Microbiota act like an endocrine organ producing dopamine, serotonin or other neurotransmitters	Clark and Mach (2016)
Inflammatory cytokine reduction	Lowers levels of inflammatory cytokines, supporting immune health.	Reininghaus et al. (2020)
Enhanced nonspecific cellular immune system	Activation of macrophages, natural killer cells and antigen-specific cytotoxic T-lymphocytes.	Ashraf and Shah (2014)
Immune system boost	Stimulates Immunoglobulin A (IgA)-producing cells and cytokine-producing cells, aiding in immune response.	Ashraf and Shah (2014)
Gut barrier integrity improvement	Reduces intestinal leakage and maintains tight junction proteins, protecting against microbial invasion.	(Haidmayer et al., 2020; Horvath et al., 2020a; Leblhuber et al., 2018; Szulinska et al., 2018b)
Blood-brain barrier strengthening	Reduces neurotoxicity risk by enhancing barrier integrity through microbial metabolite production and neurotransmitter production.	(Loh et al., 2024; Parker et al., 2020)
Reduced inflammatory activation in organs	Limits unnecessary immune activation, preventing tissue damage in liver, spleen, kidneys, and other organs, by reducing secretion of inflammatory molecules.	(Cristofori et al., 2021; Daniel et al., 2021; Niers et al., 2009; Phillippi et al., 2022; Singer et al., 2018; Strauss et al., 2023)
Improved glucose & lipid cycles	Improves insulin sensitivity, hyperglycaemia and lipid metabolism parameters.	(Alokail et al., 2013; Horvath et al., 2020b; Kobyliak et al., 2016; Mazruei Arani et al., 2019; Sabico et al., 2019; Sabico et al., 2017; Skrypnik et al., 2018; Szulinska et al., 2018a; Szulinska et al., 2018b; Zhang et al., 2016)
Antioxidant protection	Protects against oxidative damage by activating host antioxidant enzymes and preventing lipid peroxidation.	(Baralic et al., 2021; Lamprecht et al., 2012; Oumeddour et al., 2019; Tong et al., 2020; Yu et al., 2017)
Detoxification of heavy metals and toxic chemicals	Binds, absorbs or degrades toxic substances (e.g., lead, cadmium, copper, mercury, mycotoxins, benzo[a] pyrene, phthalates, bisphenol A), eliminating or reducing their toxicity.	(Alcantara et al., 2020; Baralic et al., 2020; Baralic et al., 2021; do Nascimento et al., 2019; Jones et al., 1988; Ju et al., 2019; Karazhiyan et al., 2016; Kyrila et al., 2021; Lili et al., 2017; Majlesi et al., 2017; Zhao et al., 2013; Zhao et al., 2020; Zoghi et al., 2014)
Pathogen growth suppression and immune modulation	Inhibits pathogen growth by producing antimicrobials, competing for colonization, reducing adherence to the mucosa, co-aggregating, and modulating the immune system.	(Campana et al., 2017; Müller, 2016)

protecting the reproductive system, where MNPs have been detected in tissues such as the placenta, semen, endometrium and testis (Braun et al., 2021; Liu et al., 2023; Montano et al., 2023; Ragusa et al., 2022; Ragusa et al., 2021; Sun et al., 2024a; Zhao et al., 2023b; Zhu et al., 2023).

Probiotics may also reduce the toxic impact of MNP-bound heavy metals, like lead, cadmium, mercury and copper (Baralic et al., 2021; Lili et al., 2017; Majlesi et al., 2017), mycotoxins (Karazhiyan et al., 2016; Lili et al., 2017), benzo[a]pyrene (Zhao et al., 2013), phthalates (Baralic et al., 2020; Baralic et al., 2021; Zhao et al., 2020) and bisphenol A by binding these toxins through components like mannan oligosaccharides or peptidoglycans, absorbing them through their cell walls (Alcantara et al., 2020; do Nascimento et al., 2019; Jones et al., 1988; Zoghi et al., 2014) or degrading them (Ju et al., 2019; Kyrila et al., 2021). Multi-species probiotics show enhanced efficacy in toxin removal due to their varied mechanisms of binding, absorbing, or degrading noxious substances (Hamad et al., 2018; Hatab et al., 2012; Kasmani and Mehri, 2015).

Another way of tackling MNPs inside the human gut could be plastic accumulation by probiotics via biofilm-production. Some human lactic acid bacteria are able to absorb MNPs from food by aggregating them on their surfaces. The adsorption effects seem to work stronger, the smaller the particle size (Zhao et al., 2023a), possibly diminishing effects of NPs or degradation products of MPs. Extracellular polymeric substances produced by different bacterial strains are able to accumulate MNPs via their adhesive properties (Liu et al., 2021; Sun et al., 2023b). Liu et al. (2021) even genetically engineered a bacterial strain with a trap and release mechanism for MP, proving the applicability of bacterial biofilms for this purpose. Especially bacteria already found in the human gut, with biofilm-forming properties, could prove useful for that purpose. Due to the uncertainty about the maximum size of particles being able to pass the gut epithelium, this path needs to be carefully examined, to avoid the uptake of bigger aggregates.

6. Conclusion

The interactions between MNPs and the human body are multifaceted and potentially harmful. Recent research suggested that bacteria may prove helpful in more than one way to diminish the negative effects of MNPs. It was hypothesized, that bacterial enzymes to degrade MNPs could be utilized to degrade MNPs in the human gut. However, high substrate specificity of the enzymes stands in contrast to the enormous variety of polymers that humans are exposed to. Many factors can affect microbial degradation of plastics, including polymer characteristics, environmental factors and additives or chemical reagents. An additional problem is the long degradation time needed for full mineralization of plastics, being far too long, to be done inside the human gut, even if the conditions would allow optimal enzyme activity and stability. In the time available in the gut, plastic particles would only be degraded partly, leading to smaller particles, with a possibly higher bioavailability and potentially higher toxicity. A recent publication (Zhang et al., 2024c) reviewed health implications of polymer degradation products and highlighted concerns for liver and kidney damage due to accumulation of succinic acid or butanediol, degradation products of polybutylene succinate. PE degradation products may impact people with

allergies, as in animal experiments they led to allergic reactions and issues with the respiratory system. Succinic acid and butanediol derived from PP led to immune responses and nervous system damage in animals, suggesting similar implications for human health. The review also highlights the need for long-term evaluations of these products. Microorganisms might metabolize plastics into less harmful particles, or also produce even more toxic intermediate products. Literature already suggests that degradation products might be as toxic as the polymer itself (Martinez et al., 2024). Microplastic degradation by bacteria will therefore not be able to solve the problem of MNPs in the human gut in the near future.

Understanding the interactions of MNPs with the human body at the cellular level is crucial for decoding the immunotoxic effects of these particles and developing strategies to mitigate their impact on human health. While current research has provided valuable insights into the effects of MNPs on the human gut, the gut microbiota and the immune system, there are still many unknown variables regarding the complexity of polymer particles and their interactions with the human body.

According to our literature review we hypothesize that attempts to modulate the human gut microbiome with probiotics may be an additional possibility to counteract MNP effects indirectly. In this regard, we propose that further studies are necessary to understand if the use of probiotics might reduce the toxicity of these materials in humans. The use of biofilm producing probiotic bacteria could additionally lead to an agglomeration of small plastic particles inside the human gut, increasing their size and preventing them from passing through the gut barrier, eventually leading them towards the natural exit from the human body. This could eventually help to prevent negative health effects from occurring altogether, by encapsulating polymer particles and preventing them from being taken up and also shielding them from interactions with the gut microbiome.

The probiotic list compiled in this review includes bacteria that do not show plastic degrading effects, making them possible candidates for future experiments, without the dangers of possibly producing undesirable degradation products.

CRediT authorship contribution statement

Christian Pacher-Deutsch: Writing – review & editing, Writing – original draft, Visualization, Investigation, Data curation, Conceptualization. **Natascha Schweighofer:** Writing – original draft. **Mark Hanemaaijer:** Writing – original draft, Investigation. **Wioleta Marut:** Writing – original draft. **Kristina Žukauskaitė:** Visualization, Data curation. **Angela Horvath:** Visualization, Funding acquisition, Conceptualization. **Vanessa Stadlbauer:** Writing – review & editing, Funding acquisition, Conceptualization.

Availability of data and materials

All data generated or analysed during this study are included in this published article and its supplementary information files.

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

During the preparation of this work the authors used ChatGPT in order to summarize highlights and improve overall readability. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: N.S. is employee of Institut AllergoSan (Institut AllergoSan manufactures and markets probiotics). M.H. and W.M. are employees of Winclove Probiotics (Winclove Probiotics manufactures and markets probiotics). The content of this study was neither influenced nor constrained by this fact. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Abbreviations:

BBB	blood-brain barrier
DCs	Dendritic cells
MNPs	Micro- and nanoplastics
MPs	Microplastics
NETs	Neutrophil extracellular traps
NPs	Nanoplastics
PCL:	Poly(ε-caprolactone)
PE	Polyethylene
PET	Polyethylene terephthalate
PLA	Poly(lactic acid)
PMMA	Poly(methyl methacrylate)
PP	Polypropylene
PS	Polystyrene
PU	Polyurethane
PVC	Polyvinyl chloride;
ROS	Reactive oxygen species
SCFAs	Short chain fatty acids
TLR	Toll-like receptor

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envpol.2024.125437.

Data availability

Data will be made available on request.

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