Italian Journal of Animal Science

ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/tjas20

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**To cite this article:** Julia Eichinger, Marco Tretola, Jana Seifert & Daniel Brugger (2024) Review: interactions between microplastics and the gastrointestinal microbiome, Italian Journal of Animal Science, 23:1, 1044-1056, DOI: 10.1080/1828051X.2024.2377642

**To link to this article:** https://doi.org/10.1080/1828051X.2024.2377642



Published online: 15 Jul 2024.

ITALIAN JOURNAL OF ANIMAL SCIENCE
2024, VOL. 23, NO. 1, 1044–1056
https://doi.org/10.1080/1828051X.2024.2377642

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REVIEW ARTICLE

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# Review: interactions between microplastics and the gastrointestinal microbiome

Julia Eichinger[a], Marco Tretola[b], Jana Seifert[c,d] and Daniel Brugger[e]

[a]Ruminant Nutrition and Emissions, Agroscope, Posieux, Switzerland; [b]Swine Research Group, Agroscope, Posieux, Switzerland; [c]Institute of Animal Science, University of Hohenheim, Stuttgart, Germany; [d]HoLMiR – Hohenheim Center for Livestok Microbiome Research, University of Hohenheim, Stuttgart, Germany; [e]Institute of Animal Nutrition and Dietetics, Vetsuisse-Faculty, University of Zurich, Zurich, Switzerland

**ABSTRACT**

Plastics are ubiquitous materials in our daily lives, but their inadequate disposal has led to the widespread distribution of their micro- and nanoparticles in various ecosystems. Their detection in feed and food, as well as in livestock and human stool samples, strongly suggests a continuous circulation in the feed and food chain. The ability of plastic particles to penetrate the intestinal barrier determines their accumulation in the body and in food of animal origin. The gastrointestinal (GI) microbiome, whose fermentation activity can influence the particle size distribution of certain plastic materials and which is known to modulate the permeability of the intestinal barrier, may be a critical hub in this transfer. This review attempts to summarise research efforts to date on the interaction between microplastics (MPs) and the GI microbiome of humans, mice, chickens and aquatic animals. We have analysed the state of knowledge and identified future avenues for targeted research approaches to answer open questions regarding the interaction of plastic particles with the GI microbiome, which may help to develop predictive models for the accumulation of plastic particles from feed and food in the body and animal products, respectively.

**HIGHLIGHTS**

- Microplastics (MPs) influence the gastrointestinal (GI) microbiome.
- The microbiome may facilitate microplastic breakdown.
- This may influence the size distribution of plastic particles and their potential to penetrate the intestinal barrier.

**ARTICLE HISTORY**
Received 30 January 2024
Revised 5 June 2024
Accepted 3 July 2024

**KEYWORDS**
Synthetic polymers; microbial fermentation; resistance; plastic breakdown

## 1. Introduction

Plastics are integral materials in modern life, with an approximate annual global production of 390 million tonnes by 2021 (Statista 2023). Unfortunately, only 14% of plastics are collected for recycling (Bachmann et al. 2023), resulting in extensive plastic pollution in both marine and terrestrial ecosystems (Hurley et al. 2020; Thushari and Senevirathna 2020). The primary source of plastic waste, predominantly in the form of macroplastics (>5 mm), undergoes fragmentation through weathering and degradation processes, resulting in the formation of microplastics (MPs; 1 µm–5 mm) and even nanoplastics (NPs; <1 µm) (Horton et al. 2017; Hartmann et al. 2019). These small particles are easily transported through different ecosystems (Da Costa et al. 2019), even through the air (Wang et al. 2023), and are recognised as hazardous substances due to their persistence in the environment, thereby posing environmental and potential health risks (Dong et al. 2023). Most studies on MPs have focused on the marine ecosystem (Li et al. 2018). Due to the complexity of the flow and distribution of plastic particles in the agricultural environment, there are several targets with insufficient knowledge (Zhang et al. 2020). The main sources of plastics in the agricultural system are soil amendments such as compost and sewage sludge, packaging materials, silage and mulch film (Zhang et al. 2020). Several recent reports show the presence of MPs in plant- and animal-based food, as well as in human and animal stool samples (Schwabl et al. 2019; Oliveri Conti et al. 2020; Clere

CONTACT Julia Eichinger julia.eichinger@agroscope.admin.ch; Daniel Brugger dbrugger@nutrivet.uzh.ch

et al. 2022; van der Veen et al. 2022), suggesting their continuous circulation in the food chain. For instance, MPs have been found in raw manure of pigs (Yang, Li, et al. 2020; Yang, Wang, et al. 2020), poultry (Wu et al. 2021) and sheep (Beriot et al. 2021). Furthermore, for the Chinese Mainland it has been estimated that MPs range between 144 and 150 particles/kg of manure fertilisers from pigs, chickens and goats (Zhang et al. 2022). Whether or not plastic particles can penetrate cells is a question of their size, with a recent study finding that 4 μm particles are more effectively taken up by intestinal cells than 1 μm and 10 μm particles, respectively (Barboza et al. 2018). This may be determined by the interaction of plastic particles with the digestive system (Krasucka et al. 2022), including the gastrointestinal (GI) microbiome (Nugrahapraja et al. 2022). On the one hand, the GI microbiome can modulate GI permeability (Kaczmarczyk et al. 2021). In addition, certain microorganisms have been shown to degrade a variety of plastic materials (Mohanan et al. 2020), potentially reducing the size of larger particles. These interactions could affect the amount of plastic particles transferred across the gut barrier, as well as the spectrum and size distribution of particles that accumulate in manure and are redistributed to agricultural land.

This review examines the current state of knowledge on the interaction between MPs and the GI microbiome of humans and animals, with particular emphasis on their potential ability to promote plastic transfer across the gut barrier through particle size reduction and impairment of gut mucosal permeability.

## 2. Interaction between microplastics and the gastrointestinal microbiome

### 2.1. Effects of microplastic on the microbial taxonomy and function

Several studies have shown that MPs can affect both the composition and diversity of microbial communities in the gastrointestinal tract (GIT) of humans, mice, chickens and aquatic animals (Qiao et al. 2019; Tamargo et al. 2022; Yin et al. 2023) (Table 1). To the best of our knowledge, data on terrestrial domesticated animals, except chickens, are not available until now.

#### 2.1.1. Changes in microbial taxonomy

The addition of MPs leads to microbial dysbiosis in humans, mice, chickens and aquatic animals, with Tamargo et al. (2022), Lu et al. (2018), Li et al. (2020), Wan et al. (2019), Yin et al. (2023) and Zhu et al. (2018) reporting an increase in commensal and pathogenic bacteria, such as Chloroflexi, Cyanobacteria, Desulfobacterota (*Bilophila*), Firmicutes (*Bacillaceae*, *Staphylococcus*, *Lactococcus, Phascolarctobacterium, Lachnoclostridium* and *Megasphaera*), Fusobacteria, Melainabacteria, Peptostreptococcaceae and Proteobacteria (*Vibrio, Acinetobacter, Haemophilus, Neisseria, Legionella, Ottowia, Pseudomonas, Polynucleobacter* and *Methyloversatilis*). In contrast, the beneficial bacteria, mainly from Bacteroidetes (*Dysgonomonas, Parabacteroides, Bacteroides, Muribaculum, Clostridiales, Akkermansia* and *Alistipes*), Butyricicoccaceae, Erysipelatoclostridiaceae, Lacetospirillum, Lactobacillus and Actinobacteria (*Bifidobacterium* spp.) decreased. As shown by Lu et al. (2018), this change in bacterial abundance in mice is also strongly dependent on the particle size and MP concentration used. Recent own preliminary data suggests that MP particles of several potential fermentable and non-fermentable plastic species increase the ruminal ratio of Firmicutes:Bacteriodetes and may further promote ruminal Proteobacteria *ex vivo* (Eichinger et al. 2024, 2023) (not shown in Table 1).

Overall, earlier studies showed limitations in their experimental designs, such as the lack of homogeneity of MP particles when applied *via* drinking or swimming water of fishes and their unknown MP intake *via* water, lacking replicates, and evaluating the microbiome data solely based on phylum level and selected number of genera. These analyses neglected the specificities of single species within a genus, such as *E. coli* strains, which can be pathogenic or commensal, respectively. It is therefore, difficult to interpret the overall effects of MP on the GI microbiome based on the available literature.

#### 2.1.2. Host-microbiome-interaction

In addition to the effect of MPs on the composition of the GI microbiome, they may also have adverse effects on the host, particularly on the intestinal epithelial tissues (Table 1). Plastic particles come into contact with various complex matrices, such as feed or food residues, saliva, gastric and intestinal juices before reaching the GI barrier. Possible changes in the particle characteristics could include a decomposition resulting in decreased size and/or shape of particles. Zauner et al. (2001) showed a size-dependent uptake of polystyrene (PS) micro and nanoparticles in different cell lines. In an *in-vitro* study, five different particulate plastic materials, PS, polyethylene (PE), polypropylene (PP), polyethylene terephthalate (PET) and polyvinylchloride

**Table 1.** Effect of different types of microplastics in different doses and particle sizes on the gastrointestinal microbiota as well as on their host[a].

| Host | Microplastic treatment | | | Effects | | |
|---|---|---|---|---|---|---|
| | Type | Dose | Particle size | Microbiota (GI sample type) | Host | Reference[b] |
| Human | PET | 4–5 intakes of 166 mg | 160 μm ± 110 μm | Feces (*in vitro*):<br>Increase: Firmicutes (*Phascolarctobacterium*, *Lachnoclostridium* and *Megasphaera*), Proteobacteria and Desulfobacterota (*Escherichia/Shigella* and *Bilophila*)<br>Decrease: Bacteroidetes (*Bacteroides*, *Parabacteroides* and *Alistipes*) and Actinobacteria (*Bifidobacterium* spp.) | Not tested | Tamargo et al. (2022) |
| Mouse | PS | 1000 μg/L and 100 μg/L | 0.5 and 50 μm | Feces (*in vitro*):<br>All MP groups: Decrease: Firmicutes, α-Proteobacteria and Bacteroidetes<br>50 μm groups:<br>Decrease: Actinobacteria<br>1000 μg/L of 0.5 μm group:<br>Increase: Proteobacteria and Actinobacteria<br>Decrease: Verrucomicrobia<br>1000 μg/L groups:<br>Increase: *Parabacteroides, Prevotella, Dehalobacterium, Ruminococcus, Bilophila, Bifidobacterium, Adlercreutzia, Plesiomonas, Halomonas* and *Acinetobacter*<br>Decrease: *Oscillospira* and *Anaerostipes* | Decreased body, liver and fat weights; decreased serum and hepatic TG and TCH; increased hepatic pyruvate; decreased mucin secretion in the colon | Lu et al. (2018) |
| Mouse | PE | 100 mg/kg/d | 45–53 μm | Small intestine and caecum:<br>Increase: Actinobacteria, *Adlercreutzia, Butyricimonas* and *Parabacteroides* | Not tested | Deng Y. et al. (2020) |
| Mouse | PE | 6, 60 and 600 μg/d | 10–150 μm | Feces (*in vitro*):<br>All MP groups:<br>Increase: Firmicutes (*Staphylococcus*)<br>Decrease: Bacteroidetes (*Parabacteroides*)<br>60 and 600 μg/d group:<br>Decrease: Bacteroidetes (*Bacteroides, Muribaculum, Clostridiales* and *Akkermansia*) | All MP groups:<br>Increased serum interleukin-1α<br>600 μg/d group:<br>Higher TLR4, AP-1 and IRF5 | Li B et al. (2020) |
| Chicken | PS | 1, 10 and 100 mg/L | 5 μm | Caecum content:<br>All MP groups:<br>Increase: Peptostreptococcaceae<br>Decrease: Butyricicoccaceae, Erysipelatoclostridiaceae, Lachnospiraceae and Lactobacillaceae<br>10 mg/L group:<br>Increase: Oscillospiraceae<br>Decrease: Enterobacteriaceae, Eggerthellaceae and Erysipelotrichaceae<br>100 mg/L group:<br>Increase: Actinomycetes, Enterobacteriaceae, Eggerthellaceae, Erysipelotrichaceae and Proteobacteria<br>Decrease: Oscillospiraceae | Inflammation and tissue damage in duodenum and caecum, shorter and less intestinal and caecal crypts, less caecal submucosa, increased intestinal permeability, disruption of hepatic lipid metabolism | Yin et al. (2023) |
| Adult zebrafish | PS | 500 μg/L of water | 5 μm | Feces (*in vitro*):<br>Increase: Fusobacteria<br>Decrease: Proteobacteria | Intestinal villi and epithelial damage, oxidative stress, impaired amino acid and lipid metabolism | Qiao et al. (2019) |

(*continued*)

**Table 1.** Continued.

| Host | Microplastic treatment | | | Effects | | Reference[b] |
|---|---|---|---|---|---|---|
| | Type | Dose | Particle size | Microbiota (GI sample type) | Host | |
| Larval zebrafish | PS | 1000 mg/L of water | 5 and 50 µm | Total larvae:<br>All MP groups:<br>Increase: Firmicutes<br>Decrease: Bacteroidetes, Proteobacteria and *Methylobacterium*<br>5 µm group:<br>Increase: *Methyloversatilis, Polynucleobacter, Legionella* and *Ottowia*<br>Decrease: *Sphaerotilus, Haliangium* and *Leptothrix*<br>50 µm group:<br>Increase: *Flectobacillus* and *Methylophilus* | Changed metabolite profile (energy, glucose and lipid metabolism) | Wan et al. (2019) |
| Soil collembo-lan[c] | PVC | 1 g/kg dry soil | 80–250 µm | Gut:<br>Increase: Firmicutes (*Bacillaceae*)<br>Decrease: Bacteroidetes | Not tested | Zhu D et al. (2018) |
| Crab (Eriocheir sinensis) | PS | 40 mg/L | 5 µm | Gut:<br>Increase: Fusobacteria, Proteobacteria (*Pseudomonas*), Cyanobacteria and Chloroflexi<br>Decrease: Firmicutes and Bacteroidetes (*Dysgonomonas*) | Decrease of alkaline phosphatase, phenoloxidase, lysozyme and acid phosphatase | Liu et al. (2019) |

[a]GIT: gastrointestinal tract; MP: microplastic; PVC: polyvinylchloride; PS: polystyrene; PET: polyethylene terephthalate; PE: polyethylene; TG: triglyceride; TCH: total cholesterol.
[b]All listed studies used Next Generation Sequencing (amplicon sequencing of the *16S rRNA* gene).
[c]Folsomia candida.

(PVC) were subjected to an artificial *in-vitro* digestion. Changes in particle sizes and shapes were investigated. The study demonstrated a high resistance of all plastic particles to the artificial digestive juices and that the main digestive compartments of the human GIT do not decompose the particles *in-vitro* (Stock et al. 2020). This *in-vitro* test, however, does not consider the complexity in physiology of the GI tract and the activity of the intestinal microbiome. To the best of our knowledge, no *in-vivo* study evaluated the effects of digestion on MPs size and shape yet.

Qiao et al. (2019) studied the inflammatory and oxidative stress response in the intestinal epithelial tissues of PS-treated zebrafish (5 µm beads; 500 µg/L of water), leading to gut wall thinning, villi damage and epithelial damage. Inflammation and increased oxidative stress in the GIT have been earlier associated with intestinal microbiota dysbiosis and metabolic disorders (Furukawa et al. 2004; Goyette et al. 2007). Reduced mucus secretion was observed in PS treated mice by Lu et al. (2018) (0.5 or 50 µm PS beads; 100 or 1000 µg/L of drinking water) and Zhai et al. (2023) (5 µm PS beads; 10 mg/L*$d^{-1}$ of drinking water, which is comparable with the percentual minimum daily intake of humans). In addition, Yin et al. (2023) found less caecal submucosa in PS treated chickens (5 µm PS beads; 1, 10 or 100 µg/L of drinking water), which was associated with a reduction in caecal Lactobacteriaceae and Lacetospirillaceae abundance, which produce butyrate and other short-chain fatty acids that increase mucosal mucin production. The depletion of the mucin layer as a direct effect of MP exposure further hinders biofilm formation, as indicated by a reduced abundance of certain intestinal bacteria such as *Akkermansia muciniphila* (Lu et al. 2018). Another problem associated with an impaired mucus layer is the maintenance of the intestinal barrier. Zhai et al. (2023) recognised a parallel reduction of tight junction proteins ZO-1, Occuldin and Claudin3 in PS-treated mice. The resulting reduction in intestinal permeability, which was also observed in the aforementioned study by Yin et al. (2023) in chicken intestine, has been linked to several pathological events, including inflammatory bowel disease, microbial infection and microbial dysbiosis (Schroeder et al. 2018).

Imbalances in the composition of the microbiota, particularly the demonstrated change in the Firmicutes:Bacteroidetes ratio (Zhu et al. 2018; Wan et al. 2019; Li et al. 2020; Eichinger et al. 2023), have been earlier associated with the development of metabolic disorders (Tamargo et al. 2022) and obesity (Turnbaugh et al. 2006). The aforementioned study by

Qiao et al. (2019) showed impaired amino acid and lipid metabolism in intestines of PS-treated zebrafish, Wan et al. (2019) described changes in energy, glucose and lipid metabolism in PS-treated zebrafish larvae (Table 1), Deng et al. (2017) showed a change in lipid metabolism in serum of PS-treated mice (5 or 20 μm; 0.01–0.5 mg MP/d by oral gavage) and Yin et al. (2023) showed hepatic lipid metabolism changes in chicken (Table 1). This may be related to the aforementioned effects on the GIT microbiome and mucus. An increased abundance of *Fusimonas intestini*, a commensal species of *Lachnospiraceae*, and a decrease in *Bifidobacteriacea* were shown in PS treated mice in the study of Zhai et al. (2023). The abundance of *Fusimonas intestini* is positively correlated with obesity by excessive production of long-chain fatty acids (Takeuchi et al. 2023). In contrast, *Bifidobacteriacea* have been shown to be negatively correlated with obesity (Zhai et al. 2023). The studies by Lu et al. (2018) and Yin et al. (2023) suggest a direct link between certain microbial metabolites of the GIT and hepatic lipid metabolism in mice and chickens, respectively. Specifically, Lu et al. (2018) suggest the downregulation of peroxisome proliferator-activated receptor (PPAR) γ, a key transcription factor in lipid metabolism and adipogenesis, and genes involved in triglyceride synthesis, such as Gpat, Dgat1 and Dgat2 in epididymal fat as pathways for the disruption of hepatic lipid metabolism. Yin et al. (2023) found an increase of Proteobacteria in the caecum of PS-treated mice, which are the main source of intestinal endotoxin. These endotoxins contributed to the observed mitochondrial damage, endoplasmic reticulum stress, and hepatocyte apoptosis in this study (Table 1). As a result, lipid metabolism, primarily carried out in the mitochondria, was disrupted by increased lipid synthesis and inhibited β-oxidation of fatty acids, causing lipid deposition in the liver. Li et al. (2020) showed a decreased abundance of *Clostridiales* in mice receiving PE-supplementation (10–150 μm; 60 or 600 μg/d) within their basal feed, which are mainly involved in the production of short-chain fatty acids in the GIT (Ferrario et al. 2014). In addition, members of the *Clostridiales* have been proposed to be involved in the maintenance of the metabolism of fatty acids, sugars and cholesterol through enzymatic bile acid production (Setälä et al. 2014). In the same study, *Parabacteroides* producing the bile acids lithocholic acid (LCA) and ursodeoxycholic acid (UDCA) in the GIT were reduced (Li et al. 2020). These bile acids can reduce hyperlipidaemia by activating glycogen synthesis, fatty acid metabolism and regeneration in the liver, as well as improving GI barrier integrity (Wang et al. 2019; Katafuchi and Makishima 2022). In summary, the data discussed above on the interaction of MPs with lipid metabolism are mostly correlative. An in deep analysis of mode-of-action has yet not occurred, therefore it is not clear how much resembles direct functional interaction or to which degree these associations are the result of autocorrelation or even technical artefacts.

The gut microbiome has been suggested to affect host immunity and generate immune responses through its metabolites (Arnolds and Lozupone 2016; Gu et al. 2020). During the microbial dysbiosis induced by MPs, anti-inflammatory signalling was reduced whereas pro-inflammatory stimuli predominate as a result of the reduced ratio of beneficial:pathogenic bacteria at the mucosal barrier (Furukawa et al. 2004; Goyette et al. 2007; Krishnan et al. 2018). Bifidobacteria have been shown in various studies to have health-promoting properties (Bae et al. 2002) through aromatic amino acid metabolism, producing aromatic lactic acids, indolelactic acid and phenyllactic acid, which have anti-inflammatory and antimicrobial properties (Krishnan et al. 2018). For example, *Bifidobacterium longum* has been shown to have inhibitory effects against several Gram-negative bacteria, such as *Salmonella* and *Escherichia coli*, due to its preventive effect on the mucosal adhesion of Gram-negative pathogens and its ameliorative effect on intestinal tight junctions (Inturri et al. 2016). An improvement of immune functions by Bifidobacteria has been suggested to enhance host production of pro-inflammatory cytokines such as interferon-γ (IFN-γ), interleukin 12 (IL12), and immunoglobulin and natural killer cell activity (Lee et al. 2017). *Bacteroides, Parabacteroides* and *Alistipes*, which were shown to decrease in the human microbiome with PET supplementation, are essential members of a balanced microbiome and are involved in maintaining health by strengthening the epithelial barrier, reducing inflammation by producing anti-inflammatory molecules, such as polysaccharide A and sphingolipids, and producing antimicrobial molecules against exogenous bacteria (Hiippala et al. 2020; Parker et al. 2020). The reduction of *Parabacteroides* in the human and mouse microbiome by the addition of PE and PET has been associated with intestinal inflammation (Li et al. 2020), as seen in patients with ulcerative colitis and irritable bowel syndrome (Noor et al. 2010). The reduction of these taxa by the addition of MPs may impair the maintenance of GI immune homeostasis and barrier function (Hiippala et al. 2020). In addition, MPs, such

as PS particles have been shown to promote the abundance of several pathogenic bacteria belonging to the phyla Firmicutes and Proteobacteria in adult zebrafish, including *Staphylococcus, Aeromonas, Actinobacillus, Vibrio, Acinetobacter, Haemophilus* and *Neisseria* in zebrafish intestinal cells, which are associated with the production of inflammatory cytokines and the induction of immune responses, particularly of phagocytes and lymphocytes (Gu et al. 2020). Gu et al. (2020) showed that M1 macrophages produce pro-inflammatory cytokines and initiate immune responses upon PS addition (Gordon and Martinez 2010). High abundance of *Staphylococcus* was associated with superantigen-induced inflammation and inflammatory bowel disease (Collado et al. 2008) and expression of the pro-inflammatory cytokine IL-1a (Kielian et al. 2004). In addition, Proteobacteria and Desulfobacterota were associated with GI inflammation (Shin et al. 2015), and some members, such as *Escherichia/Shigella* and *Bilophila*, have pro-inflammatory effects, promoting TH1 immunity and colitis in mice (Kamada et al. 2013). Furthermore, several studies indicated immune system dysfunction or damage due to MP. Gu et al. (2020) found that PS inhibited genes related to phagosomes and regulation of immune system processes in M1 macrophages, suggesting immune system dysfunction. Consistently, the downregulation of alkaline phosphatase, phenoloxidase, lysozyme and acid phosphatase in the haemolymph and hepatopancreas of crabs (Eriocheir sinensis) after PS addition (5 μm; 40 mg/L) indicated immune system damage (Liu et al. 2019). In addition, IgA production in B cells was downregulated in the presence of PS (Gu et al. 2020), resulting in reduced antigen-specific defence and impaired GI defence functions (Kubinak et al. 2015; Zhao and Elson 2018). The aforementioned study of Li et al. (2020) showed a decrease in the granulocyte colony-stimulating factor (G-CSF) due to PE supplementation of mice, indicating impaired immunoprotection. It further increased specific cytokines in the presence of 600 μg/d of PE intestinal inflammation through toll-like-receptor (TLR) signalling.

Overall, MPs have been shown to modulate the growth of specific microorganisms and the composition of the GI microbiota in general. This may have consequences for the inflammatory status and the GI lymphoid tissue of the host. Detailed knowledge about the mode-of-action has to be elucidated in future research. In addition, most observations of impaired immune function during MP exposure were not made under additional pathogen-challenged conditions. Therefore, it remains unclear whether MP-treated animals are truly less responsive to pathogenic challenge.

### 2.1.3. Microplastics and resistance mechanisms

The process of physicochemical ageing and depolymerisation of plastics by mechanical abrasion or solar radiation increases their surface area and thereby changes their physicochemical properties, in particular altering the concentration of external contaminants on the plastic surface and promoting microbial adhesion and biofilm formation (Tuvo et al. 2023). The biofilm formed on the surface of different MPs, such as PS (Zhu et al. 2023), PP (Cheng et al. 2022) or PET (Lu et al. 2019) by selected, mostly pathogenic, microbial communities is a factor in the transport of substances, such as heavy metals, persistent organic pollutants or antibiotics between and within environments (Anand et al. 2021; Tuvo et al. 2023). It has been observed, that MPs coated with a biofilm have higher affinities to pollutants than virgin MPs, which may pose more serious consequences. Moreover, biofilms release quorum-sensing signals to attract organisms to ingest MPs, making the entrance of the sorbed pollutants to the food chain easier. More detailed information about the impact of biofilm-developed MPs as vectors of pollutants has been provided by Wang et al. (2021).

These external chemicals on the plastic surface and plastic additives alter the membrane permeability of microorganisms and promote the horizontal gene transfer (HGT) of antibiotic resistance genes (ARGs) (Yuan et al. 2022). In particular, the chemicals released during MP ageing under light irradiation increased the susceptibility of recipient cells to HGT. This occurred through both increased membrane permeability and the activation of genes linked to gene uptake in *E. coli* (Yuan et al. 2022).

Landfills receiving sewage sludge contain antibiotics, toxic organic pollutants, plastic particles and pathogenic microbes that have a high potential for spreading ARGs (Anand et al. 2021). The dispersal of these particles in the soil environment facilitates their migration into the feed and food chain, affecting the GI microbiota of livestock and humans (Zainab et al. 2020; Tuvo et al. 2023). In addition to the transfer of ARGs to the plastic surface in the soil or marine environment, this effect can also occur within the GIT of an organism. Several studies have shown that especially pathogenic species, such as *Escherichia coli*, *Pseudomonas aeruginosa* and *Staphylococcus epidermidis* within the GIT of different organisms are able to adhere and form biofilms on different plastic particles, such as PE, PP or PS present in the GIT (Hoellein et al. 2017; Tamargo et al. 2022).

A recent study has shown that PS (0.05–0.1 μm; 2 ppm in drinking water) can influence antibiotic resistance in the mouse, rather than acting as a carrier (Gao et al. 2023). Specifically, increased sulphonamide resistance was observed, mediated by the alternative dihydropteroate synthase (DHPS) genes sul1, sul2 and sul3, which reduce the affinity for sulphonamides (Changkaew et al. 2014). Furthermore, tetracycline resistance mainly through activation of efflux pumps, production of ribosomal protective proteins and enzymatic degradation has been reported in PS-treated mice (Gao et al. 2023; Grossman 2016).

### 2.2. Effects of microbial fermentation on microplastic particles

Rumen microbes express a diverse set of carbohydrate – active enzymes and cutinases for synergistic degradation of naturally occurring (hemi-) cellulose and cutin in plant biomass (Quartinello et al. 2021). The ability of some microbes to degrade plastics may be related to the structural or chemical similarity of plastics to these biopolymers (Lear et al. 2021). Several authors have already isolated putative plastic-degrading microbes from the GIT such as from aquatic or soil organisms, ruminants and humans (Lear et al. 2021; Yang et al. 2015; Quartinello et al. 2021; Tamargo et al. 2022). Inhibition of plastic degradation after antibiotic treatment of the host has been observed in numerous studies, suggesting that especially the microbiomes and their enzymes within organisms, such as mealworms and larvae of the greater wax moth, are capable of degrading plastic and using it as a carbon or energy source (Yang et al. 2015; Cassone et al. 2020; Yang, Li, et al. 2020; Yang, Wang, et al. et al. 2020). Quartinello et al. (2021) demonstrated the ability of hydrolytic enzymes within the bovine ruminal microbiome to degrade PET, polybutylene adipate terephthalate (PBAT), and PE furanoate (PEF) polyesters *in-vitro* as well as the high polyester hydrolysing activity and the synergistic effects of different esterases, lipases or cutinases within the ruminal fluid compared to published data obtained with pure enzymes. In accordance, Cassone et al. (2020) found that plastic degradation is highly dependent on the synergisms and microbial interactions of different microbial members within the GI community, while individual bacterial species, such as *Acinetobacter* are only capable of degrading plastics at a very slow rate.

After adhesion and biofilm formation, microbial degradation of plastics begins with surface erosion as microbial enzymes are unable to diffuse into the polymer matrix due to their size. The microbial hydrolases, esterases, lipases and cutinases depolymerise the polymer into shorter chains followed by further intracellular metabolism to $CO_2$ and $H_2O$ (Haider et al. 2019). These enzymes have been identified in different ruminal species (Quartinello et al. 2021). *Pseudomonas* spp. and *Acinetobacter* were detected in ruminal fluid samples degrading synthetic polymers (Cassone et al. 2020; Quartinello et al. 2021). But, Quartinello et al. (2021) used ruminal fluid from the slaughterhouse for the study and all subsequent experimental steps were done under aerobic conditions. Thus, the results are not transferable to the rumen microbial activities and are probably artefacts due to the aerobic handling. Despite numerous studies focusing on the effects of MPs on the gut microbial community in monogastric animals, there is a noticeable lack of information regarding the potential influence of microorganisms present in the GIT of monogastric animals on the characteristics of MPs. However, Actinobacteria, a Gram-positive bacterial group with aerobic, facultatively anaerobic or anaerobic metabolism, has emerged as a promising candidate against MPs. This taxonomic group is also found in the GIT of monogastric animals (e.g. *Bifidobacterium sp.*) and possesses the ability to produce a diverse range of hydrolytic enzymes and bioactive compounds, enabling them to thrive on various polymers. Studies have demonstrated that Actinobacteria are among the few microorganisms capable of exhibiting excellent biodegradation capacity towards various types of MPs, including PP, polylactic acid polymer, polyurethane and PE.

Among the eukaryota present in the rumen, *Penicillium* (Yamada-Onodera et al. 2001; Liebminger et al. 2009), *Beauveria* (Almansa et al. 2009), *Acidovorax* (Atanasova et al. 2021) and *Aspergillus* (Sáenz et al. 2019) species have been shown to degrade polyesters and other synthetic polymers in different ecosystems. Finally, also own preliminary data suggest that several plastic species (polylactide [PLA], polyhydroxy butyrate [PHB], high-density PE [HDPE], PVC and PP) can be degraded quite efficiently by ruminal communities *ex vivo* (Eichinger et al. 2023, 2024).

However, microbial degradation of MPs may increase the pathophysiological effects of such contaminants, as discussed in the next section.

## 3. Microplastics accumulation in animal tissues and animal products

The microbial degradation process of MPs may lead to the formation of smaller particles that could penetrate

the GIT epithelium of farm animals and potentially accumulate in various tissues and organs, potentially increasing the risk for pathophysiological effects (Ramachandraiah et al. 2022).

PET and polycarbonate plastic particles larger than 150 μm cannot penetrate an intact epithelium (Zhang et al. 2021), but their appearance in the GIT can lead to local inflammation, as recently shown in mice (Hirt and Body-Malapel 2020).

Various mechanisms contribute to the size-dependent absorption of nano and microparticles. These include (i) endocytosis by enterocytes, (ii) transcytosis through microfold cells or M-cells, which are a specialised subset of intestinal epithelial cells found in gut-associated lymphoid tissue, (iii) persorption, which involves passage through gaps at the tip of villi following the loss of enterocytes, and (iv) paracellular uptake. Peyer's patches, which contain a high proportion of M-cells, are considered as the primary site for MP penetration (Powell et al. 2010). Furthermore, research has shown that macrophages play a role in the intestinal uptake of MP particles *in-vitro* (Stock et al. 2019). After penetration, small plastic particles are transported *via* the lymphatic system (Hussain et al. 2001) or blood to various body tissues, such as organs and all edible muscles (e.g. tenderloin, fine ribs and steak) of livestock (van der Veen et al. 2022) or can potentially accumulate in human tissues after ingestion of contaminated food such as animal products (Leslie et al. 2022).

The accumulation of plastic particles in tissues is highly dependent on particle size (Xu et al. 2021), with plastics of 4–20 μm showing higher accumulation than smaller or larger particles, respectively, which may be due to different penetration pathways through the intestinal epithelial lining and the different abilities of immune cells to capture particles of different sizes (Dong et al. 2023). Similarly, Stock et al. (2019) showed that only plastic particles smaller than 20 μm can penetrate tissue, and Barboza et al. (2018) showed that 4 μm particles are taken up by intestinal cells more effectively than 1 μm and 10 μm particles. The higher uptake of 4 μm particles compared to 1 μm and 10 μm particles have been explained by considering that cells can take up entities between 0.5 and 10 μm by phagocytosis, while particle uptake by pinocytosis and macropinocytosis may happen for particle sizes above 1 μm. Thus, 1 μm particles are likely absorbed by phagocytosis only, while 4 μm particles might be absorbed by phagocytosis and pinocytosis/micropinocytosis (Stock et al. 2019).

Exposure of food-producing animals to such particles poses the risk of accumulation in edible animal products, as demonstrated by the identification of MP in beef (van der Veen et al. 2022). Plastic particles have also been found in raw milk, branded milk (pasteurised or ultra-high temperature processed) from Mexico and milk powder (Kutralam-Muniasamy et al. 2020; Da Costa Filho et al. 2021). The accumulation of MPs in marine organisms has been extensively documented. For example, MPs were extracted from the digestive tracts of 180 specimens representing six different species of fish (both pelagic and demersal) from the northern Adriatic Sea. Among these fish, typically consumed whole without evisceration, an average of 4 particles per individual was found (Mistri et al. 2022). Apart from the GIT, MPs have also been identified in the skin, muscle, gills and liver of commercially important species of demersal and pelagic fish (*Platycephalus indicus*, *Saurida tumbil*, *Sillago sihama*, and *Cynoglossus abbreviateus*) (Abbasi et al. 2018). The presence of MPs behind the gut barrier suggests potential translocation following ingestion or significant involvement of non-ingestive mechanisms such as adherence. The occurrence of MPs in non-digestive organs poses the risk of inducing toxic effects on individuals and serves as an exposure route for humans who consume contaminated fish (Abbasi et al. 2018).

However, studies showing the localisation and distribution of plastics in cows, pigs and chickens and their accumulation in their fresh meat or adipose tissue prior to artificial processing and packaging are still lacking (Dong et al. 2023). Reliable transfer studies on plastic particles in livestock systems are yet hampered by the absence of quality-controlled reproducible approaches for the quantification of such materials in relevant biological matrices.

## 4. Conclusion and outlook

The exposure to MPs may lead to changes in the microbial communities of the GIT, as shown in several studies in different animal species. However, these studies have only scratched the surface by focusing mainly on phyla level and a few genera. Further studies with a sufficient number of replicates and a deeper analysis of metagenomes or activity-based metabolomics are therefore needed to define models for the accumulation of plastic particles from feed and food to human and animal and to improve the interpretability of microbiome changes associated with MPs. This is of great significance, because such changes have the potential to promote inflammatory

conditions and immunological dysfunction in the GIT, as well as the transfer and spread of ARG. In addition, bacteria, archaea and fungi could form a network capable of degrading MPs present in the GIT into smaller plastic particles, facilitating their penetration and accumulation in the animal body and consequently the contamination of animal products.

There is an urgent need for in-depth *in vivo* and *ex vivo* studies in farm animals to map the transfer, effects and behaviour of MPs in and into different matrices, especially in livestock. This will provide important knowledge to understand the uptake, distribution, metabolism, excretion and elimination of plastic particles with respect to factors, such as plastic type, dose, size, shape, additives and co-contaminants. Furthermore, plastic flows in the agro-ecosystem need to be analysed and quantified, including the plastic background in feed and plant tissues in order to determine relevant plastic doses for animal testing. Standardised sampling protocols and reproducible analytical methods for the detection, extraction, separation, identification and quantification of MPs are a crucial prerequisite for carrying out these investigations and need to be developed in the medium term. In conclusion, this review describes the interaction of MPs with the GI microbiome, especially its potential to shape the particle size distribution, and highlights the lack of in-depth studies and analytical methods for MPs in farm animals which merit further investigation due to their importance for human and animal health.

## Acknowledgements

The authors report there are no competing interests to declare.

## Author contributions

All authors contributed equally to this manuscript.

## Disclosure statement

No potential conflict of interest was reported by the author(s).

## ORCID

Julia Eichinger http://orcid.org/0009-0002-2549-0811
Marco Tretola http://orcid.org/0000-0003-3317-4384
Jana Seifert http://orcid.org/0000-0002-7690-8539
Daniel Brugger http://orcid.org/0000-0001-7267-0335

## Data availability statement

Data sharing is not applicable to this article as no new data were created or analysed in this study.

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