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NARRATIVE REVIEWS

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A Comprehensive Narrative Review of Potential Gastrointestinal Adverse Effects From Micro(nano) Plastic Exposure

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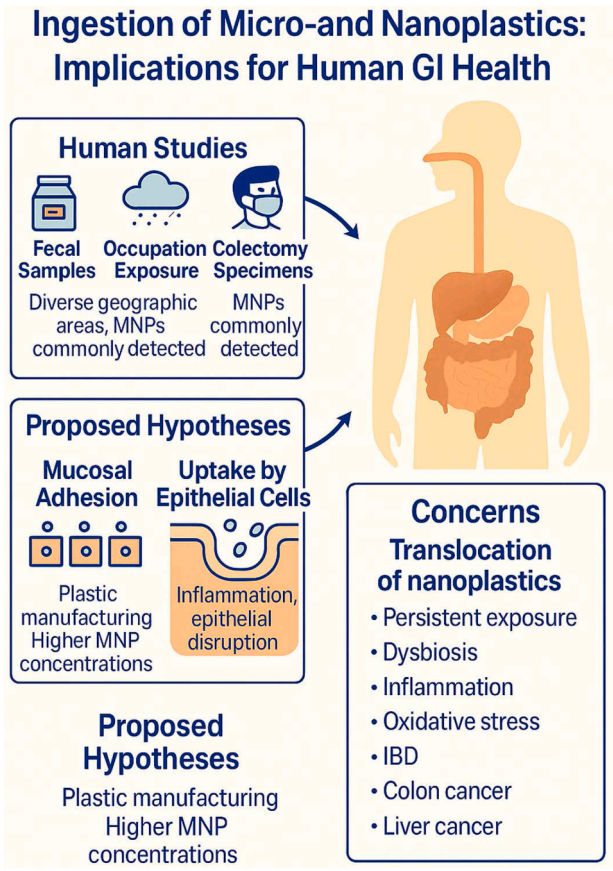

Exposure to plastics is unavoidable part of everyday life due to its ubiquitous use in consumer products, containers, and packaging. Discarded plastics degrade into microplastics (<5 μm) and nanoplastics (< 1μm), which are released into water, soil, and air, thereby reaching most life forms. In humans, these particles are primarily ingested, entering the gastrointestinal (GI) tract, where they interact with gut microbiota, immune tissue, and mucosal barriers, contributing to inflammation and neoplastic effects. To date, exposure to micro- and nanoplastics (MNPs) has been primarily linked to inflammatory bowel disease, metabolic dysfunction–associated steatotic liver disease, and multiple GI cancers. These processes are driven by MNPs induced inflammation, immune

Abbreviations used in this paper: AMR, antimicrobial resistance; BPA, bisphenol A; CRC, colorectal cancer; DPT, disposable plastic tableware; EOCRC, early-onset colorectal cancer; GI, gastrointestinal; IBD, inflammatory bowel disease; MNP, micro- and nanoplastic; MP, microplastic; NP, nanoplastic; PVC, polyvinyl chloride.

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dysregulation, and carcinogenesis. This review covers the existing evidence regarding MNP exposure and GI harm, while also highlighting the need for further research, increased public awareness, and developed of public health strategies to mitigate plastic exposures and health impacts.

Keywords: Microplastics; Nanoplastics; Gut Dysbiosis; Inflammatory Bowel Disease; Metabolic-Associated Steatotic Liver Disease; Systemic Inflammation; Oxidative Stress; Tumorigenic Immunity.

Introduction

Micro- and nanoplastics (MNPs) are synthetic solid polymers with dimensions ranging from 1 to 5 μm and 1 to 1000 nm, respectively (Figure 1).[1] These particles originate from the degradation of larger plastic debris via mechanical, chemical, or biological processes and from the intentional production of microscopic particles, such as microbeads used in personal care products. Contamination of water, soil, and air by MNPs occurs via industrial drainage, wastewater discharge, and atmospheric deposition.[2] Once released, these MNPs become bioavailable to plants via root uptake and translocation to stems and leaves. Humans and wildlife are then exposed through ingestion and inhalation, evidenced by MNP detection in samples of blood, feces, urine, and placenta.[3,4]

Geographic factors influence exposure to MNPs through variations in environmental conditions, human activities, and ecological characteristics. Urban and industrial areas exhibit elevated concentrations due to waste production and poor waste management. Industrial effluents and urban runoff introduce plastics into aquatic systems.[5] Coastal regions are susceptible to MNP pollution from maritime transport, fishing, and tourism. Oceanic plastics degrade into smaller particles, impacting marine ecosystems.[5,6] Additionally, MNPs accumulate in soils through plastic-containing fertilizers and atmospheric deposition, adversely affecting soil health.[6]

Emerging research indicates that chronic exposure to MNPs may contribute to systemic inflammation, oxidative stress, and altered gut microbiota, all of which are implicated in disease such as inflammatory bowel disease (IBD), liver disease, and possibly cancer. While the particles themselves are not currently classified as human carcinogens, they can adsorb and transport toxic compounds such as heavy metals and pollutants, which are known to increase cancer risk.[7] The relationship between MNP exposure and human health outcomes is complex and not yet fully understood. As global plastic production and environmental contamination continue to rise, understanding the biological effects of MNPs is crucial for guiding public health interventions and regulatory measures. A recent report has highlighted some of the potential GI effects of MNP exposure.[8] This comprehensive analysis highlights the urgent need to address MNP pollution and mitigate its effects, particularly in vulnerable populations disproportionately affected by the environmental plastic burden.

Global Burden of MNPs

Microplastics (MPs), defined as plastic particles smaller than 5 mm, are persistent pollutants affecting ecosystems and human health.[9] Research into their presence and impact has grown substantially. MPs originate from degrading plastic wastes, industrial processes, and everyday products.[9,10] They exist in aquatic systems, soils, atmosphere, and human food chains through ingestion and inhalation.[11–13] The Mediterranean Sea is a MP pollution hotspot due to surrounding countries' waste activities.[14] Marine animals' ingestion of MPs is concerning, as these particles carry harmful pollutants.[14] While research focuses on aqueous systems, studies of MPs in soils remain limited.[15] Airborne MPs pose health risks through inhalation.[13] MPs transport toxic chemicals and are absorbed into organisms, affecting physiological processes.[16] Studies suggest correlations between MPs and human gut microbiome changes.[11] Current efforts include regulatory measures and bioengineered remediation strategies. The pervasive nature of MPs requires continued research and management policies globally.

Presence of MNPs in Human Gut: Evidence From Fecal and Tissue Analysis

MNPs have become ubiquitous in the environment, with growing evidence suggesting adverse impacts on human health. After consumption, the GI tract is the primary location where MNPs are deposited; however, the extent and nature of the distribution of MNPs remains an area of ongoing investigation, despite being a highly alarming issue. Upon ingestion, MNPs interact with the gut microbiome, immune system, and intestinal tissues, contributing to dysbiosis, inflammation, and possibly carcinogenesis. As a result, public health is increasingly at risk as a matter of concern over MNP exposure through profiling anthropogenic food chains.

Detecting MPs and in particular, nanoplastics (NPs) in tissues and body fluids samples poses analytical challenges, as these are not seen on standard histologic imaging. The visualization of these microscopic particles is limited to specialized equipment and analysis used in laboratory settings by researchers. Recognizably, however, there are variables for accuracy, in particular due to lack of standardized protocols, extensive sample preparation, and small particle size detection. Please see Table 1[17–19] for the detection and identification of MPs

Figure 1. Size range of MNPs compared with other structures.

in environmental samples. Research has demonstrated the presence of MPs in various tissues, including the liver, blood, heart, placenta, breast milk, meconium, sputum, urine, and semen.[20,21] Unfortunately, at present time, there is no commercial test proven to be accurate particularly for MNPs in tissue or fluids.

Several human studies have verified the presence of MNPs in feces, lending support to the concept that the GI tract is a critical location of MNPs in the human body. This theory is supported by a prospective case series with 8 healthy Japanese volunteers and discovered MPs in all fecal samples; their findings point to a large presence of MNPs in the human GI tract as a result of dietary intake, with packaged seafood and beverages contributing the exposure.[22] Similarly, a cross-sectional study in China analyzing fecal samples among 26 healthy adults identified high concentrations of polypropylene particles, which are common packaging materials, confirming ingestion as the likely source.[23] The study also found correlations between MNP content and consumption of bottled water, suggesting that plastic packaging of the bottled water, easily accessible to everyone, may be a key vector for MNP ingestion.[23] These findings are complemented by analysis of nasal and fecal sampling in individuals with long-term occupational exposure in a plastic manufacturing environment.[24] Participants exhibited higher concentrations of MNPs compared with control subjects. Importantly, this study indicates that occupational settings may further amplify MNP burden in the human gut through combined inhalation and ingestion routes.

A more comprehensive study analyzing fecal samples from 50 healthy individuals and 52 patients with IBD found MNPs in both groups, but there was a significant elevation in the IBD group, supporting the theory that MNPs alter the activity of the gut and intestine.[25] The results demonstrate a positive link between the concentration of fecal MNPs and the intensity of IBD. This association implies that exposure to MNPs might be connected to the disease's progression, or that IBD could intensify the retention of MNPs. Further research is needed to explore the underlying mechanisms. Further expanding the understanding of MNP gut distribution beyond fecal analysis, examination of colectomy specimens using spectroscopic techniques detected a variety of MP particles embedded in intestinal tissue.[26] This evidence suggests MNP penetration into tissue, rather than just in the tract lumen, is a potential pathogenic driver disrupting intestinal homeostasis.

While these studies do not experimentally define mechanisms, consistent findings of MNPs in feces and histologic samples support mechanisms for MNP entrance into human tissues through mucosal adhesion, paracellular uptake, and endocytosis by intestinal epithelial cells. Principal pathways of entry include mucosal adhesion, paracellular uptake, endocytosis by

Table 1. Key Methods of Detection and Identification of Microplastics in Environmental Samples

Different Methods	
Infrared spectroscopy	Micro-Fourier transform infrared spectroscopy identifies polymer types of microplastics down to 20 μm and differentiates synthetic from natural particles.
Raman spectroscopy	Micro-Raman spectroscopy identifies microplastic types and provides detailed information on polymer chemical structure.
Microscopy	Stereo microscopy identifies and quantifies microplastics through visual inspection, though less effective for very small particles.
Fluorescence technique	Frequency-domain fluorescence lifetime imaging microscopy distinguishes plastics from natural materials using different excitation wavelengths in terrestrial samples.
Mass spectrometry	Mass spectrometry with thermal analysis or gas chromatography identifies polymer components through mass spectra analysis.
Spectroscopic and microscopic analysis	Combining spectroscopic methods improves identification and quantification.

intestinal cells, and inhalation or ingestion through food and water.[20,27,28] MNPs adhere to mucous membranes and enter cells via endocytosis or traverse between cells through paracellular uptake. The size of NPs influences gut barrier permeability, with smaller particles (approximately 50 nm) penetrating the intestinal mucus barrier more effectively than larger ones.[29,30] Interestingly, a recent brief communication on MNPs identified MNPs in all examined organs, with the brain showing higher concentrations than the liver and kidney.[31] The presence of MNPs in brain tissues indicates their ability to cross the blood-brain barrier, although the mechanism of delivery to the brain remains unknown. However, the study suggests that processes similar to clathrin-dependent endocytosis and micropinocytosis facilitation of MNP translocation within the intestine may be involved, as seen in *Daphnia magna* (a type of water flea, valuable model organism used in scientific research, particularly in ecotoxicology and aquatic ecosystem dynamics).[32] The researchers hypothesize that lipid uptake might enable NP transfer into the brain. This trend raises questions about these particles' environmental presence and health impact.[31] All these studies lead to one commonality, which is that MNP exposure is primarily from environmental exposure, including food/beverage packaging, preparation and storage, workplace plastics contamination, and regional plastic pollution.

Effects of MNPs on Gut Microbiota

The gut microbiota exerts significant influence across various animal species. Research indicates that exposure to MNPs diminishes microbiota diversity, alters the Firmicutes:Bacteroidetes ratio, and disrupts bacterial equilibrium.[33] Studies have reported that MNPs, particularly polystyrene, promote biofilm formation, thereby enhancing antimicrobial resistance (AMR). MNPs may favor bacteria with superior biofilm-forming capabilities, resulting in increased resistence.[34] The research team led by Yang et al[35] demonstrated that MNPs serve as surfaces for bacterial growth and influence gene expression to facilitate the spread of AMR, with significant implications for countries with inadequate waste management. This research underscores the importance of plastic management in controlling AMR. While animal studies have shown adverse effects, human studies have not demonstrated significant changes in microbial composition due to MNPs; however, specific MNPs have been associated with the genera *Roseburia*, *Clostridium*, and *Prevotella*.[11] The interaction between MNPs and bacterial taxa is complex and influenced by various environmental factors.

The physical properties of MNPs affect gut microbiota composition and health risks. Size, shape, and surface properties influence interactions with gut microorganisms. Small MNPs may penetrate the gut epithelium, causing oxidative stress and disrupting intestinal homeostasis,[36] while larger particles affect intestinal walls and surface microbial communities, leading to adsorption of toxic substances and heavy metals through physical abrasion and leaching of adsorbed substances.[37] Genes for plastic-degrading enzymes in the human gut microbiome suggest adaptation to MP contamination.[11] Different animal species show varying susceptibilities to MP toxicity. Mice showed heightened susceptibility, experiencing decreased gut microbiota diversity, destabilized microbial networks, and disrupted beneficial-harmful bacteria balance.[33] This suggests MNPs interact differently with gut environments across species, likely due to varying physiology and microbial composition. Duration of exposure has been identified as key role in MP-induced gut microbiota changes, emphasizing the importance of both physical properties and exposure duration in health risk assessment.[33] Studies have shown that MP exposure leads to gut microbiota dysbiosis, tissue inflammation, and plasma lipid metabolism disorders.[38] The interaction between gut microbiota and immune cells can lead to the formation of inflammasomes, protein complexes that play a crucial role in the inflammatory response. Inflammasomes regulate the release of proinflammatory cytokines such as interleukin-1β and interleukin-18, which are critical in maintaining immune balance and can contribute to chronic inflammatory diseases.[39]

Emerging evidence highlights the significant impact of MNPs on the gut-brain axis, a complex bidirectional communication network, which is heavily influenced by the intestinal microbiota.[40] This is particularly concerning given the established role of the gut-brain axis in neurodegeneration.[40] The ability of to disrupt the gut microbiota composition may have far-reaching consequences, as the microbiota plays a crucial role in producing and modulating neurotransmitters such as GABA, serotonin, and dopamine.[41] The ingestion of MNPs through contaminated food and water sources may be contributing to neurological disorders by disrupting the delicate balance of the gut-brain axis. Understanding the effects of MNPs on the gut-brain axis is crucial for informing future research and policy changes aimed at mitigating adverse consequences on neurological health.[42]

MNPs in Relation to IBD

IBD, which includes Crohn's disease and ulcerative colitis, is a chronic inflammatory disease of the GI tract. Environmental factors, such as dietary allergens, additives, contaminants, and, more recently, MNPs are increasingly being linked to its pathogenesis.[43,44] Systemic plastic-related exposures are associated with shifts in immunological markers, including cytokines and metabolic byproducts.[43] Emerging data indicate that MNPs may damage gut barrier integrity, modify

immunological responses, and alter gut microbiota, hence possibly causing or aggravating IBD.[43,45,46] High-throughput sequencing has identified gut microbiota changes in IBD patients, showing decreased beneficial bacteria like Firmicutes and increased harmful bacteria from Proteobacteria phylum.[47] This microbial imbalance worsens intestinal inflammation, affecting the immune system and metabolic pathways, leading to opportunistic pathogen colonization that furthers IBD progression.[48] Treatments targeting gut microbiota, including probiotics, prebiotics, and fecal microbiota transplantation, show promise in restoring microbial balance and improving outcomes by increasing diversity and beneficial bacteria.[49]

Evidence supporting a pathogenetic role of MNPs in the development of IBD comes from a quasi-experimental investigation examining the effects of thermal exposure to disposable plastic tableware (DPT) on the gut microbiota of 60 healthy subjects.[45] Results showed DPT exposure increased the amount of MNPs in feces and dramatically affected the composition of gut microbiomes, likely through the release of monomers and additives such as bisphenol A (BPA), phthalates, and flame retardants.[24] These changes were characterized by an increase in pro-inflammatory bacteria and a decrease in beneficial strains, implying that DPT-derived MNPs may disrupt microbial balance and contribute to gut inflammation, which is a known catalyst for IBD aggravation. Fecal analysis comparing IBD patients to healthy individuals revealed a 49% higher concentration of MNPs among IBD patients.[25] Importantly, there was a positive correlation between fecal MNPs content and IBD severity, Analysis of the MNPs subtypes showed predominantly polyethylene terephthalate and polyamide, which originate from food packaging and environmental dust. Furthermore, among patients with Crohn's disease, serum samples from 200 patients were evaluated for endocrine-disrupting chemicals, including BPA; patients with colonic Crohn's disease had higher BPA levels than those with ileal involvement, indicating a region-specific impact of plastic-related chemicals.[43] There were also significant alterations in butyrate levels, a short-chain fatty acid known for its role in influencing gut microbial activity related to mucosal healing and inflammation control. This decrease could potentially affect the integrity of the intestinal barrier and immune regulation, as butyrate plays a crucial role in sustaining these functions.[43]

Impact of MNPs on Liver Health

The liver is a primary site where MNPs accumulate. As a result, recent studies have raised concerns about hepatotoxicity due to MNP accumulation. MNPs can lead to oxidative stress, causing liver damage through energy redistribution, cell death, and autophagy in hepatocytes.[50,51] MNPs stimulate immune responses and inflammation, contributing to liver diseases such as metabolic dysfunction–associated steatotic liver disease, fibrosis, and cirrhosis. They also disrupt the microbiome and gut-liver axis, potentially leading to metabolic dysfunction–associated steatotic liver disease.[50,52]

In addition to the previous mentioned role for ingested and inhaled sources of MNPs, analysis of postmortem tissues from patients with hip or knee replacements and found metallic and polyethylene wear particles in the liver, spleen, and abdominal lymph nodes.[53] The prevalence was higher in patients with failed implants, suggesting systemic dissemination of prosthetic debris. Interestingly, a recent study found MNPs in liver tissues of patients with cirrhosis but not in individuals without liver disease.[54] These findings raise questions about whether MNP accumulation contributes to liver pathology or results from existing hepatic conditions.

Looking more closely at the mechanisms of hepatotoxicity, MNPs can disrupt mitochondrial function in adipose tissue, leading to oxidative stress and inflammation.[54] These processes, termed *oxinflammation*, may contribute to the progression of metabolic dysfunction–associated steatotic liver disease by altering mitochondrial bioenergetics and dynamics; subsequent impacts include impaired energy homeostasis, promotion of insulin resistance, and increased lipid accumulation in the liver.[54] Animal studies have shown that MNPs cause biochemical and structural liver damage, including dysfunctions of the excretory and reproductive systems. In fish, MPs cause oxidative damage to liver and reproductive tissues,[55] causing structural liver damage and disrupting metabolic balance.[52] MPs accumulate in reproductive organs, leading to abnormal testicular and ovarian tissues and reduced sperm vitality in mammals.[56] The extent of damage depends on particle size, concentration, type, and exposure duration.[52,57] The ingestion of MNPs through food and water, particularly via poultry and marine products, poses additional environmental toxicity risks with eventual health impacts on humans suggested through various modeling approaches.[58]

In summary, studies suggest significant negative effects of MNPs on liver health, impacting both structure and metabolic functions. Despite these findings, there remains a need for clearer and more extensive studies to better characterize the long-term health consequences across different animal models and extrapolate the potential risks to human health effectively.

Carcinogenic Potential of Specific MNPs

One of the most significant health concerns associated with MNPs is the potential link to cancers, particularly GI cancer. Studies show that MNPs can penetrate cells, disrupt biological processes, and create carcinogenic conditions.[59] The primary exposure occurs

through ingestion of MNPs in contaminated food and water, accumulating in the GI tract. MNPs can alter gut microbiome composition and promote biofilm formation.[60] Biofilms enhance microbial survival through antibiotic resistance and immune evasion, leading to chronic inflammation and cancer risk.[61] Dysbiosis, an imbalance in gut microbiota, leads to increased intestinal permeability ("leaky gut"), allowing microbial molecules to enter the bloodstream and cause inflammation. These inflammatory conditions create an environment conducive to cancerous growths[62–64] Chronic inflammation is a known cancer risk factor, leading to DNA damage, tumor growth, and cancer cell invasiveness.[65,66] Additionally, the gut microbiome metabolizes dietary components into various metabolites that can protect from or promote carcinogenesis. Such a balance can be disrupted by these metabolites, increasing carcinogenic risk.[67] A key mechanism for carcinogenesis is disruption of immune surveillance in which the immune system identifies and destroying cancerous cells, facilitating cancer progression through modulation of local and systemic immune responses.[68] Additionally, MNPs can serve as vectors for various contaminants, including endocrine-disrupting chemicals, which have been linked to cancer and other health issues.[69]

Emerging experimental data and animal models have started to explore the carcinogenic potential of MNPs in the GI tract with a particular concern relating to colorectal cancer (CRC). MNPs, especially polystyrene particles, have been detected in CRC tissues using laser infrared chemical imaging.[70] These particles were shown to promote tumor progression and resistance to chemotherapy by activating autophagy via the mTOR/ULK1 axis, which is a pathway known to support tumor survival under stress conditions. In vivo models, further support this connection, where mice exposed to MNPs exhibited increased intestinal inflammation and dysbiosis the ingestion of MNPs disrupted gut homeostasis, altered microbial composition, and elevated proinflammatory cytokines, all of which are known contributors to tumorigenesis.[71] These studies have emphasized MNPs' ability to act as carriers for carcinogens like polycyclic aromatic hydrocarbons and heavy metals, which may compound their tumorigenic effects. It is increasingly apparent, that MNPs are not inert contaminants, but rather are biologically active agents capable of influencing cancer development through inflammation, oxidative stress, and immune modulation.[21]

A potential role of MNPs has been suggested as an environmental factor contributing to rising early-onset colorectal cancer (EOCRC) in individuals under 50 years of age. Despite decreased overall colorectal cancer incidence due to improved screening and lifestyle changes, EOCRC has increased worldwide over 2 decades.[72,73] Epidemiological data imply an environmental cause, with a cohort effect since 1960 corresponding to widespread plastic use and pollution. MNPs are ingested by humans from early age and accumulate over time. As they traverse the GI tract, MNPs may interact with colonic mucus layer, reducing its protective function and potentially increasing colorectal cancer risk.[74] While genetic predisposition poses risk, most cases are sporadic, likely driven by environmental exposures like MNPs.[75,76] The impact of MNPs may relate to microbiome changes or chronic inflammation in colorectal cancer pathway.[77] Chronic MNP exposure may reduce beneficial short-chain fatty acids through microbiota alteration, weakening mucosal defenses.[78] The timing of increased MNP pollution coincides with rising EOCRC trends, suggesting a possible correlation requiring investigation.

Certain types of MNPs, particularly polyvinyl chloride (PVC), have been associated with increased liver cancer risk, including angiosarcomas and hepatocellular carcinomas.[78] A study of 5,498 male UK vinyl chloride workers from 1940 to 1974 showed excess primary liver tumors.[79] Eleven deaths occurred from non-secondary liver tumors, with 7 from angiosarcomas. Angiosarcoma deaths were confined to autoclave workers, showing a 25-year median latency from initial exposure,[79] supporting liver toxicity is most robust in PVC exposure. A recent literature review of 34 published studies addresses worker exposures to MNPs across 6 chemical types, with half involving PVC exposure.[78] The review shows that PVC exposures are linked to liver toxicity and increased risk of liver cancers, including angiosarcomas and hepatocellular carcinomas.[78] This evidence demonstrated that PVC-related liver cancers had extended latency periods up to 56 years, affecting health surveillance of exposed populations.[78] Please see Table 2[23,24,26,46,54,78–88] for a summary of the previous study findings.

In conclusion, the presence of MNPs in human tissues, the capacity to disrupt cellular processes, and impact on the gut microbiome all suggest a possible role in cancer development. Further research is essential to fully comprehend the long-term health consequences of this increasingly prevalent exposure and to develop effective strategies for mitigating these risks[59,60,69]

Minimization Strategies

The widespread occurrence of MNPs in ecosystems and human tissues presents health concerns, affecting gut health through ingestion. Public health interventions and regulatory reforms are needed. Annually, 400 million tons of plastic are produced globally, with 14% entering aquatic ecosystems as MNPs.[89] MNPs have been detected in both plastic and glass bottles, with bottle caps identified as the main contamination source.[90] Policies promoting biodegradable alternatives and banning single-use plastics can reduce MNPs. The EU's 2021 ban on oxo-degradable plastics reduced MP leakage by 500,000 tons per year.[89] The use of HEPA

Table 2. Summary of Studies Investigating the Potential GI and Hepatic Effects of MNPs

Authors	Study Design	Specimen	Size Classifications (MPs vs NPs)	Type of Exposure	Main Study Findings
Ibrahim et al[1]	Cohort study	Colectomy specimen	MP	Ingestion and inhalation	MPs are ubiquitously present in the human colon.
Zhang et al[2]	Cross-sectional	Nasal, stool, and environmental	MP	NR	Alters the gut microbiota by increasing the abundance of intestinal microbiota, which is positively associated with digestive tract diseases.
Zhang et al [3]	Cross-sectional	Fecal samples	MP	Ingestion	An abundance of MP in feces.
Urban et al[4]	Cross-sectional	Lymph nodes	Both	NR	Dissemination of polyethylene particles in the liver, spleen, and abdominal lymph nodes of patients undergoing hip or knee arthroplasty.
Xue Zhang et al[5]	Quasi-experimental	Fecal samples	MP	Ingestion	Alters gut microbiota and raises microplastic levels, impacting health.
Jun Xhao et al[6]	Cross-sectional	Surgical Excisions of Tumors	MP	NR	MPs were found in various tumor tissues, notably pancreatic cancer, in which they may worsen the tumor immune microenvironment.
Jones RD et al[7]	Cohort study	NR	Both	NR	A significant increase in nonsecondary liver tumors, with 11 deaths, of which 7 were angiosarcomas.
Liss et al[8]	Cohort study	Liver biopsy	Both	NR	15 patients were found to have chemical liver injury, 27 patients had nonchemical liver disease, and 25 patients were normal.
Du C et al[9]	Cohort study	Liver biopsy/imaging	Both	NR	A significantly increased risk of primary liver cancer, liver cirrhosis, and other chronic diseases.
Ward et al[10]	Cohort study	Imaging and liver biopsy	Both	NR	53 deaths from primary liver cancer and 18 incident cases of liver cancer were identified, including 37 angiosarcomas, 10 hepatocellular carcinomas, and 24 liver cancers of other and unknown histology.
Hsiao et al[11]	Cohort study	Imaging	Both	NR	Significantly increased risks of developing liver fibrosis were found in workers who had a history of high exposure when compared with workers who did not have history of high-exposure jobs.
Maroni et al[12]	Cross-sectional	Imaging and LFTs	Both	NR	Periportal fibrosis.
Attarchi et al[13]	Cross-sectional	LFTs	Both	NR	Liver effects were reported by increased levels of ALP and GGT

Table 2. Continued

Authors	Study Design	Specimen	Size Classifications (MPs vs NPs)	Type of Exposure	Main Study Findings
Hsieh et al[14]	Cohort study	Imaging	Both	NR	A dose-response trend between plastic (MP/NP) exposure and liver fibrosis.
Gennaro et al[15]	Cohort study	NR	Both	NR	Increased risk of liver tumors and cirrhosis.

ALP, alkaline phosphatase; GGT, gamma-glutamyltransferase; LFT, liver function test; MP, microplastic; NP, nanoplastic; NR, not reported.

Table 3. Public health and Dietary Strategies to Minimize MNP Use

Problem	Intervention/Strategies
Public health interventions	
Reduction in plastic usage	- Promote biodegradable alternatives - Ban single-use plastics
Advanced water filtration	- Use ultrafiltration/reverse osmosis/activated carbon filters - Boil + filter tap water
Wastewater management	- Upgrade to tertiary treatments like membrane bioreactors and dissolved air flotation
Public education	- Awareness programs on microplastic sources and effects - Promote reusable alternatives
Dietary strategies	
Avoid/minimize - Processed foods - Beverages/foods in plastic	- Choose fresh, whole foods - Use nonplastic containers - Heating/freezing/storing/preparation food or beverages
Organic and hydroponic produce	- Prioritize organic farming - Choose hydroponic-grown crops
Gut barrier support	- Probiotics and prebiotics - Omega-3s (eg, flaxseed) - Zinc-rich foods - High fiber diet

filters and wet mopping can diminish airborne MP exposure.[91] Public education campaigns, conducted through schools and healthcare facilities, aim to increase awareness about MP pollution, focusing on sources, health effects, and exposure reduction (Table 3). To reduce MNP consumption, avoid plastic-bottled drinks, use glass/ceramic containers,[92] choose sustainable seafood, and minimize plastic packaging. Research on MNP removal technology at present is in the preliminary stages, with many limitations such as type and size of target particles.[93] Approaches currently include physical methods (filtration), chemical approaches (eg, coagulation/flocculation), and biological processes (eg, biodegradation and bioabsorption).[94] Probiotics, omega-3, and zinc-rich foods may protect against MNP toxicity,[63] while fibers offer protection.[95] Ulusoy et al[96] compared plastic-free diet, diet education, and BPA exposure feedback effects on adolescents' BPA levels. Subjects with high plastic product usage were assigned to intervention groups (n = 108). Post-tests followed a plastic-free diet in intervention 1, diet education and BPA feedback in interventions 1 and 2, and BPA feedback in intervention 3. All interventions reduced BPA levels and improved eating attitudes.[96]

Regulatory measures are vital in addressing MNP pollution. The EU's 2022 Nanomaterials Regulation mandates labeling for particles under 100 nm. Bans on microbeads in personal care products have reduced MP release. Following the EU's 2021 E171 ban, France reported a 25% decrease in pediatric inflammatory bowel disease cases linked to TiO_2.[97] Managing MNPs necessitates implementing the 4Rs (reduce, reuse, recycle, recover), promoting a circular economy, and developing sustainable alternatives.[98]

At present, there are no proven methods or approaches for removing MNPs from tissues. Clearly, more information and research is needed. Where there are luminal particles evident, accelerating excretion or

favorable effects on GI microbiome (eg, high-fiber diet) of dietary fibers in protecting health.[95] Recently, preliminary study found that extracorporeal apheresis could effectively eliminate MNP-like particles from the bloodstream.[99] The study examined 21 individuals with myalgic encephalomyelitis/chronic fatigue syndrome using double filtration apheresis. Analysis identified 14 substances including polyamide 6 and polyurethane. This method offers potential for reducing MNP-related health risks, though further validation is needed. Notably, dialysis does not remove MNPs and the repeated usage of plastic tubing used, increases exposure to these patients.[100]

Study Limitations

During our review, we identified several limitations: (1) **inconsistency in detection methods**, as current analytical techniques for identifying MNPs show variability in sensitivity and reliability, challenging result comparison across studies; (2) **a lack of longitudinal human studies**, as most research focuses on short-term findings, limiting understanding of chronic MNP exposure effects; (3) **limited human-specific data**, as most MNP exposure literature relies on animal models or in vitro experiments, limiting result generalizability; and (4) **multiple exposure sources**, as humans are exposed to MNPs through food, water, air, and consumer products. Due to their ultra-small size and chemical diversity, NPs often evade detection, potentially underestimating exposure levels.

Conclusion and Future Directions

MNPs are pervasive contaminants in human environments, detected in biological matrices including feces, blood, placenta, and lung tissue. Their impact on the GI system is concerning, as MNPs disrupt the gut microbiome, compromise epithelial barriers, and induce oxidative stress and inflammation. These effects are linked to IBD, liver disease, and GI cancer. However, knowledge remains limited due to research gaps, lack of epidemiological studies, and inadequate standardized detection methods. While evidence suggests MNPs' link to GI disturbances, definitive conclusions require more research. Addressing these gaps requires investment in interdisciplinary human studies to quantify exposure and trace health outcomes. Advanced technologies, improved protocols, and harmonized monitoring systems could enhance research reliability. As plastic production increases coordinated efforts are essential to eliminate risks to human and planetary health. This issue demands immediate action through reducing plastic production, enhancing waste management, and upgrading filtration infrastructure. Public education is needed to minimize MNP intake. Both clinicians and patients need to me aware of this ubiquitous threat to GI health that we are only beginning to understand.

Supplementary Material

Note: To access the supplementary material accompanying this article, visit the online version of *Clinical Gastroenterology and Hepatology* at www.cghjournal.org, and at https://doi.org/10.1016/j.cgh.2025.11.002.

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Conflicts of interest
The authors disclose conflicts.