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Review Article

Micro/Nanoplastics and Cancer: Focus on Gastrointestinal Malignancies — A Narrative Review

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Abstract

Micro- and nanoplastics (MNPs) are plastic particles smaller than 5 mm and 1 μm, respectively, and are emerging environmental pollutants with growing implications for human health. These particles stem from either 'primary sources', such as intentionally manufactured microbeads and industrial abrasives, or 'secondary sources', where larger plastic items break down into smaller fragments over time. Human exposure primarily occurs through ingestion and inhalation, with contaminated seafood and plastic-laden food packaging representing key routes of entry. Once ingested, MNPs can cross the intestinal barrier, accumulate in gastrointestinal (GI) tissues, and trigger biological responses. Mechanistic studies reveal that MNPs induce oxidative stress, DNA damage, chronic inflammation, and endocrine disruption, all of which are hallmarks of carcinogenic pathways. They also alter gut microbiota, potentially promoting dysbiosis and immune dysregulation. The GI tract is particularly vulnerable to these effects due to direct luminal mucosal contact and high epithelial turnover. Epidemiological data remain limited, but early evidence supports a plausible link between MNPs exposure and GI malignancies. Such findings are particularly concerning given the increasing global incidence and early age presentation of colorectal and esophageal cancers. Given that MNPs may represent a modifiable environmental risk factor in GI cancer prevention, public health strategies must prioritize reducing plastic exposure, promoting antioxidant-rich diets, and improving environmental monitoring. This review explores the potential carcinogenic effects of microplastics while also examining their emerging roles in cancer therapeutics. It highlights critical avenues for future investigation and underscores the importance of cross-disciplinary efforts to tackle this growing global health concern.

Introduction

Recent research has highlighted plastics as a rapidly escalating threat to both human and planetary health, with consequences spanning from infancy to old age and disproportionately affecting vulnerable populations. The exponential rise in plastic production, from two megatons (Mt) in 1950 to a projected 1,200 Mt by 2060, has led to over 8,000 Mt of waste polluting the planet, with less than 10% being recycled. These health-related impacts are estimated to cost over US$1.5 trillion annually. UN member states recognized the need to address this rising threat and, in 2022, decided to develop the Global Plastics Treaty, a legally binding instrument addressing the full lifecycle of plastics.[1] The most recent attempt at developing an international consensus, however, was unsuccessful.

Micro- and nanoplastics (MNPs) are pervasive environmental contaminants defined by their size. Microplastics range from 1 μm to 5 mm, while nanoplastics are smaller than 1 μm. Primary MNPs are intentionally manufactured for products such as cosmetic microbeads, industrial abrasives, and synthetic textiles, whereas secondary MNPs result from the breakdown of larger plastic debris under mechanical, chemical, or photo-degradation processes. Despite their differing origins, both types accumulate in air, soil, freshwater, and marine ecosystems, ultimately entering

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human food chains and potable water sources.[2] Their resistance to biodegradation allows them to persist for decades, raising urgent questions about long-term health impacts, especially carcinogenic risks.[3,4] Humans are exposed to MNPs through multiple interconnected pathways. Dietary ingestion constitutes the largest route, in which contaminated seafood, shellfish, and sea salt bioaccumulate MNPs and then transfer to humans upon consumption. Plastic packaging and food containers further leach MNPs into beverages and foods upon contact, heating, or mechanical stress. Inhalation of airborne microplastic fibers and fragments, mostly originating from tire wear, synthetic textiles, and atmospheric deposition, adds a respiratory dimension to MNPs uptake.[5] Although dermal absorption remains less well quantified, occupational and recreational handling of plastic powders and industrial pellets can deliver particles to the skin surface, where they may penetrate micro-abrasions or enter via hair follicles.[6] Collectively, these routes result in continuous, low-dose exposure that likely interacts with other environmental and lifestyle carcinogens.

Once ingested, MNPs transit the gastrointestinal (GI) tract, confronting the mucosal barrier that normally regulates absorption and defends against pathogens. Laboratory studies demonstrate that chronic exposure to MNPs can disrupt tight junction proteins, undermining barrier integrity and increasing intestinal permeability. Such "leaky gut" conditions permit smaller particles and associated chemical additives like plasticizers, stabilizers, and adsorbed pollutants to translocate across the epithelium into the lamina propria.[2] From there, particles can access the portal vein and migrate to the liver, while lymphatic uptake via M cells in Peyer's patches delivers them to mesenteric lymph nodes. Animal models and *in vitro* investigations have confirmed MNPs accumulation in hepatic tissue and lymphoid organs, highlighting a systemic distribution that extends beyond the gut.[7]

At the cellular and molecular levels, several overlapping mechanisms suggest how MNPs exposure may promote carcinogenesis (Fig. 1). First, microplastics stimulate chronic low-grade inflammation through particle-induced activation of nuclear factor kappa B (NF-κB) and pro-inflammatory cytokine release, fostering a microenvironment conducive to DNA damage and uncontrolled cell proliferation. Second, the oxidative stress generated by reactive oxygen species (ROS) on particle surfaces oxidizes nucleic acids and lipid membranes, resulting in mutagenic lesions and genomic instability. Third, MNPs alter gut microbial communities, a phenomenon known as dysbiosis, shifting the balance toward pro-inflammatory and potentially carcinogenic metabolites such as secondary bile acids and hydrogen sulfide.[7] Finally, endocrine-disrupting chemicals like bisphenol A, phthalates, and flame retardants that adsorb onto or leach from plastic particles exert genotoxic effects through hormone receptor modulation and direct DNA adduct formation. These converging pathways reinforce each other, amplifying the risk of cellular transformation in exposed tissues.[7]

GI cancers are of particular concern in the context of MNPs exposure because they represent the first sites of contact and absorption. The expansive surface area and rapid epithelial turnover of the GI tract render it highly vulnerable to persistent irritants and mutagens. In the colon and esophagus, repeated physical abrasion by particles, compounded with chemical insults from plastic additives, can accelerate epithelial cell proliferation, a known risk factor for malignant transformation.[8,9] Worldwide, colorectal cancer ranks among the leading causes of cancer mortality, with incidence rising sharply in both developed and developing regions. Esophageal cancer, which is linked to dietary carcinogens and chronic inflammation, similarly displays alarming upward trends in many

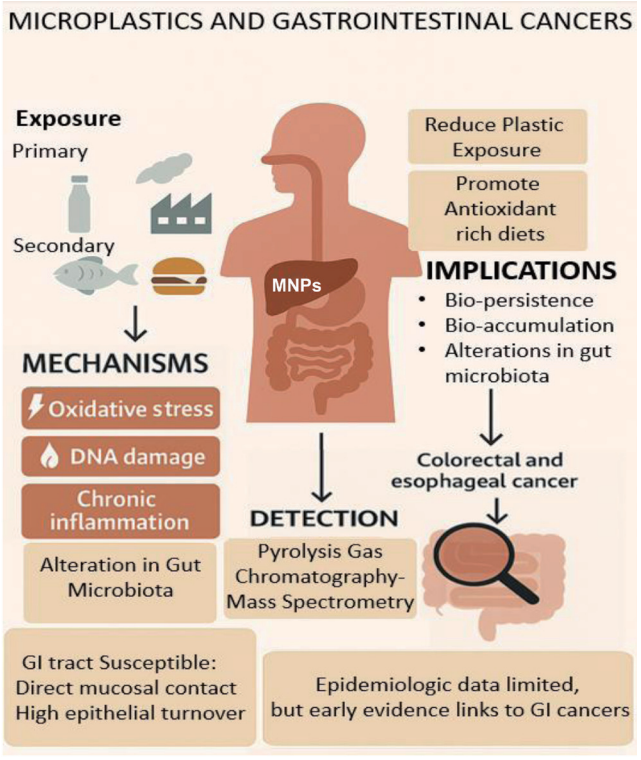

Fig. 1. Summary of the contribution of micro- and nanoplastics (MNPs) exposure from food, water, and the environment to gastrointestinal (GI) cancer risk. MNPs induce oxidative stress, DNA damage, and chronic inflammation, leading to gut microbiota alterations and bioaccumulation. These mechanisms may promote colorectal and esophageal cancers, though current epidemiologic evidence remains limited. Preventive strategies include reducing plastic exposure and promoting antioxidant-rich diets.

parts of Asia, Africa, and Latin America. The direct luminal exposure to MNPs, coupled with regional dietary habits and plastic use patterns, frames the GI tract as a critical battleground where plastic pollution may translate into elevated cancer risk. From an epidemiological standpoint, the global footprint of MNPs contamination is staggering. Surveys estimate that humans ingest tens of thousands to hundreds of thousands of MNPs particles annually,[10] with higher loads reported among populations consuming large quantities of seafood or using plastic-lined food containers. Urban residents face greater exposure through inhaled particles, with indoor air studies detecting average microplastic concentrations several times higher than in rural areas.[11]

Detection of MNPs in human stool samples confirms routine ingestion, while emerging reports describe their presence in colon biopsies and liver tissue obtained during surgical resections. It is important to understand, however, that, as opposed to standard histology, the visualization of these microscopic particles is limited to advanced and highly technical images by specialized equipment and analysis, such as spectroscopy, chromatography, or pyrolysis, used in laboratory settings by researchers.[12–14] Geographic variations in exposure, shaped by local plastic usage, waste management practices, and dietary customs, point to region-specific cancer risks that remain largely unexplored. Behavioral factors, such as reliance on bottled water and single-use plastics, further modulate individual MNPs burdens.[3]

Although experimental data increasingly suggest a carcinogenic potential of MNPs, direct epidemiological evidence linking their exposure to GI cancers is still limited. In response, this review aimed to consolidate current insights on the distribution, interactions, and biological impacts of MNPs within the GI system, with particular emphasis on their involvement in cancer-related mechanisms. It further delineates critical knowledge gaps and proposes directions for future multidisciplinary research to clarify this emerging environmental health concern.

MNPs accumulation in the GI tract

The human GI tract has emerged as a primary reservoir for MNPs, largely due to chronic ingestion through contaminated food, water, and packaging materials. Recent studies demonstrated that MNPs are not only internalized by intestinal epithelial cells but are also retained and passed on during cell division, suggesting long-term persistence within the GI mucosa and potential implications for tumorigenesis. These particles evade lysosomal degradation and accumulate intracellularly, raising concerns about chronic toxicity and inflammatory responses.[11,15,16] The widespread accumulation of MNPs in the body is supported by the detection of MNPs in a variety of biological specimens, including blood, stool, colonic mucosa, and hepatic tissues.[17,18] Notably, tissues with pathological disease, such as inflamed intestines or fibrotic liver, exhibited significantly higher MNPs loads compared to healthy tissues, suggesting a possible role in disease exacerbation.[19] Post-mortem analyses also demonstrate bioaccumulation of synthetic polymers such as polystyrene, polyethylene terephthalate, and polyacrylonitrile in the liver, kidney, and even brain tissues.[20] These findings underscore the systemic distribution of ss and their ability to cross biological barriers, likely via transcytosis or paracellular via systemic bloodborne transport. Moderate evidence also links them to structural damage in the colon and small intestine, including altered cell growth and death. These effects suggest a potential role of microplastics in promoting GI dysfunction and cancer risk.[21]

Effect of MNPs on upper GI carcinogenesis

MNPs are increasingly implicated in upper GI carcinogenesis through a convergence of exposure, persistence, and pro-tumor mechanisms. First, MNPs are now detectable in human gastric matrices and tissues, confirming direct contact with the gastric mucosa and potential for local bioaccumulation.[22]

Upon ingestion, sub-micron particles can traverse epithelial barriers, be internalized by gastric and esophageal epithelial cells, and localize to organelles, where they provoke ROS generation and oxidative DNA damage, suppress homologous recombination repair, and activate pro-proliferative signaling (e.g., MAPK, NF-κB) (Fig. 2).[23]

In esophageal models specifically, polystyrene and polyvinyl chloride nanoplastics trigger oxidative stress, DNA damage responses, apoptosis/pyroptosis, and epithelial barrier injury. These are the lesions that can set the stage for dysplasia under chronic exposure.[23] In the stomach, MNPs have been shown to promote malignant phenotypes in gastric cancer models, including enhanced migration/invasion and epithelial-mesenchymal transition, suggesting direct tumor-progression effects once neoplasia emerges.[24]

Beyond intrinsic particle toxicity, MNPs can carry adsorbed co-contaminants e.g., "polycyclic aromatic hydrocarbons, plasticizers, and metals, compounding genotoxic and endocrine-disrupting signals that foster chronic inflammation and immune evasion in the tumor microenvironment.[25]

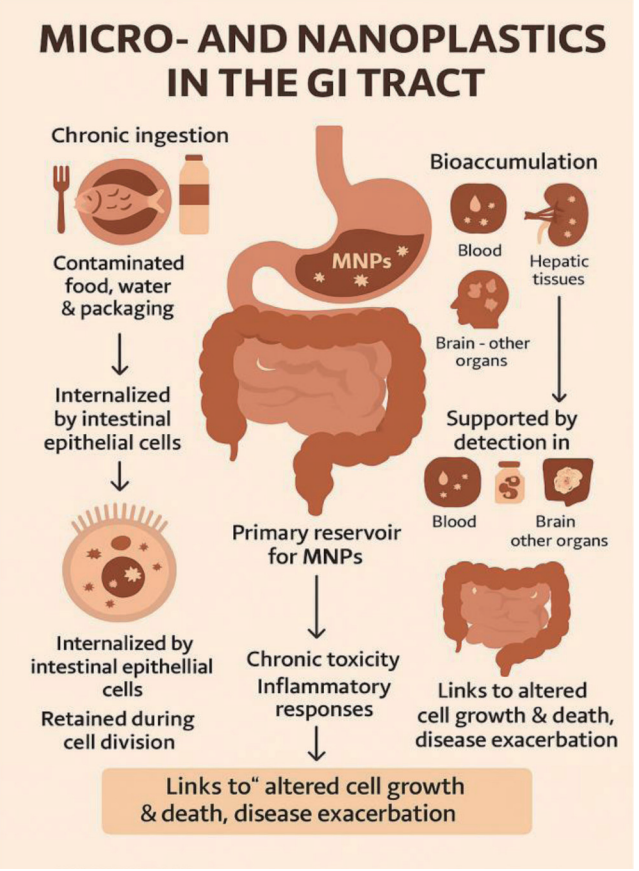

Fig. 2. Summary of the absorption, bioaccumulation, and retention of micro- and nanoplastics (MNPs) following chronic ingestion from contaminated food, water, and packaging. These processes trigger chronic inflammation and toxicity, contributing to altered cell growth, cell death, and disease exacerbation.

Parallel disturbances of the gastric–esophageal microbiome and tight-junction integrity facilitate endotoxin translocation, further amplifying ROS and cytokine cascades "interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α)" that link persistent injury to carcinogenesis. Human tumor data now report microplastics within resected cancers and associations with an altered tumor immune microenvironment, raising concern that retained particles may blunt antitumor surveillance and reshape therapy responses (Fig. 3).[22,24]

Current evidence supports a plausibility chain for gastric and esophageal cancer risk: Ubiquitous exposure → mucosal contact/uptake → oxidative and DNA damage + barrier failure → dysbiosis/inflammation/immune modulation → pro-oncogenic signaling and, in established disease, enhanced aggressiveness.

While longitudinal human studies are still limited, these mechanistic and early translational findings justify precautionary exposure reduction and targeted research in populations with reflux disease, *H. pylori*, tobacco–alcohol use, or occupational plastic dust exposure, where co-risks could synergize with MNPs injury.

MNPs and colorectal cancer

Emerging experimental data and animal models have started to ex-

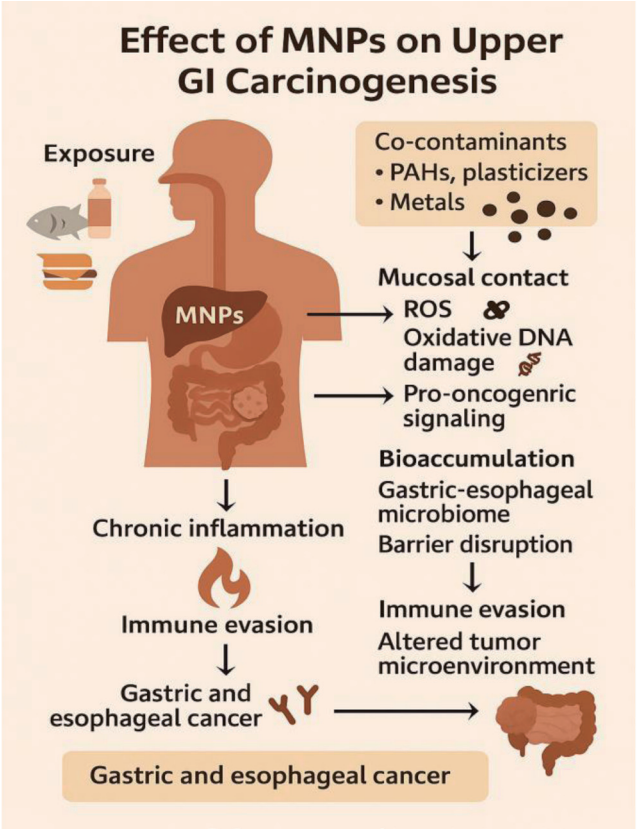

Fig. 3. Summary of the potential contribution of micro- and nanoplastics (MNPs) to gastric and esophageal carcinogenesis. MNPs and their co-contaminants (PAHs, plasticizers, metals) induce oxidative stress, DNA damage, and pro-oncogenic signaling. Disruption of the gastric–esophageal microbiome and barrier integrity leads to chronic inflammation, immune evasion, and tumor microenvironment alterations, linking persistent exposure to cancer development. GI, gastrointestinal; PAHs, polycyclic aromatic hydrocarbons; ROS, reactive oxygen species.

plore the carcinogenic potential of MNPs in the GI tract, with particular concern relating to colorectal cancer. A recent study demonstrated that MNPs, especially polystyrene particles, were present in human colorectal cancer tissues using laser infrared chemical imaging.[26] These particles were shown to promote tumor progression and resistance to chemotherapy by activating autophagy via the "mechanistic target of rapamycin/Unc-51 like autophagy activating kinase 1 (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[27]; the ingestion of MNPs disrupted gut homeostasis, altered microbial composition, and elevated pro-inflammatory cytokines, all of which are known contributors to tumorigenesis.[27] Complementary reviews have emphasized MNPs' ability to act as carriers for carcinogens like polycyclic aromatic hydrocarbons and heavy metals, which may compound their tumorigenic effects. These findings collectively suggest that MNPs are not inert contaminants, but biologically active agents capable of influencing cancer development through inflammation, oxidative stress, and immune modulation.[28]

In vitro studies have provided mechanistic insights into how MNPs interact with colorectal cells. One investigation revealed that MNPs are readily internalized by colon cancer cell lines, triggering mitochondrial ROS production, disrupting membrane potential, and activating the NLRP3 inflammasome, which are clear indicators of cellular stress and transformation.[29] MNPs appear particularly potent, inducing higher rates of cell migration and proliferation. This size-dependent uptake suggests that nanoplastics may pose an even greater risk than larger particles. Transcriptomic analyses show upregulation of autophagy-related genes such as ULK1, LC3, and SQSTM1, reinforcing the role of MNPs in promoting survival pathways in malignant cells. Moreover, MNPs were found to interfere with epithelial integrity by downregulating E-cadherin and upregulating Ki67, a proliferation marker. These changes mimic early oncogenic transformation and suggest that chronic MNPs exposure could prime normal colorectal cells for malignant conversion.[30] The cumulative evidence from these cell-based assays underscores the plausibility of MNPs contributing to colorectal tumorigenesis through direct cellular reprogramming.[26]

Finally, recent studies have begun correlating microplastic load with tumor biomarker expression in GI cancers.[31] In colorectal cancer tissues, higher concentrations of MNPs were associated with elevated levels of Ki67, mTOR, and LC3. These markers are linked to proliferation, autophagy, and poor prognosis. Immunohistochemical analyses revealed that tumors with greater MNPs burden exhibited more aggressive phenotypes and enhanced chemoresistance.[26] Additionally, MNPs were found to co-localize with inflammatory markers such as IL-6 and TNF-α, suggesting a synergistic role in shaping the tumor microenvironment. These findings hint at the potential of MNPs quantification as a novel biomarker for tumor aggressiveness and treatment response.[3] While still in early stages, this biomarker-MNPs relationship opens avenues for integrating environmental exposure metrics into cancer diagnostics. Future studies may validate MNPs load as a predictive tool for GI malignancy progression and therapeutic stratification.

MNPs and hepatocellular cancer

A primary location for MNPs deposition is within hepatic tissue, raising concerns about the role of MNPs in hepatocellular carcinoma (HCC) development. MNPs act as carriers for carcinogenic additives like phthalates and bisphenols, which can leach into hepatic tissues and activate oncogenic pathways such as PI3K/AKT/mTOR and Wnt/β-catenin. The cumulative evidence suggests that MNPs accumulation in the liver is not merely incidental but may actively contribute to hepatocarcinogenesis through inflammation, metabolic disruption, and genotoxicity.[32] Other supporting evidence comes from evidence linking concentrations of polyvinyl chloride and polystyrene particles to the induction of oxidative stress and DNA damage in liver cells, increasing cancer risk.[33] *In vivo* studies using rat models of cirrhosis-associated hepatocarcinogenesis revealed altered biodistribution of magnetic nanoparticles, suggesting impaired hepatic clearance and increased retention in fibrotic livers.[34] Moreover, polyethylene terephthalate microplastics were shown to disrupt lipid metabolism and elevate insulin levels in piglets, indicating metabolic dysfunction, which is a known precursor to metabolic-associated steatotic liver disease and subsequent risk of progression to cirrhosis and HCC.[35] These findings align with epidemiological data linking environmental pollutants to rising HCC incidence, especially in regions with high plastic exposure (Fig. 4).[36]

MNPs and pancreatic cancer

Although research on MNPs in pancreatic tissues is still nascent,

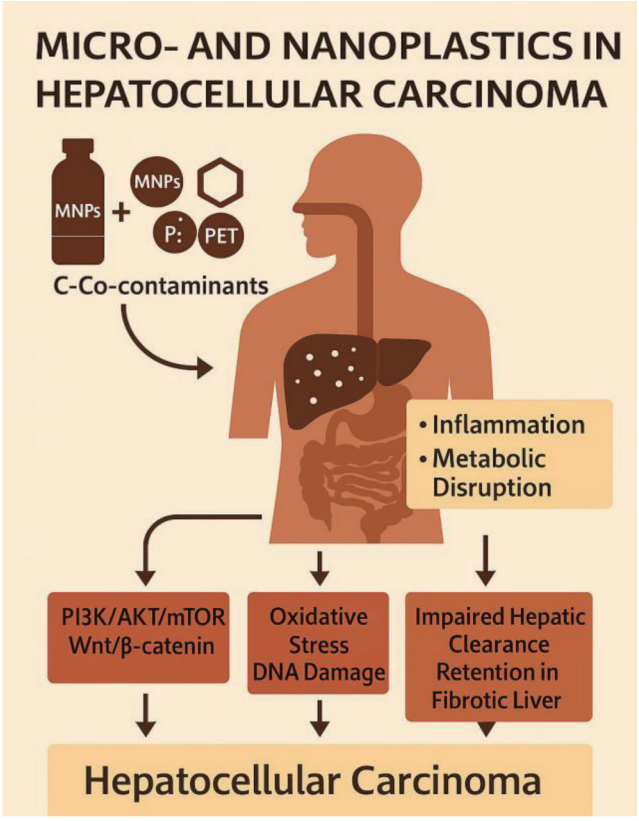

Fig. 4. Summary of the potential contribution of micro- and nanoplastics (MNPs) and their co-contaminants (phthalates, bisphenols, and PET) to hepatocellular carcinoma. MNPs accumulation in the liver activates oncogenic pathways (PI3K/AKT/mTOR, Wnt/β-catenin), induces oxidative stress and DNA damage, disrupts metabolism, and impairs hepatic clearance in fibrotic liver tissue, collectively promoting liver carcinogenesis. PET, polyethylene terephthalate.

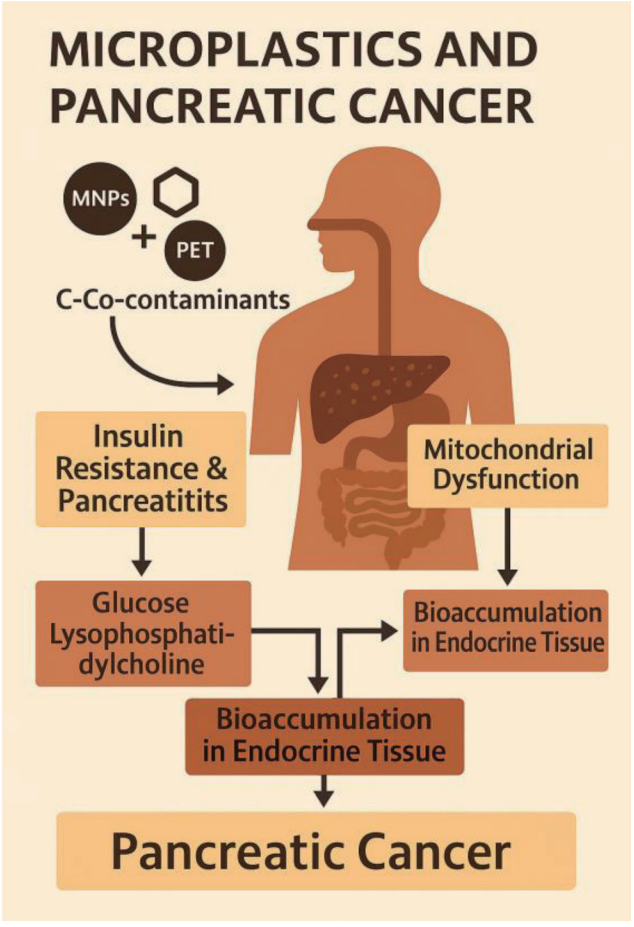

Fig. 5. Summary of the potential contribution of micro- and nanoplastics (MNPs), particularly polyethylene terephthalate (PET), to pancreatic carcinogenesis. MNPs exposure induces insulin resistance, pancreatitis, mitochondrial dysfunction, and inflammatory changes, leading to metabolic disruption and bioaccumulation in endocrine tissues. These processes collectively promote chronic inflammation and increase the risk of pancreatic cancer.

early findings are concerning. Polyethylene terephthalate microplastics have been shown to increase insulin resistance and trigger pancreatitis in animal models, with elevated levels of glucose, lysophosphatidylcholine, and inflammatory markers in pancreatic tissue.[37] These metabolic shifts are closely linked to pancreatic cancer risk, particularly in the context of chronic inflammation. Mechanistic studies also suggest that MNPs may interfere with mitochondrial function and activate stress pathways like the NLRP3 inflammasome, which are implicated in pancreatic tumorigenesis (Fig. 5).[29] Furthermore, the ability of MNPs to bioaccumulate in endocrine tissues raises concerns about their impact on insulin-producing β-cells and the tumor microenvironment.[38] While direct evidence of MNPs-induced pancreatic cancer remains limited, the emerging biochemical and inflammatory profiles warrant deeper investigation into their carcinogenic potential.

Gut microbiome, immune surveillance, and carcinogenesis

The gut microbiome, a dynamic consortium of trillions of microorganisms, orchestrates a multitude of physiological processes, including immune modulation, metabolic regulation, and epithelial integrity maintenance. Perturbations in microbial equilibrium, termed dysbiosis, have emerged as pivotal contributors to carcinogenesis, particularly within the GI tract. Dysbiosis denotes a pathological imbalance between commensal and pathogenic microbes, often characterized by reduced microbial diversity and overrepresentation of pro-inflammatory taxa. These shifts disrupt mucosal homeostasis, compromise barrier function, and facilitate translocation of microbial antigens, thereby inciting chronic inflammation.[39,40] Moreover, microbial metabolites like secondary bile acids and lipopolysaccharides potentiate genotoxic stress and oxidative damage, further exacerbating tumorigenic potential.

Recent studies suggest that micronutrient powders, while designed to combat malnutrition, may inadvertently influence gut microbial composition, particularly in vulnerable populations. These nutritional powders often contain iron, which has been shown to alter microbial ecology by favoring the growth of pathogenic taxa such as Enterobacteriaceae at the expense of beneficial commensals like Lactobacillus and Bifidobacterium. This iron-induced dysbiosis can exacerbate mucosal inflammation and oxidative stress, both of which are implicated in GI carcinogenesis.[41]

Microbial dysbiosis reconfigures the tumor microenvironment by skewing immune cell polarization and dampening antitumor surveillance. For instance, altered microbial metabolites sup-

press cytotoxic $CD8^+$ T cell activity while promoting regulatory T cells and myeloid-derived suppressor cells, fostering immune evasion. Dysbiotic microbiota also upregulate toll-like receptors, activating NF-κB and STAT3 pathways. These are the key drivers of inflammation and oncogenic signaling. This immunological reprogramming culminates in a milieu conducive to neoplastic transformation, angiogenesis, and metastasis.[39,41–43] The GI mucosa serves as a sentinel interface, orchestrating tolerance and defense through a multilayered immune architecture. Disruption of goblet cell function and mucin biosynthesis, particularly MUC2, compromises the mucus barrier, facilitating microbial translocation and epithelial stress. Impaired zonulin regulation and tight junction integrity further exacerbate permeability, triggering aberrant antigen presentation and loss of immune quiescence. Peyer's patches and intraepithelial lymphocytes, essential for antigen sampling and IgA secretion, exhibit diminished responsiveness under dysbiotic conditions. This erosion of mucosal immunity fosters a permissive niche for neoplastic transformation and chronic inflammation.[44,45]

Finally, cytokine disequilibrium skews the immunological landscape, undermining tumor surveillance and promoting oncogenesis. Elevated pro-inflammatory mediators like IL-6, TNF-α, and IL-1β activate NF-κB and STAT3 pathways, sustaining epithelial proliferation and inhibiting apoptosis. Concurrently, downregulation of anti-inflammatory cytokines like IL-10 and TGF-β impairs regulatory T cell function, disrupting immunoediting and enabling immune escape. This imbalance reshapes the tumor microenvironment, favoring angiogenesis and immune evasion. Targeting cytokine crosstalk between epithelial and immune cells is emerging as a rational therapeutic strategy in GI malignancies.[46–48]

MNPs and carcinogenic risk factors

Dietary patterns and alcohol consumption can amplify carcinogenic risk when combined with environmental pollutants such as MNPs and persistent organic compounds. High-fat diets facilitate lipophilic pollutant absorption, enhancing bioavailability and retention in GI and hepatic compartments. Ingested pollutants synergistically activate cytochrome P450 and aryl hydrocarbon receptor pathways, intensifying mutagenic activities.[49] Genetic polymorphisms in detoxification enzymes like GSTs and CYPs modulate individual vulnerability to carcinogens. Individuals with reduced GST activity exhibit impaired clearance of xenobiotics, heightening cancer risk when exposed to alcohol or dietary toxins. Chronic inflammation, often fueled by dysbiosis or autoimmune conditions, creates a pro-tumorigenic microenvironment. Gene-environment interactions that particularly involve inflammatory cytokines and DNA repair genes drive oncogenic transformation in predisposed individuals.[50,51] Although early data do not provide a clear clinical correlation, the current evidence supports that future research may better identify specific enzymatic and genetic mutations, which can enhance personalized medication and potentially mitigate cancer risk for certain individuals.

Minimization strategies for reducing microplastic exposure

The quick infiltration of MNPs into natural habitats and human biological systems has sparked intense scrutiny over their potential health consequences. Among various bodily systems, the GI tract bears the brunt, given its frontline role in nutrient intake and digestion. To curb the escalating exposure, reinforcing public health initiatives, promoting mindful eating practices, enacting stringent environmental policies, and harnessing the power of media to raise awareness can drive behavioral change.

Reduction in plastic usage

Over 400 million tons of plastic are produced annually, with 14% entering aquatic environments and breaking down into harmful MNPs. Policy measures like promoting biodegradable packaging and banning single-use plastics help curb primary MNPs emissions. A notable example is the European Union's directive implemented in 2021, which prohibited oxo-degradable plastics and consequently decreased microplastic release by an estimated 500,000 tons per year.[52] Global plastic waste is estimated to reach 1.7 billion metric tons by 2060.

Similarly, silicon dioxide (E551) and titanium dioxide (E171), frequently used in powdered food products, have been shown to negatively affect gut health by altering microbial diversity and triggering inflammation. After the EU banned E171 in 2021, France observed a notable 25% drop in pediatric cases of inflammatory bowel disease associated with titanium dioxide exposure. These findings highlight the need for comparable restrictions in regions like Asia and North America to help curb related health risks.[53] The Lancet Countdown on health and plastics monitors progress through geographically and temporally representative indicators, offering a data-driven framework to mitigate plastic-related harms through evidence-based policy interventions.[1]

Advanced filtration systems

Conventional water treatment plants remove only 70–90% of MNPs. Installing ultrafiltration membranes or activated carbon filters in households can reduce nanoplastics by 95%.[54] Certain filtration technologies, particularly reverse osmosis and activated carbon systems, have proven highly effective in eliminating microplastics from drinking water. Integrating these into both residential setups and municipal water infrastructures could significantly reduce human exposure.

Opting for filtered tap water over bottled options can dramatically cut microplastic intake. One study found this switch could drop annual consumption from around 90,000 particles to just 4,000. Furthermore, in regions with hard water, a simple process of boiling followed by filtration has been shown to eliminate up to 90% of these particles.[55,56] Practical strategies like installing HEPA filters, wet mopping floors, and maintaining a "no-shoes" policy indoors can substantially reduce the accumulation of these particulates. Recent Studies reported graphene oxide–based membranes can achieve high-efficiency microplastics separation, and recent applied membrane designs aim to remove targeted contaminants while retaining key nutrients in water. This supports their potential for drinking-water applications.[57]

Enhancing wastewater management through advanced tertiary treatments like membrane bioreactors and dissolved air flotation has shown remarkable effectiveness. It has the potential to eliminate up to 99% of MNPs from industrial runoff. As an example, textile sectors in India have used it, which led to an 80% reduction in MNP emissions. This shift has contributed to preserving gut microbiome health among populations situated downstream of these facilities.[53]

Public education

Community education initiatives are instrumental in building public understanding of MNPs pollution (Table 1). Well-structured outreach programs can illuminate the origins of MNPs and their potential effects on health, especially gut-related risks. Leveraging

Table 1. Public health interventions to reduce microplastic exposure

Problem	Intervention
Reduce plastic usage	Promote biodegradable and compostable alternatives; Minimize or avoid single-use plastics; Encourage industry packaging reform (e.g., paper, glass, metal); Avoid food preparation/storage involving plastics
Water and air quality	Use advanced filtration (ultrafiltration 0.01–0.1 µm, reverse osmosis, activated carbon); Boil and filter tap water before use; Improve indoor air quality through ventilation and filtration; Monitor MNPs levels in municipal supplies
Wastewater management	Upgrade to tertiary treatments (Membrane Bioreactors, Dissolved Air Flotation); Implement microplastic capture technologies at treatment plants; Encourage decentralized wastewater treatment in rural/under-resourced areas; Monitor and regulate effluent standards for MNPs discharge
Public education	Awareness programs on MNPs sources, exposure, and health effects; Promote reusable, sustainable alternatives (cloth bags, refillable bottles); Community outreach via schools, health fairs, and social media; Collaborate with NGOs and policymakers to push behavior change campaigns

MNPs, micro- and nanoplastics; NGO, non-governmental organizations.

trusted spaces like schools, health clinics, and local community hubs enables direct engagement and fosters long-term behavioral change.

Dietary modifications to reduce MNPs intake

Processed foods contain two to ten times more MNPs than fresh produce due to plastic packaging and additives like TiO_2 (E171) and SiO_2 (E551).[58] A study revealed that people who primarily ate less processed foods had significantly less microplastics in their stool compared to those with more processed diets.[59] This suggests that fresh, minimally handled foods are less likely to carry plastic contaminants, since packaging and industrial processing often introduce these particles. Choosing whole grains, fruits, and vegetables over packaged items can help limit intake. Also, the way food is cooked matters; using plastic containers in microwaves or ovens can cause microplastics to make their way into meals. Safer options include glass or stainless-steel cookware.[53]

Evidence from animal studies shows that increasing intake of certain fibers (e.g., cellulose, wheat bran, chitosan) can speed GI transit, increase fecal bulk, and thereby enhance the excretion of ingested particles.[60]

MNPs in soil cling to edible plants, especially leafy greens and root vegetables.[53] By avoiding synthetic fertilizers, organic farming helps cut down MNPs contamination in the soil by significantly. Hydroponic systems can further decrease MNPs exposure; these systems use plastic-free growing mediums, so crops like tomatoes raised hydroponically have shown less microplastic attachment compared to those grown in traditional soil.[53,61]

Certain probiotics, along with prebiotic fibers such as inulin, help reinforce the gut's protective lining by tightening junctions between intestinal cells. A healthy barrier makes it harder for MNPs to pass into the bloodstream. Lab-based studies have shown that probiotics can cut titanium dioxide–related gut leakiness.[62] In addition, foods rich in omega-3s, like flaxseeds and walnuts, offer antioxidant support that helps reduce the oxidative stress MNPs can trigger in colon cells (Table 2).

Regulatory policies targeting industrial plastic release

Strengthening environmental policies and enforcing stricter industrial standards are vital to limiting MNPs release at the source. Collaborative efforts between governments, industries, and scientific communities can accelerate the development of safer production practices and drive innovation in clean technologies.

A major hurdle in regulating MNPs is the absence of a standard definition, which leads to fragmented approaches. For example, the European Union's 2022 "Nanomaterials Regulation" requires labeling for particles smaller than 100 nanometers, while the U.S. Food and Drug Administration evaluates materials based on their intended use, not size. Aligning global standards, such as through the Organization for Economic Co-operation and Development Guidance on Nanomaterials, could simplify regulations, improve industry compliance, and help reduce international contamination. Widening these restrictions to include other unnecessary plastic additives could further cut down environmental microplastic release.[52,63] Notably, however, the most recent international consensus in Geneva, Switzerland, involving 600 participants, including delegates from 183 countries, ministers, and observers from hundreds of organizations, failed to reach consensus statements for restrictions.

Media's role in shaping public perception and behavior

Traditional and digital media can shift public behavior by linking MNPs to health risks and promoting awareness by prioritizing the clinical implications of quality-derived evidence. Investigative journalism also plays a key role in exposing environmental violations and driving policy reform. Unfortunately, in present times, social media is far more pervasive in shaping both personal habits. Social media platforms like Instagram and Twitter help amplify

Table 2. Dietary strategies for reducing microplastic ingestion

Problem	Strategy	Key findings/benefits
Eat healthy	Choose fresh, whole foods; Avoid microwaving in plastic; Use glass/steel containers for storage	Processed foods contain 2–10× more MNPs; Whole food diets = 40% lower fecal MNPs; Plastic-free cookware prevents leaching
Soil to gut	Prioritize organic farming; Choose hydroponic-grown crops; Wash fruits/vegetables thoroughly	Organic soil = 30% less MNPs; Hydroponic tomatoes = 90% less MNPs adhesion; Proper washing reduces particle load
Gut barrier support	Probiotics and prebiotics; Omega-3s (e.g., flaxseed, fish oil); Zinc- and antioxidant-rich foods	Probiotics reduce gut permeability by 60%; Omega-3s mitigate oxidative stress; Promotes microbiome balance and resilience

MNPs, micro- and nanoplastics.

movements like *#PlasticFreeJuly*, yet they also act as hotspots for misinformation. However, a study showed that nearly 30% of posts about MNPs contained misleading claims, such as saying "all MNPs are toxic", which oversimplifies the nuanced relationship between dosage and biological impact.[64] To ensure accuracy, these campaigns need to engage researchers to help craft more informed narratives, including distinctions between harmless and harmful changes to gut microbiota triggered by MNPs.

Future directions

Despite mounting experimental evidence, direct epidemiological links between MNPs exposure and GI cancer incidence remain scarce. To close this gap, future research must employ quantitative biomonitoring approaches, coupling high-precision mass spectrometry and microscopy to map particle types, sizes, and associated chemical payloads in human tissues. Large-scale cohort studies tracking dietary plastic intake alongside cancer outcomes are needed to establish exposure–response relationships. Mechanistic investigations at the cellular level should elucidate dose thresholds and co-carcinogen interactions, while *in vivo* models can clarify long-term effects of chronic low-dose exposure. Interdisciplinary efforts that integrate environmental science, toxicology, microbiology, and oncology will be essential to understand how this novel class of contaminants influences tumor initiation and progression.

At the same time, several prevention steps can be taken to reduce exposure, such as improving the safety of food packaging, regulating harmful plastic additives, strengthening waste management, and encouraging simple everyday practices like avoiding heating food in plastic containers, choosing filtered water, and cutting down on single-use plastics. Together, these efforts can help reduce potential risks while the scientific community works toward clearer answers.

Conclusions

MNPs in modern ecosystems have become a pressing biomedical concern, particularly for GI health and cancer risk. From plastic-laden seafood to polymer accumulation in tissues, environmental pollutants are infiltrating the human body with oncogenic consequences. The synergy between MNPs and established carcinogens is concerning, as alcohol, poor diet, and genetic predisposition compound malignancy risk through inflammation and impaired detoxification. These particles contribute to endocrine disruptors and heavy metals, exacerbating preexisting vulnerabilities. The epidemiological landscape remains nascent, requiring large-scale biomonitoring studies to delineate dose-response relationships and MNPs accumulation dynamics. Future research must integrate molecular toxicology, microbiomics, immunology, and oncology to understand MNPs' health impact. Public health interventions should focus on exposure reduction through regulations, education, and sustainable packaging. Clinicians must stay vigilant when managing GI pathologies in high-exposure populations. Viewing MNPs as modifiable risk factors could potentially reduce the risk of GI cancer. The intersection of plastic pollution and GI malignancies shows that environmental health directly impacts human biology. To address this more clearly, data on MNPs' effects requires both scientific research and evidence-based societal action.

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Conflict of interest

The authors have no conflicts of interest to disclose.

Author contributions

Study concept and design, drafting and revision of the manuscript (SR, PD, AM, AV, EO, DJ). All authors have approved the final version and publication of the manuscript.

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