Journal of Enterocolitis

Review

Microplastics and Inflammatory Bowel Disease: From Environmental Exposure to Clinical Uncertainty

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Cite this article as: Günegül Y, Şenocak İE, Kav T. Microplastics and Inflammatory Bowel Disease: From Environmental Exposure to Clinical Uncertainty. *J Enterocolitis.* 2026;5(2):33-42.

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Received: July 29, 2026 **Accepted:** August 26, 2026

DOI: 10.14744/Jenterocolitis.2026.38359

Abstract

Microplastics and nanoplastics are persistent environmental contaminants of increasing relevance to human health, particularly because ingestion places the gastrointestinal tract at the center of exposure. In inflammatory bowel disease (IBD), in which epithelial barrier dysfunction, dysbiosis, immune activation, and environmental factors intersect, these particles represent a biologically plausible but incompletely characterized exposure. This narrative review summarizes the current evidence on microplastics, intestinal inflammation, and IBD, including exposure routes, particle definitions, measurement challenges, biological mechanisms, experimental data, and human studies. Experimental evidence suggests that microplastics may disrupt the mucus–epithelial barrier, promote oxidative stress, alter microbial and metabolite profiles, and amplify inflammation in susceptible or inflamed intestinal environments. Human evidence remains limited and observational, with fecal, serum, and tissue-based studies reporting associations between microplastic burden and IBD-related features, including disease activity, fecal calprotectin levels, and fibrostenotic tissue changes. Current data do not establish microplastics as causal drivers of IBD or clinically useful biomarkers. The most balanced interpretation is that they may act as environmental disease modifiers or markers of altered intestinal handling rather than as primary initiators of IBD. Standardized analytical methods and longitudinal human studies are needed to clarify their clinical relevance.
Keywords: Inflammatory bowel diseases, intestinal mucosa, microplastics, nanoplastics.

INTRODUCTION

Plastics have become integral to modern life because of their low cost, durability, and versatility, with widespread use in packaging, textiles, and consumer products. However, the physicochemical stability that makes plastics useful also contributes to their environmental persistence, as plastic debris progressively fragments into smaller particles that disperse throughout terrestrial, aquatic, and atmospheric environments.[1,2] Microplastics and nanoplastics are increasingly recognized not only as environmental contaminants but also as potential human health hazards, with the gastrointestinal tract representing a principal interface of exposure through ingestion and, indirectly, inhalation.

Inflammatory bowel disease (IBD), which includes Crohn's disease and ulcerative colitis, is a chronic immune-mediated disorder arising from interactions among genetic susceptibility, epithelial barrier dysfunction, alterations in the microbiota, and environmental exposures. Its rising incidence in newly industrialized regions has drawn attention to environmental triggers associated with urbanization and dietary westernization.[3] Compared with acute or predominantly functional gastrointestinal disorders, IBD provides a particularly informative model for studying environmental pollutants because it is characterized by chronic mucosal inflammation, measurable disease activity, and opportunities for longitudinal assessment using stool, blood, endoscopic, and tissue samples. Within this context, microplastics represent a biologically plausible but incompletely characterized exposure. Whether these particles act as causal contributors to IBD, consequences of intestinal inflammation, or modifiers of disease activity remains unresolved, and the available human data remain limited.

Several recent reviews have summarized the analytical challenges, experimental findings, and potential gastrointestinal effects of microplastics and nanoplastics, including their possible relationship with intestinal inflammation and IBD.[4-6] However, the rapid emergence of human and disease-relevant translational data warrants an updated, clinically oriented appraisal that more clearly distinguishes biological plausibility from evidence of causation.

Accordingly, this review updates the evidence through August 2026 and organizes it around a three-part interpretive framework: whether microplastics may represent causal contributors to IBD, consequences or markers of altered intestinal handling, or modifiers of established disease. The review also considers recent human and translational evidence, the possibility of reverse causality, clinically relevant confounding factors, and the implications of current uncertainty for patient counseling and future research.

METHODS

This narrative review aimed to synthesize the most relevant evidence regarding human exposure to microplastics and nanoplastics, their measurement in gastrointestinal samples, and their relationship with intestinal inflammation and IBD. Relevant literature was identified through searches of major electronic databases, including PubMed/MEDLINE, Scopus, and Web of Science, from inception through August 2026. The search strategy used a combination of Medical Subject Headings (MeSH) terms and free-text keywords, including but not limited to "microplastics," "nanoplastics," "polystyrene," "polyethylene," "plastic particles," "inflammatory bowel disease," "Crohn's disease," "ulcerative colitis," "intestinal barrier," "intestinal permeability," "mucus layer," "gut microbiota," and "intestinal inflammation." Reference lists of relevant articles and previous reviews were hand-searched to identify additional sources, and ClinicalTrials.gov was searched to identify registered ongoing studies.

The literature was selected based on its relevance to the central question of whether microplastics and nanoplastics act as causal contributors to IBD, represent consequences of altered intestinal handling, or function as environmental disease modifiers. Selected sources included human observational studies reporting microplastic burden in stool, serum, or intestinal tissue; in vitro and organoid-based epithelial models; animal models of intestinal injury and colitis; and methodological reports addressing particle detection, polymer identification, and contamination control. Environmental and production-related data were incorporated selectively to establish the scale and routes of human exposure. Priority was given to recent, high-quality, peer-reviewed articles, while earlier methodological and mechanistic studies were retained when they provided foundational context for current analytical and biological interpretations. Conference abstracts and registered studies were considered selectively when they provided recent IBD-specific human evidence not yet available as full peer-reviewed publications. Sources without clear relevance to gastrointestinal exposure or intestinal inflammation, as well as duplicate or less complete reports of the same study, were excluded. Study selection and thematic organization were based on author consensus, consistent with a narrative review design rather than a formal systematic review process.

Global Plastic Burden and Routes of Human Exposure

The global burden of plastic pollution has increased in parallel with the expansion of plastic production and consumption. Since the mid-20th century, plastics have been produced at an accelerating rate, and a substantial proportion of cumulative plastic waste has either been landfilled or released into the environment rather than effectively recycled.[1,7] Global emissions inventories indicate that mismanaged plastic waste remains an important source of environmental leakage, with marked regional variation related to consumption patterns, waste management capacity, and disposal practices.[8]

Plastic pollution is not restricted to marine environments. Fragmented plastic particles have also been documented in freshwater and polar systems, illustrating their environmental persistence and capacity for long-range transport.[9] Larger plastic items may fragment through mechanical, photochemical, and thermal degradation into smaller particles that persist and circulate within environmental systems, providing the basis for recurrent human exposure to microplastics and nanoplastics.[2,10]

Human exposure occurs through multiple pathways, of which ingestion is the most directly relevant to the gastrointestinal tract (Figure 1). Microplastics have been reported in drinking water, including tap and bottled water, seafood, salt, and other food products, and food processing and packaging may further contribute to dietary exposure.[11-14] Studies of commercially available table salts further illustrate that microplastic contamination may affect everyday dietary products, including sea, lake, and rock salts.[15] Particle release from plastic containers may increase with heat, prolonged storage, mechanical stress, and repeated use, although the extent of exposure varies according to polymer type and handling conditions. Inhalation represents an additional indirect route, as airborne fibers and fragments from synthetic textiles, household dust, urban air pollution, and cigarette smoke-related exposure may undergo respiratory deposition and, in part, mucociliary clearance followed by swallowing.[16,17]

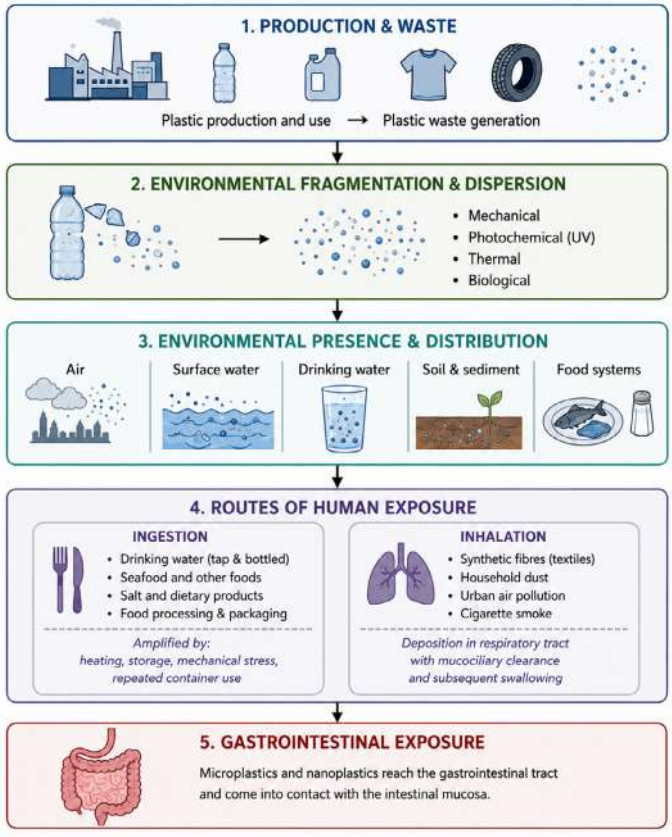

Figure 1. Routes of human exposure to microplastics and nanoplastics through the gastrointestinal tract. Ingestion and inhalation represent two major exposure pathways. Dietary exposure may occur through water, seafood, salt, processed foods, and packaged foods, whereas inhaled particles from textile fibers, household dust, urban air, or cigarette smoke may reach the gut through mucociliary clearance and swallowing. Particle release from food-contact materials may increase with heating, prolonged storage, mechanical stress, and repeated container use.

This exposure landscape is particularly relevant to gastroenterology because the intestinal mucosa represents a chronically exposed biological surface. In IBD, the parallel increase in environmental plastic exposure and disease incidence in industrializing regions should not be interpreted as evidence of causality; however, it supports the rationale for examining microplastics within the broader environmental context in which intestinal inflammation develops and persists.[4,5]

Definitions and Classification of Microplastics and Nanoplastics

Microplastic research has long been limited by inconsistent terminology and the absence of a universally accepted classification framework.[18]

The most widely used consensus definition characterizes microplastics as synthetic solid particles ranging from 1 µm to 5 mm in size, of either primary or secondary origin.[19] Nanoplastics generally refer to plastic particles smaller than 1 µm, although some nanomaterial-based frameworks restrict the term to particles between 1 and 100 nm.[18,20] In this review, we use the term nanoplastics in the broader sense to describe plastic particles smaller than 1 µm, while acknowledging that analytical detection becomes increasingly challenging within this size range.

Beyond size, particles are classified according to origin, shape, and polymer composition, with polymer type influencing density, surface chemistry, and degradation behavior.[18] Primary microplastics are manufactured at small dimensions, such as industrial pellets and microbeads, whereas secondary microplastics arise from the fragmentation of larger plastic items through mechanical, photochemical, and thermal degradation.[19] Shape—whether fragment, fiber, film, or bead—is not merely descriptive: experimental studies have demonstrated shape-dependent gut accumulation, with fibers retained to a greater extent than fragments or beads and associated with more pronounced mucosal damage and increased intestinal permeability.[21] Shape may also influence analytical detectability because fibers and fragments may be identified with different sensitivities across analytical methods.

Plastic particles should not be considered inert materials. They may contain additives such as plasticizers and stabilizers, and their surfaces can adsorb environmental pollutants or microorganisms.[18] Therefore, their potential intestinal effects may reflect not only the physical presence of the particles but also the chemical and microbial exposures they carry. This composite nature provides an important bridge to the mechanistic pathways discussed in the following sections.

Measurement of Microplastics in Gastrointestinal Samples

Measuring microplastics in gastrointestinal samples is technically challenging. Stool and intestinal tissues contain complex organic material, mucus, and cellular debris, whereas the near-ubiquitous presence of plastic in laboratory environments creates a substantial risk of contamination. Therefore, contamination control is a critical methodological concern regardless of sample type. Rigorous protocols require the use of glass or metal labware rather than plastic, cotton laboratory garments, procedural and airborne blanks, and blank-corrected reporting; without these precautions, reported particle concentrations are difficult to interpret.[22,23]

Several analytical techniques are available for microplastic analysis and polymer identification, each with distinct strengths and limitations in terms of sensitivity, size range, and chemical specificity.[24] Fourier-transform infrared (FTIR) spectroscopy is widely used for polymer identification but loses resolution for very small particles. Raman spectroscopy can detect smaller particles with chemical specificity but may be affected by fluorescence and matrix interference. Pyrolysis–gas chromatography/mass spectrometry provides robust polymer mass quantification but does not preserve information on particle count, size, or morphology (Table 1).

Two biological matrices are commonly used in gastrointestinal research. Stool sampling is noninvasive and allows repeated measurements, making it the most feasible approach for clinical cohorts, although its composition is influenced by diet and intestinal transit time.[25-27] Tissue-based assessment using endoscopic biopsy or surgical specimens provides more direct information on mucosal or tissue-associated particles but is invasive and more susceptible to procedural contamination.[28,29] In IBD research, stool sampling has been the more practical approach to date, whereas tissue-based evidence remains comparatively limited.

These findings highlight the need to interpret gastrointestinal microplastic measurements in relation to the biological matrix being analyzed. Stool-based recovery may be confounded by diarrhea, mucus, blood admixture, diet, and transit time, whereas tissue-based detection may be influenced by sampling site, disease activity, fibrosis, and procedural contamination. This raises an interpretive question central to this review: does a measured microplastic burden reflect ambient exposure, altered intestinal handling, or a downstream consequence of active disease? Disentangling these possibilities requires longitudinal studies pairing fecal and tissue sampling with disease activity indices, an issue revisited in later sections.

Biological Mechanisms Linking Microplastics to Intestinal Inflammation

The biological link between microplastics and intestinal inflammation

Table 1. Comparison of analytical techniques for microplastic detection and polymer identification in gastrointestinal samples

Technique	Principle	Effective size range	Polymer/chemical identification	Key strengths	Key limitations
Fourier-transform infrared (FTIR) spectroscopy	Vibrational absorption spectroscopy	Generally limited to particles ≳20 µm	Yes—established polymer spectral libraries	Widely available and well validated for polymer identification	Reduced resolution and sensitivity for particles smaller than approximately 20 µm
Raman spectroscopy	Vibrational/inelastic light scattering	Can resolve particles down to approximately 1 µm	Yes—high chemical specificity	Detects smaller particles than FTIR and provides polymer-specific signals	Susceptible to fluorescence interference and signal masking by organic or biological matrices
Pyrolysis–gas chromatography/mass spectrometry (Py-GC/MS)	Thermal decomposition followed by chromatographic and mass spectrometric analysis	Not particle-resolved; bulk sample analysis	Yes—robust polymer mass and chemical quantification	Provides quantitative data on polymer mass and composition	Destructive; does not preserve information on particle count, size, or morphology

FTIR: Fourier-transform infrared; Py-GC/MS: pyrolysis–gas chromatography/mass spectrometry.

is best interpreted in terms of plausibility rather than established causality. Current evidence suggests that microplastics and nanoplastics may interact with several pathways already central to IBD pathogenesis, including mucus and epithelial barrier function, oxidative stress, dysbiosis, mucosal immune activation, and exposure to plastic-associated chemicals.[4,5]

The mucus–epithelial interface is the first major barrier encountered by ingested particles and is increasingly recognized as a key determinant of whether such particles interact with the mucosal surface, are retained, or cross the epithelial barrier.[30] Human intestinal mucus appears to limit particle migration and cellular damage, while organoid-derived epithelial models, including systems incorporating microfold cells, and gut barrier platforms suggest that particle size, surface characteristics, epithelial context, and specialized particle-sampling pathways may further influence uptake, toxicity, and inflammatory responses.[31]

Experimental evidence also indicates that ingested nano- and micro-sized polystyrene particles can cross the intestinal barrier and accumulate systemically, and advanced epithelial barrier models have similarly demonstrated particle passage across lung and gut systems.[32,33] These findings are relevant to IBD because the mucus layer and epithelial barrier are frequently disrupted during active inflammation. However, barrier passage in experimental models should not be interpreted as evidence of clinically meaningful translocation in humans.

Oxidative stress and epithelial injury represent additional plausible pathways. Experimental studies suggest that microplastics, particularly polystyrene particles, can induce reactive oxygen species generation, mitochondrial stress, and cytotoxicity.[34] Importantly, aged or contaminant-loaded particles, such as benzo[a]pyrene-loaded aged polystyrene microplastics, may exert greater biological effects than pristine particles, promoting colonic barrier injury through oxidative stress-mediated Notch signaling.[35] In IBD, such epithelial stress may amplify existing mucosal injury rather than act as an independent disease trigger.

The gut microbiota provides another mechanism through which microplastics may influence intestinal inflammation. Microplastics may alter the intestinal microenvironment, interact with microbial communities,

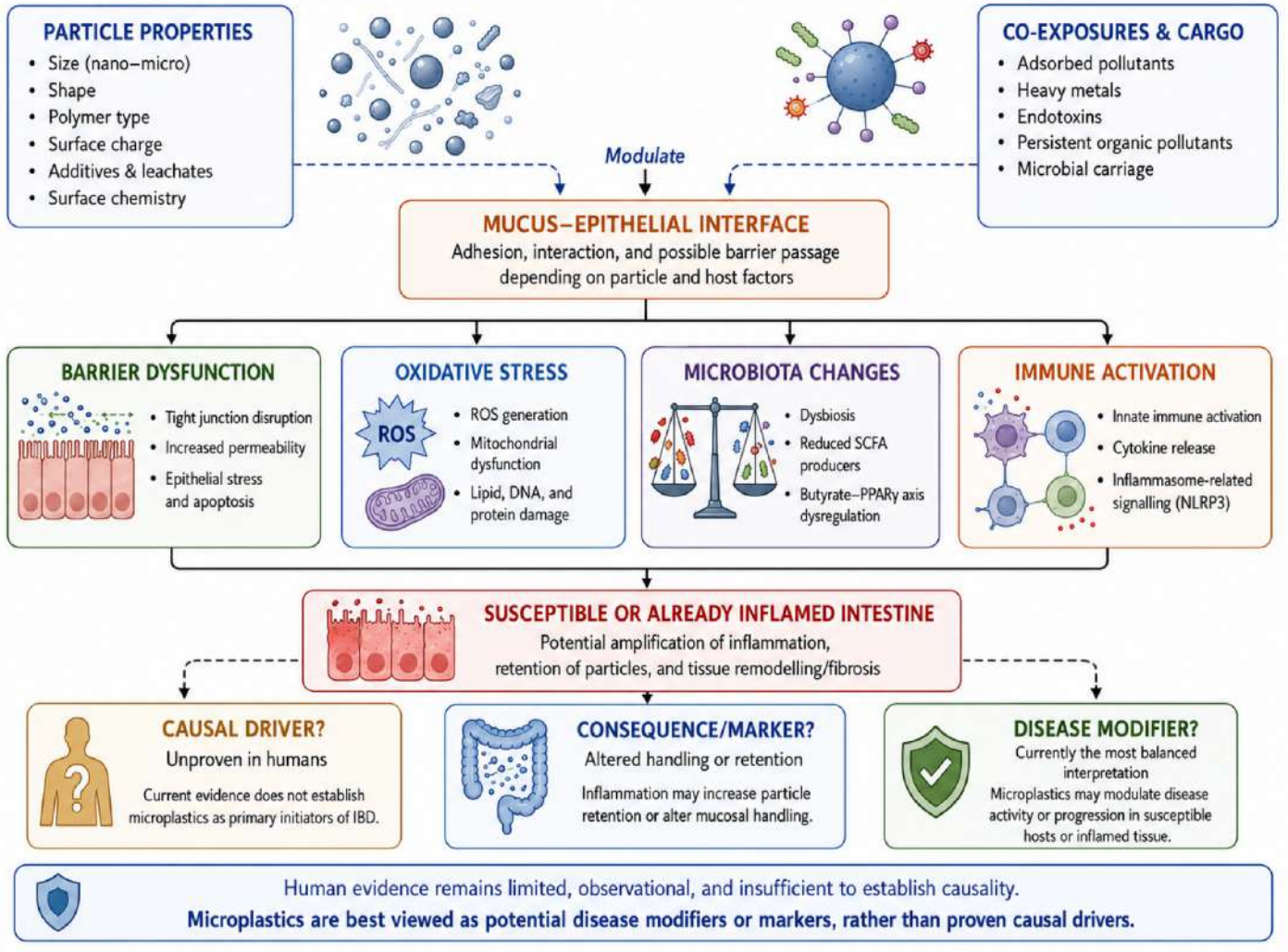

Figure 2. Proposed mechanisms linking microplastics to intestinal inflammation and IBD. Particle properties and co-exposures may influence interactions at the mucus–epithelial interface, contributing to barrier dysfunction, oxidative stress, alterations in the microbiota, and immune activation. In susceptible or already inflamed intestinal tissue, these pathways may amplify inflammation, particle retention, and tissue remodeling. Current evidence supports a disease-modifier model rather than a primary causal role for microplastics in IBD.

Table 2. Representative experimental studies linking micro- and nanoplastics to intestinal inflammation

Mechanistic domain	Representative studies	Model and exposure	Main experimental findings	Relevance to IBD
Mucus–epithelial barrier interaction	van Wijngaarden et al[31] Donkers et al.[33] Lehner et al.[42]	Human intestinal mucus model; advanced epithelial lung/gut barrier models; 3D intestinal barrier model	Human intestinal mucus model; advanced epithelial lung/gut barrier models; 3D intestinal barrier model Human intestinal mucus limited microplastic migration, uptake, and cellular damage; advanced barrier systems demonstrated particle passage under experimental conditions; 3D models enabled evaluation of epithelial and immune responses	Supports the mucus–epithelial interface as a key determinant of particle exclusion retention, and barrier interaction
Human-derived epithelial models	Cortes et al.[43] Chen et al.[44] Kharaghani et al.[45]	Human Caco-2 intestinal epithelial cells; human intestinal organoid-derived epithelial models and without M cells; with healthy and Crohn's disease-derived human duodenum-chip models exposed to polystyrene microplastics and nanoplastics	Polystyrene microplastics and nanoplastics showedsize- and concentration-dependent epithelial uptake or injury, inflammatory cytokine induction, and differential responses between healthy and disease-relevant epithelial systems	Provides human-relevant evidence that particle size, epithelial model complexity, and disease-associated epithelial context may influence toxicity and absorption
Epithelial barrier injury and junction disruption	Luo et al.[46] Sung et al.[47] Xie et al.[48]	DSS-induced colitis mouse models exposed to polystyrene or polyethylene modifying	Microplastics worsened colitis severity, shortened colon length, increased histological injury, impaired epithelial junctions, reduced mucus-related protection, and increased intestinal permeability	Supports a disease-modifying effect in an already inflamed intestine rather than a purely disease-initiating role
Inflamed-tissue susceptibility and particle retention	Lamprecht et al.[49] Busch et al.[50]	TNBS-induced colitis model exposed to fluorescent polystyrene particles; triple-culture in vitro model (Caco-2/HT29-MTX-E12/THP-1) under healthy versus inflamed conditions	Particles preferentially accumulated in ulcerated and inflamed colonic mucosa compared with healthy tissue, with greater retention of smaller particles; in vitro, particle-associated cytotoxicity and cytokine responses were more evident under inflamed culture conditions	Provides experimental support for the disease-modifier hypothesis, suggesting that the inflamed inflamed intestinal environment may alter alter particle retention and biological responses
Oxidative stress, apoptosis, and epithelial injury	Jia et al.[51] Zeng et al.[52] Shaoyong et al.[35] Hwang et al.[34]	Mouse and cellular models exposed to polypropylene or polystyrene microplastics, including aged or contaminant -loaded particles	Microplastics induced oxidative stress, mitochondrial injury, colonic apoptosis, cytotoxicity, and barrier dysfunction; benzo[a]pyrene-loaded aged particles promoted colonic barrier injury through oxidative stress-mediated Notch signaling; polystyrene microplastics activated NF-κB/NLRP3/IL-1β-related pathways	Links microplastics to epithelial stress pathways, inflammasome-related signaling, and barrier damage relevant to intestinal inflammation
Mucus secretion and microbiota–metabolite alterations	Li et al.[53] Sun et al.[54] Qiao et al.[55] Huang et al.[36] Ghosal et al.[37] Zhai et al.[38]	Mice orally exposed to polyethylene or polystyrene microplastics/nanoplastics; in vitro gut simulation/ Caco-2 models; IBD-like experimental models; murine colitis models	Microplastic exposure altered gut microbiota composition, reduced colonic mucin release, affected microbial metabolism, induced dysbiosis-like shifts, impaired gut barrier function, and disrupted the gut microbiota–butyrate –PPARγ axis	Connects particle exposure to dysbiosis, mucus barrier dysfunction, microbial metabolites, and short-chain fatty acid-related pathways central to IBD biology
Immune activation and host susceptibility	Liu et al.[56] Schwarzfischer et al.[57] Choi et al.[58]	Mice with intestinal immune imbalance; nanoplastic exposure in an IBD mouse model; mice exposed to polystyrene microplastics	Microplastics aggravated inflammatory damage in immune-imbalanced mice, affected macrophage-mediated inflammation inflammation and intestinal health, and induced inflammatory responses in the colon	Suggests that host immune status may modify the biological effects of microplastic and nanoplastic exposure
Particle size, polymer type, and exposure context	Li et al.[59] Schwarzfischer et al.[32] Xu et al.[60]	IBD-like zebrafish and mouse models; mice exposed to nano- and microsized polystyrene particles; nanoplastic exposure models	Microplastics and nanoplastics produced distinct adverse effects; particles crossed the intestinal barrier and accumulated in tissues; surface-labeled nanoplastics induced Crohn's ileitis-like features through epithelial injury pathways	Highlights that biological effects depend on particle size, surface properties, polymer type, and experimental context
Environmental aging and co-exposure	Shaoyong et al.[35] Huang et al.[36]	Aged polystyrene microplastics loaded with benzo[a]pyrene; co-exposure to microplastics and tetrabromobisphenol A in human gut simulation /Caco-2 models	Contaminant-loaded particles or co-exposures produced barrier injury, oxidative stress, altered epithelial responses, or gut microbiota-related effects	Emphasizes that real-world microplastic toxicity may reflect mixed exposure to particles, additives, and adsorbed pollutants

3D: three-dimensional; DSS: dextran sodium sulfate; IBD: inflammatory bowel disease; IL-1β: interleukin-1β; M cell: microfold cell; NF-κB: nuclear factor kappa B; NLRP3: NOD-like receptor protein 3; PPARγ: peroxisome proliferator-activated receptor gamma; TNBS: 2:4:6-trinitrobenzenesulfonic acid.

or deliver adsorbed chemicals that affect bacterial composition and function. In vitro gut simulation and experimental studies have linked microplastic exposure to dysbiosis-like shifts and impaired epithelial–microbial homeostasis.[36,37] More recent work has implicated disruption of the microbiota–butyrate–PPARγ axis as a potential pathway through which microplastics may exacerbate colitis-like inflammation.[38] Conversely, certain gut bacteria may also contribute to the degradation or biological mitigation of ingested microplastics, suggesting a dynamic and potentially bidirectional host–microbiota–microplastic relationship.[39] These findings are biologically relevant because reduced microbial diversity and altered short-chain fatty acid metabolism are common features of IBD; however, their translation to human disease remains uncertain.

Microplastics may also modulate mucosal immune responses indirectly through barrier injury and dysbiosis or directly through interactions with epithelial and immune cells. Such interactions may promote innate immune activation, cytokine production, epithelial–immune signaling, and inflammasome-related pathways, particularly in susceptible hosts.[40] The extent to which this occurs in human IBD is unknown, but the concept is consistent with the broader model of IBD as an exaggerated immune response to luminal and microbial stimuli.

Finally, microplastics should not be considered inert particles. They may contain plastic additives and adsorb pollutants, metals, and microorganisms onto their surfaces.[13] Upon contact with biological fluids, plastic particles may acquire a coating of proteins, lipids, and other biomolecules, often termed a biocorona, which can alter their surface properties, cellular recognition, and biological effects in ways not fully captured by studies using pristine particles.[41]

In addition, microorganisms carried on environmentally exposed plastic surfaces may potentially influence the local microbial balance independently of the particles themselves.[39] Therefore, intestinal effects attributed to microplastics may reflect a combination of particle-related injury, polymer-specific properties, biocorona formation, environmental aging, chemical co-exposures, and microbial carriage. Taken together, these mechanisms provide biological plausibility for microplastics as potential modifiers of intestinal inflammation in susceptible or already inflamed mucosal environments, although direct clinical causality remains unproven (Figure 2).

Experimental Evidence from Cellular and Animal Models

Experimental studies have provided most of the mechanistic evidence linking microplastics and nanoplastics to intestinal inflammation. These studies include intestinal epithelial cell systems, mucus and barrier models, organoid-derived epithelial tissues, gut-chip platforms, and animal models, which are summarized by mechanistic domain in Table 2.[31-38,42-60] Overall, these findings support biological plausibility but should not be interpreted as direct evidence that microplastics cause IBD in humans.

In vitro and translational models suggest that microplastics and nanoplastics can interact with intestinal epithelial surfaces and, under certain experimental conditions, cross epithelial barriers.[31,33,44] Disease-relevant human duodenum-chip models further suggest that healthy and Crohn's disease-derived epithelial systems may differ in their absorption and toxicity responses to polystyrene microplastics and nanoplastics.[45] These models provide useful human-relevant information on mucus and epithelial barrier function, but they do not fully reproduce the chronic immune, microbial, vascular, and treatment-related complexity of IBD.

Animal studies, most commonly using DSS-induced colitis models, have primarily examined whether microplastic exposure can aggravate an already inflamed intestine. Across these models, exposure has been associated with more severe colitis, increased intestinal permeability, impaired epithelial junctions, reduced mucus-related protection, and greater inflammatory injury.[46-48] Similar effects have been reported with different polymer types, including polystyrene, polyethylene, and polypropylene, suggesting that intestinal toxicity is not restricted to a single type of plastic.[47,51,54] Overall, these findings are more consistent with the amplification of pre-existing inflammation than with microplastics acting as isolated initiating causes.

Across animal and in vitro models, microplastic exposure has also been associated with dysbiosis, altered microbial metabolism, reduced mucin-related protection, oxidative stress, epithelial apoptosis, and NF-κB/NLRP3/IL-1β-related signaling.[37,38,51,52,56,57] These converging pathways are relevant to IBD but remain dependent on experimental context, dose, polymer type, and host susceptibility.

Experimental evidence nevertheless has important limitations. Many studies use selected polymers, defined particle sizes, high concentrations, short exposure periods, or pristine particles that may not reflect mixed real-world exposures. Animal colitis models reproduce only selected features of Crohn's disease and ulcerative colitis. Therefore, experimental data support biological plausibility and disease-modifying potential but do not establish clinical causality.

Human Evidence in IBD

Experimental and mechanistic data support the biological plausibility of microplastic-related intestinal injury; however, direct human evidence in IBD remains limited and predominantly observational (Table 3). Only a small number of studies have measured microplastics in the stool, serum, or intestinal tissue of patients with IBD.

The principal stool-based evidence in IBD builds on the broader observation that microplastics can be detected in human stool under controlled analytical conditions.[25] This methodological foundation was extended to an IBD population in a case-control study of 52 patients with IBD and 50 healthy controls, which reported significantly higher fecal microplastic concentrations in patients with IBD, with polyethylene terephthalate and polyamide among the most abundant polymers. Particles smaller than 50 μm were disproportionately enriched in the IBD group, and fecal microplastic burden was positively correlated with clinical disease activity.[26] However, this was a cross-sectional study using a nitric acid-based extraction protocol that may alter or degrade certain particles, and causality cannot be inferred from the results. A key limitation is that fecal microplastic burden may reflect ambient exposure, altered intestinal transit, disease-related retention, or active inflammation itself rather than a causal exposure–disease relationship. Complementary pilot data from the ongoing PLANET Study similarly link fecal microplastic burden to fecal calprotectin, independent of IBD status (Spearman's r=0.65, P=.04), although this finding is based on a small sample (n=11) and has been reported only in abstract form to date.[61]

Although more limited, tissue-based evidence provides a complementary line of investigation. Microplastics have been detected in fibrotic intestinal tissue and adjacent mesenteric adipose tissue from patients with Crohn's disease, with particle concentrations reported to correlate with the severity of intestinal fibrosis.[28] A subsequent 2026 pilot study found higher mucosal microplastic concentrations in patients with ac-

Table 3. Summary of human studies evaluating microplastics and nanoplastics in IBD and related intestinal inflammation

Study	Study design and population	Sample type	Analytical method	Main findings	Key limitations
Yan et al.[26]	Cross-sectional case-control study; 52 patients with IBD and 50 healthy controls	Feces	Multistep Fenton reagent and nitric acid digestion followed by micro-Raman spectroscopy	Mean fecal MP concentration was higher in patients with IBD than in healthy controls (41.8 vs. 28.0 particles/g dry weight). Smaller particles were enriched in the IBD group, and MP burden correlated positively with clinical disease activity.	Cross-sectional design and single fecal measurement; acid-based processing may alter some particles; residual confounding and reverse causality
Lykkemark et al.[61]	Pilot analysis within the prospective PLANET Study; 11 pregnant women, including 4 with CD	Feces collected during the third trimester	Plastic-free collection; multistep enzymatic and oxidative treatment; μ-FTIR imaging for particles >10 μm; fecal calprotectin measured by ELISA	MPs were detected in all samples. MP count correlated with fecal calprotectin after adjustment for IBD status (Spearman's r=0.65, P=.04), whereas the association with total MP mass was not statistically significant.	Pilot sample of 11 pregnant women, only 4 of whom had CD; pregnancy-specific population and single-time-point analysis
Wu et al.[28]	Cross-sectional paired-tissue study; 10 patients undergoing surgery for fibrostenotic CD	Involved and adjacent uninvolved ileum; creeping fat and adjacent mesenteric adipose tissue	Laser direct infrared imaging spectroscopy	MPs were detected in intestinal and mesenteric adipose tissues. Concentrations were higher in affected regions and correlated positively with the severity of intestinal fibrosis.	Phenotype-selected surgical cohort restricted to fibrostenotic CD; no non-IBD control group; possible inflammation-related particle retention
Cui et al.[62*]	Pilot cross-sectional study; 20 patients with active CD, 20 with CD in remission, and 10 healthy controls	Colonoscopic mucosal biopsies	High-resolution digital imaging, μ-FTIR spectroscopy, and Raman imaging	Median mucosal MP concentration was higher in active CD than in remission and healthy controls (162.7 vs. 87.31 and 61.28 particles/g, respectively) and correlated with SES-CD in active disease (r=0.628, P<.01).	Cross-sectional pilot design with limited subgroup sizes; possible sampling contamination and inflammation-related particle retention
Du et al.[63†]	Exploratory paired-tissue study; 6 individuals with colonic or terminal ileal erosions	Inflamed erosive tissue and adjacent noninflamed tissue	Histopathological confirmation of inflammation followed by Py-GC/MS quantification of PE, PVC, and PS	Total MNP mass was higher in inflamed tissue than in adjacent noninflamed tissue (1758.15 vs. 611.49 mg/kg, P<.05).	Exploratory paired-tissue study involving 6 non-IBD participants; no healthy control group; Py-GC/MS does not provide particle count, size, or morphology.
Huang et al.[64*]	Two-center prospective observational study; 241 patients with CD and 43 healthy controls; 167 patients with nonstricturing, nonpenetrating CD and no prior intestinal resection followed for a mean of 27 months	Serum	Py-GC/MS quantification of 11 MNP polymers	Serum MNP burden was higher in patients with CD than in healthy controls. Among prospectively followed patients, a higher baseline total MNP burden was independently associated with subsequent disease progression (HR 2.95, 95% CI 1.57–5.57).	Selected follow-up population; residual confounding and reverse causality; serum measurements require validation and do not establish temporality for IBD onset

*Currently reported as a conference abstract. †Supportive non-IBD-specific human study. CD: Crohn's disease; CI: confidence interval; ELISA: enzyme-linked immunosorbent assay; HR: hazard ratio; IBD: inflammatory bowel disease; MP: microplastic; MNP: microplastic and nanoplastic; PE: polyethylene; PS: polystyrene; PVC: polyvinyl chloride; Py-GC/MS: pyrolysis–gas chromatography/mass spectrometry; SES-CD: Simple Endoscopic Score for Crohn's Disease; μ-FTIR: micro-Fourier-transform infrared spectroscopy.

For clinicians, the main practical implication is communication rather than intervention. Questions about whether plastic exposure contributes to IBD may increasingly arise in clinical practice as public awareness of microplastics grows. The most accurate response is that microplastics are being investigated as potential environmental modifiers of intestinal inflammation, but they are not established causes of IBD. There is currently no evidence that reducing plastic exposure improves IBD outcomes; therefore, microplastics cannot be considered a validated therapeutic target.

Specific microplastic-avoidance measures, particularly burdensome or restrictive strategies, currently have no evidence-based role in IBD management, either as adjuncts to or alternatives to established treatment. Nevertheless, general exposure-reduction measures may be reasonable from a broader environmental and public health perspective, particularly when they are low-cost and low-risk. These may include avoiding heating food in plastic containers, reducing unnecessary use of single-use plastics, and using glass or stainless steel containers when feasible. Two existing guideline-based recommendations provide an additional complementary rationale in this context. Smoking cessation is already recommended for all patients with IBD by current national guidelines, and emerging evidence of smoking-associated microplastic exposure in the lower respiratory tract provides a further environmental rationale for avoiding tobacco exposure.[17,66] Similarly, IOIBD dietary guidance recommends limiting ultra-processed foods and selected additives, including nanoparticles, independent of any microplastic-specific concern, and highly processed or heavily packaged foods may also contribute to dietary microplastic exposure through packaging and processing.[12,13,67] However, in both cases, these associations do not demonstrate that plastic-derived particles directly influence IBD outcomes.

Evidence in pediatric IBD is particularly limited, and current data do not support pediatric-specific microplastic testing, dietary restriction, or avoidance strategies. Counseling for children and their families therefore remains centered on established nutritional and disease-management principles rather than unproven microplastic-focused restrictions. Importantly, these discussions should not distract from established IBD management, including objective monitoring of inflammation, adherence to prescribed therapy, nutritional optimization, smoking cessation, and timely treatment escalation when necessary. Currently, microplastics remain a research question rather than a clinical management tool for IBD.

Knowledge Gaps and Future Directions

Several important knowledge gaps limit the interpretation of the current literature. First, human data remain sparse, largely cross-sectional, and heterogeneous in terms of sampling and analytical protocols. Standardized methods are needed for the collection and processing of stool, blood, and intestinal tissue; sample digestion; polymer identification; particle counting; size reporting; and contamination control, including more robust approaches to quantifying inhalational exposure, which remains particularly poorly standardized.[24,68] Without harmonized protocols, comparisons across studies and populations will remain difficult.

Second, clinically integrated longitudinal designs are essential for moving beyond simple detection. Prospective cohorts are needed to determine whether microplastic burden precedes disease onset, increases during flares, decreases during remission, or reflects altered intestinal handling during active inflammation. Where clinically feasible, paired stool, blood, and intestinal tissue samples collected at the same time points would permit comparison of luminal, systemic, and mucosal particle burdens and their integration with endoscopic activity, histology, fecal calprotectin, CRP, disease phenotype, medication exposure, diet, smoking status, and environmental exposure assessments. Newly diagnosed, treatment-naïve patients may be particularly informative because they allow assessment before long-term treatment, dietary adaptation, and disease-related behavioral changes.

Encouragingly, this field is beginning to move in this direction. Registered and ongoing studies are assessing multi-matrix microplastic burden in pediatric IBD populations, segmental distribution in inflamed and noninflamed Crohn's disease biopsy samples (the OPTIC study), and integrated microbial, immune, and barrier function profiles (ClinicalTrials.gov identifiers: NCT07141238 and NCT07630883). These efforts represent an important step toward pairing exposure measurements with clinical and immunological outcome data, although the findings remain emerging and hypothesis-generating.

Third, the relationship between microplastics and specific IBD phenotypes requires further clarification. Current human evidence suggests possible associations with disease activity and fibrostenotic Crohn's disease; however, it remains unknown whether microplastic burden differs between Crohn's disease and ulcerative colitis, inflammatory and stricturing phenotypes, active and quiescent disease, or treatment-responsive and treatment-refractory patients.

A broader unanswered question is whether microplastics represent one of the unintended environmental costs of modernization. Industrial development, urbanization, and dietary westernization have improved many aspects of human life while introducing persistent synthetic materials into food systems, water sources, and air. Future reductions in plastic use may provide a natural experiment for evaluating whether lower environmental exposure has measurable effects on gastrointestinal or immune-mediated diseases. Such ecological trends should be interpreted cautiously; however, they may help generate hypotheses for IBD and other chronic inflammatory disorders.

Fourth, experimental models should better reflect real-world exposure. Many studies have used pristine particles, selected polymers, high concentrations, or short exposure periods. Two decades of broader microplastic research have underscored that methodological rigor, rather than additional descriptive detection studies alone, is now the primary need for translating environmental plausibility into clinically actionable evidence.[69] Future models should incorporate environmentally aged and mixed-polymer particles under chronic, low-dose exposure conditions that more closely approximate human exposure, ideally using disease-relevant human organoid, gut-chip, or mucosal biopsy-based systems.

Finally, intervention research requires a staged approach. After analytical validity and longitudinal associations have been established, initial feasibility studies can test whether predefined, low-risk exposure-reduction measures produce measurable changes in biological particle burden. Subsequent controlled studies, if justified, may evaluate effects on objective inflammatory biomarkers, endoscopic activity, and clinical outcomes. A reduction in measured particle burden alone would not establish therapeutic benefit. Until such evidence becomes available, microplastics remain a research target rather than an established clinical determinant of IBD.

Limitations

Several limitations should be considered when interpreting this narra-

tive review. Although the search followed a predefined strategy in terms of databases, terminology, and time frame, it was not conducted as a systematic review; source selection ultimately reflected author consensus rather than formal eligibility scoring, which introduces the possibility of selection bias and means that the emphasis placed on individual reports may not correspond to the weight they would receive in a formal evidence synthesis. The search was restricted to English-language publications, and relevant work published in other languages may have been missed. In a field expanding as rapidly as this one, positive and mechanistically striking findings are also more likely to be published and cited than null results, and this asymmetry cannot be excluded from the evidence summarized here. Furthermore, the methodological heterogeneity described above constrains not only comparisons among individual studies but also any attempt to synthesize them collectively, and the small, predominantly cross-sectional human evidence base does not permit inferences about temporality. The interpretations advanced in this review should therefore be regarded as provisional and are likely to require revision as standardized analytical methods and longitudinal human data become available.

CONCLUSION

Microplastics and nanoplastics are widespread environmental exposures with plausible relevance to intestinal inflammation. Experimental evidence suggests that these particles may disrupt the mucus–epithelial barrier, promote oxidative stress, alter microbial and immune homeostasis, and amplify inflammation, particularly in susceptible or inflamed intestinal environments. In contrast, human evidence in IBD remains limited, observational, and insufficient to establish microplastics as causal drivers or clinically useful biomarkers.

The most balanced interpretation is that microplastics may represent environmental disease modifiers or markers of altered intestinal handling rather than primary initiators of disease. As microplastic detection methods become more standardized, accessible, and integrated into human studies, their relationship with IBD may be clarified with greater precision. Until stronger longitudinal human evidence becomes available, microplastics should be regarded as an important research priority rather than a basis for disease-specific clinical recommendations.

Peer-review: Externally peer-reviewed.

Use of AI for Writing Assistance: Artificial intelligence–assisted tools were used during the preparation of this manuscript. Figures 1 and 2 were designed with the help of ChatGPT (version 5.6, OpenAI). ChatGPT and Claude (Opus 4.8 and Opus 5, Anthropic) were additionally used for English language editing and for assistance with manuscript structuring and formatting to meet journal requirements. No AI tool was used to conduct the literature search, select the included sources, interpret the evidence, or generate the scientific arguments presented in this review. The authors reviewed and edited the resulting text and take full responsibility for the content of the manuscript.

Author Contribution: Concept – Y.G.; Design – Y.G.; Supervision – T.K.; Analysis and/or Interpretation – Y.G., İ.E.Ş., T.K.; Literature Review – Y.G., İ.E.Ş.; Writing – Y.G.; Critical Review – Y.G., T.K.

Conflict of Interest: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

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