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Microplastic and Nanoplastic Exposures, Gut Microbiome Disruption, and Nutritional Health: A One Health Systematic Review of Human and Translational Evidence-Systematic Review

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Abstract: ***Background**: Food, drinking water, food-contact materials, and infant-feeding products are important interfaces for exposure to microplastics, nanoplastics, and associated contaminants. Their effects on the gut microbiome and host metabolism create a One Health problem linking nutritional, human, animal, and ecosystem health. Objective: To synthesize recent human and translational evidence on microplastic exposure, emphasizing gut microbiome disruption, nutritional pathways, One Health, and multi-omics. **Methods**: PubMed/MEDLINE and Europe PMC were searched for English-language reports published from January 1, 2021, through September 12, 2026; supplementary citation searching identified additional reports. Eligibility was restricted to primary studies examining a defined microplastic or nanoplastic exposure together with host-associated intestinal microbiota, experimentally generated omics data relevant to host response, or a human nutritional-exposure pathway. Environmental and mechanistic publications not meeting these primary criteria were retained only for contextual interpretation. **Results**: The reconstructed evidence set comprised 29 primary studies and 16 contextual sources. Primary evidence described context-dependent dysbiosis, altered microbial metabolites, epithelial injury, oxidative stress, inflammation, and perturbation of gut-organ axes. Food processing, heating, storage, and infant-feeding materials emerged as potentially modifiable exposure points. Fiber-, prebiotic-, probiotic-, and polyphenol-oriented strategies remain preclinical or hypothesis-generating; no omics-guided or dietary treatment is validated for routine care. **Conclusions**: Current evidence supports exposure reduction, standardized measurement, and prospective human research. Multi-omics may improve mechanistic characterization and trial stratification, but clinical recommendations must not be inferred directly from animal, aquatic, or in-vitro findings.*

Keywords: microplastics; gut microbiome; nutritional exposure; multi-omics; One Health; precision prevention

1. Introduction

Plastic pollution is no longer solely an environmental-waste problem. Fragmentation, abrasion, weathering, and industrial use generate microplastics, generally defined as plastic particles smaller than 5 mm, and nanoplastics, commonly operationalized in the submicrometre range. Humans and animals encounter these particles through ingestion, inhalation, and contact with contaminated matrices. Particles may also carry additives, adsorbed metals, persistent organic pollutants, antimicrobials, and microorganisms, making the biologically relevant exposure a dynamic particle-chemical-microbial complex rather than an inert polymer alone [1-4].

The gastrointestinal tract is a primary interface for ingested particles. Experimental studies indicate that polymer chemistry, size, shape, weathering, surface charge, protein or eco-corona, dose, diet, age, and baseline microbiota can modify toxicity. Reported consequences include epithelial injury, mucus alteration, oxidative stress, inflammatory signaling, microbial community shifts, and changes in microbial and host metabolites [5-16]. The direction of individual taxonomic changes is not universal, but disruption of community function and metabolite production is more consistent than any single microbial signature.

Nutrition is relevant at three levels: foods and beverages can carry particles; preparation, heating, and storage can add particles from contact materials; and dietary pattern can modify barrier integrity, microbial metabolism, inflammation, and metabolic susceptibility. Early-life nutrition deserves particular attention because breastmilk, formula, storage bags, bottles, and complementary foods may contribute to exposure during a vulnerable developmental window. These observations support exposure reduction without undermining breastfeeding or dietary adequacy.

Microplastic exposure is embedded within a wider environmental exposome. Air pollution, metals, pesticides, per- and polyfluoroalkyl substances, endocrine-disrupting chemicals, pharmaceutical residues, and antimicrobial contamination can affect microbiota and host pathways independently, while plastics can act as vectors or interactive surfaces. Such mixtures complicate conventional single-agent toxicology and may amplify inflammatory, endocrine, metabolic, and antimicrobial-resistance effects [6,10,17-20].

One Health recognizes that the health of humans, domestic and wild animals, plants, and ecosystems is interdependent. This framework is particularly appropriate for plastic pollution because the same particles circulate through

wastewater, soil, crops, food webs, livestock, wildlife, indoor air, and human tissues. Omics technologies can connect these compartments by measuring microbial genes, host responses, metabolites, proteins, lipids, and epigenetic changes. The clinical promise is substantial, but the distinction between biomarker discovery and validated treatment must remain explicit.

This review aimed to answer four questions: how do microplastics and major environmental co-exposures affect microbiomes and host systems; how do food, food-contact materials, and nutritional status shape exposure and susceptibility; how should One Health organize prevention, surveillance, and research; and how can omics contribute to nutritional risk stratification and future therapy without overstating current clinical readiness?

2. Methods

2.1 Design and reporting

The review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 framework [21]. The protocol was developed a priori but was not registered in PROSPERO. A restricted PECO framework was applied: populations comprised humans, human-derived gastrointestinal models, and translational animal models directly relevant to host intestinal microbiota or nutrition; exposures were defined microplastics or nanoplastics, alone or with co-contaminants; comparators were unexposed, lower-exposure, or alternative-polymer groups; and eligible outcomes were host-associated intestinal microbiota, experimentally generated omics profiles relevant to host response, nutritional exposure, epithelial-barrier injury, or linked immune and metabolic outcomes.

2.2 Information sources and search strategy

PubMed/MEDLINE and Europe PMC were searched from January 1, 2021, through September 12, 2026. Reference lists of eligible reviews and key studies were also examined to identify reports not captured in the electronic-search export. The electronic strategy combined microplastic and nanoplastic terms with microbiome, omics, One Health, barrier, inflammatory, and nutritional concepts. The complete reproducible electronic strategies and date limits are provided in Appendix 1.

2.3 Eligibility and selection

Primary peer-reviewed studies were eligible when they evaluated a defined exposure to microplastics or nanoplastics and reported at least one core outcome: host-associated intestinal microbiota or microbiome; experimentally generated metagenomic, transcriptomic, proteomic, metabolomic, lipidomic, epigenomic, or integrated multi-omics data relevant to host response; or microplastic exposure through foods, beverages, food-contact materials, breastmilk, infant formula, or related nutritional pathways. Human studies and human-derived gastrointestinal models were prioritized. Animal studies were eligible only when they directly linked exposure to intestinal microbiota, host-response omics, barrier or metabolic outcomes, or a defined dietary intervention. Purely environmental occurrence studies, plastisphere studies without host-health relevance, plant and soil studies without a direct nutritional pathway, single-organ toxicology without a core review-domain outcome, reviews, editorials, protocols without results, and non-peer-reviewed reports were excluded from the primary synthesis. Selected non-primary sources were retained separately for methodological and One Health context. Records were deduplicated sequentially by PubMed identifier, digital object identifier, and normalized title. During revision, a rule-based, AI-assisted audit was used to reconcile candidate citations with the screening register and to flag records for eligibility review; AI output did not independently determine inclusion. Both authors reviewed and validated the 45 retained-report decisions against the restricted eligibility criteria and resolved classification differences by consensus. Primary and contextual evidence were recorded separately in a structured screening register maintained by the authors.

2.4 Data extraction and quality appraisal

Data-extraction fields comprised year, country, design or model, polymer, particle dimensions, exposure route, dose, duration, co-contaminants, analytical method, microbiome platform, omics layer, nutritional pathway, barrier and immune endpoints, principal findings, and limitations. Risk of bias was evaluated by design: human observational and quasi-experimental studies were assessed using JBI/Newcastle-Ottawa domains; animal experiments were assessed using SYRCLE domains; and in-vitro and analytical exposure studies were evaluated using predefined domains for particle characterization, blank and contamination control, replication, dose relevance, standardized outcome assessment, and completeness of reporting. Judgments were based on accessible full reports and authoritative article records; safeguards not explicitly reported were rated unclear rather than assumed. Newcastle-Ottawa and predefined in-vitro judgments were categorized as low, moderate, or high risk, whereas SYRCLE domains were categorized as low, high, or unclear risk. No study was excluded solely because of risk of bias. Meta-analysis was not undertaken because exposure metrics, polymers, particle sizes, models, sequencing pipelines, and outcomes were not sufficiently comparable. Study-level characteristics are reported in Supplementary Table S1 and the complete domain-level risk-of-bias judgments in Supplementary Table S2.

3. Results

3.1 Study selection and evidence landscape

The electronic searches identified 7,736 records, comprising 3,916 from PubMed/MEDLINE and 3,820 from Europe PMC. Citation searching added 11 reports. After removing 3,513 duplicates, 4,234 unique records were evaluated under the restricted criteria; 4,189 were excluded and 45 reports were retained. Twenty-nine primary studies entered the systematic synthesis and 16 non-primary publications were used only for contextual interpretation. Both authors validated the retained-report decisions.

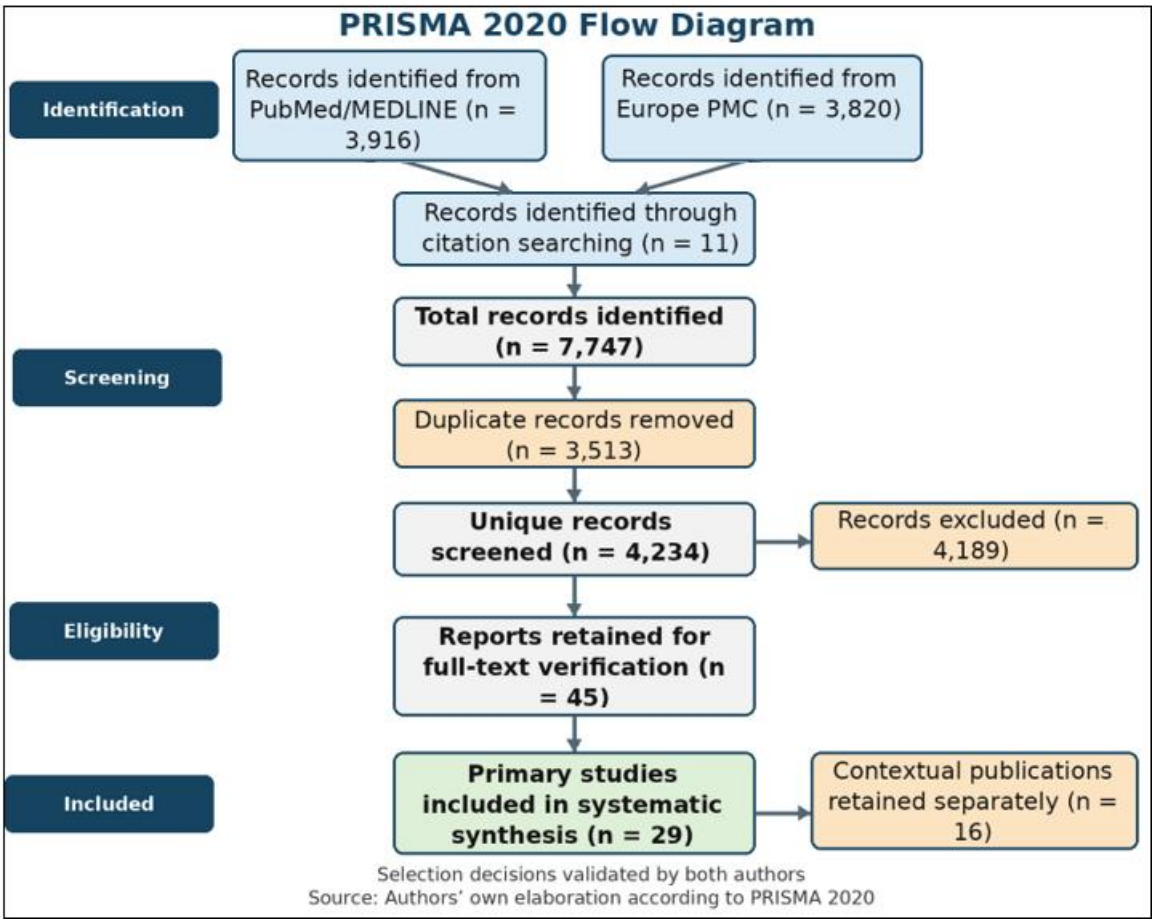

Figure 1: PRISMA 2020 flow diagram of study selection
Source: Authors' own elaboration according to PRISMA 2020.

Most primary evidence came from controlled animal, aquatic, or human-derived in vitro models. Human studies were fewer and included exposure detection, cross-sectional microbiome associations, pregnancy and infant-feeding biospecimens, food-contact exposure, and prospective clinical associations. Polystyrene was the most frequently studied polymer, followed by polyethylene, polyethylene terephthalate, polyvinyl chloride, polylactic acid, polycaprolactone, and mixed particles. Study heterogeneity was substantial and precluded pooled effect estimates.

Risk-of-bias assessment identified recurrent limitations across study designs. Among the seven human observational or quasi-experimental studies in the primary synthesis, the prospective carotid-plaque cohort was rated low risk and the remaining studies moderate risk on the Newcastle-Ottawa framework, principally because of small or selected samples, contamination-sensitive exposure assessment, nonrandomized designs, and residual confounding. The 13 controlled animal studies were rated unclear overall because sequence generation, allocation concealment, random housing, and blinded assessment were generally not reported, although baseline comparability and outcome completeness were usually adequate. The nine in-vitro or analytical studies met the predefined threshold for low risk, while still showing recurring limitations in contamination-control reporting, dose relevance, or external validity. These judgments informed interpretation but were not used as exclusion criteria. Complete study-level and domain-level assessments are provided in Supplementary Table S2.

Table 1: Evidence domains and translational relevance

Domain	Typical evidence	Strength	Main limitation
Human observational	Internal exposure; fecal, placental, meconium, nasal or tissue measurements; clinical associations	Direct relevance; real-world mixtures	Small cohorts; contamination risk; cross-sectional design; uncertain dose-response
Human-derived models	Adult, toddler, infant or simulated gastrointestinal systems; organoids and cell co-cultures	Controlled mechanistic testing with human material	Short duration; simplified exposure; donor effects
Mammalian in vivo	Gut barrier, microbiota, liver, brain, lung, cardiovascular and reproductive outcomes	Whole-organism pathways and organ axes	Often high doses; mostly polystyrene; limited chronic low-dose designs
Aquatic and terrestrial sentinels	Fish, invertebrates, soil organisms and plants; co-exposure ecology	Strong One Health relevance; ecosystem function	Species-specific translation; variable environmental realism
Environmental microbiomes	Plastisphere, soil, wastewater and resistance-gene ecology	Tracks transport, biofilms and antimicrobial resistance	Association dominates; functional validation uncommon

Source: Authors' own elaboration based on the evidence included in this systematic review.

3.2 Microbiome and intestinal barrier effects

Human-derived gastrointestinal models consistently showed that particles can modify microbial community structure and function, although responses depended on polymer and donor. Polyethylene exposure in adult and infant colon models altered luminal more than mucosal communities, reduced selected short-chain fatty acids, changed volatile organic compounds, and modified aryl-hydrocarbon-receptor activity [11,12]. Polyethylene terephthalate, polylactic acid, and polycaprolactone exposures produced polymer-specific changes in diversity, taxa, and predicted metabolic pathways [13,14]. A preschool pilot study associated fecal microplastics with altered gut microbial composition, but its observational design could not establish directionality [15]. Placental and meconium studies suggested that maternal-fetal exposure and early microbial ecosystems may be linked, yet contamination control, small samples, and temporal ambiguity remain major concerns [16, 22].

Across mammalian studies, recurring functional patterns included reduction of mucus or tight-junction integrity, increased permeability, oxidative stress, inflammatory cytokine signaling, altered bile-acid homeostasis, and changes in taxa involved in short-chain fatty acid metabolism [7-10,23-26]. Taxonomic findings were inconsistent: Firmicutes, Bacteroidota, Proteobacteria, Desulfobacterota, Lactobacillus, Akkermansia, and other groups moved in different directions across experiments. This variability argues against using a single taxon or the Firmicutes-to-Bacteroidota ratio as a diagnostic biomarker. Metagenomic functions, metabolites, barrier markers, and host-response modules are more promising endpoints.

3.3 Environmental mixtures and the Trojan horse problem

Microplastics can release additives and adsorb co-contaminants, including phthalates, bisphenols, brominated flame retardants, metals, pesticides, and antimicrobials. In vitro co-exposure to polyethylene particles and tetrabromobisphenol A modified both Caco-2 responses and human gut microbiota [6]. Phthalates released from particles suppressed microbial metabolic activity and differently affected luminal and mucosal communities [10]. Animal and aquatic studies reported that combined exposure to tetracycline, sulfamethoxazole, arsenic, cadmium, or brominated compounds can intensify intestinal injury, oxidative stress, metabolic disruption, and microbial restructuring [17-20].

The plastisphere adds a microbial dimension. Plastic surfaces select biofilms, can concentrate pathogens, and may facilitate persistence or horizontal transfer of antimicrobial-resistance genes in wastewater, agricultural soil, and aquatic systems. The strength and direction of this effect depend on polymer aging, nutrient availability, contaminant loading, hydrodynamics, and the surrounding microbial pool. Thus, plastics should be treated as mobile ecological habitats and chemical vectors, not only as particles.

3.4 Systemic effects and organ axes

Experimental evidence links gut disruption with downstream organ effects. The gut-liver axis is the best characterized: microplastic exposure has been associated with altered bile acids, hepatic lipid accumulation, cholestatic signaling, oxidative injury, and inflammatory responses [8,23,24]. Studies of the gut-brain describe changes in neurotransmitter-related metabolites, neuroinflammation, behavior, and vulnerability to neurological pathology, but most use doses or particle preparations that limit human inference [25,26]. Gut-lung and gut-reproductive pathways are emerging, with immune and metabolic intermediates proposed as links.

Human tissue-detection studies support internal exposure but do not by themselves demonstrate disease. Microplastics have been reported in blood, lung tissue, placenta, meconium, and atherosclerotic plaques [27-31]. A prospective study of carotid plaque reported an association between detected micro- and nanoplastics and subsequent cardiovascular events [30]. This clinically important observation remains observational; residual confounding, exposure measurement, laboratory contamination, and replication must be considered before causal or therapeutic conclusions are drawn.

3.5 What multi-omics adds

Table 2: Omics contributions to microplastic and environmental health research

Omics layer	Measurement	Principal value	Key caveat
Metagenomics and metatranscriptomics	Taxa, genes, pathways and active microbial functions	Functional dysbiosis; resistance and degradation genes	Batch effects; compositionality; incomplete databases
Host transcriptomics and epigenomics	Stress, barrier, immune and endocrine regulation	Response signatures and susceptible phenotypes	Tissue specificity; causal direction; cell-mixture effects
Proteomics	Enzymes, cytokines, structural proteins and pathway activation	Closer to phenotype than RNA alone	Dynamic range; platform variability
Metabolomics and lipidomics	SCFAs, bile acids, amino acids, lipids, xenobiotic metabolites	Mechanistic links across gut-organ axes	Diet and medication confounding; annotation gaps
Exposomics and adductomics	Mixture burden and molecular fingerprints of exposure	Real-world co-exposure assessment	Cost; harmonization; uncertain reference ranges
Integrated multi-omics	Cross-layer networks and candidate causal mediators	Biomarker panels and intervention stratification	Overfitting; small samples; need external validation

Source: Authors' own elaboration based on the evidence included in this systematic review.

Integrated microbiome-metabolome studies strengthen biological interpretation by linking community shifts to bile acids, short-chain fatty acids, amino acids, lipids, oxidative-stress products, and energy metabolism. Transcriptomics and proteomics identify epithelial, immune, endocrine, and organ-specific response pathways. Exposomics can place particles within the total mixture of chemicals, diet, social conditions, and physical exposures. Cross-species integration can then test whether conserved pathways recur in humans, livestock, wildlife, plants, and laboratory models.

3.6 One Health position and operational response

The One Health response is primarily preventive and systems-based. It prioritizes upstream reduction in plastic production and unnecessary use, safer material and additive design, control of industrial and textile emissions, improved wastewater and storm-water capture, safer food-contact practices, monitoring of agricultural inputs, and protection of high-risk workers and communities. Health surveillance should connect environmental measurements with veterinary, wildlife, food-chain, and human biomonitoring rather than maintaining isolated datasets [32-35].

Table 3: One Health action matrix

Action level	Core measures	Expected contribution
Source control	Polymer and additive redesign; reduce avoidable single-use plastics; emission standards	Lower exposure across all compartments
Environmental surveillance	Air, water, soil, wastewater, food-web and plastisphere monitoring	Maps transport, hotspots, pathogens and resistance genes
Animal and ecosystem health	Sentinel species, livestock, aquaculture, crop and soil microbiome monitoring	Early warning and food-system protection
Human biomonitoring	Validated particle analytics plus exposomics and clinical phenotyping	Defines internal burden and susceptible groups
Data integration	Shared metadata, reference materials, ontologies and interoperable repositories	Enables cross-species causal inference
Risk communication	Calibrated precaution without unsupported detoxification claims	Supports feasible behavior and policy change

Source: Authors' own elaboration based on the evidence included in this systematic review.

3.7 Therapeutic and preventive implications of omics medicine

No pharmacological treatment is approved specifically to remove microplastics from the human body or reverse a defined microplastic-associated clinical syndrome. Omics-guided medicine should therefore be described as a translational pathway rather than current standard care. Its near-term value lies in identifying exposure-response phenotypes, detecting early barrier or inflammatory injury, defining susceptible subgroups, and distinguishing particle effects from co-contaminant and lifestyle effects.

Preclinical interventions include dietary fiber and prebiotics, selected probiotics or microbial consortia, antioxidant and anti-inflammatory compounds, bile-acid pathway modulation, and engineered microbial or biomaterial systems capable of binding or degrading specific polymers [36-40]. Some studies report partial restoration of microbial composition, metabolite profiles, barrier integrity, or organ injury. However, effects are strain-, polymer-, dose-, and model-specific. Human trials must establish safety, clinically meaningful outcomes, and the possibility that degradation can generate smaller or more bioavailable particles before these strategies can be recommended.

Table 4: Omics-enabled intervention pathway

Intervention domain	Omics input	Potential use	Current readiness
Exposure reduction	Exposome profile plus source attribution	Prioritize dominant routes and co-contaminants	Feasible now when analytical methods are validated
Dietary or microbiome support	Baseline microbiome and metabolome	Select and monitor fiber, prebiotic or probiotic trials	Experimental for microplastic-specific disease
Host pathway modulation	Transcriptomic, proteomic and lipidomic injury signatures	Identify oxidative, barrier, immune or bile-acid targets	Preclinical; avoid empirical off-label claims
Biodegradation or sequestration	Metagenomic enzymes and polymer-specific assays	Design microbial or biomaterial removal strategies	Environmental and preclinical stage
Precision surveillance	Integrated exposure, omics and clinical longitudinal data	Detect high-risk phenotypes and early effects	Research use; needs validated thresholds

Source: Authors' own elaboration based on the evidence included in this systematic review.

3.8 Nutritional exposure and dietary strategies

Food and nutrition are both exposure pathways and modifiers of biological response. Particles have been detected in breastmilk and infant formula, while breastmilk-storage bags can release particles during routine use [41-43]. These findings do not establish that formula feeding is safer than breastfeeding, nor do they justify restricting nutritionally important foods. They instead identify preventable contact points: limiting unnecessary heating in plastic, avoiding visibly damaged containers, following manufacturer instructions, and preferring suitable non-plastic materials when feasible and safe.

Dietary fiber may support mucus, short-chain-fatty-acid production, intestinal transit, and microbial resilience, providing a plausible countermeasure to particle-associated barrier and metabolic effects [44]. Probiotics and related microbiome-directed approaches have also been proposed and show encouraging preclinical signals [36-38,45]. Nevertheless, evidence is strain- and model-specific, and no fiber, probiotic, supplement, or restrictive diet has been

shown in clinical trials to remove microplastics or prevent a defined microplastic-associated disease. Nutritional recommendations should therefore remain consistent with established healthy-diet guidance and should not displace source control.

Nutrigenomics, metagenomics, metabolomics, and lipidomics can clarify why responses differ according to diet quality, life stage, obesity, micronutrient status, and baseline microbiota. Repeated dietary assessment linked to food-source analytics and multi-omics could distinguish exposure reduction from improved host resilience. This design is especially relevant in pregnancy, infancy, metabolic disease, and food insecurity, where poorly designed avoidance advice could cause greater harm than the uncertain exposure it seeks to reduce.

Table 5: Nutrition-focused exposure and intervention framework

Nutritional domain	Exposure or mechanism	Evidence calibrated response
Food and water	Particles in ingredients, processing, beverages, and drinking water	Quantify source contribution; improve production and filtration controls
Food contact	Packaging, storage, abrasion, and heating	Avoid unnecessary heating in plastic; replace damaged containers; validate alternatives
Early-life feeding	Breastmilk, formula, storage bags, bottles, and preparation water	Protect breastfeeding; reduce avoidable contact; standardize infant-product testing
Diet-microbiome axis	Fiber, polyphenols, fermented foods, and microbial metabolites	Support dietary adequacy and test resilience mechanisms in controlled trials
Metabolic susceptibility	Obesity, insulin resistance, bile-acid and lipid pathways	Integrate nutritional phenotype with exposomics and longitudinal outcomes
Targeted intervention	Prebiotics, probiotics, and postbiotics	Research use only until strain-specific efficacy and safety are clinically validated

Source: Authors' own elaboration based on the evidence included in this systematic review.

The integrated relationships among shared microplastic exposures, the gut–microbiome interface, systemic effects, and One Health–based prevention are summarized in Figure 2.

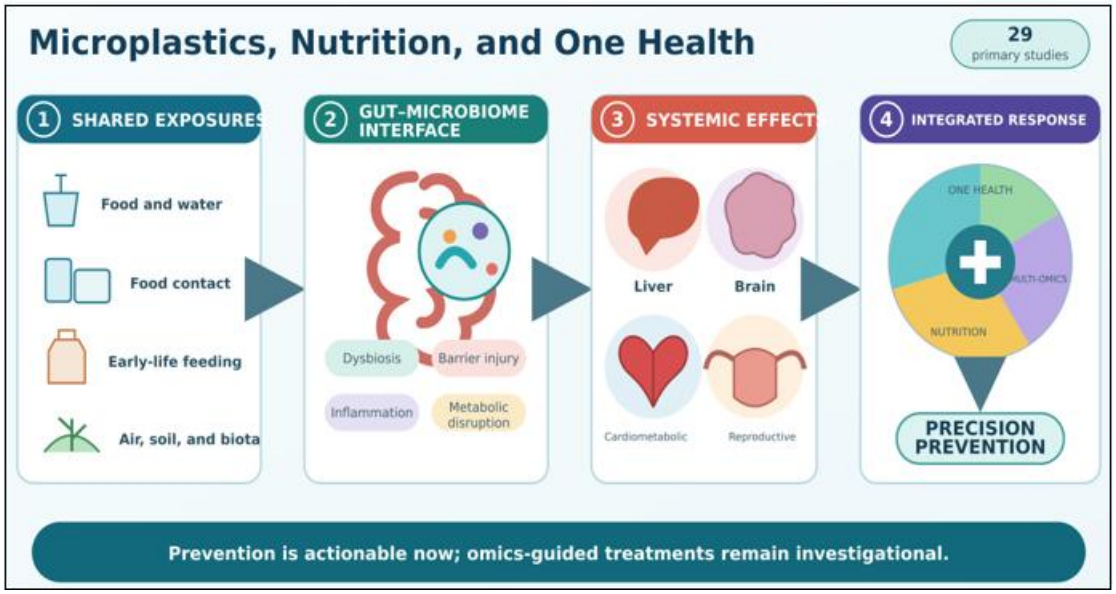

Figure 2: Integrated framework linking microplastic exposure, the gut–microbiome interface, systemic effects, and One Health prevention

Source: Authors' own elaboration for this systematic review and its 29 included primary studies.

Shared exposure pathways converge at the gut–microbiome interface, where dysbiosis, barrier injury, inflammation, and metabolic disruption may contribute to hepatic, neurological, cardiometabolic, and reproductive effects. A combined One Health, nutrition, and multi-omics response supports precision prevention, while omics-guided treatments remain investigational.

4. Discussion

This synthesis indicates that microplastics can perturb microbial ecosystems and host pathways, but certainty differs sharply by level of evidence. The most reproducible mechanistic pattern is not a universal taxonomic dysbiosis signature; it is a network of particle-dependent barrier stress, oxidative and inflammatory signaling, microbial functional change, and altered metabolite exchange. These effects plausibly connect the gut with liver, brain, lung, cardiovascular, and reproductive systems. Nevertheless, much of the evidence derives from polystyrene spheres, short experiments, and doses that may not reproduce everyday exposure [5-20,22-26,37-39].

Other environmental factors are not peripheral confounders. Plastic additives and adsorbed chemicals may account for part of the observed toxicity; diet, age, medications,

infection, socioeconomic conditions, and baseline microbiome alter susceptibility. Antimicrobial residues and plastic biofilms also connect pollution with antimicrobial resistance. A reductionist particle-only model can therefore misclassify both hazard and intervention opportunity. Mixture-aware exposomics and factorial experimental designs are required [6,9,10,18-20,32,35].

One Health contributes a governance logic as well as a biological model. Source reduction and ecosystem remediation prevent exposure more broadly than downstream clinical treatment. Sentinel organisms and environmental microbiomes can provide early signals, while shared analytical standards allow comparison across water, food, animals, and humans. The framework also guards against shifting burdens: a substitute polymer should not be considered safer merely because it is biodegradable if its fragments, additives, or degradation products disrupt microbiomes or metabolic pathways [32,35,36,40].

Omics can transform the field if used with rigorous causal design. First, exposure measurements should be linked to repeated multi-omics sampling and clinical phenotypes. Second, mediation analysis, negative controls, and experimental validation should distinguish markers from causal intermediates. Third, candidate signatures must be replicated across laboratories and populations. Fourth, machine-learning models require preregistration, appropriate validation, interpretable features, and assessment across sex, age, geography, diet, and socioeconomic strata. Finally, clinical trials should test interventions against patient-important endpoints, not only taxonomic normalization [18-20,23-26,36,40].

A nutrition-centered interpretation strengthens clinical relevance but also exposes evidence gaps. Dietary intake, food preparation, packaging, and drinking-water sources are often incompletely measured, even though they may influence both particle burden and microbiome outcomes. Future studies should combine validated dietary assessment with particle analysis, nutritional biomarkers, and multi-omics. Trials of fiber, probiotics, or other dietary strategies should include exposure measures and clinically meaningful metabolic, gastrointestinal, and inflammatory outcomes rather than assuming that microbiome change alone represents benefit [15-17,22,41-45].

4.1 Research priorities

- Establish harmonized reference materials, blanks, contamination controls, recovery experiments, and reporting standards for particle detection in biological tissues.
- Create longitudinal pregnancy, childhood, occupational, and chronic-disease cohorts with repeated exposure, microbiome, multi-omics, and clinical measurements.
- Use environmentally realistic mixtures, aged particles, fibers and irregular fragments rather than relying predominantly on pristine polystyrene spheres.
- Integrate metagenomics with metabolomics, host transcriptomics or proteomics, and validated barrier and immune outcomes.
- Develop cross-species One Health surveillance capable of linking wastewater, soil, food, animals, wildlife and human exposure.
- Test microbiome-directed interventions in phased studies with predefined safety, exposure, mechanistic, and clinical endpoints.

4.2 Clinical Interpretation

Clinicians and dietitians should neither dismiss exposure concerns nor diagnose a microplastic-related disorder from unvalidated commercial tests. At present, care should focus on established diagnoses, nutritionally adequate dietary patterns, safe food handling, and practical exposure reduction when it does not create nutritional, financial, or other health harms. Breastfeeding should remain supported. Omics results obtained in research settings should not be converted automatically into restrictive diets, supplements, antimicrobial regimens, chelation, or so-called detoxification treatments. The appropriate clinical posture is precautionary, evidence-calibrated, and attentive to vulnerable periods such as pregnancy and childhood [1,5,27-31,34,41-45].

4.3 Limitations

The review was limited to English-language literature and two biomedical databases supplemented by citation searching. Broad One Health evidence is heterogeneous and may be indexed outside biomedical databases. Publication bias toward harmful findings is likely. Polymer characterization, dose units, contamination controls, sequencing platforms, and omics pipelines varied markedly. The inclusion of diverse species improved systems-level interpretation but reduced direct clinical comparability. The rapid expansion of the literature in 2025-2026 means that conclusions, especially for human cohorts and intervention studies, require periodic updating [5,35,36,40].

5. Conclusions

Microplastics and nanoplastics interact with food systems, microbiomes, chemical co-contaminants, and host molecular pathways across human, animal, and environmental systems. Current evidence supports biological plausibility for dysbiosis, barrier dysfunction, inflammation, oxidative stress, and metabolic disturbance, while human causal inference and safe exposure thresholds remain insufficient. Nutrition is central to exposure prevention, susceptibility assessment, and intervention research, but no microplastic-specific diet or supplement is clinically validated. One Health offers the appropriate framework because prevention, monitoring, and remediation must operate across shared ecosystems and food chains. The most defensible strategy combines upstream source control, safe food-contact practices, dietary adequacy, standardized analytics, longitudinal human research, and carefully staged nutrigenomic and multi-omics trials.

Declarations

Ethics approval: Not applicable because this study synthesized previously published literature and involved no new human or animal participants.

Consent for publication: Not applicable.

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Conflicts of interest: The authors declare no conflicts of interest.

Data availability: Supplementary Table S1 reports the study-level characteristics; Supplementary Table S2 provides the complete design-specific risk-of-bias assessment. The reproducible search strategies are reported in Appendix 1. The structured screening register is retained by the authors and can be provided upon reasonable request. No new participant-level dataset was generated.

Author contributions: Vicente Manuel Martinez Cardenas: Conceptualization, Methodology, Investigation, Data curation, Writing – original draft, Visualization, and Project administration. Vivian Rosario Mena Miranda: Conceptualization, Methodology, Investigation, Validation, Data curation, Writing – review and editing, and Supervision. Both authors reviewed and validated the 45 retained-report decisions, resolved classification differences by consensus, approved the final version, and agreed to be accountable for all aspects of the work.

Abbreviations

AhR: aryl hydrocarbon receptor; AMR: antimicrobial resistance; DNA: deoxyribonucleic acid; GI: gastrointestinal; MP: microplastic; NP: nanoplastic; PA: polyamide; PCL: polycaprolactone; PE: polyethylene; PECO: Population Exposure Comparator Outcome; PET: polyethylene terephthalate; PLA: polylactic acid; PP: polypropylene; PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; PS: polystyrene; PVC: polyvinyl chloride; RNA: ribonucleic acid; SCFA: short-chain fatty acid; TBBPA: tetrabromobisphenol A.

Appendix 1 Reproducible search strategy

PubMed/MEDLINE core search: ((microplastic*[Title/Abstract] OR nanoplastic*[Title/Abstract]) AND (microbiome* [Title/Abstract] OR microbiota*[Title/Abstract] OR metagenom*[Title/Abstract] OR transcriptom* [Title/Abstract] OR proteom*[Title/Abstract] OR metabolom* [Title/Abstract] OR lipidom*[Title/Abstract] OR epigenom* [Title/Abstract] OR exposom*[Title/Abstract] OR "One Health"[Title/Abstract] OR barrier[Title/Abstract] OR inflammation[Title/Abstract])) AND ("2021/01/01"[Date - Publication] : "2026/09/12"[Date - Publication]).

Co-exposure block: AND (plasticizer*[Title/Abstract] OR phthalate*[Title/Abstract] OR bisphenol*[Title/Abstract] OR metal* [Title/Abstract] OR pesticide* [Title/Abstract] OR PFAS[Title/Abstract] OR pharmaceutical*[Title/Abstract] OR antibiotic*[Title/Abstract] OR antimicrobial resistance [Title/Abstract] OR air pollution [Title/Abstract] OR particulate matter [Title/Abstract] OR endocrine disrupt*[Title/Abstract]). Europe PMC used equivalent free-text terms and the same date limits.

Nutrition block: AND (food*[Title/Abstract] OR diet*[Title/Abstract] OR nutrition*[Title/Abstract] OR drinking water [Title/Abstract] OR dietary fiber [Title/Abstract] OR probiotic*[Title/Abstract] OR prebiotic*[Title/Abstract] OR polyphenol*[Title/Abstract] OR food contact [Title/Abstract] OR packaging [Title/Abstract] OR heating [Title/Abstract] OR breastmilk [Title/Abstract] OR infant formula [Title/Abstract] OR obes*[Title/Abstract] OR metabolic health [Title/Abstract]). Europe PMC used equivalent free-text terms and the same date limits.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation and revision of this work, the authors used OpenAI ChatGPT (Codex) to assist with language editing, document organization, eligibility-rule implementation, screening-register reconciliation, and structured risk-of-bias tabulation. The authors reviewed and edited all outputs, validated the retained-study decisions, and take full responsibility for the content of the publication. The graphical abstract was rendered from author-reviewed vector elements using SVG and Inkscape; no generative image model was used, and no scientific data were generated or altered by AI.

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Supplementary Tables

Microplastic and Nanoplastic Exposures, Gut Microbiome Disruption, and Nutritional Health

A One Health Systematic Review of Human and Translational Evidence

Supplementary Table S1 Characteristics and principal findings of the included primary studies

Study and country	Design, model, and population	Exposure characteristics	Assessment, principal findings, and limitations
Stock et al., 2022 [4]; Germany	In-vitro intestinal cell models	Polystyrene micro- and nanoplastics differing in size, surface characteristics, concentration, and dispersant; no co-exposure	Cellular, barrier, inflammatory, and oxidative-stress assessments. Effects depended on particle size, surface, concentration, and dispersant. Limitations: in-vitro design and uncertain relevance to chronic human exposure.
Huang et al., 2021 [6]; China	In-vitro Caco-2 cells and human gut microbiota	Polyethylene microplastics co-exposed with	Microbiota and cellular oxidative, mitochondrial, and inflammatory assessments. Combined exposure altered

Study and country	Design, model, and population	Exposure characteristics	Assessment, principal findings, and limitations
		tetrabromobisphenol A	microbial composition and intensified cellular injury. Limitations: short-term exposure and simplified intestinal environment.
Djouina et al., 2022 [7]; France	Controlled mouse study	Polyethylene microbeads, approximately 36 and 116 µm; 100 µg/g of food orally for 6 weeks; no chemical co-exposure	Gut histology, immune markers, and microbiota profiling. Exposure altered intestinal morphology, immune responses, and microbiota in a size-dependent manner. Limitations: animal model and experimental dose.
Luo et al., 2022 [8]; China	Dextran-sulfate-sodium experimental colitis mouse model	Orally administered polystyrene microplastics	Gut microbiota, intestinal-barrier, and hepatic inflammatory assessments. Exposure aggravated colitis, dysbiosis, barrier dysfunction, and hepatic inflammation. Limitation: disease-specific animal model.
Wang et al., 2023 [9]; China	Controlled mouse study	Oral exposure to polystyrene microplastics combined with tetracycline	Intestinal histology and gut microbiota profiling. Combined exposure produced intestinal injury and microbial alterations differing from either exposure alone. Limitations: animal model and uncertain environmental relevance of doses.
Yan et al., 2022 [10]; China	In-vitro human luminal and mucosal intestinal microbiota model	Microplastics releasing dimethyl, dibutyl, and diethylhexyl phthalates	Microbial composition and metabolic activity. Released phthalates inhibited microbial metabolism and affected luminal and mucosal communities differently. Limitations: in-vitro design and absence of systemic responses.
Fournier et al., 2023 [11]; France	Adult human in-vitro gastrointestinal and colonic models	Repeated polyethylene-microplastic exposure without additional co-exposure	16S rRNA sequencing, short-chain fatty acids, gases, volatile metabolites, and epithelial-barrier assays. Exposure produced donor-dependent microbial and metabolic changes. Limitations: few donors and artificial conditions.
Fournier et al., 2023 [12]; France	Infant human in-vitro gut model containing immature microbiota	Repeated polyethylene-microplastic exposure without additional co-exposure	16S rRNA sequencing, fermentation metabolites, and epithelial-barrier assessment. Exposure altered immature microbial communities and metabolic activity. Limitations: in-vitro model and small donor representation.
Tamargo et al., 2022 [13]; Spain	Simulated human gastrointestinal digestion and fecal fermentation	Polyethylene terephthalate microplastics subjected to simulated digestion	16S rRNA sequencing and microbial metabolic assessment. Digested particles modified microbial communities and showed possible polymer transformation. Limitations: simulated conditions and need for in-vivo confirmation.
Jiménez-Arroyo et al., 2023 [14]; Spain	Simulated gastrointestinal digestion and dynamic human colonic model	Polylactic-acid biodegradable microplastics subjected to gastrointestinal digestion	16S rRNA sequencing and short-chain-fatty-acid analysis. Exposure changed microbial composition and fermentation. Limitations: in-vitro design and absence of systemic outcomes.
Ke et al., 2023 [15]; China	Cross-sectional pilot study of preschool children in Xiamen	Fecal microplastics from real-world dietary and environmental exposure; multiple polymers and sizes	Spectroscopic identification and fecal 16S rRNA profiling. Microplastic burden was associated with differences in gut microbial diversity and composition. Limitations: small sample, cross-sectional design, and residual confounding.
Liu et al., 2023 [16]; China	Observational study of paired human placentas and neonatal meconium	Microplastics detected following maternal dietary and environmental exposure	Particle characterization and microbiota profiling. Microplastic occurrence was associated with differences in placental and meconium microbiota. Limitations: small sample, contamination risk, and no causal inference.
Liu et al., 2023 [17]; China	Prospective pilot mother-infant study	Microplastics assessed in placenta, meconium, infant feces, breastmilk, and infant formula	Spectroscopic particle identification. Multiple polymers were detected across maternal, neonatal, and nutritional matrices. Limitations: small sample and absence of downstream clinical outcomes.
Li et al., 2025 [18]; China	Controlled zebrafish study	Polystyrene nanoplastics at 1 or 10 mg/L plus arsenic at 10 µg/L; waterborne exposure for 21 days	Physiological assessment, 16S rRNA profiling, and metabolomics. Nanoplastics intensified arsenic-associated oxidative stress, dysbiosis, and metabolic disturbance. Limitations: aquatic model and comparatively high concentrations.
Chen et al., 2025 [19]; China	Controlled mouse study	Polystyrene microplastics, 10 mg/day, plus cadmium chloride, 4.8 mg/kg/day; oral exposure for 42 days	Colonic histology, microbial-diversity analysis, and untargeted metabolomics. Co-exposure aggravated injury, dysbiosis, and lipid- and bile-acid-related alterations. Limitations: animal model and high doses.
Zheng et al., 2025 [20]; China	Acute controlled tilapia study	Polystyrene microplastics co-exposed with sulfamethoxazole and BDE-153	Integrated microbiome, transcriptomic, and metabolomic analyses. Triple exposure produced synergistic intestinal toxicity. Limitations: acute aquatic model and complex mixture.
Zhang et al., 2024 [22]; China	Quasi-experimental study of 60 adults	Hot meals from disposable plastic tableware for 1 month; thermal food-contact exposure	Fecal microplastic assessment, 16S rRNA sequencing, and urinary metabolomics. Exposure altered gut microbiota and urinary metabolites. Limitations: nonrandomized design, short follow-up, and dietary confounding.
Wen et al., 2024 [23]; China	Controlled mouse study	Environmentally relevant oral concentrations of microplastics; no	Gut microbiota, bile-acid, metabolomic, and hepatic assessments. Exposure caused cholestasis and disturbed

Study and country	Design, model, and population	Exposure characteristics	Assessment, principal findings, and limitations
		additional co-exposure	bile-acid metabolism through the gut-liver axis. Limitation: animal-to-human extrapolation.
Zha et al., 2024 [24]; China	Controlled mouse study	Polystyrene micro- and nanoplastics with full-exposure and exposure-reduction groups	Immune markers, gut microbiota, and metabolomics. Partial exposure reduction improved immune, microbial, and metabolic abnormalities. Limitations: animal model and short follow-up.
Shi et al., 2024 [25]; China	Controlled female-mouse study	Polystyrene nanoplastics in drinking water for 49 days; dose-level comparison	Integrated transcriptomics and metabolomics. Exposure altered oxidative-stress, immune, inflammatory, and energy-metabolism pathways. Limitations: high exposure, female-only animals, and no human validation.
Medriano et al., 2025 [26]; Republic of Korea	Chronic adult-zebrafish experiment	Polyethylene- and polyester-based microplastics; polymer comparison	Gut microbiome and untargeted metabolomics. Both polymers produced dysbiosis and metabolic alterations with polymer-specific responses. Limitations: aquatic model and controlled particles.
Ragusa et al., 2021 [29]; Italy	Exploratory observational study of six human placentas	Environmentally acquired microplastics in placental tissue; dose and duration not measurable	Raman microspectroscopy. Twelve fragments were identified in four placentas. Limitations: very small sample, contamination risk, and absence of biological outcomes.
Marfella et al., 2024 [30]; Italy	Prospective observational study of patients undergoing carotid endarterectomy	Polyethylene and polyvinyl-chloride micro- and nanoplastics detected in carotid plaques	Pyrolysis-GC-MS, electron microscopy, inflammatory markers, and follow-up. Plaque particles were associated with inflammation and cardiovascular events. Limitations: observational design, confounding, and selected population.
Li et al., 2025 [37]; China	Controlled experimental model of microplastic-induced intestinal injury	Microplastic exposure with Clostridium dalinum probiotic intervention	Gut microbiota, metabolomics, barrier proteins, and inflammatory markers. The probiotic mitigated injury through microbiota-metabolism-barrier interactions. Limitations: preclinical model and strain-specific intervention.
Zhao et al., 2025 [38]; China	Polystyrene-nanoplastic-exposed colitis mouse model	Polystyrene nanoplastics with Lactiplantibacillus plantarum ZP-6	16S rRNA profiling, metabolomics, barrier markers, and hepatic assessment. The probiotic reduced intestinal and hepatic injury through the gut-liver axis. Limitations: disease-specific animal model and no human validation.
Deng et al., 2025 [39]; China	Acute honeybee exposure model	Size-dependent polystyrene nanoplastics with jute nanocrystalline cellulose	Gut microbiota, metabolomics, histology, oxidative stress, and apoptosis. The intervention reduced injury and modulated Gilliamella apicola and lipid metabolism. Limitations: invertebrate model and acute exposure.
Ragusa et al., 2022 [41]; Italy	Observational study of breastmilk from 34 women	Environmentally acquired microplastics in breastmilk; dose and duration not measurable	Raman microspectroscopy. Microplastics were detected in 26 of 34 samples. Limitations: small sample, contamination risk, and absence of infant outcomes.
Liu et al., 2023 [42]; China	Laboratory simulation and infant-intake assessment	Microplastics released from commercial breastmilk-storage bags under different conditions	Particle counting, spectroscopy, and intake estimation. Bags released microplastics and may contribute to infant ingestion. Limitations: laboratory simulation and no biological confirmation.
Kadač-Czapska et al., 2024 [43]; Poland	Analytical study of 30 commercial infant formulas	Multiple polymers detected in powdered formula; packaging- and preparation-related exposure	Particle isolation, microscopy, and spectroscopic identification. Microplastics were found in infant formulas. Limitations: uncertain source attribution and absence of health outcomes.

Abbreviations: BDE-153, 2,2′,4,4′,5,5′-hexabromodiphenyl ether; Caco-2, human colorectal adenocarcinoma cell line; GC-MS, gas chromatography-mass spectrometry; rRNA, ribosomal ribonucleic acid.
Source: Authors' own elaboration based on the 29 primary studies included in the systematic review [4,6–20,22–26,29,30,37–39,41–43].

Supplementary Table S2 Study-level risk-of-bias assessment of the included primary studies
A Human observational and quasi-experimental studies assessed with the Newcastle-Ottawa Scale

Study	Selection	Comparability	Exposure or outcome	Total score	Overall risk
Ke et al., 2023 [15]	**3/4**	**1/2**	**2/3**	**6/9**	**Moderate**
Liu et al., 2023 [16]	**2/4**	**1/2**	**2/3**	**5/9**	**Moderate**
Liu et al., 2023 [17]	**2/4**	**0/2**	**2/3**	**4/9**	**Moderate**
Zhang et al., 2024 [22]	**3/4**	**1/2**	**2/3**	**6/9**	**Moderate**
Ragusa et al., 2021 [29]	**2/4**	**0/2**	**2/3**	**4/9**	**Moderate**
Marfella et al., 2024 [30]	**4/4**	**2/2**	**3/3**	**9/9**	**Low**
Ragusa et al., 2022 [41]	**2/4**	**0/2**	**2/3**	**4/9**	**Moderate**

Interpretation: 7–9 points, low risk; 4–6 points, moderate risk; 0–3 points, high risk.

B Controlled animal studies assessed with the SYRCLE risk-of-bias tool
B1 Selection and performance domains

Study	SG	BC	AC	RH	BP
Djouina et al., 2022 [7]	U	L	U	U	U
Luo et al., 2022 [8]	U	L	U	U	U

Study	SG	BC	AC	RH	BP
Wang et al., 2023 [9]	U	L	U	U	U
Li et al., 2025 [18]	U	L	U	U	U
Chen et al., 2025 [19]	U	L	U	U	U
Zheng et al., 2025 [20]	U	L	U	U	U
Wen et al., 2024 [23]	U	L	U	U	U
Zha et al., 2024 [24]	U	L	U	U	U
Shi et al., 2024 [25]	U	L	U	U	U
Medriano et al., 2025 [26]	U	L	U	U	U
Li et al., 2025 [37]	U	L	U	U	U
Zhao et al., 2025 [38]	U	L	U	U	U
Deng et al., 2025 [39]	U	L	U	U	U

B2 Detection, attrition, reporting, and overall judgment

Study	ROA	BOA	IOD	SR	OB	Overall
Djouina et al., 2022 [7]	U	U	L	U	L	Unclear
Luo et al., 2022 [8]	U	U	L	U	L	Unclear
Wang et al., 2023 [9]	U	U	L	U	L	Unclear
Li et al., 2025 [18]	U	U	L	U	L	Unclear
Chen et al., 2025 [19]	U	U	L	U	L	Unclear
Zheng et al., 2025 [20]	U	U	L	U	L	Unclear
Wen et al., 2024 [23]	U	U	L	U	L	Unclear
Zha et al., 2024 [24]	U	U	L	U	L	Unclear
Shi et al., 2024 [25]	U	U	L	U	L	Unclear
Medriano et al., 2025 [26]	U	U	L	U	L	Unclear
Li et al., 2025 [37]	U	U	L	U	L	Unclear
Zhao et al., 2025 [38]	U	U	L	U	L	Unclear
Deng et al., 2025 [39]	U	U	L	U	L	Unclear

Judgments: L, low risk; H, high risk; U, unclear risk. Domains: SG, sequence generation; BC, baseline comparability; AC, allocation concealment; RH, random housing; BP, blinding of caregivers or investigators; ROA, random outcome assessment; BOA, blinding of outcome assessors; IOD, incomplete outcome data; SR, selective reporting; OB, other bias.

C In-vitro and analytical studies assessed with predefined criteria

C1 Particle characterization, contamination control, replication, and controls

Study	Particle characterization	Contamination control	Independent replication	Appropriate controls
Stock et al., 2022 [4]	2	2	2	2
Huang et al., 2021 [6]	2	1	2	2
Yan et al., 2022 [10]	2	1	2	2
Fournier et al., 2023 [11]	2	2	2	2
Fournier et al., 2023 [12]	2	2	2	2
Tamargo et al., 2022 [13]	2	2	2	2
Jiménez-Arroyo et al., 2023 [14]	2	2	2	2
Liu et al., 2023 [42]	2	2	2	2
Kadač-Czapska et al., 2024 [43]	2	2	2	1

C2 Dose relevance, outcome assessment, and overall judgment

Study	Dose relevance	Standardized or blinded assessment	Total	Overall risk
Stock et al., 2022 [4]	1	2	11/12	Low
Huang et al., 2021 [6]	1	2	10/12	Low
Yan et al., 2022 [10]	1	2	10/12	Low
Fournier et al., 2023 [11]	1	2	11/12	Low
Fournier et al., 2023 [12]	1	2	11/12	Low
Tamargo et al., 2022 [13]	1	2	11/12	Low
Jiménez-Arroyo et al., 2023 [14]	1	2	11/12	Low
Liu et al., 2023 [42]	1	2	11/12	Low
Kadač-Czapska et al., 2024 [43]	1	2	10/12	Low

Scoring: 2, adequately reported; 1, partially reported; 0, absent or inadequately reported. Interpretation: 10–12 points, low risk; 6–9 points, moderate risk; 0–5 points, high risk.

Source: Authors' own elaboration based on independent assessments performed using the Newcastle-Ottawa Scale, the SYRCLE risk-of-bias tool, and predefined criteria for in-vitro and analytical studies.