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Dietary Microplastics: Exposure Pathways, Hotspots, and Organ-Specific Health Effects

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A B S T R A C T

Microplastics (MPs) and nanoplastics (NPs) are pervasive environmental contaminants that increasingly infiltrate the human food chain, making dietary intake a continuous and unavoidable route of exposure. However, the integration of food-chain sources, exposure hotspots, biological absorption, and organ-specific health effects remains fragmented across the literature. This umbrella review synthesizes recent review-level evidence published between 2022 and 2025, focusing on farm-to-fork pathways of dietary microplastics, high-risk food contact materials—particularly tea bags and disposable cups—and mechanistic links to human health outcomes. Evidence indicates that microplastics originate from environmental contamination, food processing, packaging, and consumer-stage practices, with hot beverages representing a major but under-recognized exposure hotspot. Following ingestion, microplastics interact with the gastrointestinal tract, disrupt barrier integrity, and translocate systemically, accumulating in target organs including the liver, kidneys, endocrine organs, and reproductive tissues. Mechanistic pathways consistently involve oxidative stress, inflammatory signaling, endoplasmic reticulum stress, and endocrine disruption, often exacerbated by plastic-derived chemical additives. Despite growing concern, substantial methodological heterogeneity in detection and reporting limits quantitative risk assessment. Overall, dietary microplastics should be regarded as a multi-organ toxicological concern, underscoring the urgent need for standardized analytical frameworks and regulatory attention to food-contact materials and hot beverage consumption practices.

1. Introduction

Microplastics (MPs), defined as plastic particles smaller than 5 mm, and nanoplastics (NPs), typically below 1 µm, have emerged as ubiquitous contaminants across environmental and biological systems (Eze et al., 2024; Kadac-Czapska et al., 2024). While inhalation and dermal contact contribute to human exposure, dietary intake is increasingly recognized as a dominant and chronic exposure route due to the continuous consumption of contaminated foods and beverages (Shukla et al., 2024; Udovicki et al., 2022).

Microplastics enter the human diet through interconnected farm-to-fork pathways, beginning with plastic production and environmental leakage, followed by fragmentation and dispersion in terrestrial, aquatic, and atmospheric compartments (Jayasinghe et al., 2023; McHale & Sheehan, 2024). These particles are transferred through aquatic and terrestrial food chains, accumulating in seafood, crops, livestock, and processed food products (Jadhav & Medyńska-Juraszek, 2024; Unuofin & Igwaran, 2023). In parallel, food processing, packaging, and storage have emerged as critical contributors to dietary microplastic contamination, particularly where plastic contact, heat, and mechanical stress occur (Hee et al., 2022).

Recent evidence highlights food-contact materials as major and modifiable sources of exposure, especially in the context of hot beverages. Synthetic tea bags, disposable paper cups with plastic linings, and other single-use containers can release substantial quantities of microplastics and nanoplastics when exposed to hot liquids (Joseph et al., 2023; Mei et al., 2022; Yousefi et al., 2024). These findings challenge the traditional emphasis on seafood as the primary dietary source of microplastics and suggest that everyday consumer practices may significantly shape exposure profiles (Bai et al., 2022; Lam et al., 2024).

Following ingestion, microplastics interact with the gastrointestinal tract, where particle size, shape, and surface chemistry influence absorption and biological behavior (Prata, 2023). Experimental and human-relevant evidence suggests that microplastics can disrupt intestinal barrier integrity, alter gut microbiota composition, and translocate into systemic circulation (Wang et al., 2024; Yin et al., 2022). Accumulation of microplastics has been reported in human tissues, including liver, kidney, placenta, and blood, raising concerns regarding long-term organ-specific toxicity (Horvatits et al., 2022; Vdovchenko & Resmini, 2024).

Mechanistic studies increasingly link microplastic exposure to oxidative stress, inflammatory signaling, endoplasmic reticulum stress, mitochondrial dysfunction, and endocrine disruption, either through particle-driven effects or via associated plastic additives such as phthalates and per- and polyfluoroalkyl substances (PFAS) (Jahedi et al., 2025; Ojo et al., 2025). However, existing reviews often address exposure sources, toxicological mechanisms, or target organs in isolation, limiting integrated risk assessment and policy relevance (Bhavsar et al., 2023; Canga et al., 2024). Therefore, this umbrella review aims to synthesize recent review-level evidence into an integrated framework linking dietary sources, exposure hotspots, absorption pathways, and organ-specific health effects.

2. Methodology

Study design

This study was conducted as an umbrella review (systematic review of reviews), synthesizing narrative reviews, scoping reviews, systematic reviews, and meta-analyses addressing dietary microplastics and human health (Heo et al., 2025; Udovicki et al., 2022).

Eligibility criteria

Eligible publications were published between 2022 and 2025 and addressed at least one of the following domains: (i) microplastics or nanoplastics in foods, beverages, drinking water, or food-contact materials; (ii) food-chain transfer and dietary exposure pathways; or (iii) human health impacts, toxicological mechanisms, or exposure assessment related to dietary intake (Eze et al., 2024; Kadac-Czapska et al., 2024). Studies focusing exclusively on environmental compartments without relevance to food or human exposure were excluded.

Data extraction and synthesis

Data were extracted into predefined domains, including source and product category, food-chain stage, particle characteristics (polymer type, size, morphology), exposure context, and reported health outcomes or mechanisms. Evidence was synthesized narratively and thematically, with emphasis on exposure hotspots, absorption pathways, and organ-specific toxicity (Canga et al., 2024).

Reporting

Study identification, screening, eligibility assessment, and inclusion followed the PRISMA 2020 guidelines. The study selection process is summarized in a PRISMA flow diagram (Figure 1) (Page et al., 2021).

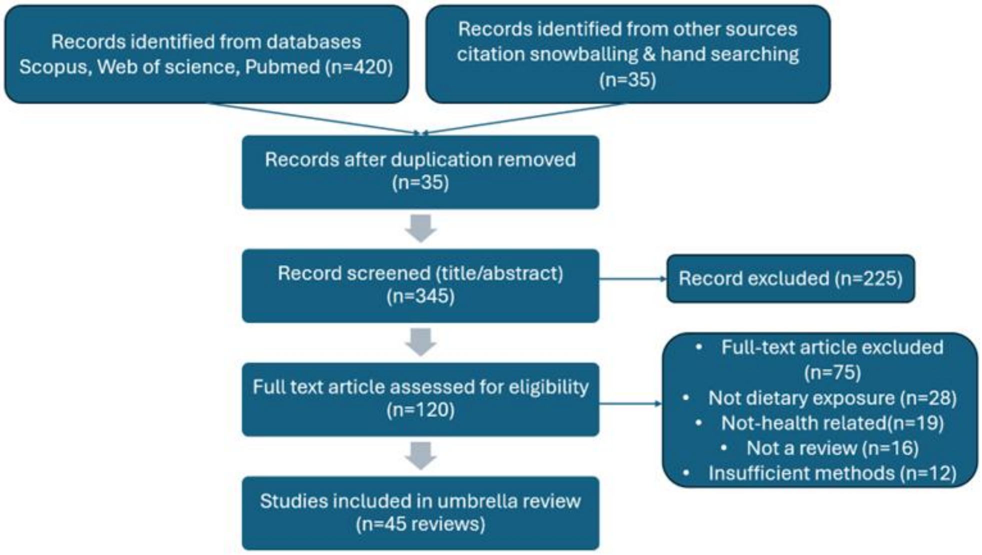

Figure 1. Prisma 2020 Diagram : Umbrella reviewdietary microplastics and human health (2022-2025)

3. Result and Discussion

Dietary sources and farm-to-fork pathways of microplastics

Dietary microplastics originate from multiple interconnected stages along the farm-to-fork continuum, including upstream plastic production, environmental contamination of soil and aquatic systems, food-chain transfer, processing, packaging, and consumer-stage practices (Eze et al., 2024; Jayasinghe et al., 2023). Aquatic food chains remain a major exposure route due to bioaccumulation and trophic transfer in seafood species (Unuofin & Igwaran, 2023), while terrestrial pathways involve contaminated agricultural soils, irrigation water, and livestock feed (Jadhav & Medyńska-Juraszek, 2024).

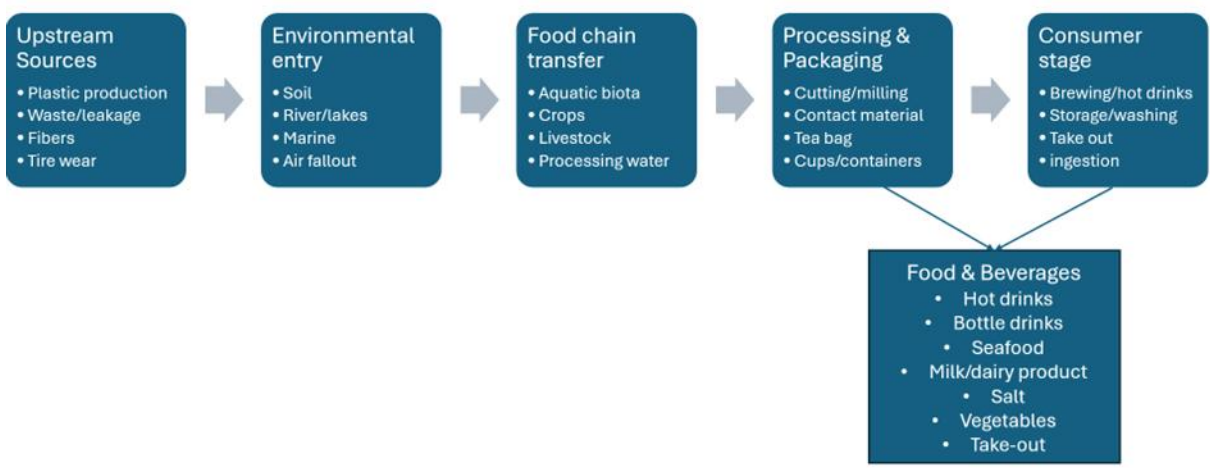

Figure 2. Farm to Fork Pathways of Microplastic Into Food and Beverages

In addition to environmental sources, food processing and packaging represent critical points of microplastic release, particularly under conditions involving heat and mechanical stress (Hee et al., 2022). Hot beverages have emerged as a prominent exposure hotspot, as synthetic tea bags, disposable paper cups, and plastic-lined containers release microplastics and nanoplastics during brewing and consumption (Joseph et al., 2023; Mei et al., 2022; Yousefi et al., 2024). The integrated pathways of dietary microplastic contamination are summarized in Figure 2.

Characteristics of dietary microplastics

Polyethylene (PE), polypropylene (PP), polyethylene terephthalate (PET), and polystyrene (PS) were the most frequently reported polymers in food and beverage matrices, with fibers and irregular fragments dominating particle morphology (Siddiqui et al., 2023; Vitali et al., 2023). Substantial heterogeneity exists in analytical approaches, detection limits, and reporting units, complicating cross-study comparison and exposure estimation (Bhavsar et al., 2023; Canga et al., 2024).

Health impacts and mechanistic pathways

Following ingestion, microplastics interact with the gastrointestinal tract, where particle size and surface properties influence uptake and biological effects (Prata, 2023). Experimental evidence indicates disruption of intestinal barrier integrity, gut microbiota dysbiosis, and translocation into systemic circulation (Wang et al., 2024; Yin et al., 2022). The liver and kidneys consistently emerge as primary target organs, reflecting their roles in detoxification and filtration (Horvatits et al., 2022; La Porta et al., 2023). Mechanistic pathways include oxidative stress, inflammatory signaling, immune cell activation, and endoplasmic reticulum stress, while endocrine and reproductive systems are increasingly implicated through hormone disruption and developmental effects (Inam, 2025; Jahedi et al., 2025; Zhang et al., 2025). A conceptual overview of absorption routes and organ-specific mechanisms is presented in Figure 3.

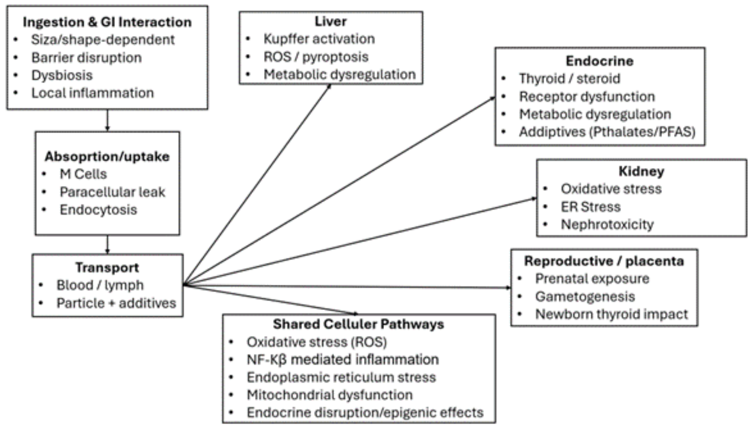

Figure 3. Mechanism of Action (MOA) and Target Organ Impacts of Dietary Microplastics

Discussion

This umbrella review integrates evidence across food-chain pathways, consumer practices, and biological mechanisms, demonstrating that dietary microplastics represent a systemic health concern rather than a localized exposure issue. Identification of hot beverages and disposable food-contact materials as major exposure hotspots expands current understanding beyond traditional seafood-centered paradigms (Joseph et al., 2023; Yousefi et al., 2024). *Mechanistic convergence on oxidative stress, inflammation, and endocrine disruption provides biological plausibility for observed organ-specific toxicity, particularly in the liver, kidneys, and reproductive system* (Inam, 2025; La Porta et al., 2023; Wang et al., 2024).

Nevertheless, methodological inconsistency in sampling, particle identification, and reporting remains a major limitation, restricting quantitative risk assessment and dose–response analysis (Bhavsar et al., 2023; Canga et al., 2024). *Standardized analytical frameworks and regulatory attention to food-contact materials—especially those used with hot liquids—may offer immediate opportunities for exposure reduction while epidemiological evidence continues to develop* (Xu et al., 2025).

4. Conclusion

Dietary microplastics and nanoplastics arise from interconnected farm-to-fork pathways, with beverages and disposable food-contact materials representing prominent and modifiable exposure sources. Accumulating evidence supports biologically plausible mechanisms linking ingestion to multi-organ toxicity, underscoring the need for standardized risk assessment and regulatory oversight to protect human health.

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