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From environment to dental plaque: The emerging impact of microplastics on oral cavity

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ABSTRACT

Microplastics (MPs) have emerged as pervasive environmental contaminants, raising increasing concern for human exposure and health risks. These particles are now detected in multiple daily sources, including drinking water, food products, indoor and outdoor air, and certain oral-care formulations, making the oral cavity one of the earliest and most consistent points of contact. Evidence from environmental, marine, and gastrointestinal microbiology demonstrates that MPs readily act as abiotic substrates capable of supporting microbial attachment, promoting biofilm formation, and altering microbial behavior. Recent studies suggest that similar processes may occur within the oral environment, where MPs can integrate into dental plaque, modify its structural organization, and influence interactions among bacterial and fungal communities. MPs may also affect microbial virulence, extracellular polymeric substance production, and horizontal gene transfer, raising concerns regarding their potential to exacerbate cariogenic or periodontal dysbiosis. Despite these emerging insights, current knowledge remains limited by the lack of *in vivo* studies, insufficient mechanistic investigation in oral tissues, and major analytical challenges in reliably detecting MPs within complex oral samples. These gaps hinder the ability to assess causal pathways and clinical significance. This review synthesizes current interdisciplinary evidence on the interaction between MPs and oral biofilms and highlights priority areas for translational research. A deeper understanding of these interactions is essential to clarify the role of MPs as novel environmental determinants of oral disease and to inform future preventive and regulatory strategies.

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1. Introduction

Microplastics (MPs), typically defined as synthetic polymer particles ranging from 1 µm to 5 mm, and nanoplastics (NPs), measuring <1 µm, have become pervasive contaminants in natural and human-made environments due to widespread plastic degradation and industrial production processes [1]. Increasing concern has emerged regarding human exposure to MPs, as numerous studies have detected them in drinking water, seafood, salt, packaged foods, and atmospheric fallout, indicating that ingestion and inhalation are major exposure pathways [2-4]. The oral cavity, as the initial interface for food and air intake, is therefore a primary site of MP contact, where particles may interact directly with oral epithelial surfaces, saliva, and the resident microbiota [5].

Dental plaque represents a complex multispecies biofilm composed of microbial communities embedded within an extracellular polymeric matrix, contributing to the pathogenesis of dental caries, gingivitis, and periodontal disease [6]. Environmental microbiology research has demonstrated that MPs readily serve as substrates for microbial colonization, forming biofilm-rich "plastisphere" communities with altered metabolic activity and enhanced pathogenicity [7,8]. However, the implications of such interactions within the oral cavity remain largely unexplored.

Given the increasing levels of human exposure and the recognized role of biofilms in oral disease, investigating the potential for MPs to influence dental plaque structure and microbial ecology is an emerging research priority. Understanding these interactions may reveal new environmental determinants of oral disease and guide future public health strategies.

2. Sources and routes of microplastic exposure

MPs are ubiquitous contaminants that enter the human body through multiple pathways, with the oral cavity being a major point of contact. The primary route of exposure is ingestion, but inhalation, as well as certain oral-care products and dental materials, also contribute to MP exposure. As MPs accumulate in the oral cavity, they present potential risks for interactions with the oral tissues and microbiota, affecting oral health [9].

2.1 *Ingestion*

Ingestion is a major pathway of microplastic exposure, with studies documenting MP contamination in bottled water, tap water, sea salt, seafood, and even tea brewed from plastic-infused tea bags [3,4,10,11]. Analytical approaches such as Fourier-transform infrared (FTIR) spectroscopy and Raman spectroscopy have been widely used to identify polymer composition and particle size distribution in these food and water sources [12,13].

These findings indicate that oral ingestion represents a continuous and unavoidable route of MP entry into the oral cavity.

2.2 *Inhalation*

Airborne MPs, originating from synthetic textiles, tire wear, indoor dust, and environmental fragmentation processes, have been detected in both indoor and outdoor air [14,15]. Inhaled particles can deposit within the oral and nasopharyngeal regions prior to reaching the respiratory tract, suggesting that the oral cavity is a significant site of initial exposure [16]. The presence of MPs in indoor aerosols highlights additional relevance for occupational and high-exposure environments.

2.3 *Oral-care products and dental materials*

Polyethylene (PE) microbeads were historically incorporated into toothpaste formulations until regulatory restrictions led to their phase-out, though residual reports persist in older products and regions without bans [17]. Wear particles generated from resin-based composites, polishing devices, and other dental materials may also contribute polymeric microparticles to the oral cavity, as noted in recent dental materials research [18]. These sources underscore the potential for both environmental and clinical contributions to intraoral MP burden. As illustrated in Figure 1, MPs can enter the oral cavity through ingestion, inhalation, and exposure to oral-care products.

3. Physicochemical determinants of microplastics-biofilm interactions

The propensity of MPs to become colonized by microbial communities is strongly influenced by their inherent material properties and how these properties evolve in the environment. In natural settings, MPs rapidly acquire an organic conditioning layer that alters their surface chemistry, facilitating the initial attachment of microorganisms and subsequent biofilm development [19].

3.1 *Size, surface area, and roughness*

Smaller MPs and NPs possess a disproportionately high surface area-to-volume ratio, which increases available attachment sites and promotes bacterial adhesion, as shown extensively in marine plastisphere studies [7,8]. Surface roughness, intensified through fragmentation or mechanical abrasion, further enhances microbial colonization [20].

3.2 *Surface charge and hydrophobicity*

Most common polymers such as PE, polypropylene (PP), and polystyrene (PE) exhibit hydrophobic surfaces that favor the attachment of primary colonizer, including oral species such as streptococci and *Actinomyces*, and *Fusobacterium* [21].

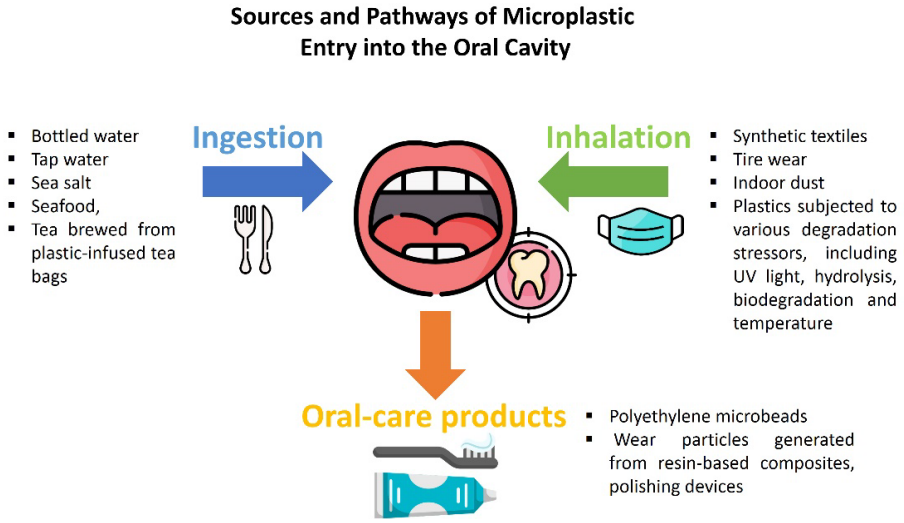

Figure 1. Schematic illustration showing the primary routes through which microplastics reach the oral cavity, including ingestion from contaminated food and drinking water, inhalation of airborne particles from indoor and outdoor environments, and exposure through oral-care products and dental materials. These pathways represent the earliest points of contact between microplastics and oral tissues, saliva, and resident microbiota.

Hydrophobic interactions, van der Waals forces, and electrostatic properties collectively influence microbial affinity and subsequent biofilm development [22].

3.3 *Weathering and saliva-induced aging*

Environmental weathering processes such as UV exposure, thermal degradation, and oxidative reactions alter MP surface chemistry and porosity, increasing their reactivity and capacity to bind organic matter and microorganisms [23]. Within the oral cavity, saliva can modulate MP surfaces through enzymatic activity, pH fluctuations, and protein adsorption, forming a salivary conditioning film analogous to those observed in marine biofilms [24]. These transformations may enhance bacterial adhesion and influence downstream biofilm architecture.

4. Microplastics as substrates for oral biofilm formation

MPs have the capacity to function as supplementary attachment surfaces for microorganisms, a behavior well characterized in aquatic ecosystems and increasingly relevant to the oral cavity. Once present in the mouth, MPs can be rapidly conditioned by salivary proteins and enzymes, creating a surface that favors the adhesion of early colonizing bacteria. This secondary substrate may integrate into developing plaque, modifying its spatial arrangement and enabling the accumulation of additional microbial layers [25].

4.1 *Parallels with environmental microbiology*

MPs rapidly develop microbial biofilms upon entering water systems, forming a distinct ecological niche known as the “plastisphere.” Aged MPs, owing to their increased surface roughness, hydrophobicity changes, and biofilm coverage, serve as stable substrates for diverse microbial communities, including potentially pathogenic bacteria and viruses. This colonization parallels natural environmental biofilms, exhibiting similar succession patterns, extracellular polymeric substance production, and horizontal gene transfer. However, unlike natural substrates that eventually degrade, recalcitrant MPs act as long-term vectors, facilitating microbial survival, dispersal across water bodies, and enhanced resistance to disinfection processes [26]. These findings provide important biological parallels that may extend to oral microbial communities.

4.2 *Relevance to the oral biofilm*

Dental plaque is a structurally complex and dynamic biofilm that develops through bacterial co-adhesion, metabolic cooperation, and extracellular polymeric substance (EPS) production [6]. The ability of MPs to support biofilm growth in other environments suggests a high likelihood that similar interactions could occur in the oral cavity, given comparable microbial traits such as hydrophobic adhesion, quorum sensing, and EPS matrix formation [27,28].

4.3 *Environmental evidence*

MPs in aquatic environments facilitate biofilm formation, which can interact with pollutants and act as a habitat for microorganisms. Recent studies have shown that MP biofilms play a significant role in the spread of pathogens and antibiotic resistance genes (ARGs) to aquatic organisms. The adsorption of antibiotics by MPs is enhanced after biofilm formation, and ARGs and pathogens are selectively enriched in biofilms. These biofilms promote the transfer of ARGs through horizontal and vertical gene transfer, leading to

the emergence of antibiotic-resistant pathogens, posing threats to aquatic ecosystems and human health [28]. Furthermore, MPs enhanced bacterial adhesion and may selectively enhance the adhesion of cariogenic and periodontal pathogens such as *Streptococcus mutans* [29].

4.4 Structural modifications of dental plaque

By providing additional abiotic surfaces, MPs may act as nucleation sites within dental plaque, altering its architecture and stability. Their integration into plaque could result in denser or more resilient biofilms with increased resistance to mechanical removal or antimicrobial agents [30,31]. These structural effects represent a potential mechanism through which environmental contaminants may influence oral health and disease susceptibility [30].

5. Microplastics effects on oral tissues and microbial ecology

MPs and nanoplastics induce multiple biological disturbances in mammalian cells. Studies using epithelial and fibroblast models demonstrate that PS and PE particles generate significant reactive oxygen species, disrupt mitochondrial membrane potential, and impair antioxidant defenses [32-34]. These findings parallel toxicology research in gut and airway systems, suggesting similar vulnerabilities in oral mucosa. MP exposure also triggers pro-inflammatory signaling. Experimental studies show activation of NF-κB pathways and elevated levels of IL-6, IL-1β, and TNF-α following PS or polyamide exposure. This is particularly relevant to gingival inflammation and periodontal disease [35-37].

Beyond host responses, MPs influence microbial virulence and gene expression. Environmental microbiology studies report that MP-associated biofilms exhibit elevated virulence markers, stress-response genes, and antimicrobial resistance determinants [7,38]. Although direct oral evidence remains limited, these mechanisms may contribute to dysbiosis in dental plaque. Additionally, MPs enhance the thickness and cohesion of the EPS matrix, increase aggregation, and reinforce biofilm adhesion to polymeric surfaces [39]. These effects can further stabilize and intensify plaque biofilms, increasing their resistance to shear forces and antimicrobial challenges.

6. Microplastics as vectors for oral pathogens

MPs are recognized as carriers of microbial communities in aquatic systems. PE, PP, and PS particles accumulate diverse taxa including pathogens such as *Vibrio* spp. and *Pseudomonas* spp. and facilitate long-distance microbial transport [7,40-42]. MP-associated biofilms frequently harbor ARGs and mobile genetic elements (MGEs), enabling enhanced horizontal gene transfer among microbial species [42]. Because oral biofilms are already hotspots for genetic exchange, MPs may amplify HGT by creating new micro-niches within plaque. MPs may also promote cross-kingdom interactions. Polymer surfaces enhance co-adhesion between bacteria and fungi, such as *Candida albicans* with *Streptococcus* species, potentially increasing mixed-species virulence in dysbiotic oral biofilms [43]. Figure 2 illustrated how MPs may modify plaque architecture and microbial behavior through several interconnected mechanisms.

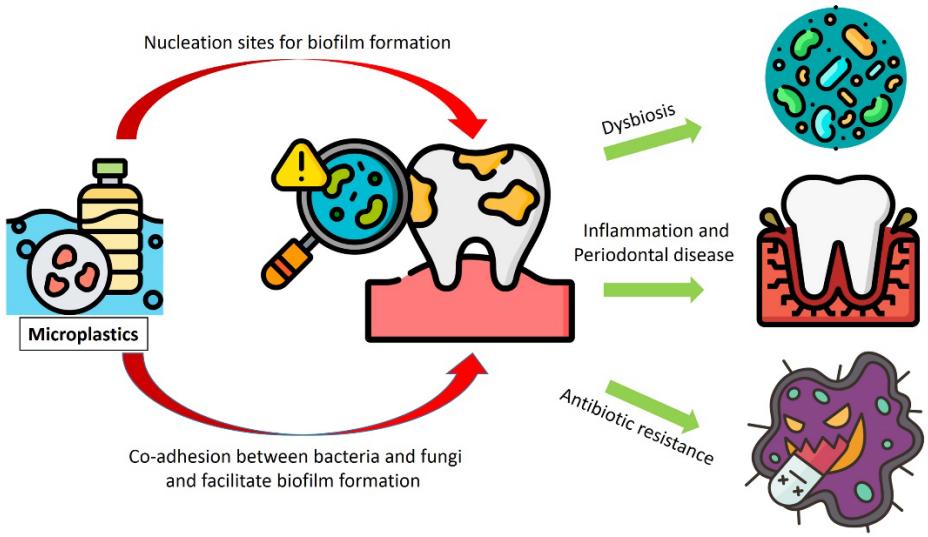

Figure 2. Mechanisms by which microplastics influence oral biofilm development and oral health. Schematic illustration showing how microplastics (MPs) enter the oral cavity and integrate into dental plaque. MPs act as additional adhesion surfaces, enhance bacterial attachment, stimulate extracellular polymeric substance (EPS) production, inflammation, horizontal gene transfer, and promote multispecies co-aggregation, including bacteria-fungi interactions. These processes contribute to altered plaque structure, increased microbial resilience, and potential risks for caries, periodontal disease, and peri-implant complications.

7. Clinical implications for oral health

Growing experimental evidence suggests that MPs may influence key pathological processes in oral disease. In periodontal disease, MPs may contribute to dysbiosis and exaggerated host inflammatory responses. They induce oxidative stress and elevate IL-6 and TNF-α in epithelial cells, which may worsen gingival inflammation and facilitate progression to periodontitis [44,45].

MP-associated biofilms often carry virulence and antimicrobial resistance genes, raising concerns for subgingival ecosystems [38].

In peri-implant disease, polymer particles enhance microbial adhesion to titanium and zirconia, potentially increasing the risk of peri-implant mucositis and peri-implantitis [46,47]. Children may face greater susceptibility due to higher exposure per body weight and the developmental vulnerability of oral microbiota and dentition.

8. Methodological challenges in studying microplastics

Studying MPs in oral samples is technically challenging. Saliva and plaque have viscous, protein-rich matrices that complicate particle extraction and may cause underestimation [48]. Nanoplastics are especially difficult to detect due to limited resolution in standard spectroscopic methods. FTIR and Raman spectroscopy are widely used but have reduced discriminatory capacity for small or heavily weathered particles [49].

Pyrolysis–GC/MS provides detailed chemical composition but is destructive and lacks morphological data [50]. A major limitation is the absence of standardized protocols for sampling, contamination control, and quantification in human oral studies, which restricts cross-study comparability and slows clinical translation.

9. Conclusion

MPs are emerging contaminants capable of interacting with oral biofilms and affecting key biological processes. Evidence from environmental and *in vitro* studies shows that MPs can alter plaque architecture, modify microbial composition, promote virulence traits, and induce inflammatory responses. Although clinical data remain limited, mechanistic evidence provides strong biological plausibility that MPs may contribute to caries, periodontal disease, and peri-implant complications. Advancing analytical methods and establishing standardized protocols will be essential for accurately characterizing MPs in oral samples. Integrating clinical, environmental, and materials science research may improve preventive strategies and inform public health policies addressing microplastic exposure.

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Authors' contributions

SRH: developed the concept of the review, outlined its objectives, and lead the project. SS: conducted the detailed literature search, extracted relevant data, and structured the manuscript's thematic framework. SS, and SRH: contributed to analyzing the data, integrated the main insights, and prepared the first draft of the manuscript. SRH: contributed to critical revision of the manuscript. All authors reviewed and approved the final version of manuscript.

Conflict of interest

No potential conflict of interest was reported by the authors.

Ethical declarations

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