Oral microbiome and inflammatory bowel disease: A scoping review

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Abstract: Purpose: To examine the association between the oral microbiome and IBD in terms of microbiota diversity, composition, function, and diagnostic potential through a scoping review of the existing literature. **Methods:** This scoping review was structured following the PRISMA-ScR guidelines. Literature searches were conducted in PubMed, Embase, and the Cochrane Library, with the time frame restricted from database inception to November 17, 2025. **Results:** This review included 31 studies, among which seven topics for patients with periodontitis and IBD-related periodontitis, three studies assessed single bacterial species, and the remaining 21 articles explored the diversity and phylum-level taxonomic shifts of oral microbiota in IBD patients, functional and metabolic pathway alterations associated with oral dysbiosis, as well as the diagnostic potential and biomarkers derived from oral microbial profiles. (*Am J Dent* 2026;39:253-258).

Clinical significance: The oral microbiota of IBD patients exhibits characteristic dysbiosis, manifested by the enrichment of pathogenic taxa, depletion of anti-inflammatory species, and metabolic dysfunction, which are closely associated with gut microbial alterations and systemic inflammatory status. The salivary microbiome demonstrates significant diagnostic potential for IBD.

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Introduction

Inflammatory bowel disease (IBD), a chronic immune-mediated disorder, comprises two principal subtypes: Crohn's disease (CD) and ulcerative colitis (UC). Emerging evidence has established that the pathogenesis of IBD is related to host genetic predisposition, environmental triggers, immune abnormalities, and the gut microbiota,[1,2] with intestinal dysbiosis serving as a critical effector mechanism driving disease initiation and progression. The gastrointestinal tract is densely colonized by microbial consortia. Disruption of the microbial equilibrium triggers a cascade of pathophysiological alterations, including compromised mucosal barrier integrity, dysregulated immune homeostasis, and metabolic dysfunction.[3,4]

The oral cavity harbors one of the most diverse and densely populated microbial ecosystems in the human body, second only to the gastrointestinal tract, and consists of 774 bacterial species that form a highly dynamic ecosystem.[5,6] Disruption of oral microbial homeostasis (dysbiosis) predisposes individuals not only to localized pathologies, such as dental caries and periodontal diseases, but also to systemic disorders, including malignancies,[7] cardiovascular pathologies,[8] and adverse pregnancy outcomes.[9]

Emerging evidence indicates an association between oral microbial dysbiosis and disease severity in IBD.[10] On one hand, IBD patients exhibit marked compositional and functional imbalances in the oral microbiota, termed dysbiosis; on the other hand, an overabundance of opportunistic pathogenic oral commensals has been observed within their gut microbial communities.[11] Furthermore, various oral pathologies, including angular cheilitis and periodontitis, represent common extra-intestinal manifestations of IBD, constituting dual microbiome and immune mechanisms bridging oral and gut health.[12,13] Collectively, these findings suggest interconnected inflammatory processes at oral and intestinal mucosal interfaces, with oral microbiota demonstrating the capacity for intestinal expansion and colonization. However, the precise mechanisms governing oral microbial gut colonization and their specific causal links to IBD pathogenesis remain incompletely elucidated.

This study employed the PRISMA-ScR (Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews)[14] as its methodological framework to comprehensively examine published literature on oral microbiota and IBD. For the first time, it systematically synthesized evidence linking the oral microbiome to IBD from perspectives including microbial diversity, compositional characteristics, functional profiles, and diagnostic potential. Through scoping mapping of existing evidence, this review proposes a forward-looking perspective: the oral cavity may serve not only as an "early-warning window" for IBD but also as a potential "novel interventional target". This study systematically evaluated the potential of specific oral micro-organisms (such as Leptotrichia and Prevotella in saliva) as tools for non-invasive diagnosis, disease activity assessment, and prognosis prediction in IBD, thereby providing a reference for future research.

Materials and Methods

Identify the research question - The research questions of this study were formulated through a comprehensive literature review: (1) How does the oral microbiota of IBD patients differ from that of healthy individuals? (2) Can oral microbiota be used as an indicator for the diagnosis of IBD?

Search strategy and data sources - A systematic search of electronic databases (PubMed, Embase, and the Cochrane Library) was conducted from their inceptions until December 7th, 2024. The following search terms were included: ("mouth" OR "mouth mucosa" OR "oral" OR "saliva" OR "salivary" OR "subgingival" OR "supragingival" AND "microbio*" OR "bac-

teria" OR "dysbiosis" OR "microorganism" OR "microflora" AND "Inflammatory Bowel Diseases" OR "inflammatory bowel disease" OR "IBD" OR "colitis" OR "Crohn's disease" OR "ulcerative colitis") NOT ("review" OR "letter" OR "editorial" OR "case reports").

Inclusion and exclusion criteria - Inclusion criteria: (1) Studies involving participants diagnosed with IBD; (2) Investigations focused on the oral microbiome; (3) Literature examining microbial diversity or relative abundance of oral microbiota in IBD populations. Exclusion criteria: (1) Studies with full-text unavailable; (2) Publications containing incomplete or irretrievable data; (3) Duplicate publications (retaining only the most comprehensive version); (4) Non-primary research articles including reviews, news articles, conference abstracts, and commentaries.

Literature screening and data extraction - The search results were imported to EndNote X9.3.3 and duplicates were deleted. Two authors independently screened titles and abstracts according to inclusion and exclusion criteria; then, potentially eligible articles were screened by full text reading. Reasons for excluding papers in the full-text screening stage were recorded. Each cross-checked after completion, and any disagreements were discussed and resolved or referred to a third author. Inter-rater agreement at the full-text screening stage was assessed by calculating Cohen's kappa coefficient, which yielded a value of 0.769 (95% CI: 0.618-0.920), indicating a substantial level of agreement between the reviewers. Data extraction from the included articles was completed independently by two authors using a data extraction form. Extracted data included the first author, year of publication, country, aims, study type, study design, sample size, oral microbiological sequencing, and the main findings. Any discrepancies in this process were discussed and resolved with the third author.

Quality assessment - The methodological quality of each cohort study was evaluated according to the Newcastle-Ottawa Scale (NOS)[15] item-by-item, and a total score was calculated, which was proportional to the quality of the study. The NOS scoring was done by two evaluators separately. Disagreements were resolved by further reference to the original text and by consensus, and if not, a qualified third person was asked to evaluate the study.

Results

Literature search results - According to the search strategy, a total of 6,845 articles were retrieved, and 6,582 articles remained after removing duplicates. 72 articles were screened after reading the titles and abstracts. The above 72 papers were re-screened after reading the full text in detail, finally, 29 articles met inclusion and exclusion criteria and are included in this review. The literature search and screening process was updated on November 17, 2025, which yielded two additional articles for inclusion, bringing the total number of included studies to 31. The literature screening process and results are shown in the Figure.

Characteristics and methodological quality of the included studies - Among the 31 studies included in this review, except for one article[16] published in 1986, the remaining studies were published between 2010 and 2025, by authors from 14 different countries, mostly from China (9/31, 29%); followed by USA (6/31, 19%); Germany (4/31, 13%); and Australia (2/31, 6%). The remaining 10 studies were published by Iranian, Japanese, Polish, Korean, Spanish, Singaporean, British, Emirati, Irish, and Brazilian researchers. Regarding study designs, one article[17] was a prospective cohort study, another[18] employed a hybrid case-control and prospective cohort design, while the remaining studies exclusively utilized case-control methodologies. The sample sizes across studies ranged from 18[19] to 398 participants,[20] with notably unreported control group sample size in one study.[16] Regarding the sequencing methodologies for oral microbiota, 21 studies employed 16S ribosomal RNA (rRNA) sequencing, three utilized metagenomic sequencing via the Illumina HiSeq platform, and one study applied both 16S rRNA sequencing and bft toxin detection. The remaining investigations applied the following techniques: dark-field microscopy, matrix-assisted laser desorption/ionization with a time-of-flight analyzer (MALDI-TOF), polymerase chain reaction (PCR), multilocus sequence typing (MLST), Human Oral Microbe Identification Microarray (HOMIM), the checkerboard DNA-DNA hybridization technique. The included studies demonstrated satisfactory overall methodological quality, with 24 articles achieving a total score of 7 or higher. The detailed characteristics and quality assessment results of the included literature are available upon request from the first author.

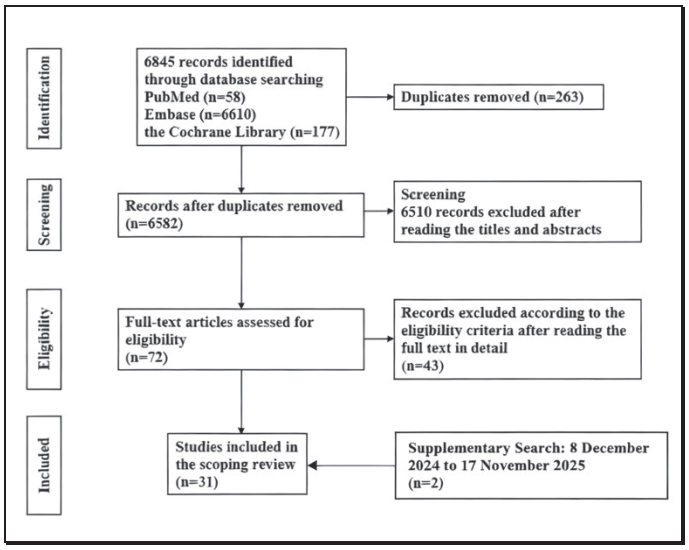


Figure. Flow diagram. Selection of articles for the scoping review.

The composition and diversity in the oral microbiome between periodontitis and IBD-associated periodontitis - Among the 31 studies included, seven[16,18,19,21-24] focused on patients with periodontitis and IBD-related periodontitis. Researchers in all four of the studies[18,19,23,24] reported differences in microbial diversity between the periodontitis group and IBD-associated periodontitis, although the results reported were not entirely consistent between studies. Interestingly, in one study,[23] subgingival plaque and saliva samples were collected, and the results showed that the subgingival plaque had greater microbial diversity than saliva in CD-associated periodontitis. An earlier study[16] noted that the periodontal flora of IBD-affected patients is composed of predominantly small, motile, gram-negative rods, which were most consistent with the genus *Wolinella*. The bacterial load in the gingival sulcus in patients

with periodontal disease-free IBD was significantly lower than in the IBD group with periodontal disease, however, the microbiota composition was similar. Only one research group[21] reported the differences in microbial community structure observed in unstimulated saliva, stimulated saliva, gingiva, and stool samples. The main differences among the four samples were between active IBD (aIBD) and healthy control subjects (HC). In addition, aIBD saliva exhibited higher relative abundances of putative oral pathobionts from the genera *Streptococcus*, *Granulicatella*, *Rothia*, and *Actinomyces* compared to HC. One study[22] investigated concentration and prevalence of selected periodontal pathogenic bacteria (*Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, *Prevotella intermedia*, *Parvimonas micra*, *Fusobacterium nucleatum*, *Campylobacter rectus*, *Eubacterium nodatum*, *Eikenella corrod*ens, and *Capnocytophaga* species) within a group of patients with IBD and compared the results with a group of HC, showing that the prevalences of *Eubacterium nodatum* and *Eikenella corrodens* were significantly lower in IBD compared to HC. However, further statistically significant differences in selected bacteria between IBD and HC groups could not be found.

Oral microbiota and IBD - Microbial diversity encompass both alpha diversity and beta diversity. Alpha diversity reflects the species' richness and evenness within individual samples, with commonly employed indices including the Chao index, Shannon index, and Simpson index. Beta diversity quantifies the dissimilarity in microbial community composition across different samples, typically measured using Bray-Curtis dissimilarity and UniFrac distance. Most studies have demonstrated that patients with aIBD (particularly CD) exhibit significantly lower Chao index and phylogenetic diversity (PD) compared to those in remission phase and HC, suggesting diminished species richness.[20,25-29] However, three studies[26,30,31] have reported non-significant differences in both the Shannon index and/or Simpson index. Patients in remission phase demonstrate no statistically significant differences in oral alpha diversity (as exemplified by Chao1) compared to HC.[26] Principal coordinate analysis (PCoA) and constrained principal coordinate analysis (CPCoA) revealed significant segregation between IBD patients and HC in oral microbial profiles - including saliva and dental plaque samples - based on both unweighted and weighted UniFrac distance metrics.[20,25,26,28,29,32-34] Microbial community structure differed less between UC and CD, but both were significantly different from HC.[35]

Multiple studies have documented alterations in microbiota composition. At the phylum level, decreased relative abundance of *Bacillota* has been observed in IBD (particularly during active phases), with concomitant increases in *Bacteroidota*, *Pseudomonadota*, and *Actinomycetota*.[26,33,36] Conversely, conflicting evidence indicates elevated *Bacillota*[25,28,29] reduced *Fusobacteriota*,[25,28,33,36] diminished *Actinomycetota*,[27,28] and significant enrichment of candidate phyla *Saccharibacteria* and *Absconditabacteri*[34] in IBD patients compared to controls.

At the genus/species level, researchers reported enrichment of *Prevotella*,[25,26,34,35,37] *Veillonella*,[25,29,35,38] *Saccharibacteria* phylum-related taxa (e.g., *Capnocytophaga*, *Leptotrichia*),[17,34,39] and Neisseria[28,40] in IBD patients. Conversely, several other investigations revealed that *Fusobacterium*[25,28,33,36] and *Neisseri*[26,29,34,35] demonstrated depletion in both UC and CD cohorts. While four studies[31,34,35,40] observed reduced *Streptococcus* abundance in IBD populations,[31,34,35,40] two independent studies[29,41] reported contradictory findings.

Three studies conducted assessments focusing on single bacterial species. *Campylobacter* species were detected in 97% of healthy individuals and 100% of IBD patients' saliva.[42] Another investigation[43] revealed that 87.5% of individuals harboring Class I *C. concisus* exhibited inflammatory bowel diseases, a prevalence significantly higher than other subgroups. Notably, EICC oral strains were identified in 50% of IBD patients, whereas none were detected in control subjects. Although no significant difference in *Bacteroides fragilis* abundance was observed between HC and UC patients, ETBF colonization was significantly elevated in UC patients compared to HC.[44]

Oral microbial dysbiosis-induced perturbations in functional and metabolic pathways - Oral microbial gene processes were highly abundant, predominantly involving metabolism, environmental and genetic information processing. Dysregulation of metabolic pathways was evidenced by downregulation of fundamental functions including ribosome biogenesis and energy metabolism,[25,26,29] concurrent with upregulation of pro-inflammatory pathways (e.g., enhanced carbohydrate metabolism and virulence factor-associated pathways),[28,29,34,35] alongside an enhanced acidogenic capacity coupled with diminished butyrate synthesis,[30,41] and reduced antioxidant capacity.[45] Oral microbial dysbiosis may serve as a biomarker of systemic inflammatory responses, characterized by elevated levels of inflammatory cytokines and immunoglobulin A, contrasted with reduced lysozyme activity.[27,29,34,35,38,45] Notably, salivary and gut microbiomes share partial taxonomic overlap (e.g., *Streptococcus* spp.), suggesting the oral cavity may act as a potential reservoir for enteropathogenic colonization.[25]

Diagnostic value of oral microbiology - Six studies reported the diagnostic potential of oral microbiota. The salivary microbiome demonstrated optimal performance in differentiating IBD patients from HC and was correlated with therapeutic responses.[32,33,46] Random Forest models or machine learning approaches achieved AUC values ranging from 0.762 to 0.966 for distinguishing IBD from HC.[17,20,30,33,41] Similarly, salivary microbial profiles exhibited favorable accuracy and AUC values in distinguishing CD from UC.[20]

Diagnostic biomarkers of the oral microbiome were additionally reported. Specific taxa such as the *Saccharibacteria*, *Veillonella*, and *Prevotella* exhibited potential as biomarkers for IBD.[17,25,34,37] The oral microbiota demonstrating significant divergence between active and remission phases of IBD was *Leptotrichia*, which showed elevated abundance during remission.[27] Distinct microbial enrichment patterns were observed in UC and CD patients, with *Leptotrichia* predominating in UC and *Bulleidia* in CD.[34]

Discussion

The pathogenesis of IBD is intricately linked to gut microbial dysbiosis.[47] Emerging evidence highlights significant alterations in the oral microbiome during IBD, suggesting its

potential involvement in disease pathogenesis and progression via the "oral-gut axis".[48] However, the extent of its contribution, as well as its applicability in disease monitoring and therapeutic strategies, remains insufficiently explored.

Consistent with previous findings,[49] patients with highly active IBD showed significant differences in Shannon diversity of the salivary microbiome compared with controls. However, these differences diminished during remission, suggesting that disease stage may reversibly affect microbial composition. Notably, among the included studies, some failed to detect alpha diversity variations, potentially attributable to confounding factors such as sample size, disease phase heterogeneity, or sequencing depth. Two principal DNA sequencing methodologies for characterizing uncultured oral microbial communities are 16S rRNA amplicon sequencing and whole metagenome shotgun sequencing (WMS).[50] While 16S amplicon sequencing enables rapid and precise microbial identification at the genus level, it lacks resolution for species- or subspecies-level classification. Conversely, WMS provides comprehensive genomic data, facilitating both taxonomic profiling and biological functional maps of microbial communities, albeit with higher sequencing costs compared to 16S approaches.[50,51] Future research should prioritize establishing standardized methodologies to enable reliable cross-study data comparisons while minimizing technical bias.

The oral microbiome is drawn largely from a few dozen genera across seven phyla: *Actinomycetota, Bacteroidota, Bacillota, Fusobacteriota, Pseudomonadota, Saccharibacteria* and *Spirochaetota*.[5] No conclusive inferences can be drawn regarding specific bacterial genus-level changes in IBD patients, as the included studies reported heterogeneous findings (e.g., concerning the abundance of the *Bacillota*, *Actinomycetota*, and *Streptococcus*). This heterogeneity may stem from divergent sampling sources (e.g., saliva, tongue, buccal mucosa), as oral niches exhibit distinct microbial diversity profiles despite sharing compositional similarities.[52] Furthermore, the medications utilized by patients, such as immunosuppressants, may also interfere with the microbiota,[53] leading to a reduction in microbial concentration within the gut while facilitating the intestinal colonization of oral microbes.[54] The selection of sequencing platforms and target regions constitutes another significant source of variability. This is evidenced by the suboptimal amplification of *Actinomycetota* by the common V1-V3 primers 27F/534R, potentially underestimating its abundance,[55] and by the reported biases of the V3-V4 primers 341F/805R, which can over-represent *Bacillota* (e.g., *Staphylococcus*) while underrepresenting *Bacteroidota*.[56,57] High-quality, large-scale cohort studies are required to delineate the changes in oral microbiota during different stages of the disease.

IBD exhibits intricate associations with compromised diversity in both oral and gut microbial ecosystems, driving fluctuations in the abundance of specific taxa. The transfer of oral microorganisms from the oral cavity to the intestinal mucosa is generally thought to take two routes: hematogenous and enteral, although the mode of transfer is uncertain.[5] Results from distinct studies show that in IBD, and particularly when associated with periodontitis, even low-abundance oral bacteria can translocate to the gut and contribute to inflammatory processes through both oral and intestinal immunity.[58] The interaction between the oral cavity and the intestinal tract seems to be bidirectional, the oral-gut axis is a key driver of immune dysregulation in IBD. Central to this process is the periodontitis-induced breakdown of the oral barrier, which enables oral pathobionts (e.g., *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, *Klebsiella* spp.) to translocate and colonize the gut. There they not only directly impair the intestinal epithelium but also, via pathogen-associated molecular patterns, activate innate and adaptive immunity, culminating in the pathological activation of migratory Th17 and Th1 cells that fuel chronic intestinal inflammation.[58] Advancing this field requires the development of standardized, multi-omics (metabolomic, immunomic) workflows for concurrent oral-gut microbiome analysis to improve data comparability and integration.

Although the distribution of oral and gut microbiomes is well separated due to the oral-intestinal barrier, studies have shown that gut enrichment of oral-associated bacteria is a potential microbial signature in patients with IBD.[59] Comparing human reference oral microbiome (HROM) with reference genomes of the gut microbiome, HROM contains 42 ectopic oral species, and their relative abundance in gut microbiota is predictive of intestinal diseases.[60] The present review confirms that, despite interstudy discrepancies, it is certain that the oral microbiome can be used as a diagnostic marker for IBD, including UC and CD. Oral microbial communities exhibit niche-specific stratification, while compositionally similar across oral sites, significant divergence exists between distinct microenvironments (e.g., dental plaque, tongue dorsum, and keratinized gingiva).[50,52] The saliva microbiome has great potential as a non-invasive diagnostic tool,[46] and there is a standard set of procedures and requirements for collecting saliva samples.[61]

The present review remains inconclusive regarding specific salivary biomarkers for IBD, underscoring the need for dedicated high-quality research. Periodontitis is established to link with IBD via direct and indirect mechanisms, including the systemic trafficking of activated T cells and the enteric transmission of inflammatory bacteria.[62] The utility and reliability of specific salivary biomarkers from periodontitis patients for diagnosing IBD, however, await further verification. Future directions should prioritize prospective cohort studies that recruit high-risk or incident IBD cases, with synchronized saliva collection at multiple pre- and post-diagnosis timepoints. This design will allow for establishing the temporal relationship between oral microbiome dynamics and disease onset and for identifying predictive pioneer species. Ultimately, constructing integrated risk prediction models using machine learning, which combine oral microbial features with clinical indicators, will be crucial for the early detection of at-risk individuals.

Some limitations of this scoping review must be acknowledged. Some high-quality studies only provide abstracts without accessible full texts, which has resulted in their exclusion from this discussion and may impact on the current findings. While we have not presented all results in this paper due to space limitations, we have endeavored to elaborate on them as thoroughly as possible in the results section. The

methodological quality of the included studies was limited, mainly due to the predominant use of hospital-based controls, posing a risk for selection bias, and inadequate accounting for key confounding variables. We consequently advise the adoption of community controls and rigorous control for confounders in subsequent research. Additionally, significant clinical and methodological heterogeneity was present throughout the studies, necessitating cautious interpretation of collective evidence. Future research should focus on the clinical validation of the oral microbiome as a non-invasive biomarker, the development of diagnostic tools based on oral microbial markers, an in-depth investigation of the specific mechanisms of the oral-gut axis (such as immune regulation and metabolite exchange), and the formulation of intervention strategies targeting the oral microbiota (such as probiotics and oral hygiene management) to enhance treatment outcomes in IBD.

In conclusion, dysbiosis of the oral microbiome has been implicated in the pathogenesis of multiple localized and systemic diseases, although specific causal relationships and underlying mechanisms remain conjectural. Current research paradigms are shifting from a "pathogen-centric" perspective to an "ecological network regulation" framework, with the broader implications of oral microbial research extending beyond oral health. IBD-associated oral microbial communities display characteristic dysbiotic features, marked by enrichment of pathogenic taxa, depletion of anti-inflammatory bacteria, and dysregulated metabolic functions, and are closely linked to intestinal microbiome alterations and systemic inflammatory responses. The salivary microbiome demonstrates significant potential as a non-invasive biomarker for IBD diagnosis, disease staging, and therapeutic monitoring; however, standardization of analytical protocols and cross-regional validation are critically required. Future research should prioritize multi-omics convergence to overcome existing technological limitations and facilitate the translation of foundational discoveries into clinical applications.

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