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Review article

The oral microbiome of root caries: A scoping review

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ABSTRACT

Objective: This review characterizes shifts of the oral microbial community on carious root surfaces compared to sound root surfaces.

Data and sources: A systematic search of English-language publications on clinical studies evaluating oral microbiomes in patients with root caries using high-throughput sequencing technologies published before April 1, 2025, was included. The search was conducted on PubMed, Cochrane Central Register of Controlled Trials, Web of Science, Embase, and Scopus. Gray literature was searched in ClinicalTrials.gov and Google Scholar.

Study selection/Results: Based on the eligibility criteria, 1133 publications were screened, and 465 duplicates were removed. Of the remaining 16 studies assessed for full-text review, eight investigating the oral microbiome of saliva, carious roots, or dental plaque in patients with root caries were included. These studies reported the intra-community species diversity (alpha-diversity, 4/8 studies), inter-community compositional diversity (beta-diversity, 4/8 studies), dominant microbial genera/species (8/8 studies), and functional pathways (1/8 studies) of the microbial community in root caries patients. Alpha-diversity showed no significant difference between root caries and sound root surfaces in three studies, but root caries exhibited a significantly lower alpha diversity in one study. Beta-diversity differed significantly between root caries and sound root surfaces in three studies, with one study reporting no difference. The dominant microbial species in root caries varied among the included studies. However, *Lactobacillus* spp., *Prevotella denticola, Propionibacterium acidifaciens, Streptococcus mutans*, and *Veillonella parvula/dispar* were frequently identified in the root caries-associated microbiota. Furthermore, root caries-associated bacteria altered the predicted functional pathways, promoting organic acid production and accelerating collagen degradation.

Conclusion: Root caries microbiomes exhibit distinct compositional profiles, dysbiotic species predominance, and a shift in predicted functional pathways compared to healthy root surfaces.

Clinical significance: This review provides valuable insights into root caries' microbial landscape, potentially guiding future preventative and therapeutic strategies.

1. Introduction

With global demographic shifts toward aging populations and improved dental retention rates, root caries is emerging as a critical concern in modern dental practice. Root caries refer to carious lesions beneath the cementoenamel junction (CEJ), which is more prevalent among the older population [1]. As a subtype of dental caries, root caries is a non-communicable disease caused by tooth-adherent cariogenic bacteria that metabolize fermentable carbohydrates to generate acid, leading to the gradual demineralization of the tooth structure over time [2,3]. Due to differences in anatomy, chemical composition of the tissues, and histology, root surfaces are at a greater risk of developing caries compared to coronal surfaces [4]. Epidemiological evidence from a recent meta-analysis indicates a significant disease burden of root caries, with an annual incidence rate reaching 18.25 % [5]. The impact of root caries extends beyond dental hard issues. They commonly coexist with periodontal disease and are significantly associated with diabetes, creating a complex interplay that affects both oral and general health in older adults [6,7].

Given the multifaceted impacts of root caries, it is essential to develop effective strategies for its prevention and management. Non-restorative management should be prioritized over restorative treatment due to the latter's poor prognosis [8]. To support the development of nonrestorative strategies, studying the root caries microbiome is critical,

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as microorganisms are the primary drivers of the disease. A recent study has demonstrated that microbial community shifts during root caries remineralization could inform the development of novel therapeutic approaches [9]. Therefore, a deeper understanding of the root caries microbiome can elucidate its pathogenic mechanisms and facilitate the development of advanced targeted therapies.

Research on root caries microbiology has evolved from culture-dependent methods to advanced molecular technologies. Initially, studies relied on culture-dependent methods, which captured only a fraction of microbial diversity, thereby limiting our understanding of the root caries microbiome [10]. More recently, the development of high-throughput sequencing technologies has revolutionized the field, enabling the identification of novel caries-related species and the elucidation of intricate microbial interactions within the root caries microbiome [11,12]. Notably, studies have revealed that the microbial composition differs significantly between the coronal and root surfaces, with root caries comprising a richer and more complex microbiome [13, 14].

Although molecular-based studies have investigated the microbiome associated with root caries, consolidated knowledge of its microbial communities remains limited. Therefore, the objective of this review is to systematically analyze the differences in oral microbial communities between root caries and sound root surfaces, aiming to elucidate alterations of microbial community in root caries to advance the understanding of the root caries microbiome and provide a foundation for developing strategies of prevention and management for root caries.

2. Methods

2.1. Protocol and registration

This review adhered to the Preferred Reporting Items for Systematic Reviews and Meta Analyses Extension for Scoping Reviews (PRISMA-ScR) guidelines [15] and was registered on the Open Science Framework (Registration DOI: 10.17605/OSF.IO/QT52B).

The research question of this review was constructed following the PICO (Patient, Intervention, Comparison, Outcome) format: How do changes in the oral microbial community (I) in patients with root caries (P) compare to sound root surfaces (C) in terms of microbial composition and diversity (O).

2.2. Information sources and search strategy

The search for literature was carried out on Dec 30, 2024 and updated on April 1, 2025. Databases, including PubMed, the Cochrane Central Register of Controlled Trials, Web of Science, Embase, and Scopus, were utilized for the publication searches. The detailed search strategy is presented in **Supplementary Table 1**, and the simplified search strategy is as follows:

#1 “root caries” OR “root surface caries” OR “root decay” OR “cementum caries” OR “root cavit*”

#2 “microbiota” OR “microbiome” OR “microbial” OR “biofilm” OR “microorganism” OR “mycobiome” OR “microflora” OR “virome”

#3 [1] AND [2]

In addition, searches for gray literature were conducted using Google Scholar and ClinicalTrials.gov. There were no restrictions on the publication date during the literature search. Duplicate entries were removed. Following the initial screening, the reference lists of the chosen publications were manually reviewed to identify any additional relevant studies.

2.3. Study selection and eligibility criteria

The selection of publications was independently carried out by two researchers (RRZ and JSZ). Any disagreements during the selection process were resolved through consultation with a third author (OYY). Publications were included only if all reviewers agreed unanimously.

The titles and abstracts of the publications were reviewed. Publications were included if they:

(1) were original studies;
(2) were in English;
(3) used healthy root as control;
(4) used samples collected from human root caries; and
(5) adopted molecular-based assessment of the oral microbial community.

Subsequently, the full texts of the remaining publications were examined. Publications were excluded if the studies:

(1) were case reports;
(2) were reviews;
(3) were not relevant to root caries;
(4) used samples obtained from non-human or in vitro models; or
(5) did not assess the entire microbial community of root caries.

2.4. Data extraction and synthesis

Two researchers (RRZ and JSZ) collaboratively identified the variables to be extracted and created the data-charting form based on these variables. Data extraction was performed independently by both researchers. The collected information included general details about the selected articles, including the first author’s name, year of publication, country, patient age, sample size (patient), diagnostic criteria, sample size/N (tooth), type of sample, study design, storage medium, storage temperature, DNA extraction kit, sequencing technique, and amplicons. The results of each study were further reviewed and sequentially extracted. Outcomes were reported according to the following aspects of the root caries microbiome: microbial community diversity (alpha-diversity and beta-diversity), the distributions of significant genera or species based on abundance/prevalence level, and the shift of the predicted functional pathways. Besides, the predominant genera and species in root caries among the included studies were also investigated.

3. Results

3.1. Study inclusion

The literature search and selection process are displayed in a flow diagram in Fig. 1. A comprehensive search strategy yielded 1133 publications from databases including PubMed, the Cochrane Central Register of Controlled Trials, Web of Science, Embase, Scopus, Google Scholar, and ClinicalTrials.gov. Eight publications were included after removing duplicates and screening with exclusion and inclusion criteria. An additional search of the reference lists of the selected articles identified two more publications, which were subsequently excluded after eligibility screening. Ultimately, with the consensus of all reviewers, eight original studies focusing on the microbiome of root caries were included in this scoping review.

3.2. Characteristics of the included studies

3.2.1. Study population

The general details of the eight included studies are summarized in Table 1. Six out of eight studies focused on older adults aged over 59 [16–21], while two studies targeted adults aged 48 to 73 and 53 to 88, respectively [22,23]. Regarding the source of the population, three out of eight studies recruited patients from nursing homes [17,19,20], three from hospitals [18,22,23], one from dentate institutionalized individuals [21], and one lacked information on the recruitment source [16]. In terms of population size, six out of eight studies recruited around 21 to 42 participants [17–21,23], while two out of eight studies

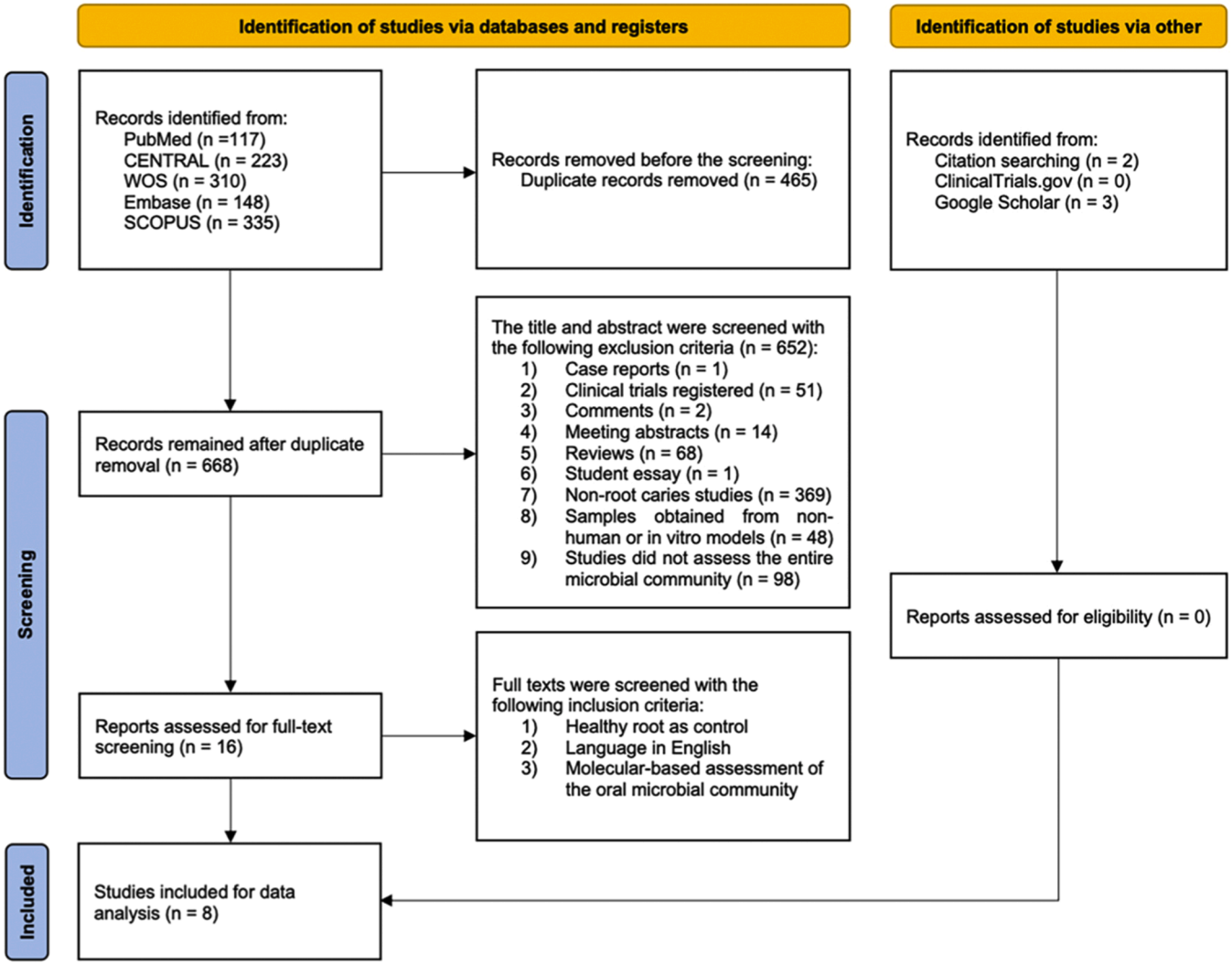

Fig. 1. Flow diagram for study selection.

recruited 6 to 7 participants [16,22], resulting in a total of 219 participants across all studies.

3.2.2. Diagnostic criteria

Regarding the diagnostic criteria of root caries, four out of eight studies applied the World Health Organization (WHO) criteria [17,19,20,23], two studies used the International Caries Detection and Assessment System (ICDAS) criteria [18,21], and two studies did not specify the diagnostic criteria [16,22].

3.2.3. Sample source

Five out of eight studies collected samples from plaque [16,17,21–23], while two studies obtained samples from both plaque and carious dentin in the root caries group [19,20]. Additionally, one study included samples from saliva [18]. The included studies varied widely in terms of sample size, with numbers ranging from 12 to 130.

3.2.4. Study design

All included articles were cross-sectional studies. There were several ways to group the included studies. The first comparison was made between patients with root caries and the sound root surfaces of healthy individuals without root caries (SR-Health), with samples coming from plaque (3/8), carious dentin (2/8), and saliva (1/8). The second comparison was made between root caries and sound root surfaces within the same individuals who had root caries (SR-Patient), with samples derived from plaque (7/8) and carious dentin (2/8). Additionally, Hashimoto et al. [22] compared the supragingival and subgingival plaque community of SR-Patient. Usuga et al. [21] and Ji et al. [23] classified root caries based on different stages of lesions and conducted comparisons of the microbial community of root caries at different stages.

3.2.5. Sample storage

The storage conditions for samples differed across studies. Samples were stored in TE (Tris, EDTA) buffer (3/8), phosphate-buffered saline (PBS) (2/8), potassium phosphate buffer (1/8), and reduced transport fluid buffer (1/8). Only one study did not report the storage medium [18]. Seven out of the eight studies stored their samples at −80 °C [16–21,23], while one study incubated its samples in an anaerobic glove box at 37 °C for one week [22].

3.2.6. Sample processing

Regarding the DNA extraction kits, four out of the eight studies used the QIAamp DNA Mini Kit (Qiagen, Hilden, Germany), the QIAamp DNA Mini Kit (Qiagen, USA), or the QIAamp DNA Mini Kit (Qiagen, California, USA) [18–21]. The other three studies employed the InstaGene Matrix Kit (Bio-Rad Laboratories, California, USA), the MaterPure™ DNA Purification Kit (Epicentre, Wisconsin, USA), and the DNA Extraction Kit (Magen, Guangzhou, China), respectively [16,17,22]. One study described the DNA extraction kit as the Qiagen Blood and

Table 1
General information of the included studies.

Author, Year	Country	Age (Range, Mean)	Sample size (Patient)	Diagnosis criteria	Sample size/N (Tooth)	Type of sample	Study design/n	Storage medium	Storage temperature	DNA extract kit	Sequencing technique	Amplicons
Preza et al. [1]	Norway	82–98, 89	21	WHO	43	Plaque, Dentin	RC plaque=11 RC dentin=11 SR-Patient=11 SR-Health=10	Tris EDTA buffer	−80 °C	QIAamp DNA mini kit (Qiagen, Hilden, Germany)	16S rRNA gene sequencing	N/A
Preza et al. [2]	Norway	70–101, N/A	41	WHO	83	Plaque, Dentin	RC plaque=21 RC dentin=21 SR-Patient=21 SR-Health=20	Tris EDTA buffer	−80 °C	QIAamp DNA mini kit (Qiagen, Hilden, Germany)	16S rRNA gene microarray	N/A
Hashimoto et al. [3]	Japan	48–73, 65.5	6	N/A	12	Plaque	RC=6 SR-Patient/ Supragingival=4 SR-Patient/ Subgingival=5	Potassium phosphate buffer	37 °C	InstaGene Matrix kit (Bio-Rad Laboratories, California, USA)	16S rRNA gene sequencing	V1-V9
Chen et al. [4]	China	66–82, N/A	42	WHO	63	Plaque	RC=21 SR-Patient=21 SR-Health=21	Reduced transport fluid buffer	−80 °C	MaterPure™ DNA purification kit (Epicentre, Wisconsin, USA)	16S rRNA gene sequencing	V3-V5
Abram et al. [5]	USA	65–83, N/A	7	N/A	14	Plaque	RC=7 SR-Patient=7	Phosphate-buffered saline	−80 °C	Qiagen blood and tissue DNA purification kit	16S rRNA gene sequencing	V6
Li et al. [6]	China	60–85, N/A	30	ICDAS	30	Saliva	RC/Initial Active=15 SR-Health=15	N/A	−80 °C	QIAamp DNA mini kit (Qiagen, California, USA)	16S rRNA gene sequencing	V3-V4
Usuga-Vacca et al. [7]	Colombia	>59, N/A	42	ICDAS	130	Plaque	RC/Initial Inactive=23 RC/Initial Active=18 RC/Moderate Inactive=2 RC/Moderate Active=16 RC/Extensive Active=26 SR-Patient=45	Phosphate-buffered saline	−80 °C	QIAamp DNA mini kit (Qiagen, USA)	16S rRNA gene sequencing	V4
Ji et al. [8]	China	53–88, 69.9	30	WHO	90	Plaque	RC/Superficial=30 RC/Deep=30 SR-Patient=30	Tris EDTA buffer	−80 °C	DNA Extraction Kit (Magen, Guangzhou, China)	16S rRNA gene sequencing	V1-V9

Abbreviations: N/A, not applicable; WHO, World Health Organization criteria; ICDAS, International Caries Detection and Assessment System; RC, root caries; SR, sound root.

Tissue DNA Purification Kit [23]. In terms of sequencing techniques, only one study utilized a 16S rRNA gene microarray, whereas the other seven studies performed amplicon sequencing of the V1-V9, V3-V4, V3-V5, V4, or V6 regions of the 16S rRNA gene.

3.2.7. Outcome measures

The primary outcomes measured in the included studies were microbial community diversity, bacterial diversity, and shift of the predicted functional pathways in root caries, as displayed in Table 2. Alpha diversity and beta diversity serve as two indices of microbial community diversity. Specifically, alpha diversity refers to the diversity within a single sample, whereas beta diversity indicates the differences between multiple samples. Furthermore, bacterial diversity can be categorized into abundance and prevalence levels. Prevalence refers to the proportion of samples in which a specific bacterial species is detected. In contrast, relative abundance denotes the proportion of sequencing reads attributed to a particular bacterial species compared to the total reads of all detected bacterial species.

3.3. Main findings of the included studies

3.3.1. The microbial community diversity in root caries

Regarding intra-community species diversity, one out of eight studies found that alpha diversity was significantly lower in root caries compared to sound root surfaces [21], whereas three out of eight studies reported no difference in alpha diversity between the two groups [17,18,23]. As for inter-community microbial compositional dissimilarity, three out of eight studies demonstrated that beta diversity in root caries significantly differed from that in sound root surfaces [17,18,21], while one out of eight studies found no difference in beta diversity between the two groups [23].

3.3.2. The microbial genera/species with shifted abundance in root caries samples

3.3.2.1. Microorganisms with increased abundance. Compared to the sound root surfaces of SR-Patient, five genera were predominant in root caries: *Lactobacillus* (4/8), *Prevotella* (2/8), *Propionibacterium* (3/8), *Streptococcus* (4/8), and *Veillonella* (2/8). Furthermore, when comparing root caries to the sound root surfaces of SR-Health, two dominant genera, *Prevotella* (2/8) and *Veillonella* (2/8), exhibited an increase.

Root caries exhibited higher levels of five dominant species compared to the sound root surfaces of SR-Patient: *Lactobacillus crispatus* (2/8), *Prevotella denticola* (4/8), *Propionibacterium acidifaciens* (3/8), *Streptococcus mutans* (5/8), and *Veillonella parvula/dispar* (4/8). Additionally, in comparison to the sound root surfaces of SR-Health, three notable species including *Prevotella denticola* (2/8), *Streptococcus mutans* (2/8), and *Veillonella parvula/dispar* (2/8), were identified as increased in root caries.

3.3.2.2. Microorganisms with decreased abundance. Based on the results of the included studies, root caries featured a decrease in two bacterial genera when contrasted with the sound root surfaces of SR-Patient: *Actinomyces* (2/8) and *Capnocytophaga* (3/8). Likewise, in contrast to the sound root surfaces of SR-Health, the genera *Neisseria* (2/8) and *Porphyromonas* (2/8) exhibited a reduction in their levels in root caries.

Root caries exhibited lower levels of *Capnocytophaga* sp. (2/8) and *Corynebacterium matruchotii* (2/8) compared to the sound root surfaces of SR-Patient. Besides, in contrast to the sound root surfaces of SR-Health, *Corynebacterium matruchotii* (2/8) was observed to decrease in root caries.

3.3.3. The microbial genera/species prevalent in root caries samples

One out of eight studies reported the predominant genera in root caries, such as *Bifidobacterium, Cryptobacterium,* and *Lactobacillus* [17]. Regarding species, root caries showed increased levels of *Lactobacillus casei/paracasei/rhamnosus* (2/8) compared to the sound root surfaces of SR-Patient. In addition, compared to the sound root surfaces of SR-Health, there was an increase in three dominant species in root caries: *Lactobacillus casei/paracasei/rhamnosus* (3/8), *Lactobacillus* spp. (2/8), and *Streptococcus mutans* (2/8).

3.3.4. Shift of the predicted functional pathways in root caries

Only one out of eight included studies reported the shift in the predicted functional pathways associated with root caries (Table 2). Hashimoto et al. [22] found that root caries-related bacteria could promote organic acid production and accelerate collagen degradation, enhancing the pathogenesis of root caries.

4. Discussion

This review compares the microbial communities of root caries and sound root surfaces using sequencing technologies, offering systematic evidence that the microbial composition of root caries differs from that of healthy root surfaces. By summarizing microbial community diversity and identifying the predominant bacterial genera and/or species in root caries, this study provides new insights into microecological modulation for managing root caries, emphasizing the potential for reconstructing a healthy microbial community as a promising therapeutic strategy.

4.1. The main findings of the included studies

4.1.1. Intra-community diversity

The included studies exhibited no significant difference in alpha-diversity between root caries and sound root surfaces, suggesting that the development of root caries is not driven by the overall complexity of microbial communities. For example, while pathogenic bacteria like *Streptococcus mutans* and *Lactobacillus* spp. are more abundant in root caries, and beneficial bacteria are reduced, these specific shifts may not be reflected in alpha-diversity metrics, which measures overall community richness and evenness. Instead, functional differences in root caries-associated microbiota, such as enhanced acid production or biofilm formation, are not captured by alpha diversity metrics [24,25].

4.1.2. Inter-community diversity

Based on the results of included studies, root caries exhibited a significantly higher beta-diversity in microbial community structure compared to sound root surfaces [17,18,21], which was consistent with the prior view that caries microbiomes exhibited increased variability in community structure across samples [26]. This implies that the organismal organization of root-caries-associated microbiotas are significantly more variable, while the healthy microbiome demonstrates a greater conservation.

The inter-community variability in root caries differs across disease developmental stages [21], suggesting that the microbial community undergoes dynamics shifts during the lesion progression, potentially influencing treatment outcomes and the approach to managing root caries.

4.1.3. The dominant genera/species in root caries

Based on the included studies, we summarized the predominant genera associated with root caries, including *Bifidobacterium, Lactobacillus, Prevotella, Propionibacterium, Streptococcus*, and *Veillonella* (Fig. 2). These dominant genera generally align with the common species found in root caries, such as *Lactobacillus* spp., *Prevotella denticola, Propionibacterium acidifaciens, Streptococcus mutans*, and *Veillonella parvula/dispar*, all of which play a critical role in the pathogenesis of root caries, as will be discussed further in the following sections.

4.1.3.1. Lactobacillus spp. *Lactobacillus* spp. were strongly implicated in

Table 2
Main findings of the included studies.

Author, Year	Study design		Microbial community diversity		Bacterial diversity				Shift of the predicted functional pathways in Gp-T
	Test group (Gp-T)	Control group (Gp-C)			Distributions of abundance-based significant genera or species		Distributions of prevalence-based significant genera or species		
			Alpha diversity	Beta diversity	Increased abundance in Gp-T	Decreased abundance in Gp-T	Predominant presence in Gp-T (>30 %)	Predominant presence in Gp-C (>30 %)	
Preza et al. [1]	RC plaque	SR-Health plaque	N/A	N/A	• *Prevotella denticola* • *Selenomonas* sp. clone CS002 • *Veillonella parvula/dispar*	• *Corynebacterium matruchotii* • *Fusobacterium nucleatum* subsp. *polymorphum* • *Leptotrichia* spp. • *Selenomonas noxia* • *Streptococcus gordonii* • *Streptococcus cristatus*	• *Atopobium* spp. • *Lactobacilli* spp. • *Olsenella* spp. • *Prevotella denticola* • *Selenomonas* sp. clone CS002 • *Selenomonas* sp. strain GAA14	• *Campylobacter curvus* • *Corynebacterium matruchotii* • *Fusobacterium nucleatum* subsp. *polymorphum* • *Kingella oralis* • *Leptotrichia* spp. • *Prevotella conceptionensis* • *Selenomonas noxia* • *Selenomonas* sp. clone AA024/DS051 • *Streptococcus cristatus*	N/A
	RC plaque	SR-Patient plaque	N/A	N/A	• *Prevotella denticola* • *Selenomonas* sp. clone CS002 • *Veillonella parvula/dispar*	• *Campylobacter gracilis* • *Selenomonas sputigena*	• *Atopobium* spp. • *Olsenella* spp. • *Selenomonas* sp. clone CS002	• *Selenomonas* sp. clone FT050	N/A
	RC dentin	SR-Health plaque	N/A	N/A	• *Actinomyces* sp. clone IP073 • *Actinomyces* spp. • *Atopobium* spp. • *Enterococcus faecalis* • *Lactobacillus casei/paracasei/rhamnosus* • *Lactobacillus* spp. • *Olsenella* spp. • *Propionibacterium* sp. strain FMA5 • *Pseudoramibacter alactolyticus* • *Selenomonas* sp. clone CS002 • *Streptococcus mutans*	• *Corynebacterium matruchotii* • *Fusobacterium nucleatum* subsp. *polymorphum* • *Granulicatella adiacens* • *Leptotrichia* spp. • *Prevotella conceptionensis* • *Selenomonas noxia* • *Streptococcus gordonii* • *Streptococcus cristatus* • *Veillonella parvula/dispar* • *Veillonella* sp. clone AA050	• *Atopobium* spp. • *Lactobacillus casei/paracasei/rhamnosus* • *Lactobacillus* spp. • *Olsenella* spp. • *Propionibacterium* sp. strain FMA5 • *Selenomonas* sp. clone CS002 • *Streptococcus mutans*	• *Campylobacter curvus* • *Corynebacterium matruchotii* • *Fusobacterium nucleatum* subsp. *polymorphum* • *Kingella oralis* • *Leptotrichia* spp. • *Prevotella conceptionensis* • *Selenomonas noxia* • *Streptococcus cristatus* • *Streptococcus mitis* bv. 2 • *Streptococcus sanguinis*	N/A
	RC dentin	SR-Patient plaque	N/A	N/A	• *Actinomyces* sp. clone IP073 • *Actinomyces* spp. • *Atopobium* spp. • *Enterococcus faecalis* • *Lactobacillus casei/paracasei/rhamnosus* • *Lactobacillus* spp. • *Olsenella* spp. • *Propionibacterium* sp. strain FMA5 • *Pseudoramibacter alactolyticus* • *Selenomonas* sp. clone CS002 • *Streptococcus mutans*	• *Campylobacter gracilis* • *Fusobacterium nucleatum* subsp. *polymorphum* • *Selenomonas infelix* • *Selenomonas noxia* • *Selenomonas sputigena* • *Veillonella parvula/dispar* • *Veillonella* sp. clone AA050	• *Atopobium* spp. • *Lactobacillus casei/paracasei/rhamnosus* • *Olsenella* spp. • *Propionibacterium* sp. strain FMA5 • *Selenomonas* sp. clone CS002	• *Campylobacter gracilis* • *Leptotrichia* spp. • *Prevotella nigrescens* • *Selenomonas* sp. clone FT050 • *Selenomonas sputigena*	N/A

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Table 2 (*continued*)

Author, Year	Study design		Microbial community diversity		Bacterial diversity				Shift of the predicted functional pathways in Gp-T
	Test group (Gp-T)	Control group (Gp-C)			Distributions of abundance-based significant genera or species		Distributions of prevalence-based significant genera or species		
			Alpha diversity	Beta diversity	Increased abundance in Gp-T	Decreased abundance in Gp-T	Predominant presence in Gp-T (>30 %)	Predominant presence in Gp-C (>30 %)	
Preza et al. [2]	RC plaque	SR-Health plaque	N/A	N/A	N/A	N/A	N/A	• *Campylobacter concisus* • *Campylobacter showae* • *Campylobacter* spp. • *Capnocytophaga cluster* • *Capnocytophaga sputigena* • *Cardiobacterium hominis* • *Fusobacterium nucleatum* subsp. *polymorphum* • *Gemella morbillorum* • *Leptotrichia cluster* • *Streptococcus cristatus/ S.* sp. clone BM053	N/A
	RC plaque	SR-Patient plaque	N/A	N/A	N/A	N/A	• *Streptococcus oralis* • *Streptococcus parasanguinis*	N/A	N/A
	RC dentin	SR-Health plaque	N/A	N/A	N/A	N/A	• *Lactobacillus casei/ paracasei/rhamnosus* • *Lactobacillus* spp. • *Pseudoramibacter alactolyticus*	• *Campylobacter concisus* • *Capnocytophaga sputigena* • *Eubacterium saburreum* • *Eubacterium* spp. • *Fusobacterium nucleatum* subsp. *polymorphum* • *Gemella morbillorum* • *Leptotrichia cluster* • *Propionobacterium* spp.	N/A
Hashimoto et al. [3]	RC plaque	SR-Patient/ Supra-plaque	N/A	N/A	• *Bifidobacterium* • *Lactobacillus* • *Propionibacterium*	• *Actinomyces* • *Capnocytophaga* • *Streptococcus* • *Veillonella*	N/A	N/A	• Increased organic acids production • Increased collagen degradation
	RC plaque	SR-Patient/ Sub-plaque	N/A	N/A	• *Bifidobacterium* • *Lactobacillus* • *Propionibacterium*	• *Actinobaculum* • *Eubacterium* • *Olsenella* • *Prevotella*	N/A	N/A	N/A
Chen et al. [4]	RC plaque	SR-Health plaque	No difference	Significant difference	• *Lactobacillus crispatus* • *Prevotella denticola* • *Prevotella multisaccharivorax* • *Propionibacterium acidifaciens* • *Streptococcus mutans* • *Veillonella dispar*	• *Capnocytophaga granulosa* • *Capnocytophaga leadbetteri* • *Corynebacterium matruchotii* • *Delftia acidovorans* • *Fusobacterium* sp. • *Porphyromonas* sp. • *Prevotella intermedia*	• *Bifidobacterium dentium* • *Cryptobacterium curtum* • *Lactobacillus crispatus* • *Lactobacillus fermentum* • *Lactobacillus gasseri* • *Lactobacillus panis* • *Lactobacillus paracasei* • *Lactobacillus salivarius* • *Olsenella profusa* • *Oribacterium* sp. • *Prevotella multisaccharivorax* • *Prevotella oralis* • *Propionibacterium acidifaciens* • *Scardovia wiggsiae* • *Shuttleworthia satelles* • *Streptococcus mutans*	• *Alloprevotella* sp. • *Bacteroidetes*[G-2] sp. • *Bacteroidetes*[G-5] sp. • *Bergeyella* sp. • *Cardiobacterium valvarum* • *Catonella morbi* • *Delftia acidovorans* • *Eubacterium*[11][G-6] *nodatum* • *Fretibacterium* sp. • *Gemella sanguinis* • *Lachnoanaerobaculum* sp. • *Lachnoanaerobaculum umeaense* • *Lachnospiraceae*[G-3] sp. • *Lachnospiraceae*[G-8] sp. • *Peptococcus* sp.	N/A

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Table 2 (*continued*)

Author, Year	Study design		Microbial community diversity		Bacterial diversity				Shift of the predicted functional pathways in Gp-T
	Test group (Gp-T)	Control group (Gp-C)			Distributions of abundance-based significant genera or species		Distributions of prevalence-based significant genera or species		
			Alpha diversity	Beta diversity	Increased abundance in Gp-T	Decreased abundance in Gp-T	Predominant presence in Gp-T (>30 %)	Predominant presence in Gp-C (>30 %)	
								• *Peptostreptococcaceae*[11][G-7] sp. • *Peptostreptococcus stomatis* • *Prevotella intermedia* • *Prevotella saccharolytica* • *Propionibacterium propionicum* • *Tannerella* sp. • *Treponema* sp.	
	RC plaque	SR-Patient plaque	No difference	Significant difference	• *Lactobacillus crispatus* • *Prevotella denticola* • *Prevotella multisaccharivorax* • *Propionibacterium acidifaciens* • *Streptococcus mutans* • *Veillonella dispar*	• *Capnocytophaga granulosa* • *Capnocytophaga* sp. • *Corynebacterium matruchotii* • *Delftia acidovorans*	• *Bifidobacterium dentium* • *Cryptobacterium curtum* • *Lactobacillus crispatus* • *Lactobacillus fermentum* • *Lactobacillus gasseri* • *Lactobacillus panis* • *Lactobacillus paracasei* • *Lactobacillus salivarius* • *Olsenella profusa* • *Oribacterium* sp. • *Prevotella multisaccharivorax* • *Prevotella oralis* • *Propionibacterium acidifaciens* • *Scardovia wiggsiae* • *Shuttleworthia satelles* • *Streptococcus mutans*	• *Bacteroidetes*[G-2] sp. • *Bergeyella* sp. • *Cardiobacterium valvarum* • *Delftia acidovorans* • *Gemella sanguinis* • *Lachnospiraceae*[G-3] sp. • *Prevotella intermedia* • *Prevotella saccharolytica* • *Propionibacterium propionicum* • *Tannerella* sp.	N/A
Abram et al. [5]	RC plaque	SR-Patient plaque	N/A	N/A	• *Actinomyces* sp. • *Capnocytophaga* spp. • *Delftia* sp. • *Propionibacterium acidifaciens* • *Propionibacterium* sp. • *Selenomonas* spp. • *Streptococcus mutans* • *Streptococcus* spp. • *Veillonella* spp.	• *Capnocytophaga* sp.	N/A	N/A	N/A
Li et al. [6]	RC saliva	SR-Health saliva	No difference	Significant difference	• *Leptotrichia* • *Prevotella* • *Veillonella*	• *Neisseria* • *Porphyromonas*	N/A	N/A	N/A
Usuga-Vacca et al. [7]	RC/Initial plaque	SR-Patient plaque	Gp-*T*<Gp-C*	Significant difference	• *Prevotella* sp. HMT313	• *Leptotrichia wadei*	N/A	N/A	N/A
	RC/Moderate & Extensive plaque	SR-Patient plaque	Gp-*T*<Gp-C*	Significant difference	• *Fusobacterium nucleatum* subsp. *animalis* • *Lactobacillus crispatus* • *Lactobacillus fermentum* • *Mitsuokella* sp. HMT131 • *Prevotella denticola*	• *Fusobacterium periodonticum* • *Fusobacterium* sp. HMT203 • *Porphyromonas pasteri*	N/A	N/A	N/A

(*continued on next page*)

Table 2 (*continued*)

Author, Year	Study design		Microbial community diversity		Bacterial diversity				Shift of the predicted functional pathways in Gp-T
	Test group (Gp-T)	Control group (Gp-C)			Distributions of abundance-based significant genera or species		Distributions of prevalence-based significant genera or species		
			Alpha diversity	Beta diversity	Increased abundance in Gp-T	Decreased abundance in Gp-T	Predominant presence in Gp-T (>30 %)	Predominant presence in Gp-C (>30 %)	
	RC/Inactive plaque	SR-Patient plaque	Gp-*T*<Gp-C*	Significant difference	• *Veillonella dispar*	• *Leptotrichia wadei* • *Veillonella parvula* • *Veillonella atypica* • *Fusobacterium periodonticum*	N/A	N/A	N/A
	RC/Active plaque	SR-Patient plaque	Gp-*T*<Gp-C*	Significant difference	• *Fusobacterium nucleatum* subsp. *animalis* • *Prevotella denticola* • *Lactobacillus crispatus* • *Lactobacillus fermentum* • *Streptococcus anginosus*	• *Fusobacterium* sp. HMT203 • *Porphyromonas pasteri*	N/A	N/A	N/A
Ji et al. [8]	RC/Superficial plaque	SR-Patient plaque	No difference	No difference	• *Actinomyces* sp. HMT 448 • *Campylobacter gracilis* • *Capnocytophaga* sp. HMT 412 • *Lactobacillus pentosus* • *Prevotella denticola* • *Prevotella melaninogenica* • *Prevotella* sp. HMT 300 • *Propionibacterium acidifaciens* • *Streptococcus mutans*	• *Corynebacterium matruchotii*	N/A	N/A	N/A

Abbreviations: RC, root caries; SR, sound root; N/A, not applicable; *Capnocytophaga cluster, C. gingivalis* oral taxon 337, *C. granulosa* oral taxon 325, *C.* sp. clone AH105, *C.* sp. clone S3, *C.* sp. oral taxon 325, *C.* sp. clone BB167, *C.* sp. clone TFI Cap08; *Leptotrichia cluster*, L. *buccalis* oral taxon 563, L. sp. oral taxon 498/462/463/417, L. sp. clone IK040/GT018/GT020/ C3MK102.
*Alpha diversity: species richness.

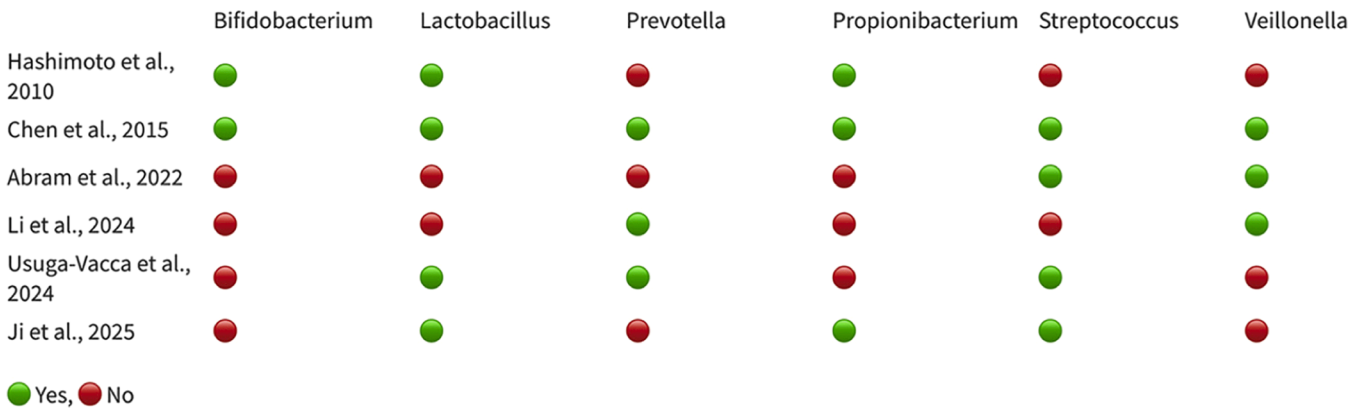

Fig. 2. Overview of the predominant genera in root caries compared with sound root surfaces among included studies.

the root-caries progression [27–29]. The pathogenesis of root caries is a continuous process, with *Lactobacillus* spp. playing a critical role during the aciduric stage, a phase characterized by the dominance of acidogenic and aciduric bacteria in the carious lesion. *Lactobacillus* spp. metabolize sugars under acidic conditions, contributing to the demineralization of tooth structures and the progression of carious lesions [10]. Their cariogenicity stems from robust acid tolerance, mediated by adaptive mechanisms, including translation and biogenesis, nucleotide transport, and energy production [30].

4.1.3.2. Prevotella denticola. *Prevotella* species, a ubiquitous group of oral bacteria detected in healthy populations, active caries, and endodontic infections [26,31,32], exhibited pronounced cariogenic potentials. Specifically, *Prevotella denticola* has been identified as a potential caries predictor [33], as it can produce acid via carbohydrate metabolism and form resilient biofilms [34]. Such a cariogenic potential is further supported by its high abundance and prevalence in root caries plaque across studies [17,19,21,23].

4.1.3.3. Propionibacterium acidifaciens. *Propionibacterium acidifaciens* has been identified as the predominant species in root caries in the included studies [16,17,23]. Its pathogenic mechanism is primarily attributed to its ability to bind to collagen and colonize dentinal tissue. Under low pH conditions, the acid production by *Propionibacterium acidifaciens* further drives the progression of caries [35].

4.1.3.4. Streptococcus mutans. *Streptococcus mutans* has been extensively studied for its role in caries development [25,36]. Evidence-based research shows that the high prevalence and abundance of *Streptococcus mutans* are strongly correlated with an increased incidence of caries [37]. Its functional adaptations, such as sugar metabolism (e.g., starch, sucrose, and lactose), heterofermentative pathways, cell-wall biosynthesis, and acid tolerance stress, are enriched on carious root surfaces, providing ecological advantages [38]. These traits enable *Streptococcus mutans* to colonize persistently, significantly elevating root caries risk [39,40].

4.1.3.5. Veillonella parvula/dispar. *Veillonella parvula/dispar* stands out as a dominant microbial species in root caries [16,17,19,21]. Its synergize with *Streptococcus mutans* to accelerate disease progression by converting lactic acid into weaker acids (acetate and propionate), facilitating mixed biofilm formation. These cross-species metabolic interactions play a key role in the transition from a healthy state to disease in dental plaque [16]. In vitro and animal studies further demonstrate that the colonization of *Veillonella parvula* enhances the abundance and virulence of *Streptococcus mutans* and *Candida albicans*, leading to polymicrobial biofilms with increased cariogenic potential and exacerbating root caries severity [18].

4.1.4. Predicted functional pathway

A pilot study on the protein-denaturing activity of plaque microflora revealed increased acid production and collagen degradation in root caries [22], suggesting a shift in the predicted functional pathway within carious lesions. This alteration was attributed to the synergistic interaction between protein-coagulating and protein-degrading bacteria in root caries. Protein-coagulating bacteria (e.g., *Actinomyces, Bifidobacterium, Lactobacillus*, and *Streptococcus*) produce organic acids denature proteins, such as the collagen in root dentin and cementum. Meanwhile, protein-degrading bacteria (e.g., *Actinobaculum, Prevotella*, and *Propionibacterium*) break down the denatured proteins into smaller peptides, contributing to the initiation and progression of root caries. Subsequently, host-derived proteases (e.g., matrix metalloproteinases) in saliva and dentin are activated in the low pH environment created by cariogenic bacteria, leading to the demineralization of the dentin matrix [41,42].

4.2. Microbial dynamics in root caries

The microbial dynamics of root caries are significantly influenced by the interaction between pathogenic and health-related species, emphasizing the critical role of ecological balance in caries prevention and management. Chen et al. [17] classified these microorganisms into pathogens and probiotics, demonstrating that among 18 root caries-related species, 10 were absent in sound root surfaces, while only 3 out of 24 health-related species were undetected in root caries. This pattern aligns with the “Specific Plaque Hypothesis”, which suggests that specific microorganisms are the primary contributors to caries development [43,44]. Concurrently, the presence of most health-related species in root caries supports the “Ecological Plaque Hypothesis”, positing that caries arise from microbial dysbiosis rather than mere pathogen presence [24,45].

4.3. Root caries and periodontal diseases

Root caries is closely associated with periodontal diseases, as periodontal pathogens can colonize root caries lesions, leading to distinct microbial variations depending on lesion location. Takenaka et al. [46] demonstrated that root caries extending beyond the gingival margin harbor a more diverse bacterial community compared to supragingival lesions, including periodontal pathogens such as *Porphyromonas, Selenomonas*, and *Tannerella*. Evidence-based studies further support the connection between root caries and periodontal diseases [47], indicating that periodontal pathogens may interact with root surfaces and contribute to carious lesion development. These findings underscore the potential synergy between cariogenic and periodontal pathogens in the etiology of root caries.

4.4. The methodological variation of included studies

In this review, the included clinical studies provided data on microbial communities and bacterial compositions associated with root caries. However, the methodologies employed in these trials varied significantly, leading to discrepancies in their conclusions. Therefore, we aim to discuss the potential reasons for these inconsistent outcomes.

4.4.1. Sample type

Variations in sample types (e.g., dentin versus plaque) across root caries studies can influence the intra-community diversity. The dentinal samples exhibited the lower diversity than plaques from sound root surfaces and root caries [19], which may be attributed to their isolated and specialized niche. This disparity suggests that sample types shape microbial community composition, highlighting the importance of standardization of sampling protocols and a careful interpretation according to sample types in the root caries microbiome studies.

4.4.2. Study design

Control group selection critically impact study outcomes, as the microbial composition may differ significantly between healthy individuals and patients with healthy sites [48,49]. Preza et al. [20] found that the variations between patients and healthy individuals were more outstanding than those observed between samples from healthy and diseased sites within the patients. Chen et al.'s [17] study also found that the variability observed in SR-Patient was moderate compared to that in root caries and SR-Health, indicating that SR-Patient was in a transitional phase between health and root caries, yet they were more prone to a healthy state.

4.4.3. Individual differences

Inter-individual variability due to genetic or immunity affects the microbiome diversity studies. A significant difference in alpha-diversity was observed between SR-Health and SR-Patient in the study by Chen et al. [17]. This suggests that individual differences in host factors, such as genetics and immune response, may play a crucial role in shaping the microbial communities present in the oral environment.

Due to such inter-individual variability, the dominant species of root caries exhibit slight differences compared to those in SR-Health and SR-Patient (Figs. 3 and 4). Only when comparing root caries with SR-Patient, the following three species were found to be predominant: *Lactobacillus crispatus, Lactobacillus fermentum*, and *Propionibacterium acidifaciens* (Fig. 4). These findings highlight the importance of considering variability among individuals when studying the microbial landscape of root caries.

4.5. Limitations and future perspectives

Following the eligibility criteria, only eight studies met the established standards. It is crucial to highlight that several limitations were identified within these studies. First, there is individual variability among the included studies, particularly concerning the population source and selection criteria of participants. The microbial composition varied among populations with different characteristics and lifestyles. Hence, the baseline conditions of the participants, such as smoking status, the presence of teeth, systemic diseases, antibiotic intake, and periodontal treatments, can influence the microbial profile associated with root caries [50–52].

Second, different pretreatment methods for sampling can significantly impact microbiome outcomes. For instance, factors such as not cleaning teeth, eating and drinking, and not use of cotton isolation can influence the results. The biofilm on root surfaces is closely related to the gingival status and crevicular fluid. Therefore, it is crucial to prevent plaque sample contamination from other sources, such as saliva.

Third, there is a notable discrepancy in the diagnostic criteria for root caries. Notably, two of the eight studies did not specify the diagnostic criteria used. Among the remaining studies, two classified root caries by severity (initial vs. moderate/extensive) and activity (inactive vs. active) based on the ICDAS criteria, while one study categorized lesions by depth (superficial vs. deep) according to the WHO criteria. Given that different stages of carious lesions can significantly influence microbial community diversity and dominant species, a clear distinction of lesion status is crucial for a comprehensive analysis of the microbial profile.

In addition, not all studies report on sample storage media and the amplicons used for sequencing, which hindered the ability to gather comprehensive information on the methods applied. Additionally, in one study, bacteria were isolated using an anaerobic glove box prior to DNA extraction [22]. This approach may significantly bias the microbial representation of the root caries microbiome, complicating cross-study synthesis. Furthermore, a limitation inherent in this scoping review stems from the potential bias introduced by using microbial reference genomes in 16S rRNA gene analysis, making it challenging to consistently synthesize findings across multiple studies.

The observed differences between carious and sound surfaces may inform non-invasive treatments, such as targeted antimicrobial therapies or remineralizing agents. However, definitive clinical recommendations are currently limited by methodological heterogeneity and small sample sizes. To translate these findings into practical treatment strategies, future research should focus on larger cohorts, clear diagnostic criteria, standardized methodologies, and longitudinal studies. Specifically, a comprehensive and standardized protocol for sample collection, DNA extraction, sequencing, database usage, and data analysis is essential to enable robust data synthesis and quantitative analysis. Such an approach would strengthen translational insights, guiding evidence-based prevention and treatment strategies.

Concurrently, conventional metagenomic approaches face technical limitations in resolving strain-level diversity and functional dynamics. To address this, we advocate for advancing novel methods enabling strain-resolved profiling and functionally annotated spatio-temporal mapping of root caries microbiomes. Integrating these innovations will bridge critical gaps in understanding microbial etiology and ecological shifts, ultimately improving clinical strategies for root caries management.

5. Conclusion

This review contrasted the microbial communities associated with root caries and those of healthy root surfaces. The microbial results from the included studies indicated that root caries exhibited distinct inter-

Fig. 3. Overview of the predominant species in root caries compared with SR-Health among included studies.

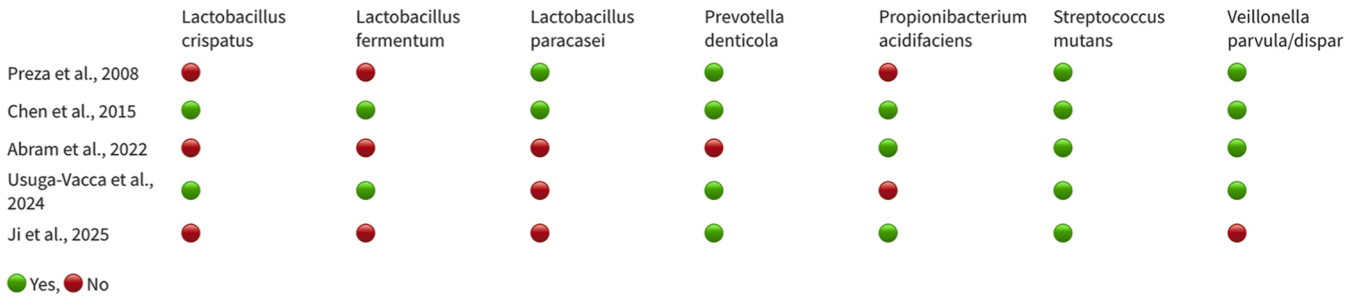

Fig. 4. Overview of the predominant species in root caries compared with SR-Patient among included studies.

community composition, shifts in predominant species (e.g., *Streptococcus mutans, Lactobacillus* spp.), and alterations in predicted functional pathways (e.g., acidogenesis, collagen degradation) when compared to sound root surfaces.

CRediT authorship contribution statement

Ronnie Ruonan Zhang: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation. **Josie Shizhen Zhang:** Validation, Methodology, Investigation, Data curation. **Shi Huang:** Writing – review & editing. **Walter Yu-Hang Lam:** Writing – review & editing, Supervision. **Chun-Hung Chu:** Writing – review & editing, Supervision. **Ollie Yiru Yu:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Methodology, Conceptualization.

Declaration of competing interest

The authors declare no conflict of interest.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jdent.2025.105899.

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