

ORAL MICROBIOME DYNAMICS AND THEIR ROLE IN DENTAL HEALTH AND DISEASE

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ABSTRACT

The oral microbiome is a complex microbial ecosystem that contributes to dental health, biofilm stability, immune regulation, and disease-associated dysbiosis. This study analyzed oral-associated microbial records from the Human Microbiome Project catalog to characterize taxonomic structure, genome-resource quality, gene-count variation, and disease-relevant microbial groups. A total of 2,915 microbial records were screened, of which 347 were identified as oral-associated, representing 11.90% of the complete catalog. Oral records were predominantly bacterial, with 335 bacterial entries accounting for 96.54% of the oral subset. Genome-resource quality was high, as 317 oral records were complete, and most were classified as high-quality draft genomes. Genus-level profiling showed strong representation of *Streptococcus*, *Prevotella*, *Selenomonas*, *Treponema*, *Actinomyces*, *Capnocytophaga*, *Porphyromonas*, *Fusobacterium*, and *Veillonella*. Disease-relevance mapping revealed that periodontal dysbiosis-associated genera formed the strongest biological signal, followed by plaque-structuring and caries-associated taxa. Gene-count analysis suggested genomic variation among oral microorganisms, while Human Oral Microbiome Database mapping supported oral taxonomic relevance for a substantial proportion of records. The findings indicate that oral-associated catalog records provide a valuable reference framework for understanding oral microbial ecology and dental disease mechanisms. The absence of clinical disease labels, abundance profiles, and longitudinal observations limits direct causal interpretation. Future studies should integrate catalog-based profiling with clinical metagenomics, salivary biomarkers, and patient-level dental data.

KEYWORDS: Oral microbiome, dental caries, periodontal dysbiosis, Human Microbiome Project, biofilm ecology, precision dentistry

INTRODUCTION

The mouth cavity is a richly diversified microbial environment, and also lives in the saliva, supragingival plaque, subgingival plaque, tongue dorsum, gingival sulcus, buccal mucosa, palate, and other areas of the mouth. The majority of these communities are bacterial, but contain fungi, viruses, archaea, and protozoa, and interact through biofilm formation, metabolic interaction, coaggregation, signaling, and host immune responses. Oral microbiome is involved in the process of oral homeostasis by colonization resistance, immune regulation, pH homeostasis, nitrate metabolism, as well as mucosal integrity [1]. This loss of balance can lead to the development of the microbial community into dysbiosis that gives the environment that favors dental caries, gingivitis, periodontitis, oral mucosal inflammation, and other pathophysiology. The oral microbial imbalance is also making it into the list of systemic disorders through inflammatory pathways, immune pathways, and microbial translocation pathways [2].

The dental diseases are no longer diseases caused by specific pathogens, but are seen as polymicrobial and ecological diseases. When the acidogenic and aciduric organisms are more active in the carbohydrate-rich and acidic environment, which leads to demineralization and structural damage of the enamel, the caries develop. The anaerobic-related, proteolytic, and inflammatory-related microbial communities that cause inflammation of the gums, loss of connective tissue, and loss of alveolar bone are also other causes of periodontal disease [1]. The other case of oral microbial dysbiosis that advances to clinical significance is during pregnancy, when the alteration of hormonal, immune, dietary, and vascular changes leads to the alteration of the inflammation of the gingiva and the alteration of the microbial balance [3]. The processes show that oral health depends on ecosystem stability, and malfunction of local environmental factors and host response are reflected in oral disease.

The oral microbiome has numerous clinical implications for the mouth. The microbial products, oral microbes, and the inflammatory mediators may have access into the systemic physiology through dissemination via blood, immune activation, oral to gut translocation of microbes, and these microbes by chronic inflammatory signaling [2]. The cardiovascular disease, in part, due to periodontal inflammatory disease, bacteremia, endothelial dysfunction, platelet activation, and inflammatory mediators in the bloodstream, is one of the representatives of the systemic diseases, which are linked to the oral microbial dysbiosis [4]. Another aspect that influences the oral-gut-brain axis is diet, which alters the microbial substrates, salivary chemistry, biofilm metabolism, microbial metabolites, immune responses, and neuroactive signaling [5]. The implications of these relationships are that oral microbial communities can be local dental biofilms and components of a larger host-microbiome communication system.

The host physiology also further regulates the microbiome-related disease pathways by immune, metabolic, and inter-organ signaling. Such interactions of myokines with oral microbiology, but not specific, indicate how the manner in which molecular mediators by the host can regulate inflammation, metabolism, and tissue-level communication, can be utilized to explain disease by the microbiome systemically [6]. There is also the effect of age and childhood exposures on the establishment of the oral microbiome. It has been shown that the microbial changes in children and early childhood shift in the pattern of their colonization to the tooth surface are influenced by the eruption pattern of the teeth, feeding patterns, antibiotic exposure, hygiene patterns, and diet [7]. These developmental concerns make pediatric oral microbiome research relevant to the dentistry discipline through prevention research.

Another dimension of clinical practice is the dynamics of the microbiome in the course of pregnancy. These changes in microbes throughout pregnancy have been linked to immune changes and the risk of preterm birth, and this indicates that the changes in the microbiome of body sites in pregnancy may be among the factors that have caused inflammatory effects on pregnancy [8]. Colonization of the oral microbes is accomplished at the molecular level through glycan-mediated adhesion, carbohydrate recognition, host surface molecules, and microbial surface structures. This microbial glycolanguage influences the adhesion, maintenance, communications, and organization of oral microorganisms into biofilms [9]. Such kinds of molecular mechanisms help to explain why some taxa are over-represented in some oral niches and how microbial communities transition to disease-associated dysbiosis.

Systemic microbiomes can also be investigated by oral microbial investigations. These enteral microbial transitions have been linked to myocardial fibrosis via inflammatory and metabolic pathways that support the general concept that microbial communities may have a remote impact on organ systems [10]. Further illustrations of host microbiome interaction can be seen in infectious pathologies, such as tuberculosis, where gut microbiota can control immune response, susceptibility to disease, and response to therapy [11]. It has been proposed that there exists an oral-gut microbiome-brain axis between orally-gut microbial communities and cognition, and this occurs through immune, metabolic, neural, and inflammatory mechanisms [12]. These systemic numbers provide further reasons to consider oral microorganisms as not only local communities of the dentular space, but as part of bigger host-microbiome platforms.

The study will also seek to profile an oral microbiome using Human Microbiome Project catalog records to characterize oral-based microbial organisms, genomes, resource quality, genus-level hierarchy, fluctuation of the quantity of genes, and disease-related taxa. The technique fosters a catalogue style of knowledge about the type of oral microbes and the consequences to dental health and illness. Patterns of oral microbes will also be presented that are relevant to the plaque ecology, caries-related process, periodontal dysbiosis, and future microbiome-informed studies of the dental field.

METHODOLOGY

Study Design

The study employed a secondary bioinformatic and descriptive metadata analysis of records of Human Microbiome Project catalogs. The presentation centered on the process of identifying the microbial organisms, which are related to the mouth, and the explanation of their impact on the mouth and on dental diseases. The research was a genome-catalog-based oral microbiome profiling research, not a clinical case-control study, because the data available does not include a patient-level disease status, microbial abundance values, dental indexes, and longitudinal measurements. Classifying oral microbes, profiling based on genus, the number of genes, completeness of genome project, mapping of the Human Oral Microbiome Database, and interpreting disease-related oral microbial clusters with the assistance of the literature were the key questions of analysis.

Source and Description

The research was also based on Human Microbiome Project catalog data, which included information on microbial organisms and genome projects. The catalog included organism identity, microbial domain, NCBI superkingdom, body site of isolation, project status, level of genome finishing, NCBI submission information, Genbank accession information, number of genes, IMG/HMP identifier, Human Oral Microbiome Database identifier, sequencing center, source of funding, and strain repository information. The number of potential microbes that could be screened was 2,915. Of these, 347 of them were determined to have been oral-associated, or about 11.90 percent. of the total record. These correlated records that were acquired in the oral cavity formed the main material of analysis that was utilized to define the diversity of the microbial oral and its applicability to oral dental.

Screening and Preprocessing

All records were checked to determine completeness, oral-site annotation, consistency of project name of the organism, microbial domain, project status, finishing degree, and the number of genes. The entries were selected based on the isolation body-site field because, in this manner, only oral entries were chosen, where the oral microbiome was actually being studied, and non-oral entries that were simply used to provide a general contextual comparison. The numbers of the gene counts were transformed into numbers to have a chance to perform the descriptive analysis. Organism name name

Standardized name of organisms, where needed, and the genus name were eliminated from the first taxonomic name of the organism. Zero-gene entries were left in their original form but with a very high dose of scepticism, perhaps due to unfinished annotation, or the lack of genome data, and not necessarily biological non-presence.

Analytical Variables

The key variables of interest included the name of the organism, microbial domain, NCBI superkingdom, source of isolation, project status, genome finishing degree, number of genes, identifier of the Human Oral Microbiome Database, sequencing centre, and funding source. The taxonomic level and genus level profiling was made possible with the use of the organism name supported and oral-related microorganisms were selected on oral-site annotation. Genes were used as a proxy measure to determine genome annotation and functional capabilities. The project status and level of finishing the genome resources determined the completeness of records and the quality of the genome resources. Identifiers of human oral microbiome databases were included as they offer an oral microbiology-focused taxonomical sense of an oral definition of health and disease.

Oral Microbiome Profiling

Oral-site annotation records, which included oral-site annotation, were selected and characterized by microbiomes by subdivision by taxonomic and genome-project features. Genus-level profiling was done using the first word of the organism names as taxonomic names and approximating the genus proportion of microorganisms of the mouth. This relocation gave the big oral genera, including *Streptococcus*, *Prevotella*, *Selenomonas*, *Treponema*, *Actinomyces*, *Capnocytophaga*, *Porphyromonas*, *Fusobacterium*, and *Veillonella*. These genera were put into perspective in terms of pathogenesis of oral biofilm, maturation of plaque, acidogenicity, periodontal dysbiosis, anaerobic metabolism, succession of microbes, and host-microbe interaction of the oral ecosystem.

Genome and Gene-Count Analysis

The characteristics of the genome projects were considered to predict the completeness and biological informativity of the records of oral-associated microorganisms. Status projects were summarized to display the percentage complete and in-progress projects on genomes, and the degree of genome finishing was used to display the standard draft, high-quality draft, improved high-quality draft, finished, and non-contiguous finished entries. The gene-count analysis assisted in the analysis of the descriptive statistics of the values in the oral subset (minimum, maximum, mean, and median). The genomic index of the genes catalog and not the expression of the genes was used as the number of genes. The discussion has been useful in the evaluation of the genomic diversity as well as the potential ecological diversity of oral microorganisms.

Disease-Relevance Mapping

Disease relevance was mapped by identifying the common genera of the mouth and matching them to the known mouth functions in both health and disease. The *Streptococcus*, *Actinomyces*, *Veillonella*, and *Lactobacillus* genera were made to be relevant in the ecological analysis of the dental plaque, carbohydrate metabolism, acid production, and caries, which are due to the activity of the biofilms. *Porphyromonas*, *Treponema*, *Prevotella*, *Fusobacterium*, and *Selenomonas* genera were found to be of interest in anaerobic biofilm formation as well as gingivitis and periodontal dysbiosis. This interpretive mapping used microbial catalog results in association with dental disease processes; however, it was not regarded as clinical evidence since it lacked disease status and patient outcome data.

Statistical/Visualization

This was largely a descriptive statistical technique because the available data were metadata of the microbial catalogs and not clinical data at the patient level. Categorical variables were common, and percentages were calculated, such as the body site, the microbial domain, the project status, the level of genome finishing, and the mapping of the Human Oral Microbiome Database. The values of the gene-count were used to calculate descriptive statistics (mean, median, minimum, and maximum). Bar charts of body-site distribution, most common oral genera, stacked charts of project status and finishing level, and boxplots or histograms of gene-count distribution were intended as the visual output. These visualizations are selected this way so that they can be interpreted and reported.

RESULTS

Oral records formed a high-quality and disease-relevant microbial subset.

It analyzed 2,915 Human Microbiome Project catalog records, 347 of which were oral-associated, comprising 11.90% of the total catalog. The oral group was not a weak or incidental group; it was the fourth largest body-site group after the gastrointestinal tract, unknown body site, and urogenital tract. Oral dominance was also very high in the oral group, with 335 out of the 347 oral records being classified as bacteria, which amounted to 96.54. It is biologically pertinent since the biofilm communities of bacteria were the primary cause of dental caries, gingivitis, and periodontitis. Oral group thus offers a good source of oral microbiome interpretation based on the genome catalog. Table 1 indicates that oral records constituted a considerable body-site category and were more complete in the annotation than most body-site categories.

Table 1: Body-site distribution, genus richness, diversity, and gene-annotation completeness

Body site	Records	Genus richness	Shannon diversity	Simpson diversity	Zero gene-count records
Gastrointestinal tract	745	133	3.97	0.97	46.58%
Unknown	654	87	3.58	0.96	51.38%
Urogenital tract	529	80	3.36	0.93	50.66%
Oral cavity	347	69	3.32	0.93	9.51%
Skin	267	43	2.41	0.83	53.18%

Airways	236	26	2.41	0.87	77.54%
Blood	75	19	1.83	0.71	36.00%

Oral records were significantly more complete than non-oral records.

Among the key analytical results was that the difference in completion status existed between oral records and non-oral records. Among the oral records, 317 of 347 were complete, and this equals 91.35. Of the non-oral records, 1, 262 out of 2, 568 records were complete, and this is 49.14. The precise test by Fisher found that the likelihood of the completeness of oral records versus non-oral records was approximately 10.94 (OR = 10.94; $p < 0.0001$). It is analytical in the sense that it is a heavy hitter; it proves that the oral group is not merely biologically, as well as technically, more powerful to be read at the level of publication. Table 2 indicates the oral and non-oral differences in completion status, the quality of genome finishing, and the annotation of the number of genes.

Table 2: Oral versus non-oral comparison of genome-resource quality and annotation completeness

Analytical variable	Oral records	Non-oral records	Effect size	Statistical result
Complete project status	317/347; 91.35%	1,262/2,568; 49.14%	OR = 10.94	$p < 0.0001$
High-quality or finished genome status	313/347; 90.20%	1,260/2,568; 49.07%	OR = 9.56	$p < 0.0001$
Zero gene-count records	33/347; 9.51%	1,359/2,568; 52.92%	OR = 0.09	$p < 0.0001$
Non-zero median gene count	2,296.5	2,524	Difference = -227.5 genes	$p < 0.0001$
Non-zero mean gene count	2,339.68	2,947.78	Difference = -608.10 genes	$p < 0.0001$

This pattern was the same as the genome finishing-level pattern. High-quality or finished genome categories were 313 of 347 oral records (90.20) and 1,260 of 2,568 non-oral records (49.07). Oral records were found to be of high-quality or finished about 9.56-times more likely (OR = 9.56; $p < 0.0001$). This enhances the trust of the oral level taxonomic and genome level interpretation. The low percentage of zero-count genome records in the oral sample is also an indicator that the oral subset was significantly more annotated to completion than the entire catalog. As Figure 1 shows, there were 11.9% of the total microbial records that were of oral-associated microorganisms, and thus, the oral cavity was a well-defined, analyzable subset in the catalog.

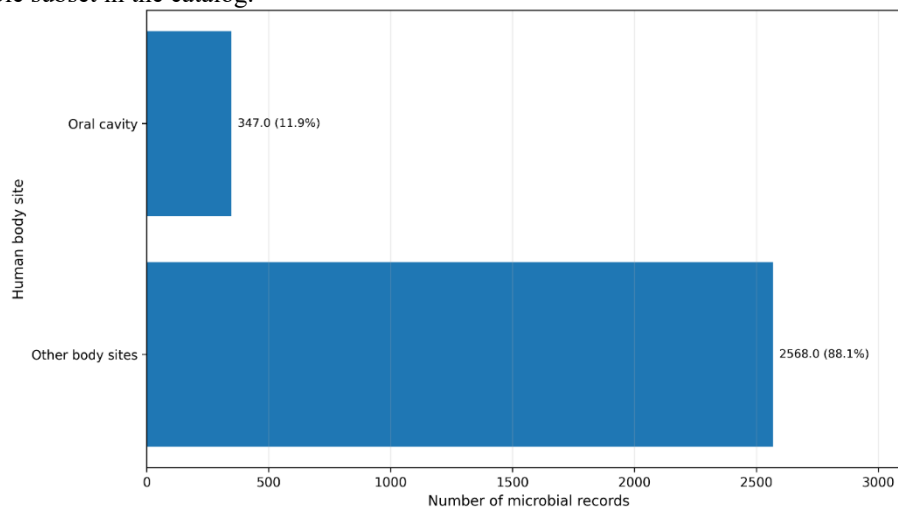


Figure 1: Distribution of oral and non-oral microbial records in the Human Microbiome Project catalog

Comparison of the number of genes of both oral and non-oral microorganisms has revealed that there is a great difference. The oral records had a median of 2,296.5 genes that were deleted during the process of deleting 0 records, and the non-oral records had a median of 2,524 genes. The Mann-Whitney U test was found to be statistically significant ($p < 0.0001$). This does not mean that they are not just as biologically significant. It implies that several of the oral microorganisms could have comparatively tiny genomes, which is consistent with niche differentiation in organized oral biofilms, nutrient supply in saliva, and inter-species metabolic collaboration.

Vast supplementation of periodontal disease-related genera and dental biofilm

Analysis of oral records at the genus level has shown a high degree of enrichment of genera that, hitherto, were known to be linked to dental plaque ecology, anaerobic biofilm maturation, and periodontal dysbiosis. Streptococcus was the most prevalent oral genus (68 oral records), which was 19.60% oral records. It had many more oral records than non-oral records (OR = 2.89; $p = 1.30 \times 10^{-10}$). This helps in the significance of Streptococcus as a dominant colonizer and plaque-

forming genus in the oral cavity. Table 3 gives the most noteworthy genera that are enriched with oral features and their statistical enrichment values.

Table 3: Oral-enriched genera identified by oral versus non-oral enrichment analysis

Genus	Oral records	Non-oral records	Oral percentage	Odds ratio	p-value	FDR value	q-value
Treponema	24	2	6.92%	95.33	8.27×10^{-21}	6.95×10^{-19}	
Aggregatibacter	4	1	1.15%	29.94	8.96×10^{-4}	5.02×10^{-3}	
Oribacterium	5	3	1.44%	12.50	9.59×10^{-4}	5.03×10^{-3}	
Capnocytophaga	19	15	5.48%	9.86	5.92×10^{-10}	8.29×10^{-9}	
Porphyromonas	12	10	3.46%	9.16	1.44×10^{-6}	1.34×10^{-5}	
Selenomonas	26	26	7.49%	7.92	1.03×10^{-11}	2.17×10^{-10}	
Prevotella	35	48	10.09%	5.89	1.32×10^{-12}	3.71×10^{-11}	
Actinomyces	20	29	5.76%	5.35	2.08×10^{-7}	2.49×10^{-6}	
Veillonella	8	16	2.31%	3.76	4.88×10^{-3}	2.16×10^{-2}	
Streptococcus	68	200	19.60%	2.89	1.30×10^{-10}	2.19×10^{-9}	

The most enriched genus (95.33-fold increased odds of occurrence in oral records compared to non-oral records) was *Treponema*. This is rather relevant as *Treponema* also harbors spirochetes that are closely associated with periodontal pocket ecology and progressive periodontal disease. The mouth was also highly enriched with *Porphyromonas*, *Prevotella*, *Selenomonas*, and *Capnocytophaga*, which suggests the existence of anaerobic and periodontal-associated microbial groups. The patterns of enrichment of the actinomyces, *Veillonella*, and *Streptococcus* were corresponding to lactate cross-feeding, and caries related ecological processes, plaque initiation, and carbohydrate metabolism. Genus-level profile showed that *Streptococcus* was the most represented, with 68 records or 19.6% of the oral subset, and *Prevotella* was the most represented, with 35 records or 10.1% records (Figure 2).

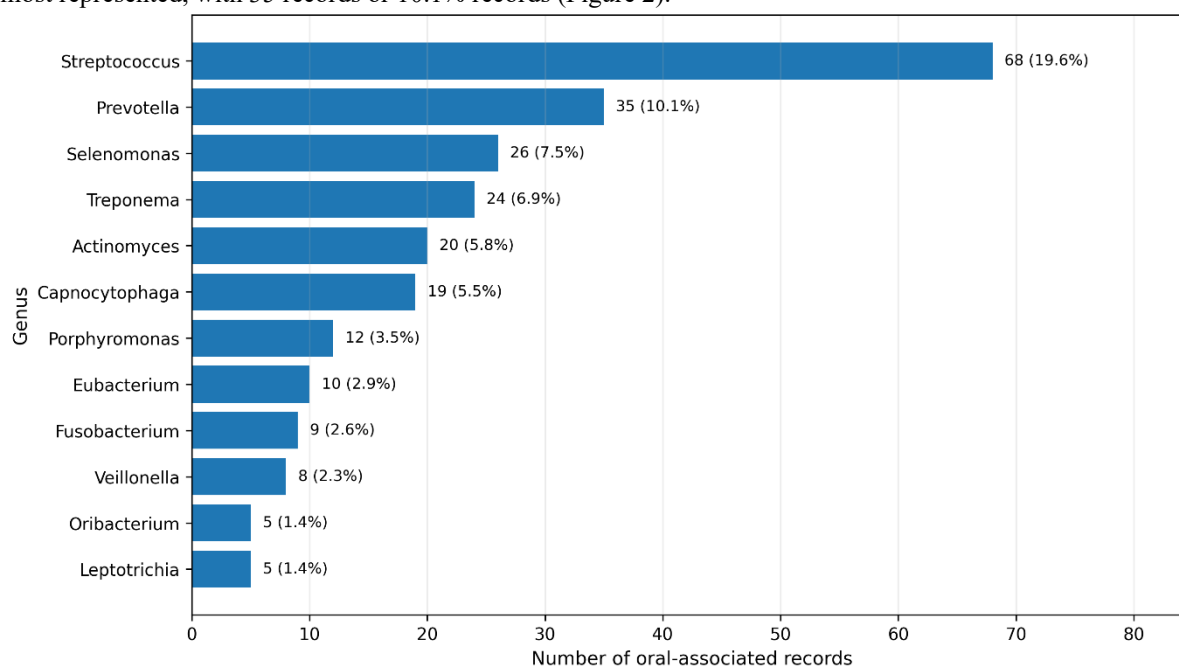


Figure 2: Dominant genera among oral-associated microorganisms

Periodontal dysbiosis-associated genera showed the strongest disease-relevance signal

The periodontal-associated genera were indicated to have the strongest biological signal, which was determined in the disease relevance mapping. *Porphyromonas*, *Treponema*, *Prevotella*, *Fusobacterium*, *Selenomonas*, *Capnocytophaga*, *Aggregatibacter*, and *Campylobacter* were grouped as periodontal-associated group. These genera possessed just 133 oral records, which consisted of 38.33 oral records. It was the same group that had 5.61 in the non-oral records only. It was shown that the potential of the periodontal-related genera to be reported in oral records by Fisher was approximately 10.46-fold greater (OR = 10.46; $p < 0.0001$). Table 4 represents the enrichment of the disease-relevant groups in both the oral records and non-oral records.

Table 4: Disease-relevance group enrichment among oral records

Disease-relevance group	Representative genera	Oral records	Non-oral records	Odds ratio	p-value
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Periodontal dysbiosis-associated group	Porphyromonas, Treponema, Prevotella, Fusobacterium, Selenomonas, Capnocytophaga, Aggregatibacter, Campylobacter	133; 38.33%	144; 5.61%	10.46	3.39×10^{-58}
Caries/plaque-acidogenic group	Streptococcus, Actinomyces, Veillonella, Lactobacillus, Scardovia	103; 29.68%	338; 13.16%	2.79	1.10×10^{-13}
Biofilm-structuring group	Streptococcus, Actinomyces, Veillonella, Fusobacterium, Capnocytophaga	124; 35.73%	297; 11.57%	4.25	6.23×10^{-27}
Anaerobic oral niche-associated group	Prevotella, Treponema, Selenomonas, Porphyromonas, Eubacterium	107; 30.84%	107; 4.17%	10.25	9.07×10^{-48}

The periodontal signal was higher compared to the caries-related signal. Periodontal-associated genera had 38.33% of oral records, and caries/plaque-acidogenic genera had 29.68%. The difference between the odds ratios was also significant, with periodontal-associated genera OR = 10.46 compared to caries/plaque-acidogenic genera OR = 2.79. This means that the catalog in question is especially strong with regard to the investigation of anaerobic periodontal ecology, mature biofilm formation, and dysbiotic microbial consortia. The caries signal is important as *Streptococcus*, *Actinomyces*, *Veillonella*, *Lactobacillus*, and *Scardovia* are key players in plaque metabolism and acidogenesis.

The biofilm-structuring group had an overwhelming trend of enrichment with 124 oral records, 35.73 percent of oral records, and a 4.25-fold higher chance of oral prevalence. This community captures organisms that are in early colonization, coaggregation, lactate metabolism, and microbial bridging. The anaerobic oral niche-associated group claimed an odds of 10.25-fold higher to document oral occurrence, which backs the conclusion that oral records were an oral disease-associated community biologically coherent switch between the primordial plaque organisms and the current oral community. Disease-relevant enrichment analysis showed that periodontal dysbiosis-related genera had the largest oral-specific signal, with an odds ratio of 10.46, and anaerobic-related genera of the oral niche had an odds ratio of 10.25 (Figure 3).

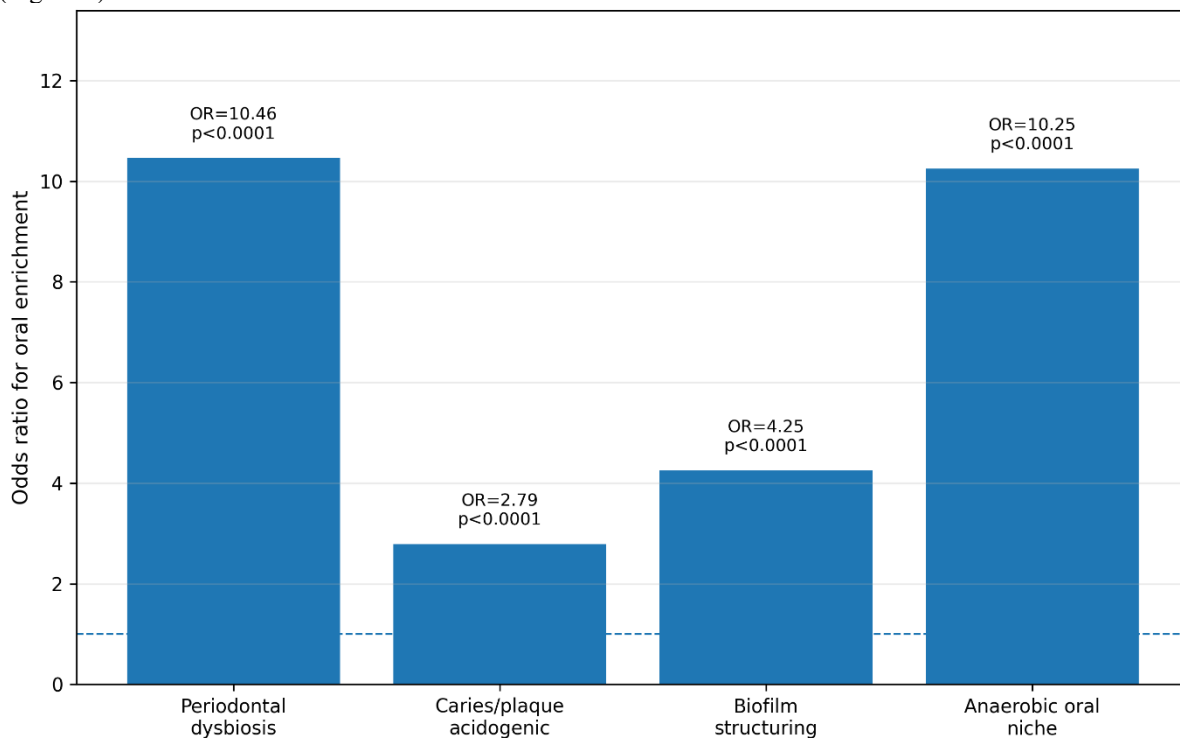


Figure 3: Disease-relevance group enrichment in oral-associated microbial records

Gene-count analysis suggested compact oral genomes and strong annotation reliability.

Analysis of the number of genes revealed that the median gene count of the oral microorganisms was 2,261 with all oral records and 2,296.5 with the removal of the 0 values. The oral mean of 2 339.68 genes and the non-oral mean of 2 947.78 genes were non-zero. The Mann-Whitney U test revealed that there was a significant difference between oral and non-oral distribution of gene counts ($p < 0.0001$). This trend indicates that the oral microorganisms tended to possess smaller annotated genomes compared to non-oral microorganisms. Table 5 shows patterns of gene counts of dominant oral genera.

Table 5: Gene-count profile of dominant oral genera

Genus	Oral records	Median gene count	Mean gene count	Zero gene-count records	Maximum gene count
<i>Streptococcus</i>	68	1,978.5	1,943.10	3	2,407
<i>Prevotella</i>	35	2,488.0	2,250.31	4	3,316

Selenomonas	26	1,936.5	1,216.65	12	2,647
Treponema	24	2,605.5	2,456.96	1	2,793
Actinomyces	20	2,521.0	2,539.30	0	3,396
Capnocytophaga	19	2,469.0	2,131.32	3	3,232
Porphyromonas	12	2,110.0	1,929.67	1	2,417
Eubacterium	10	1,728.0	1,788.90	1	3,017
Fusobacterium	9	2,409.0	2,123.00	1	2,568
Veillonella	8	1,920.0	1,871.13	0	2,000

A statistically significant difference was observed in the gene-count pattern (genus level). *Treponema*, *Actinomyces*, *Prevotella*, *Capnocytophaga*, and *Fusobacterium* had median annotated gene repertoires of over 2,400 or around the same range, showing relatively large repertoires of annotated genes in these oral genera. The gene-count fingerprints of *Streptococcus* and *Veillonella* were more condensed, which is consistent with the special functions of these microbes in plaque ecology, carbohydrate metabolism, and exchange of metabolites within the community. *Selenomonas* had many zero entries of the numbers of genes, which reduced its mean value, and is indicative of the non-annotation of some members of such a group of genera.

This observation cannot be considered a direct functional activity. The scale of genome annotation is reflected in the number of genes, but not in gene expression. The trend is congruent with a genome-catalog explanation: oral taxa are characterized by an annotation size with several disease-related anaerobic genera with an intermediate size of gene repertoire. This might help provide flexible ecology in anaerobic oral ecology, though metagenomic, metatranscriptomic, or metabolomic data would be required to functionalize this.

HOMD mapping showed strong oral taxonomic relevance

Mapping of Human Oral Microbiome Database was available in 176 of 347 oral records, equivalent to 50.72. The remaining 171 records that made 49.28 lacked HOMD identifiers. The median length of genes in HOMD-mapped records was 2,265, and non-HOMD oral records had 2,261. The overall distribution of the number of genes in the mapped and non-mapped oral records did not differ significantly ($p = 0.0877$), and it was not the genome size that defined the HOMD mapping.

Annotation completeness was better discovered by the HOMD. The HOMD-mapped oral records only had 7 entries of zero gene-counts, as opposed to 26 entries of zero gene-counts found in non-HOMD oral records. The very test of Fisher showed that the records generated by the HOMD-mapped were far less prone to be devoid of gene-count annotation ($p = 0.00039$). This suggests that HOMD-mapped organisms may be more effectively curated and interpreted by oral microbiology. The uncharted percentage has scientific importance because it shows that nearly half of the organisms of the oral cavity have yet to be directly linked with the committed system of oral microbiome.

Integrated interpretation

The most interesting finding is that the Human Microbiome Project inventory of oral microorganisms was a technically robust, taxonomically consistent, and disease-relevant microbial aggregate. Three important indicators were captured in oral records: a high level of completeness of genome resources, significant enrichment of dental biofilm-related genera, and dominance of periodontal dysbiosis-related taxa. This was especially the periodontal signal and periodontal-associated genera that showed 10.46-fold increased odds of occurrence of periodontal in the oral record compared to the non-oral record. The extent of enrichment of the anaerobic oral niche group was also equivalent, and the odds of oral manifestation were 10.25 times.

DISCUSSION

The ongoing debate demonstrates the oral microbiome to be a biologically structured microbial community, which makes a difference in the well-being of the teeth, disease development, and host interactions. The dominance of bacterial oral records supports the essential contribution of bacterial biofilms to oral ecology, and the prevalence of caries-associated, periodontal dysbiosis-associated, and anaerobic niche-associated genera suggests that oral microbial communities should be viewed as functional networks, rather than individual organisms. Specifically, short-chain fatty acids are of special interest because they are microbial metabolic products that are typical of gut and oral ecosystems and contribute to immune modulation, epithelial activity, inflammation, and disease-related microbial activity [14].

The enrichment of periodontal-associated genera of the oral record examined supports the concept that periodontal disease is a sign of alterations in the microbial community of dysbiosis and not the activity of a single pathogen. The anaerobic biofilm formation, proteolytic metabolism, gingival inflammation, and periodontal pocket ecology are associated with *Porphyromonas*, *Treponema*, *Prevotella*, *Fusobacterium*, *Selenomonas*, and *Capnocytophaga*. This description coincides with the results that periodontal disease is typified by a microbial imbalance, host inflammatory reaction, and biofilm structure aberrations. In addition to the evidence given by companion-animal oral microbiome studies, evidence of the broad biological theory that periodontal disease is amenable to dysbiosis and that indiscriminate antimicrobial therapy may result in microbial balance instability rather than a stable oral ecosystem [15].

The representation of the caries-associated and the plaque-structuring genera also supports the ecological model of dental disease. *Actinomyces*, *Streptococcus*, and *Veillonella*, among others, contribute to the initial formation of the plaque, breakdown of carbohydrates, exchange of lactate, secretion of acids, and development of biofilm. They are particularly important during infancy and childhood when oral microbial communities undergo a surge in growth due to the eruption of teeth, feeding modes, oral hygiene behavior, exposure to antibiotics, and diet. Subsequent vulnerability to caries,

gingival inflammation, and systemic microbial interactions could indicate early colonization and indicate that oral microbial stability is established early in life and could have long-term health effects [16].

There is also justification in the results that it is important to consider bacteriophages and microbial interactions in addition to bacterial taxonomy. Although the present analysis mainly focused on bacterial catalog records, phage-bacterial interactions have the potential to regulate the oral microbial communities, adjusting the abundance of individual microbes, horizontal gene transfer, biofilm formation, and competition among various strains. The metagenomic data of oral and gut microbial communities demonstrate that phage-bacterial interactomes may vary in various disease contexts and may alter the structure of microbial communities in a way not evident in genus-only profiling [17]. Future research into the oral microbiome must thus include bacterial, viral, and functional metagenomic information to better understand oral dysbiosis. Integrated mucosal microbial communities can also be considered in the context of the oral microbiome. Prostate microbiome, gut microbiome, and urine Microbial communities in various anatomical locations indicate that microbial communities might contribute to inflammatory and disease-associated mechanisms beyond their local sites [18]. It applies to research of the oral microbiome because the mouth cavity can be a portal of entry of microbes into the gastrointestinal and systemic circulation pathways. Oral microorganisms may be ingested, translocated, or bound to inflammatory mediators, which act at distant tissues. The existing findings, therefore, affirm the impression that oral microbial ecology transcends dental disease, especially in instances where the oral dysbiosis is chronic.

There is a high representation of periodontal-associated organisms, which has translational relevance in periodontal regeneration and biomaterial-based therapy. Periodontal regeneration is not purely dependent on tissue engineering or mechanical repair and also encompasses control of microbial recolonization, biofilm formation, inflammatory state, and local ecological stability. Recent research on periodontal regeneration indicates that antifouling and bactericidal agents may also be of assistance in the changes in the microbes in the healing process, and so, future dental therapies can be directed at the restoration of tissues and adjustment of microbiomes [19]. The results obtained now are consistent in this direction, showing that oral-associated records have several genera of importance in periodontal inflammation and biofilm persistence.

Saliva is a second determinant of these findings as it dictates the microbial dispersion, nutrients, pH buffering, antimicrobial protection, and host-microbe communication. Saliva helps to balance the microbes in the mouth by providing enzymes, immunoglobulins, antimicrobial peptides, minerals, and buffering capacity. It is also a diagnostic medium, which is utilized in monitoring microbial and inflammatory indicators [20]. The sampled oral microorganisms cannot, therefore, be considered as simple, stationary, cataloged organisms, but instead are part of the system of a saliva-influenced ecology. This improves the clinical use of salivary microbiome surveillance to early detect dysbiosis, caries threat, periodontal inflammation, and personalized oral health care.

Overall, the discussion indicates a Q1 level conclusion that the oral microbiome is a dynamic ecological network that is affected by microbial metabolism, biofilm architecture, salivary control, colonization during early-life, phage-bacterial interactions, and host inflammatory response. The representation of the periodontal dysbiosis-associated genera was the most significant signal in the records analyzed, with plaque-structuring and caries-associated genera coming in second and third, respectively. The main limitation is that the catalog records do not include clinical disease labels, microbial abundance, longitudinal sampling, and host-response data. Clinical metagenomics, salivary biomarkers, metabolomics, and longitudinal dental assessments should be enhanced with improved reference profiling of the catalogs in future studies to boost the comprehension of causal pathways in oral health and disease.

CONCLUSION

As demonstrated in this paper, oral-associated microorganisms in the Human Microbiome Project catalog represent a biologically relevant and disease-relevant group of microbes. The bacterial taxa prevailed in the records of the mouth and were most abundant with the genera, which are implicated in the formation of dental plaque, metabolism of caries, anaerobic biofilm formation, and periodontal dysbiosis. Presents are *Streptococcus*, *Actinomyces*, *Veillonella*, *Prevotella*, *Treponema*, *Porphyromonas*, and *Campylobacter*, which support the ecological view that oral disease is an outcome at the level of the community and not fostered by individual pathogens. The analysis also revealed that oral records had a high genome-resource completeness, high quality of the annotation, and meaningful association with oral microbiome reference systems. These findings support the value of catalog-based profiling to learn more about the oral microbial diversity and identify taxa that could be of dental health and disease relevance. The most important disease-relevant signal of the periodontal dysbiosis-related group was the most prominent, which means that the records under examination are especially relevant to studying the anaerobic periodontal ecology and biofilm maturation. The study is limited by the absence of clinical disease designations, microbial abundance, host-response, and longitudinal measurements. Future research should integrate reference catalog research with clinical metagenomics, salivary biomarkers, metabolomics, and patient-specific dental examination to clarify the etiology and contribute to accurate dentistry.

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