BMC Research Notes

RESEARCH NOTE Open Access

Salivary microbiome profile shifts after scaling in stunted children

Tasha Octaricha[1], Cimi Ilmiawati[1*] and Nila Kasuma[2*]

Abstract

Objective Stunting is a condition of impaired growth in children resulting from chronic malnutrition, characterized by shorter stature compared to peers of the same age. This condition leads to salivary gland dysfunction, which triggers oral dysbiosis and increases the risk of periodontal disease in children. Scaling and root planing (SRP) is the gold standard treatment for periodontal disease, aimed at reducing pathogenic bacterial populations. This study aimed to evaluate the effect of SRP treatment on the oral microbiome profile in the saliva of stunted children. A pre- and post-test study design was employed, involving 10 elementary school children divided into two groups: normal children and stunted children. Each participant underwent scaling, with saliva samples collected before and after the procedure. The oral microbiome profile was analyzed using next-generation sequencing, generating taxonomic data at the phylum, genus, and species level.

Result Statistical analysis revealed significant changes in the gingival index, a clinical parameter, in the normal group but not in the stunted group. Scaling resulted in shifts in the microbiome profile in both groups, with the dominant phyla identified as *Proteobacteria*, *Bacteroidota*, and *Firmicutes*. Scaling procedure alters the oral microbiome profile in stunted children without affecting the clinical parameter.

Keywords Stunting, Scaling, Saliva, Microbiome

Introduction

Stunting is a condition of impaired growth in children, primarily caused by chronic malnutrition or persistent infections [1], and is defined by a height-for-age Z-score below − 2 standard deviations based on WHO standards [1, 2]. Globally, stunting affects 149 million toddlers, with 25% of cases in Southeast Asia [1]. Indonesia has the highest prevalence in the region at 24.4%, compared to Malaysia (17%), Thailand (16%), and Singapore (4%) [3, 4].

Stunting leads to salivary gland dysfunction, causing oral cavity disorders [2, 5]. Chronic malnutrition induces pleomorphism, reduced proliferation, and increased apoptosis in acinar cells, resulting in cell atrophy and decreased saliva production [5]. This impairs the self-cleansing mechanism, reduces salivary proteins, disrupts tissue homeostasis, and lowers microbial resistance, which compromises oral hygiene [1, 5]. Consequently, stunted children face microbiome imbalances, chronic inflammation, and increased risk of periodontal diseases due to pathogens like *Porphyromonas gingivalis*, *Treponema denticola*, *Fusobacterium*, and *Tannerella forsythia* [1, 6, 7]. Periodontal disease is a chronic inflammatory

*Correspondence:
Cimi Ilmiawati
ilmiawati@med.unand.ac.id
Nila Kasuma
nilakasuma@dent.unand.ac.id
[1]Master Program of Biomedical Sciences, Faculty of Medicine, Universitas Andalas, Padang, Indonesia
[2]Department of Oral Biology, Faculty of Dentistry, Universitas Andalas, Padang, Indonesia

condition that affects the tooth-supporting tissues, including the gingiva, periodontal ligament, and alveolar bone. In severe cases, it can lead to attachment loss, tooth mobility, and bone destruction [6, 8, 9].

Pro-inflammatory cytokines, such as IL-1, IL-6, IL-8, and TNF-α, play a significant role in the pathogenesis of this condition. Additionally, stunting has been shown to exacerbate the inflammatory response, further contributing to the progression of periodontal disease [10–12]. In stunted children, the salivary microbiome profile may interact with systemic factors such as platelets, potentially linking oral health to systemic inflammation. Platelets, a cellular subgroup of blood elements with a lifespan of 8–10 days and numbering nearly one trillion in an adult, play critical roles in responding to vessel injuries, regulating angiogenesis, and mediating innate immunity. These functions make platelets key players in systemic inflammation as they respond to inflammatory signals and interact with immune cells. Dysbiosis-induced inflammation, common in stunted individuals, may lead to altered platelet activation and function, exacerbating systemic health issues and contributing to the inflammatory burden observed in periodontal disease [13].

The microbiome is an ecosystem of commensal and pathogenic microorganisms that live symbiotically within the human body. This term was introduced by Joshua Lederberg, a Nobel Prize laureate in 2001 [14]. The oral microbiome is the second most diverse microbial community in the human body, harboring more than 700 bacterial taxa [14, 15]. The oral biofilm is a complex microbial community influenced by dietary habits, oral hygiene practices, salivary flow, and salivary components [14]. A healthy oral microbiome is characterized by a balance between commensal (e.g., *Streptococcus* and *Neisseria*) and pathogenic bacteria [16, 17]. However, a pathological shift can occur, leading to periodontal disease, which is marked by the emergence of pathogenic species such as *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia*, collectively referred to as the red complex [16, 18].

The presence of pathogenic bacteria within the biofilm plays a crucial role in the progression of periodontal disease, affecting both hard and soft tissues in the oral cavity [18]. In the presence of oral pathologies, disruption of microbial balance leads to alterations in the composition and organization of biofilm, including shifts in microbial species and their interactions [19].

Scaling and root planing, a common periodontal treatment, removes plaque, calculus, and bacterial toxins from tooth surfaces, smoothing roots. It is sometimes supplemented with antiseptics or antibiotics [8]. In addition to scaling and root planing (SRP), various adjuvant methods can be employed to treat periodontal disease, including antimicrobial mouthrinses, antibiotic gels, and antibiotic microspheres placed within periodontal pockets. Oral antibiotics are also used to control bacterial infections and reduce the size of periodontal pockets over time [20, 21]. When non-surgical methods yield insufficient results, surgical interventions such as flap surgery and bone or tissue grafting may be performed. These methods aim to restore the supporting structures of the teeth [20]. Antimicrobial methods utilizing low-energy lasers have also been introduced as adjuncts to enhance the effects of scaling and root planing (SRP) [22, 23].

Dysbiosis, a microbial imbalance, is frequently observed in stunted children, with a higher *Firmicutes* population in their gastrointestinal tract, which further impairs nutrient absorption [24]. Understanding the oral microbiota in stunted children is crucial to exploring the connection between specific pathogens and their effects on health [25].

This study aimed to evaluate how scaling and root planing influences oral microbiota shifts in stunted children's saliva. By identifying these changes, the study seeks to better understand the interplay between stunting, oral health, and periodontal disease, paving the way for more targeted treatments for this vulnerable population.

Methods

Study participants

Participants were recruited from elementary schools within the Lubuk Kilangan Community Health Center's jurisdiction. Eligible participants, aged 6 to 12 years, were categorized as stunted or normal stature based on WHO height-for-age standards. All had a clinical diagnosis of gingivitis, confirmed through assessments of the gingival index and oral hygiene status.

Exclusion criteria were implemented to ensure validity and minimize confounding factors. Thorough evaluations, including alloanamnesis with legal guardians, assessed medical histories for systemic diseases, recent antibiotic use, deep dental caries, and special needs conditions like Down syndrome. Only participants meeting eligibility criteria were included.

Baseline data collection involved assessing the gingival index, oral hygiene status, and saliva sampling before scaling and root planing procedures. Height measurements were taken with a microtoise (precision 0.1 cm) and converted into Z-scores using WHO standards. These steps ensured the study's focus on eligible participants and reliable data collection.

Saliva sampling

Unstimulated saliva samples were collected at baseline and one week after treatment with the drooling method. Prior to sampling, subjects were instructed not to eat, drink, and brush their teeth for at least 30 min before the session. Saliva was collected using saliva collection kits

(SafeCollect™, Zymo Research, California, USA) according to the manufacturer's protocol. After collection, a total of two samples (baseline and one week's) from each subject were placed in an ice box and transferred into a -20℃ freezer until analysed.

Treatment protocol

All participants received oral hygiene instructions and scaling in all quadrants. Procedures were performed by a registered dentist using ultrasonic instruments. A follow-up appointment was carried out one week after treatment to assess subjects' oral hygiene, to calculate their gingival index score, and to collect their saliva sample.

PCR amplifications of the 16 S rRNA gene and sequencing

Amplification of the 16 S rRNA gene targeting the V3 and V4 regions was performed using specific forward (5'TCGTCGGCAGCGTCAGATGTGTATAAGAGACAGCCTACGGGNGGCWGCAG) and reverse (5'GTCTCGTGGGCTCGGAGATGTGTATAAGAGACAGGACTACHVGGGTATCTAATCC) primers. For amplicon PCR of the 16 S rRNA gene, KAPA HiFi HotStart ReadyMix PCR kits (Roche, Basel, Switzerland) were used according to the manufacturer's instructions. Libraries were constructed with NextEra XT DNA library preparation kits (Illumina Inc., San Diego, CA, USA). Sequencing was performed on the MiSeq Illumina platform using MiSeq v3 reagent kits (Illumina Inc., San Diego, CA, USA).

Bioinformatic and statistical analysis

FastQC software (Babraham Bioinformatics, Babraham, UK) was used to assess the quality of fastq data. Classification is based on the Silva database (http://ngs.arb-silva.de/), microbiome analysis was done using *Quantitative Insights Into Microbial Ecology* 2 (QIIME2). The output of this workflow is a classification at phylum, genus, and species level. We used paired t-test to determine the mean difference within group before and after procedure, and independent t-test to determine the mean difference between groups. Results were considered significant when p-value < 0.05.

Table 1 Characteristics of normal and stunted elementary school children

	Normal ($n=5$)	Stunted ($n=5$)	p
Sex			
• Male	3	2	
• Female	2	3	
Height (cm; mean ± SD)	136.2 ± 3.1	118.3 ± 11.8	0.01[a]
GI	**Pre-scaling**	**Post-scaling**	
• Normal stature	2.34 ± 0.35	2.04 ± 0.30	0.009[b]
• Stunted	2.30 ± 0.58	2.16 ± 0.63	0.107[b]
OHIS			
• Normal stature	2.14 ± 0.50	2.06 ± 0.55	0.455[b]
• Stunted	2.24 ± 0.48	2.02 ± 0.64	0.282[b]

[a]: independent t-test; [b]: paired t-test; GI: Gingival Index; OHIS: Oral Hygiene Index Simplified

Results

Subjects' characteristics

This was a pre-test post-test study. Table 1 shows subjects' characteristics, such as sex and body height, and clinical parameters such as gingival index (GI) and oral hygiene index simplified (OHIS).

Based on the table above, stunting was observed more frequently in girls than in boys. Additionally, stunted children were significantly shorter by an average of 17 cm (14%) compared to their non-stunted peers ($p=0.01$). The scaling procedure resulted in a significant improvement in the gingival index (GI) among normal children, while in the stunted group, only a downward trend was observed. For the oral hygiene index-simplified (OHIS), both groups exhibited a decreasing trend; however, the changes were not statistically significant.

Normal stature and stunted children's microbiome profile

Oral microbiomes in each group were compared before and after treatment, and we observed the bacteria at the phylum, genus and species level. Microbiome differences in the normal stature children group is presented in Fig. 1.

Figure 1a shows the salivary microbiome profile of normal stature children at the phylum level, revealing changes after scaling. Before treatment, *Bacteroidota* was the most abundant phylum, while *Proteobacteria* became dominant post-scaling. *Firmicutes* remained among the top three phyla.

In stunted children (Fig. 1b), *Proteobacteria* was the most abundant phylum both before and after scaling. The procedure increased the proportion of salivary bacteria in this group, except for *Proteobacteria* and *Bacteroidota*. These findings highlight distinct microbiome shifts in response to scaling between normal stature and stunted children.

At the genus level, we observed 75 genera in the saliva of normal stature children before scaling, and 81 genera were found after the procedure. We highlight three genera commonly associated with periodontal environments, which are *Prevotella*, *Veillonella*, and *Streptococcus.* Changes after the scaling procedure is presented in Fig. 2.

Significant decreases were observed in *Prevotella* and *Veillonella* composition, while *Streptococcus* showed significant increase in normal children after scaling-root planing treatment (Fig. 2a). In the stunted group (Fig. 2b), all genera composition did not differ significantly after treatment. But there was a decreasing trend

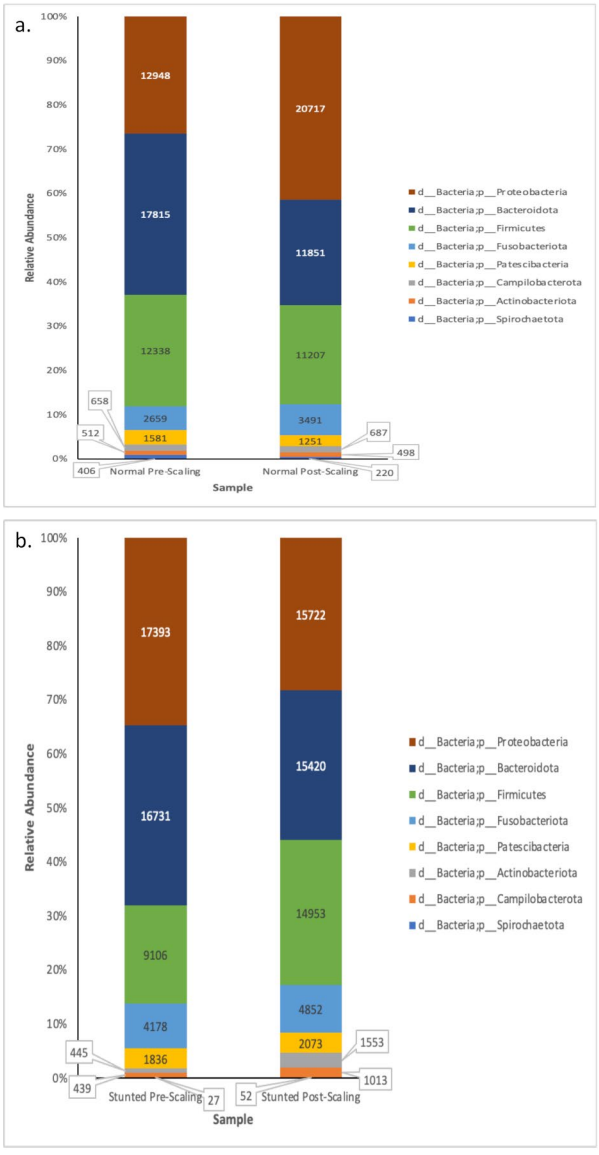

Fig. 1 Normal stature children's microbiome profile at the phylum level before and after scaling procedure **(a)**, and stunted children's microbiome profile at the phylum level before and after scaling procedure **(b)**

in all genera, except *Streptococcus*, showing a higher trend after treatment.

Bacteria found exclusively in stunted children

In this study, 165 bacterial species were identified in the saliva before scaling, and 180 species were identified one week after the procedure. Among the identified species, several bacteria were found exclusively in the stunted children group, as shown in Fig. 3.

Based on Fig. 3 there were 25 bacterial species exclusively identified in the saliva of stunted children, with the dominant phyla being *Bacteroidota*, *Firmicutes*, and *Proteobacteria*. The genus *Prevotella* had the highest number of identified species in stunted children, including *Prevotella histicola*, *Prevotella loescheii*, and *Prevotella salivae*.

Discussion

Stunting is more commonly observed in female children, potentially due to cultural biases that prioritize nutritional intake for male children [2, 26]. In many societies, male children are perceived as future economic contributors, leading to a focus on their nutrition and healthcare access, while female children often face limitations in these areas [26, 27]. Stunting is closely associated with a compromised immune system, resulting in reduced ability to eliminate pathogens and increased susceptibility to infections. This immunodeficiency is attributed to a decrease in salivary and tear immunoglobulin A (sIgA) levels and a general reduction in immune response, which may lead to long-term immunodeficiency [28]. Chronic malnutrition, a primary contributor to stunting, is also linked to salivary gland dysfunction, manifesting as reduced saliva flow and impaired oral hygiene [2, 29].

Children who are stunted often experience delays in cognitive and motor development, which negatively affect their ability to maintain proper oral hygiene [1]. This inability to perform adequate oral care leads to plaque accumulation and subsequently increases the risk of developing dental caries and periodontal disease. Furthermore, salivary gland dysfunction exacerbates oral health problems, creating a cycle of pain, discomfort, and difficulty eating. These factors further aggravate nutritional deficiencies and hinder the growth and development of stunted children [1, 30].

The impact of periodontal disease is not limited to oral health but extends to systemic health as well. For example, untreated periodontal disease is associated with adverse pregnancy outcomes, including low birth weight [31]. Mothers with untreated periodontal disease are at a higher risk of delivering low birth weight infants, which in turn increases the likelihood of stunting in these children. This establishes a cycle where a stunted mother is more likely to give birth to a low birth weight child, perpetuating stunting across generations [32].

This study identified eight bacterial phyla in the saliva of normal stature and stunted children. Scaling treatment altered oral bacterial proportions, with 165 species found before scaling and 180 species one week after. Dominant phyla in stunted children included *Proteobacteria*, *Bacteroidota*, and *Firmicutes*, with the high bacterial count linked to higher carbohydrate intake compared to protein intake [33]. An individual's dietary pattern is linked to the occurrence of caries and periodontal disease in children, as well as affecting the microbiota composition in saliva and gingival crevicular fluid [34]. Stunted children frequently consume ultra-processed foods (UPFs),

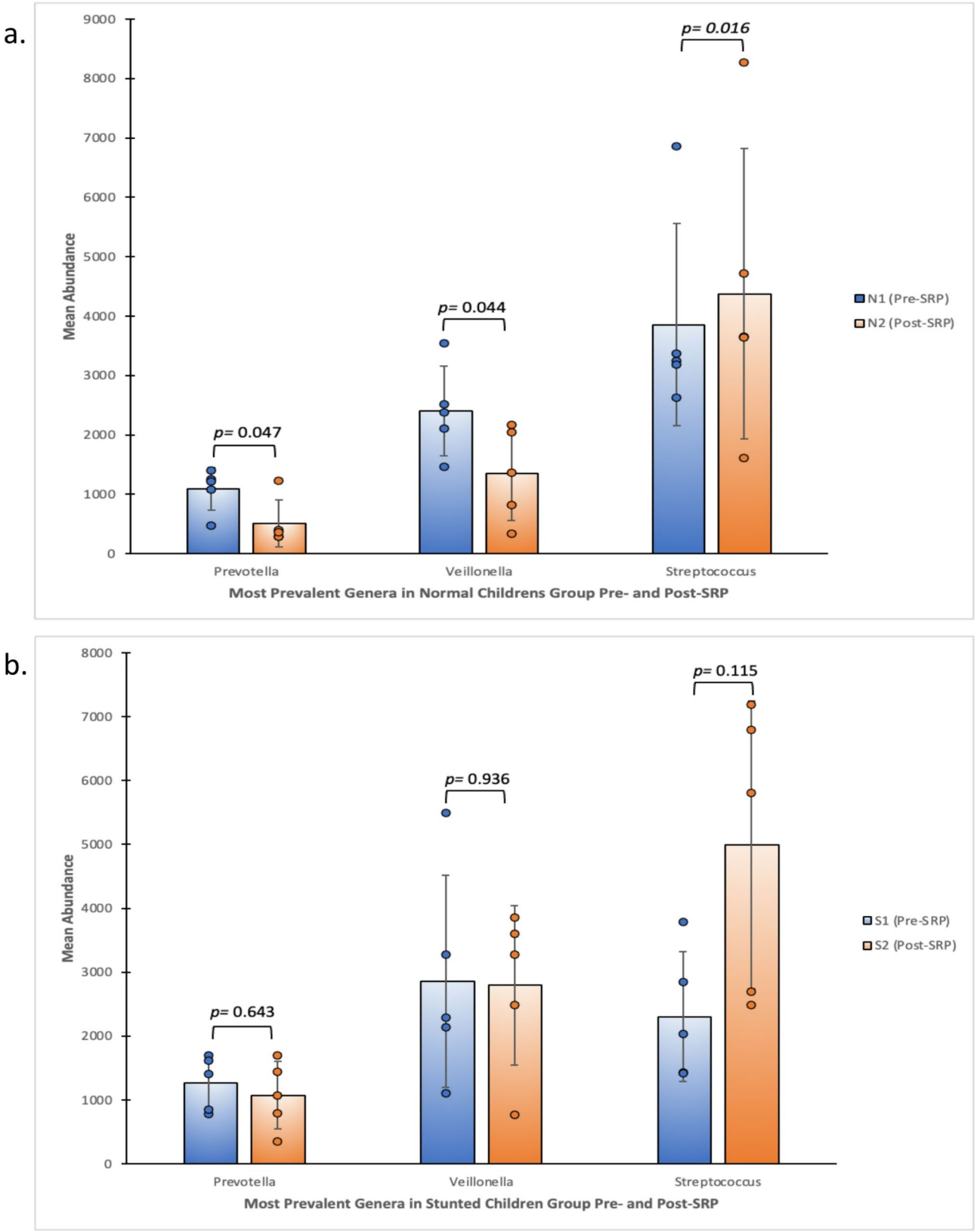

Fig. 2 Changes in genera commonly associated with periodontal environment before and after scaling procedure in normal stature children **(a)**, and in stunted children group **(b)**

	Species
1	d__Bacteria;p__Actinobacteriota;c__Actinobacteria;o__Actinomycetales;f__Actinomycetaceae;g__Actinomyces;s__Actinomyces_graevenitzii
2	d__Bacteria;p__Actinobacteriota;c__Actinobacteria;o__Micrococcales;f__Micrococcaceae;g__Rothia;s__Rothia_aeria
3	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Bacteroidales;f__Paludibacteraceae;g__F0058;s__uncultured_bacterium
4	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Bacteroidales;f__Prevotellaceae;g__Prevotella;s__Prevotella_histicola
5	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Bacteroidales;f__Prevotellaceae;g__Prevotella;s__Prevotella_loescheii
6	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Bacteroidales;f__Prevotellaceae;g__Prevotella;s__Prevotella_salivae
7	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Flavobacteriales;f__Flavobacteriaceae;g__Capnocytophaga;s__Capnocytophaga_sputigena
8	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Flavobacteriales;f__Flavobacteriaceae;g__Capnocytophaga;s__uncultured_Capnocytophaga
9	d__Bacteria;p__Bacteroidota;c__Bacteroidia;o__Sphingobacteriales;f__Lentimicrobiaceae;g__Lentimicrobium;s__unidentified
10	d__Bacteria;p__Campilobacterota;c__Campylobacteria;o__Campylobacterales;f__Campylobacteraceae;g__Campylobacter;s__Campylobacter_gracilis
11	d__Bacteria;p__Firmicutes;c__Clostridia;o__Clostridia_UCG-014;f__Clostridia_UCG-014;g__Clostridia_UCG-014;__
12	d__Bacteria;p__Firmicutes;c__Clostridia;o__Clostridia_UCG-014;f__Clostridia_UCG-014;g__Clostridia_UCG-014;s__uncultured_bacterium
13	d__Bacteria;p__Firmicutes;c__Clostridia;o__Lachnospirales;f__Lachnospiraceae;g__Catonella;s__uncultured_bacterium
14	d__Bacteria;p__Firmicutes;c__Clostridia;o__Peptostreptococcales-Tissierellales;f__Peptostreptococcaceae;g__Peptostreptococcus;__
15	d__Bacteria;p__Firmicutes;c__Negativicutes;o__Veillonellales-Selenomonadales;f__Veillonellaceae;g__Megasphaera;s__Megasphaera_micronuciformis
16	d__Bacteria;p__Fusobacteriota;c__Fusobacteriia;o__Fusobacteriales;f__Leptotrichiaceae;g__Leptotrichia;s__Leptotrichia_buccalis
17	d__Bacteria;p__Fusobacteriota;c__Fusobacteriia;o__Fusobacteriales;f__Leptotrichiaceae;g__Leptotrichia;s__Leptotrichia_hofstadii
18	d__Bacteria;p__Patescibacteria;c__Saccharimonadia;o__Saccharimonadales;f__Saccharimonadaceae;g__Candidatus_Saccharimonas;s__uncultured_bacterium
19	d__Bacteria;p__Patescibacteria;c__Saccharimonadia;o__Saccharimonadales;f__Saccharimonadaceae;g__Saccharimonadaceae;s__uncultured_bacterium
20	d__Bacteria;p__Proteobacteria;c__Gammaproteobacteria;o__Burkholderiales;f__Neisseriaceae;__;__
21	d__Bacteria;p__Proteobacteria;c__Gammaproteobacteria;o__Burkholderiales;f__Neisseriaceae;__;__
22	d__Bacteria;p__Proteobacteria;c__Gammaproteobacteria;o__Burkholderiales;f__Neisseriaceae;g__Neisseria;s__Neisseria_perflava
23	d__Bacteria;p__Proteobacteria;c__Gammaproteobacteria;o__Pasteurellales;f__Pasteurellaceae;g__Haemophilus;s__Haemophilus_sputorum
24	d__Bacteria;p__Proteobacteria;c__Gammaproteobacteria;o__Pseudomonadales;f__Moraxellaceae;g__Moraxella;__
25	d__Bacteria;p__Proteobacteria;c__Gammaproteobacteria;o__Pseudomonadales;f__Moraxellaceae;g__Moraxella;s__uncultured_Moraxellaceae

Fig. 3 Bacterial species found exclusively in stunted children

a pro-inflammatory diet associated with increased *Veillonella* populations [35]. High carbohydrate intake also correlates with elevated acidogenic bacteria, such as *Streptococcus mutans*, *Streptococcus mitis*, *Prevotella* spp., and *Actinomyces* spp., consistent with this study's findings where *Prevotella* and *Actinomyces* were exclusively identified in the stunted group [36].

The oral cavity microbiome is a complex ecosystem, with bacteria classified into color-coded complexes like purple, yellow, green, orange, and red [37]. Healthy periodontal conditions are associated with purple, yellow, and green complexes, while orange and red complexes are linked to periodontal diseases. Bacteria such as *Porphyromonas gingivalis*, *Treponema denticola*, *and Tannerella forsythia* (red complex) and *Prevotella* spp. (orange complex) are implicated in periodontitis and other pathological conditions [37, 38].

Stunted children experience decreased salivary flow due to micronutrient deficiencies, such as zinc, impairing saliva's protective properties [1]. Zinc plays a crucial role in microbial clearance by generating reactive oxygen species (ROS) that disrupt bacterial cell membranes, including *Prevotella* spp [39]. Clinically, *Prevotella* contributes to gingival tissue inflammation and bleeding on probing, key indicators of gingival disease [40, 41]. However, scaling alone did not significantly improve gingival index scores in this study, likely due to persistent *Prevotella* populations.

Prevotella, a commensal bacterium, is frequently associated with oral dysbiosis, periodontal disease, and dental caries. Its lipopolysaccharides (LPS) stimulate pro-inflammatory cytokines (TNF-α, IL-6, IL-8), disrupt phagocytosis, promote bone resorption, and collaborate with other pathogens like *Porphyromonas gingivalis* and *Treponema denticola* [40, 42]. These bacteria enhance periodontal inflammation and tissue destruction.

Similarly, *Veillonella*, a Gram-negative anaerobic bacterium, is linked to dental caries and periodontal disease. Species like *Veillonella atypica* and *Veillonella parvula* interact with periodontal pathogens such as *Porphyromonas gingivalis*, exacerbating oral pathology [43]. Scaling and root planing procedures reduce *Prevotella* and *Veillonella* populations, improving periodontal outcomes [40, 41].

Post-scaling, health-associated genera like *Streptococcus* significantly increased in normal stature children and showed a similar trend in stunted children (Fig. 2). *Streptococcus*, an early plaque colonizer, is part of the core microbiome in healthy periodontal states alongside *Neisseria* [17]. Studies in patients with generalized aggressive periodontitis (GAgP) have demonstrated that scaling and root planing can reduce periodontal pathogens while increasing health-associated microbiota such as *Neisseria*, *Streptococcus*, and *Haemophilus* [44].

Periodontal disease management is broadly classified into non-surgical and surgical therapies, both aimed at controlling infection and restoring the tooth's supporting structures. The choice of intervention depends on individual disease severity and patient-specific factors [8]. Scaling and root planing (SRP) is the cornerstone of initial periodontal therapy, focusing on the removal of dental plaque, tartar, and stains, as well as smoothing the root surface to eliminate bacterial toxins [8, 21].

Adjunctive antimicrobial therapies, such as low-level laser therapy (LLLT) and antimicrobial photodynamic therapy (aPDT), have also been integrated into periodontal disease management [22, 23]. aPDT involves the interaction between laser light and a photosensitizer (e.g., methylene blue or toluidine blue) to generate free oxygen radicals that destroy bacterial cells and promote healing [22]. Similarly, LLLT is a non-invasive technique that uses low-energy lasers to reduce inflammation and promote tissue repair, often serving as an adjunct to enhance the outcomes of SRP [23].

While aPDT and LLLT have shown promising results as adjunctive treatments, SRP remains the gold standard for managing periodontal disease due to its proven effectiveness and practicality. First, SRP effectively removes plaque and calculus from both supragingival and subgingival surfaces, which is critical for reducing the bacterial load in the oral cavity. Additionally, SRP has been consistently shown to improve key clinical parameters, such as the gingival index (GI) and bleeding on probing, further reinforcing its efficacy and reliability. Finally, SRP is a minimally invasive procedure that can be performed in a standard dental office setting, making it more accessible and cost-effective for patients [22, 23].

The established role of SRP as a primary treatment modality highlights its critical importance in the management of periodontal disease. Although innovative adjunctive therapies like aPDT and LLLT continue to evolve and provide complementary benefits, SRP remains the foundation upon which periodontal therapy is built, ensuring effective bacterial control and clinical outcomes.

Overall, the findings underscore the importance of scaling and root planing in mitigating oral dysbiosis and promoting a healthier microbiome composition, particularly in vulnerable populations like stunted children.

Limitations

This study was designed as a preliminary investigation, and as such, the findings should be interpreted with caution when considering their generalizability to broader populations. The study participants consisted of stunted children, whose nutritional and growth patterns may be influenced by a range of socio-cultural factors, including variations in dietary practices across different communities. These factors highlight the need to account for cultural diversity when interpreting the results. To ensure broader applicability of the findings, future research should include a larger, more diverse sample that adequately represents the population at large. This will help address the limitations inherent in the small sample size of this preliminary study and account for regional or cultural variability in dietary and growth-related factors.

The lack of control over participants' dietary patterns and environmental factors also become our study limitations, both of which may have influenced the outcomes. Variations in dietary intake, particularly micronutrient consumption, and differing environmental exposures, such as hygiene practices and socio-cultural influences, were not accounted for or standardized during the study. These uncontrolled variables could have introduced potential confounding factors, which should be considered when interpreting the findings. Future research should aim to incorporate these aspects to provide a more comprehensive understanding of the relationships observed.

Conclusion

Stunted children have a distinct oral salivary microbiome profile compared to normal stature children, with their saliva being dominated by bacteria from the orange complex, such as *Prevotella spp*., *Campylobacter*, and *Peptostreptococcus*. Scaling showed a promising result in reducing pathogenic bacteria such as *Prevotella* and *Veillonella* while increasing periodontal health-related bacteria such as *Streptococcus.*

Abbreviations

aPDT	antimicrobial photodynamic therapy
GAgP	Generalized Aggressive Periodontitis
GI	Gingival Index
IL-6	Interleukin-6
IL-8	Interleukin-8
LLLT	low-level laser therapy
LPS	Lipopolysaccharides
OHIS	Oral Hygiene Index Simplified
PCR	Polymerase Chain Reaction
QIIME2	Quantitative Insights Into Microbial Ecology 2
ROS	Reactive Oxygen Species
rRNA	Ribosomal Ribonucleic Acid
sIgA	Secretory Immunoglobulin A
SRP	Scaling Root Planing
TNF-α	Tumor Necrosis Factor Alpha
WHO	World Health Organization

Acknowledgements

The authors would like to thank Gian Ernesto and Dinda Ratna Juwita for their assistance with data acquisition and sample collection. We also like to thank the participants for their contributions to the completion of the intervention.

Author contributions

TO, CI, and NK were involved in the design of the research concept, data acquisition, result interpretation, and data image composition. TO and NK were involved in the sample collection and data analysis. TO and CI drafted the manuscript. CI and NK were involved in providing critical revisions of the manuscript. All authors prepare the manuscript and agree for this final version of manuscript to be submitted to this journal.

Funding

This research was partially supported by a funding from Universitas Andalas, Skim Penelitian Tesis Magister (PTM) Batch I (No. 314/UN16.19/PT.01.03/PTM/2024) to NK. The authors would like to express gratitude for the support provided.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Ethics approval for this study was granted by the Research Ethics Committee, Faculty of Medicine, Universitas Andalas (ID: 429/UN.16.2/KEP-FK/2024). Informed consent was obtained from all participants legal guardians after a thorough explanation regarding the study objectives and treatment planning. This study adhered to the principal of human study according to the Declaration of Helsinki.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

Received: 25 December 2024 / Accepted: 10 February 2025
Published online: 16 February 2025

References

1. Sadida ZJ, Indriyanti R, Setiawan AS. Does Growth Stunting Correlate with Oral Health in Children? A Systematic Review. European Journal of Dentistry. 2021;16(01):32–40. Available from: https://doi.org/10.1055/s-0041-1731887
2. Abdat M, Usman S, Chairunas C, Suhaila H. Relationship between stunting with dental and oral status in toddlers. Journal of Dentomaxillofacial Science. 2020;5(2):114. Available from: https://doi.org/10.15562/jdmfs.v5i2.1064
3. Indriyan E, Dewi YLR, Salimo H. Biopsychosocial Determinants of Stunting in Children Under Five: A Path Analysis Evidence from the Border Area West Kalimantan. Journal of Maternal and Child Health. 2018;03(02):146–55. Available from: https://doi.org/10.26911/thejmch.2018.03.02.07
4. Kemenkes RI. Buku Saku Hasil Studi Status Gizi Indonesia (SSGI) Tingkat Nasional, Provinsi, Dan Kabupaten/Kota Tahun 2021. Indonesia: Kementerian Kesehatan Republik Indonesia; 2021.
5. Tedjosasongko U, Pramudita RA, Puteri MM. Biomarker of Malnutrition in Terms of Total Salivary Protein in Stunting Children (Literature Review). International Journal of Scientific Advances. 2022;3(3). Available from: https://doi.org/10.51542/ijscia.v3i3.17
6. Al-Ghutaimel H, Riba H, Al-Kahtani S, Al-Duhaimi S. Common Periodontal Diseases of Children and Adolescents. International Journal of Dentistry. 2014; 2014:1–7. Available from: https://doi.org/10.1155/2014/850674
7. Pérez-Chaparro PJ, Gonçalves C, Figueiredo LC, Faveri M, Lobão E, Tamashiro N et al. Newly Identified Pathogens Associated with Periodontitis. Journal of Dental Research. 2014;93(9):846–58. Available from: https://doi.org/10.1177/0022034514542468
8. Octavia M, Soeroso Y, Kemal Y, Sunarto H, Bachtiar BM. Microbial effects (Porphyromonas gingivalis, Tannerella forsythia) after scaling and root planing. Journal of Physics: Conference Series. 2018;1073:062011. Available from: https://doi.org/10.1088/1742-6596/1073/6/062011
9. Bianchi S, Bernardi S, Simeone D, Torge D, Macchiarelli G, Marchetti E. Proliferation and Morphological Assessment of Human Periodontal Ligament Fibroblast towards Bovine Pericardium Membranes: An In Vitro Study. Materials. 2022;15(23):8284. Available from: https://doi.org/10.3390/ma15238284
10. Luis Muñoz-Carrillo J, Elizabeth Hernández-Reyes V, Eduardo García-Huerta O, Chávez-Ruvalcaba F, Isabel Chávez-Ruvalcaba M, Mariana Chávez-Ruvalcaba K et al. Pathogenesis of Periodontal Disease. Periodontal Disease - Diagnostic and Adjunctive Non-surgical Considerations. 2020; Available from: https://doi.org/10.5772/intechopen.86548
11. Mutasa K, Tome J, Rukobo S, Govha M, Mushayanembwa P, Matimba FS et al. Stunting Status and Exposure to Infection and Inflammation in Early Life Shape Antibacterial Immune Cell Function Among Zimbabwean Children. Frontiers in Immunology. 2022;13. Available from: https://doi.org/10.3389/fimmu.2022.899296
12. Gasner NS, Schure RS. Periodontal Disease [Internet]. StatPearls - NCBI Bookshelf. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554590/
13. Bianchi S, Torge D, Rinaldi F, Piattelli M, Bernardi S, Varvara G. Platelets' role in dentistry: From oral pathology to regenerative potential. Biomedicines. 2022;10(2):218. Available from: https://doi.org/10.3390/biomedicines10020218
14. D'Agostino S, Ferrara E, Valentini G, Stoica SA, Dolci M. Exploring Oral microbiome in healthy infants and Children: A Systematic review. International Journal of Environmental Research and Public Health. 2022;19(18):11403. Available from: https://doi.org/10.3390/ijerph191811403
15. Shaiber A, Willis AD, Delmont TO, Roux S, Chen LX, Schmid AC et al. Functional and genetic markers of niche partitioning among enigmatic members of the human oral microbiome. Genome Biology. 2020;21(292):1–35. Available from: https://doi.org/10.1186/s13059-020-02195-w
16. Lee YH, Park HJ, Jeong SJ, Auh QS, Jung J, Lee GJ et al. Oral Microbiome Profile of Gingivitis and Periodontitis by Next-Generation Sequencing. Research Square (Research Square). 2023; Available from: https://doi.org/10.21203/rs.3.rs-3530768/v1
17. Lenartova M, Tesinska B, Janatova T, Hrebicek O, Mysak J, Janata J et al. The Oral Microbiome in Periodontal Health. Do T, editor. Frontiers in Cellular and Infection Microbiology. 2021;11:1–16. Available from: https://doi.org/10.3389/fcimb.2021.629723
18. Kang Y, Sun B, Chen Y, Lou Y, Zheng M, Li Z. Dental Plaque Microbial Resistomes of Periodontal Health and Disease and Their Changes after Scaling and Root Planing Therapy. Fey PD, editor. mSphere. 2021;6(4). Available from: https://doi.org/10.1128/msphere.00162-21
19. Gerardi D, Bernardi S, Bruni A, Falisi G, Botticelli G. Characterization and morphological methods for oral biofilm visualization: where are we nowadays? AIMS Microbiology. 2024;10(2):391–414. Available from: https://doi.org/10.3934/microbiol.2024020
20. U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, National Institutes of Health. Periodontal (Gum) disease [Internet], National Institute of Dental and Craniofacial Research. 2013. Available from: https://www.nidcr.nih.gov/sites/default/files/2017-09/periodontal-disease_0.pdf
21. SDCEP. Prevention and Treatment of Periodontal Disease in Primary Care. Scotland: Scottish Dental Clinical Effectiveness Programme; 2014.
22. Malik NKA, Alkadhi OH. Effectiveness of mechanical debridement with and without antimicrobial photodynamic therapy against oral yeasts in children with gingivitis undergoing fixed orthodontic therapy. Photodiagnosis and Photodynamic Therapy. 2020;31:101768. Available from: https://doi.org/10.1016/j.pdpdt.2020.101768
23. Igic M, Mihailovic D, Kesic L, Milasin J, Apostolovic M, Kostadinovic L et al. Cytomorphometric and clinical investigation of the gingiva before and after low-level laser therapy of gingivitis in children. Lasers in Medical Science. 2011;27(4):843–8. Available from: https://doi.org/10.1007/s10103-011-0993-z
24. Surono IS, Widiyanti D, Kusumo PD, Venema K. Gut microbiota profile of Indonesian stunted children and children with normal nutritional status. Carbonero F, editor. PLOS ONE. 2021;16(1):e0245399. Available from: https://doi.org/10.1371/journal.pone.0245399
25. Sembler-Møller ML, Belstrøm D, Locht H, Enevold C, Pedersen AML. Next-generation sequencing of whole saliva from patients with primary Sjögren's syndrome and non-Sjögren's sicca reveals comparable salivary microbiota. Journal of Oral Microbiology. 2019;11(1):1660566. vailable from: https://doi.org/10.1080/20002297.2019.1660566
26. Lestari S, Fujiati II, Keumalasari D, Daulay M. The prevalence and risk factors of stunting among primary school children in North Sumatera, Indonesia. IOP Conference Series Earth and Environmental Science. 2018;125:012219. Available from: https://doi.org/10.1088/1755-1315/125/1/012219
27. Thompson AL. Greater male vulnerability to stunting? Evaluating sex differences in growth, pathways and biocultural mechanisms. Annals of Human Biology. 2021;48(6):466–73. Available from: https://doi.org/10.1080/03014460.2021.1998622
28. Bourke CD, Berkley JA, Prendergast AJ. Immune Dysfunction as a Cause and Consequence of Malnutrition. Trends in Immunology. 2016;37(6):386–98. Available from: https://doi.org/10.1016/j.it.2016.04.003
29. Alotaibi T. Malnutrition and Diet Role in Prevention of oral disease. EC Dent Sci. 2019;18(9):2206–13.
30. Van Dyke TE, Bartold PM, Reynolds EC. The Nexus Between Periodontal Inflammation and Dysbiosis. Frontiers in Immunology. 2020;11. Available from: https://doi.org/10.3389/fimmu.2020.00511
31. Sobocki BK, Basset CA, Bruhn-Olszewska B, Olszewski P, Szot O, Kaźmierczak-Siedlecka K et al. Molecular Mechanisms Leading from Periodontal Disease to Cancer. International Journal of Molecular Sciences. 2022;23(2):970. Available from: https://doi.org/10.3390/ijms23020970
32. Utami NH, Rachmalina R, Irawati A, Sari K, Rosha BC, Amaliah N, Mal J et al. Nutr. 2018 p. 11–23.
33. Masrul M, Izwardy D, Sudji IR, Purnakarya I, Syahrial S, Nindrea RD. Microbiota Profile with Stunting Children in West Sumatera Province, Indonesia. Open Access Macedonian Journal of Medical Sciences. 2020;8(E):334–40. Available from: https://doi.org/10.3889/oamjms.2020.4209

34. Millen AE, Dahhan R, Freudenheim JL, Hovey KM, Li L, McSkimming DI et al. Dietary carbohydrate intake is associated with the subgingival plaque oral microbiome abundance and diversity in a cohort of postmenopausal women. Scientific Reports. 2022;12(1). Available from: https://doi.org/10.1038/s41598-022-06421-2
35. Da Rocha IMG, Torrinhas R, Fonseca D, De Oliveira Lyra C, De Sousa Alves Neri JL, Balmant BD et al. Pro-Inflammatory Diet Is Correlated with High Veillonella rogosae, Gut Inflammation and Clinical Relapse of Inflammatory Bowel Disease. Nutrients. 2023;15(19):4148. Available from: https://doi.org/10.3390/nu15194148
36. Ribeiro AA, Azcarate-Peril MA, Cadenas MB, Butz N, Paster BJ, Chen T et al. The oral bacterial microbiome of occlusal surfaces in children and its association with diet and caries. PLoS ONE. 2017;12(7):e0180621. Available from: https://doi.org/10.1371/journal.pone.0180621
37. Singh OS, Shabina S, Shibani G. Various complexes of the oral microbial flora in periodontal disease. Journal of Dental Problems and Solutions. 2021;032–3. Available from: https://doi.org/10.17352/2394-8418.000101
38. Gambin DJ, Vitali FC, Casanova KAS, De Carli JP, Mazzon RR, De Almeida Gomes BPF et al. Prevalence of species of yellow, purple and green microbial complexes in endo-perio lesions: a systematic review. Brazilian Oral Research. 2024;38. Available from: https://doi.org/10.1590/1807-3107bor-2024.vol38.0048
39. Griauzdyte V, Jagelaviciene E. Antimicrobial Activity of Zinc against Periodontal Pathogens: A Systematic Review of In Vitro Studies. Medicina. 2023;59(12):2088. Available from: https://doi.org/10.3390/medicina59122088
40. Könönen E, Fteita D, Gursoy UK, Gursoy M. Prevotella species as oral residents and infectious agents with potential impact on systemic conditions. Journal of Oral Microbiology. 2022;14(1). Available from: https://doi.org/10.1080/20002297.2022.2079814
41. Huang S, Li Z, He T, Bo C, Chang J, Li L et al. Microbiota-based Signature of Gingivitis Treatments: A Randomized Study. Scientific Reports. 2016;6(1). Available from: https://doi.org/10.1038/srep24705
42. Zhao Y, Ye Q, Feng Y, Chen Y, Tan L, Ouyang Z et al. Prevotella genus and its related NOD-like receptor signaling pathway in young males with stage III periodontitis. Frontiers in Microbiology. 2022;13. Available from: https://doi.org/10.3389/fmicb.2022.1049525
43. Zhou P, Manoil D, Belibasakis GN, Kotsakis GA. Veillonellae: Beyond Bridging Species in Oral Biofilm Ecology. Frontiers in Oral Health. 2021;2:1–11. Available from: https://doi.org/10.3389/froh.2021.774115
44. Liu G, Luan Q, Chen F, Chen Z, Zhang Q, Yu X. Shift in the subgingival microbiome following scaling and root planing in generalized aggressive periodontitis. Journal of Clinical Periodontology. 2018;45(4):440–52. Available from: https://doi.org/10.1111/jcpe.12862

Publisher's note