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Original Article

Oral and fecal microbiota in oral lichen planus and xerostomia patients: A preliminary study

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KEYWORDS
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Abstract *Background/purpose:* The human microbiota constitutes a dynamic community of microorganisms inhabiting the body, with the gut and oral microbiotas being the most prominent. Previous studies have shown associations between oral microbiota disruption and various oral and systemic diseases, along with the involvement of the oral–gut microbiome axis. However, further investigation into the relationship between common oral conditions and microbiota changes remains needed. This study hypothesized that the distinct immune environments in oral lichen planus (OLP) and xerostomia patients result in recognizable microbiota compositions, with additional evaluation of fecal microbiota to explore the oral–gut axis.

Materials and methods: Gingival and fecal samples from 8 OLP patients, 19 xerostomia patients, and 10 healthy controls were collected and analyzed using 16S rRNA sequencing with bioinformatic analysis at the phylum level. Statistical comparisons between groups were performed using Student's T-test.

Results: Compared with healthy controls, OLP patients showed significant increases in Campylobacterota and Fusobacteria, and decreases in Actinobacteria and Proteobacteria. Xerostomia patients demonstrated a significant increase in Firmicutes. In fecal samples, both OLP and xerostomia patients exhibited significantly reduced Bacteroidetes compared with controls.

Conclusion: OLP and xerostomia are associated with distinct oral microbiota patterns, which may aid in early and non-invasive diagnosis. Fecal samples of both patient groups differed significantly from controls in Bacteroidetes, supporting the oral–gut microbiome axis and providing further evidence that oral conditions can influence systemic microbial communities. A major limitation of this study is the relatively small sample size.

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Introduction

The term microbiota refers to the diverse community of microorganisms residing within the human body.[1] The oral microbiota is the second most complex microbial ecosystem in the human body behind the gut microbiota,[2] consisting of bacteria, viruses, fungi and protozoa. The most common bacterial phyla within the oral microbiota are Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Spirochetes, and Fusobacteria.[3]

The compositional stability of the oral microbiota in a healthy individual results from a multitude of dynamic interactions between host immunity and the microbiome, a balance that helps prevent colonization by potentially pathogenic exogenous species.[4] Disruption of this balance, or dysbiosis, refers to a shift in the oral microbiota to an unusual state in terms of composition, metabolic activities, or distribution within the human body.[5] It can be caused by various factors including inflammatory diseases, poor hygiene, an unhealthy diet, smoking, excessive alcohol consumption, hormonal changes, medications, stress, genetic predisposition, etc.[6]

Various studies have pointed out the potential link between oral microbiota alterations and the development of oral diseases, including dental caries,[7] periodontal diseases,[8] and oral squamous cell carcinoma.[9,10] Numerous systemic diseases, including autoimmune disorders,[11,12] metabolic diseases,[13] cardiovascular diseases,[14] neurodegenerative diseases,[15] and malignancies,[16] were also reported to show similar relationships. Since such a wide array of conditions could be associated with oral microbiota changes, we wondered whether specific patterns of change could be discovered in the oral microbiota of each disease, thus providing the potential to serve as a tool for disease screening and early detection.

We recruited two groups of patients with diagnoses of either oral lichen planus (OLP) or xerostomia through conventional diagnostic methods. OLP is a chronic inflammatory condition related to unregulated cellular immunity, resulting in mucosal changes including ulceration, erythema, and atrophic changes.[17] Owing to its inflammatory nature, multiple studies have revealed a relationship between OLP formation and subsequent oral dysbiosis.[18–20] One of our aims was to detail the specific taxonomic changes (increase or decrease) in the microbiota of OLP patients to provide further information on potential clinical applications.

Xerostomia is defined as the subjective sensation of oral dryness, with various etiologies such as Sjogren's syndrome (SS) and medication usage.[21] The components of saliva include water, electrolytes, mucoproteins, lysozymes, lactoferrin, immunoglobulin A, etc., which all together aid in controlling the growth of microorganisms and maintaining a stable oral microbiome.[22] Thus, it is reasonable to infer that the lack of saliva observed in xerostomia may also interrupt the stability of the oral microbiome, as various studies have described.[23,24] We investigated xerostomia patients' oral microbiota composition to explore whether a common pattern could be observed that might suggest a potential clinical usage.

The oral-gut microbiome axis, consisting two of the largest habitats of microorganisms within the human body, has been recently reported to demonstrate bidirectional interplay in terms of composition and disease formation, although the detailed mechanism and causal relationship remain largely unclear.[25] The significance of this axis in further understanding various diseases, including periodontal diseases, gastrointestinal diseases, and cancers, has also been highlighted in recent studies.[25,26] For these reasons, we also aimed to investigate whether changes in the fecal microbiota could be observed in OLP and xerostomia patients in hopes of providing further information and broadening our understanding of the oral-gut-microbiome axis.

The purpose of this study was to investigate whether different oral conditions, specifically OLP and xerostomia, are associated with distinct compositional patterns in the oral and gut microbiota. This study aims to enhance our understanding of the oral—gut axis and its association with disease development and to explore potential applications in the screening and early diagnosis of OLP, xerostomia, and possibly other conditions such as precancerous lesions and systemic diseases.

Materials and methods

Study design

In this observational, cross-sectional study conducted at Chung Shan Medical University Hospital in Taichung City, we aimed to investigate whether distinct oral and gut microbiota patterns could be observed in patients with OLP and xerostomia.

Ethical statement

The study protocol was approved by the Institutional Review Board (IRB) of Chung Shan Medical University Hospital. Information on the study's objectives, risks and benefits, as well as their right to withdraw anytime was provided to all participants prior to obtaining written informed consent.

Participants

A total of 37 participants were recruited, including eight patients with OLP (N = 8), nineteen patients with xerostomia (N = 19), and ten healthy volunteers (N = 10). The

age of the OLP group ranged from 52 to 69 years, with 7 female patients and 1 male patient. The clinical diagnosis of OLP was made by experienced oral medicine doctors on the basis of the modified criteria of Van der Meij et al.[27] In accordance with Taiwan's standard clinical practice, biopsy may not be necessary for OLP unless the lesions are clinically suspicious for malignancy.[28] The age of the patients in the xerostomia group ranged from 25 to 83 years, with 17 female patients and 2 male patients. The xerostomia group comprised 14 patients diagnosed with SS and 5 patients whose xerostomia was attributed to other causes such as radiotherapy (N = 2) or medications (N = 3). The age of participants in the control group ranged from 23 to 58 years, with 6 female and 4 male volunteers. The inclusion criteria for xerostomia included the following:

A. Dry mouth with a diagnosis of SS.
B. Dry mouth with a history of taking medications, showing dry mouth side effects.
C. Patients with head and neck malignancies who received radiotherapy.

The exclusion criteria for xerostomia included the following:

A. Patients taking antibiotics.
B. Patients who smoke cigarettes.
C. Patients who were unwilling to participate in the study.
D. Patients receiving hormonal therapy.

The detailed demographic information of the participants is listed in Table 1.

Table 1 Demographic information of the study participants.

Group	Age	Gender
Oral lichen planus	66	Female
Oral lichen planus	52	Female
Oral lichen planus	52	Female
Oral lichen planus	65	Male
Oral lichen planus	67	Female
Oral lichen planus	69	Female
Oral lichen planus	53	Female
Oral lichen planus	53	Female
Xerostomia with SS	58	Female
Xerostomia with SS	83	Female
Xerostomia with SS	50	Female
Xerostomia with SS	69	Female
Xerostomia with SS	55	Female
Xerostomia with SS	42	Female
Xerostomia with SS	33	Female
Xerostomia with SS	66	Female
Xerostomia with SS	25	Female
Xerostomia with SS	63	Female
Xerostomia with SS	47	Female
Xerostomia with SS	73	Female
Xerostomia with SS	77	Female
Xerostomia with SS	52	Female
Xerostomia without SS	74	Female
Xerostomia without SS	63	Female
Xerostomia without SS	47	Female
Xerostomia without SS	58	Male
Xerostomia without SS	55	Male
Control	40	Female
Control	27	Female
Control	36	Female
Control	44	Female
Control	31	Female
Control	58	Male
Control	29	Male
Control	23	Male
Control	25	Male
Control	52	Female

Age and gender information of all the participants enlisted in the study are shown as Table 1.
SS: Sjogren's syndrome.

Sample collection

Gingival plaque samples were collected via sterile curettes and immediately placed in PowerBead tubes (Qiagen, Germantown, MD, USA). The samples were stored at 4 °C in accordance with the Human Microbiome Project Core Microbiome Sampling Protocol A (HMP Protocol #07-001, Version 12.0, July 29, 2010). After plaque collection, the participants were provided with a PowerBead tube (Qiagen) for stool sample collection at home, with instructions on proper sampling techniques. Stool samples were returned via mail and stored at 4 °C until further processing.

DNA extraction, sequencing, and bioinformatic analysis

DNA extraction was performed via the DNeasy PowerSoil Kit (Qiagen) according to the manufacturer's protocol and was described in detail as previously published.[23] The V3—V4 region of the 16S rRNA gene was amplified and sequenced via the Illumina MiSeq platform (Health GeneTech Corp., New Taipei City, Taiwan). Bioinformatic analysis involved clustering sequencing reads at 99 % similarity to define operational taxonomic units (OTUs). OTUs were taxonomically assigned by comparison with reference databases.

Statistical analysis

Thirteen bacterial phyla—Actinobacteria, Bacteroidetes, Campylobacterota, Cyanobacteria, Desulfobacterota, Firmicutes, Fusobacteria, Lentisphaerae, Patescibacteria, Proteobacteria, Spirochaetes, Synergistetes, and Verrucomicrobia—were identified and analyzed. Student's t-test was used to compare the relative abundance of each phylum between the OLP and healthy control groups, and between the xerostomia and healthy control groups, in both gingival and fecal samples at the phylum level.

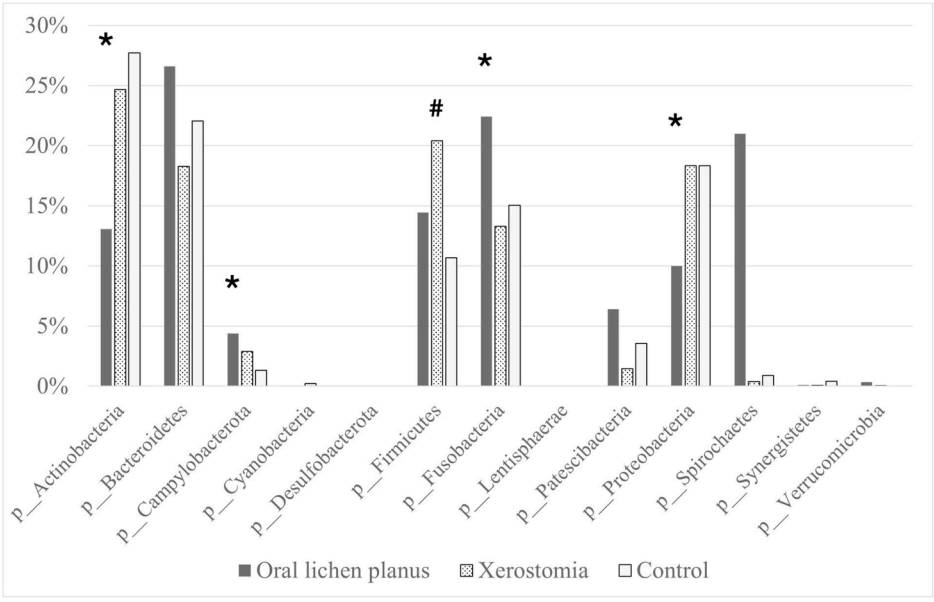

Figure 1 **Distribution of oral microbiota among OLP, xerostomia, and control groups *(bar chart)*.** Black bars: OLP (N = 8), dotted grey bars: xerostomia (N = 19), grey bars: controls (N = 10). X-axis: bacterial phyla; Y-axis: relative abundance (%). Values represent means. Student's *T*-test; $P \leq 0.05$. *: significant difference between OLP patients and controls, $P \leq 0.05$. #: significant difference between xerostomia patients and controls, $P \leq 0.05$. OLP: Oral lichen planus.

Results

Subjects with OLP, xerostomia, and healthy volunteers were recruited to provide gingival plaque and stool samples for microbiota analysis. Oral microbiota samples were successfully obtained from 8 OLP patients, 19 xerostomia patients, and 10 healthy controls, whereas fecal microbiota samples were collected from 8 OLP patients, 8 xerostomia patients, and 9 healthy controls.

Compared with those of healthy controls, the oral microbiota of the OLP group presented a significant increase in the relative abundance of Campylobacterota (4.39 % vs. 1.32 %, $P < 0.03$) and Fusobacteria (22.41 % vs. 15.04 %, $P < 0.02$), along with a significant decrease in Actinobacteria (13.08 % vs. 27.73 %, $P < 0.01$) and Proteobacteria (9.99 % vs. 18.33 %, $P < 0.05$) (Fig. 1, *). The xerostomia group presented a significant increase in Firmicutes (20.41 % vs. 10.67 %, $P < 0.04$) (Fig. 1, #). A comparison of the oral microbiota of the three groups was shown in Table 2. Distinct distribution signatures of the oral microbiota among the three groups were illustrated in Fig. 2. Subgroup analysis of xerostomia patients revealed no significant difference in oral microbiota composition between those with SS-related xerostomia and those with

Table 2 Comparison of oral microbiota composition among the OLP, xerostomia, and control groups at the phylum level.

Taxonomy	OLP (N = 8) (Mean ± SD)	Xerostomia (N = 19) (Mean ± SD)	Control (N = 10) (Mean ± SD)
Actinobacteria[a]	13.08 ± 5.49 %	24.68 ± 12.13 %	27.73 ± 12.54 %
Bacteroidetes	26.61 ± 7.40 %	18.29 ± 10.16 %	22.05 ± 10.92 %
Campylobacterota[a]	4.39 ± 2.44 %	2.88 ± 4.84 %	1.32 ± 2.62 %
Cyanobacteria	0.00 ± 0 %	0.21 ± 0.89 %	0.00 ± 0 %
Desulfobacterota	0.00 ± 0 %	0.00 ± 0 %	0.00 ± 0 %
Firmicutes[b]	14.45 ± 3.52 %	20.41 ± 14.54 %	10.67 ± 9.30 %
Fusobacteria[a]	22.41 ± 3.99 %	13.31 ± 9.39 %	15.04 ± 7.64 %
Lentisphaerae	0.00 ± 0 %	0.00 ± 0 %	0.00 ± 0 %
Patescibacteria	6.40 ± 3.78 %	1.46 ± 2.39 %	3.57 ± 7.80 %
Proteobacteria[a]	9.99 ± 5.34 %	18.33 ± 10.32 %	18.33 ± 10.03 %
Spirochaetes	2.23 ± 2.61 %	0.37 ± 1.01 %	0.90 ± 1.91 %
Synergistetes	0.09 ± 0.16 %	0.05 ± 0.20 %	0.40 ± 0.93 %
Verrucomicrobia	0.33 ± 0.91 %	0.01 ± 0.02 %	0.00 ± 0 %

OLP: Oral lichen planus. SD: Standard deviation.
[a] Significant difference between OLP patients and controls, $P \leq 0.05$.
[b] Significant difference between xerostomia patients and controls, $P \leq 0.05$.

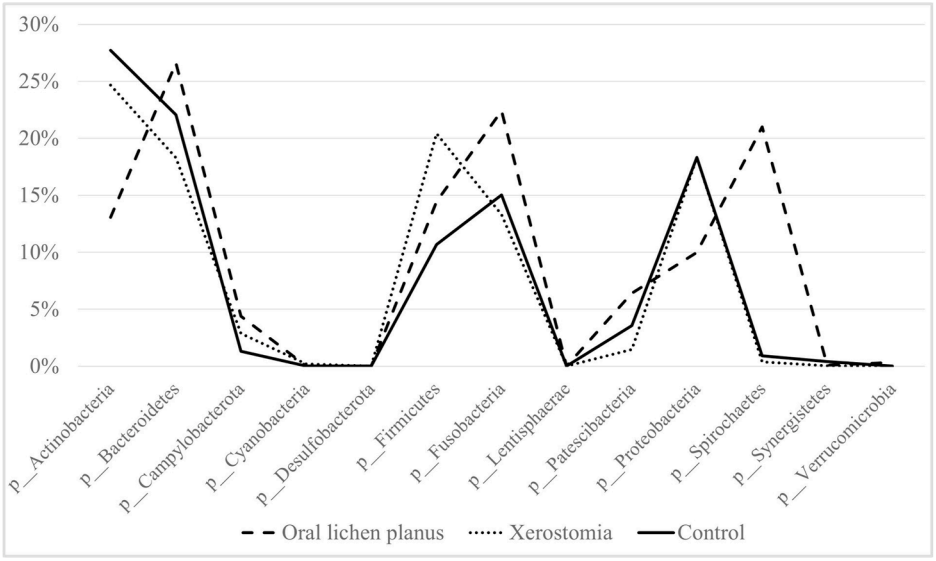

Figure 2 Distribution pattern of oral microbiota among OLP, xerostomia, and control groups *(line graph)*. Dashed line: OLP (N = 8), dotted line: xerostomia (N = 19), solid line: controls (N = 10). X-axis: bacterial phyla; Y-axis: relative abundance (%). Values represent means. OLP: Oral lichen planus.

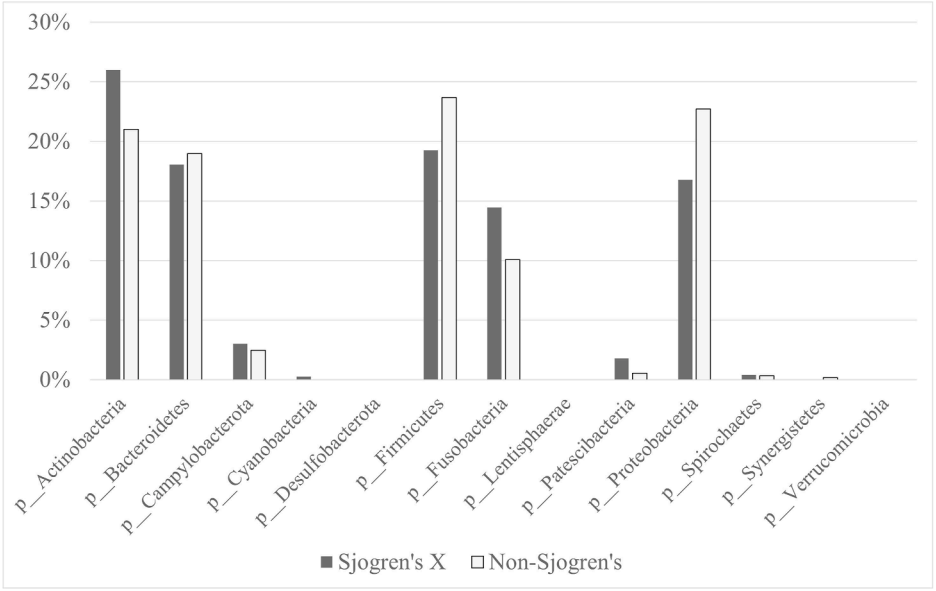

Figure 3 Oral microbiota distribution in SS-related vs non—SS—related xerostomia patients. Black bars: SS-related xerostomia (N = 14), grey bars: non-SS xerostomia (N = 5). X-axis: bacterial phyla; Y-axis: relative abundance (%). Values represent means. No significant differences were observed. SS: Sjogren's syndrome.

xerostomia due to other causes (Fig. 3). No significant differences were observed when both xerostomia subgroups were compared independently from the control group. Comparisons among the SS-related xerostomia, non-—SS—related xerostomia, and the control group are shown in Table 3.

Analysis of fecal samples revealed a significantly lower relative abundance of Bacteroidetes in both OLP patients (42.23 % vs. 48.92 %, $P < 0.05$) and xerostomia patients (41.31 % vs. 48.92 %, $P < 0.01$) than in healthy controls (Fig. 4). The distribution of the fecal microbiota among the three groups was detailed in Table 4 and similar signature patterns were illustrated in Fig. 5.

Discussion

Our data demonstrated that within the oral microbiota, OLP patients presented significant increases in Campylobacterota (4.39 % vs. 1.32 %, $P < 0.03$) and Fusobacteria (22.41 % vs. 15.04 %, $P < 0.02$), and significant decreases in Actinobacteria (13.08 % vs. 27.73 %, $P < 0.01$) and

Table 3 Comparison of oral microbiota composition between Sjogren's syndrome-related and non-Sjogren's syndrome-related xerostomia patients at the phylum level.

Taxonomy	Sjogren's syndrome-related xerostomia (N = 14) (Mean ± SD)	Non-Sjogren's syndrome-related xerostomia (N = 5) (Mean ± SD)	Control (N = 8) (Mean ± SD)
Actinobacteria	25.99 ± 12.32 %	21.00 ± 12.06 %	27.73 ± 12.54 %
Bacteroidetes	18.06 ± 7.59 %	18.96 ± 16.64 %	22.05 ± 10.92 %
Campylobacterota	3.03 ± 4.79 %	2.48 ± 5.54 %	1.32 ± 2.62 %
Cyanobacteria	0.28 ± 1.04 %	0.00 ± 0 %	0.00 ± 0 %
Desulfobacterota	0.00 ± 0 %	0.00 ± 0 %	0.00 ± 0 %
Firmicutes	19.24 ± 14.80 %	23.68 ± 14.85 %	10.67 ± 9.30 %
Fusobacteria	14.46 ± 9.37 %	10.08 ± 9.70 %	15.04 ± 7.64 %
Lentisphaerae	0.00 ± 0 %	0.00 ± 0 %	0.00 ± 0 %
Patescibacteria	1.79 ± 2.66 %	0.54 ± 1.20 %	3.57 ± 7.80 %
Proteobacteria	16.76 ± 11.20 %	22.70 ± 6.27 %	18.33 ± 10.03 %
Spirochaetes	0.39 ± 1.11 %	0.34 ± 0.76 %	0.90 ± 1.91 %
Synergistetes	0.00 ± 0 %	0.18 ± 0.04 %	0.40 ± 0.93 %
Verrucomicrobia	0.01 ± 0.02 %	0.00 ± 0 %	0.00 ± 0 %

No significant differences were noted in any phyla. SD: Standard deviation.

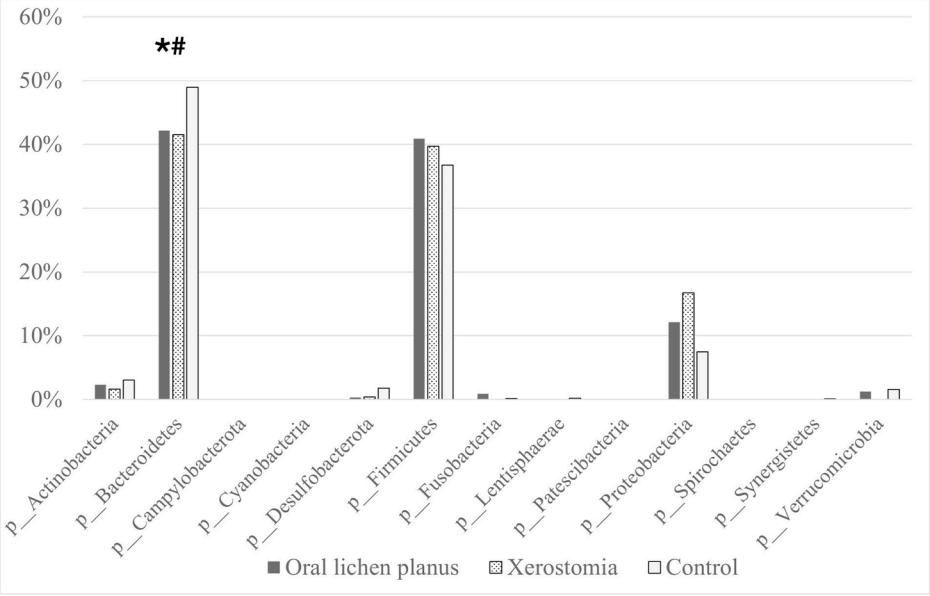

Figure 4 **Distribution pattern of fecal microbiota among OLP, xerostomia, and control groups *(bar chart)*.** Black bars: OLP (N = 8), dotted grey bars: xerostomia (N = 8), grey bars: controls (N = 9). X-axis: bacterial phyla; y-axis: relative abundance (%). Values represent means. Student's *T*-test; $P \leq 0.05$. *: significant difference between OLP patients and controls, $P \leq 0.05$. #: significant difference between xerostomia patients and controls, $P \leq 0.05$. OLP: Oral lichen planus.

Proteobacteria (9.99 % vs. 18.33 %, $P < 0.05$), compared with healthy controls. These results align with some of the discoveries in previous studies. For instance, the amount of *Fusobacterium nucleatum* was found to be greater in OLP patients in a study performed by Du et al.[29] In a study by Yan et al., Campylobacterota was noted to have a greater relative abundance in OLP patients than in healthy controls, while the control group presented greater enrichment of Actinobacteria which matches our findings.[30] However, there are also results from other studies that conflict with our findings. In the same study performed by Yan et al., a greater abundance of Fusobacteria in healthy controls and a greater abundance of Proteobacteria in OLP patients were noted. Additionally, Wang et al. noted a relative increase in Campylobacter in healthy controls compared with reticular and erosive type OLP patients.[31] The difference in results could potentially be explained by various factors, including sample size, patient background, sampling methods, and the taxonomical level analyzed. Different types of OLPs based on clinical presentation, namely reticular, papular, plaque-like, atrophic/erosive, ulcerative, and bullous,[32] might also have an impact.

Table 4 Comparison of fecal microbiota composition among the OLP, xerostomia, and control groups at the phylum level.

Taxonomy	OLP (N = 8) (Mean ± SD)	Xerostomia (N = 8) (Mean ± SD)	Control (N = 9) (Mean ± SD)
Actinobacteria	2.33 ± 1.80	1.63 ± 1.31	3.04 ± 2.44
Bacteroidetes[a,b]	42.16 ± 7.46	41.53 ± 5.60	48.94 ± 3.58
Campylobacterota	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Cyanobacteria	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Desulfobacterota	0.30 ± 0.54	0.1 ± 0.70	1.77 ± 2.61
Firmicutes	40.91 ± 8.58	39.71 ± 8.02	36.76 ± 9.44
Fusobacteria	0.86 ± 1.43	0.00 ± 0.00	0.16 ± 0.46
Lentisphaerae	0.08 ± 0.21	0.00 ± 0.00	0.19 ± 0.43
Patescibacteria	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Proteobacteria	12.13 ± 12.00	16.74 ± 10.68	7.49 ± 9.60
Spirochaetes	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Synergistetes	0.00 ± 0.00	0.00 ± 0.00	0.08 ± 0.23
Verrucomicrobia	1.23 ± 2.08	0.00 ± 0.00	1.57 ± 2.50

OLP: Oral lichen planus. SD: Standard deviation.
[a] Significant difference between OLP patients and controls, $P \leq 0.05$.
[b] Significant difference between xerostomia patients and controls, $P \leq 0.05$.

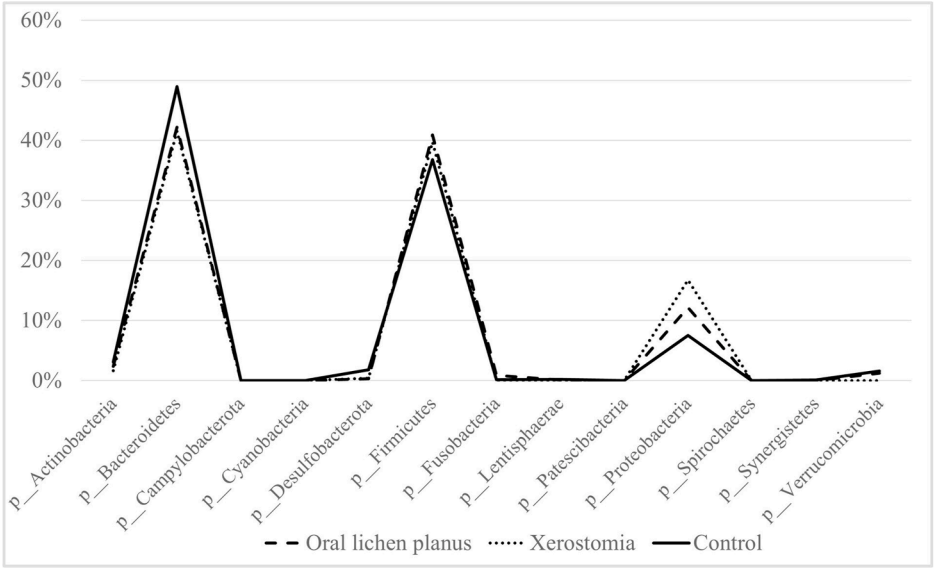

Figure 5 **Distribution pattern of fecal microbiota among OLP, xerostomia, and controls *(line graph)*.** Dashed line: OLP (N = 8), dotted line: xerostomia (N = 8), solid line: controls (N = 9). X-axis: bacterial phyla; Y-axis: relative abundance (%). Values represent means. OLP: Oral lichen planus.

In xerostomia patients, regardless of etiology, a significant increase in Firmicutes alone (20.41 % vs. 10.67 %, $P < 0.04$) was noted in the oral microbiota when compared with that in healthy controls, supporting our initial hypothesis. Notably, SS, a condition that not only results in a lack of saliva but also elicits an increased B-cell-related immune response,[33] did not significantly differ in any phyla compared with the non-SS group. Previous work by Li et al. reported a lower relative abundance in Proteobacteria in SS patients along with other discoveries,[34] yet our study revealed a slightly higher relative abundance of Proteobacteria (22.7 % in SS-related xerostomia vs. 16.76 % in non—SS—related xerostomia). These discrepancies could be attributed to the relatively small sample size and other confounding factors, such as age, sex, and family history of autoimmune diseases. Further investigations with larger sample sizes and adequate matching are needed in this respect.

Previously, Chattopadhyay et al. reported that alterations in oral commensal microbial communities have potential application as a diagnostic tool to predict oral squamous cell carcinoma.[35] Additionally, Yang et al. identified an association between oral microbiome variations and mutations in oral cancer.[36] Three years later, Su et al. demonstrated that oral microbial dysbiosis could distinguish OSCC sites from normal tissue and identified associated microbiota signatures and functional changes.[37] Consistent with previous research, our results align with

the claim that each condition may present with a distinct oral microbiota pattern, supporting its potential use for screening, early diagnosis and progression monitoring of oral diseases in the clinical setting based on oral microbiota changes.

In the fecal microbiota, similar signature patterns were observed among the groups studied. Further analysis of Bacteroidetes in the fecal samples revealed significant differences in the amount present between the two diseased groups (OLP and xerostomia) and the control group. A marked reduction in Bacteroidetes was detected in fecal samples from both OLP patients (42.23 % vs. 48.92 %, $P < 0.05$) and xerostomia patients (41.31 % vs. 48.92 %, $P < 0.01$), relative to healthy controls. The impact of various microorganisms and physiological conditions along the gastrointestinal tract, along with other factors including sample size, diet, and personal medical history may have attenuated disease-related changes in the fecal microbiota, making them less pronounced than those in the oral microbiota. Future research should expand cohort size, address confounders, and analyze additional diseases at finer taxonomic levels (genus or species) to explore disease-specific microbial distributions. Nevertheless, despite the limitations, both OLP and xerostomia cases showed significant differences from healthy controls in Bacteroidetes. This supports oral-gut axis interplay and suggests oral diseases may affect the distal GI tract. Conversely, various studies have shown that profiling the oral microbiome may offer an alternative screening method for detecting lower GI tract malignancies.[38,39] It would be interesting for future studies to include other diseases such as colorectal carcinoma to better understand the causal relationship and pathogenesis between the two environments.

This study provides preliminary evidence that identifying distinguishable changes in oral microbiota composition which may serve as an early, effective, and noninvasive method for screening and monitoring the progress of OLP and xerostomia. Additionally, despite similar patterns in the fecal microbiota, OLP and xerostomia patients differed significantly in Bacteroidetes compared to controls. These findings support the notion of an oral-gut axis, where oral conditions are associated with changes in both the oral and the gut microbiota. It would be also interesting to study the changes of fecal microbiota in GI-associated diseases, such as colorectal carcinoma. A key limitation of this study is the relatively small sample size. Future studies with larger cohorts and various disease categories are warranted to further validate these observations and elucidate the clinical relevance of oral microbiota profiles in disease diagnosis and monitoring.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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