Received: 18 July 2023 | Revised: 11 September 2023 | Accepted: 14 September 2023

DOI: 10.1111/omi.12435

REVIEW ARTICLE

WILEY

Oral microbial *biomap* in the drought environment: Sjogren's syndrome

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Funding information
Spanish Ministry of Science and Innovation, Grant/Award Number: PID2020-118557GA-I00; Plan Propio of the University of Seville, Grant/Award Number: 2021/00001194

Abstract

Sjogren's syndrome (SS) is an autoimmune disease that affects primarily the salivary glands, making perturbations in the oral ecosystem and potential factors of salivary flow that influence the onset and development of the disease. The oral cavity contains diverse microorganisms that inhabit various niches such as the oral microbial "biomap." It does not seem specific enough to establish a characteristic microbiome, given the diversity of clinical manifestations, variable rates of salivary secretion, and influential risk factors in patients with SS. This review discusses the biogeography of the oral microbiome in patients with SS such as saliva, tongue, tooth, mucosa, and gum. The microorganisms that were more abundant in the different oral niches were Gram-positive species, suggesting a higher survival of cell wall bacteria in this arid oral environment. Reduced salivary flow appears not to be linked to the cause of dysbiosis alone but influences host-associated risk factors. However, much work remains to be done to establish the role of the microbiome in the etiopathogenesis of autoimmune diseases such as SS. Future studies of the microbiome in autoimmunity will shed light on the role of specific microorganisms that have never been linked before with SS.

KEYWORDS
autoimmunity, microbial ecology, niche, oral microbiome, Sjogren's syndrome

1 | INTRODUCTION

Autoimmune diseases are a heterogeneous group of more than 100 chronic diseases according to the "Autoimmune Diseases Coordinating Committee" of the National Institutes of Health of the United States (Callejas Rubio et al., 2019). They can affect any organ of the body, so their diagnosis, treatment, and prognosis must be approached by a multidisciplinary team. The term autoimmunity implies the presence of autoreactive T or B lymphocytes in the periphery that escape the surveillance of the immune system, which can be regulatory but do not always cause all the described autoimmune diseases (Janket et al., 2015). Sjogren's syndrome (SS) is one of the rheumatic autoimmune diseases characterized mainly by oral and ocular dryness. SS is not only localized in the mouth but can affect other organs of the body, including the kidneys, lungs, liver, pancreas, and brain (Anaya, 2014). Furthermore, patients with SS are nine times more at risk for non-Hodgkin lymphoma associated with bronchial lymphoid tissue due to dysregulation of the immune system (Brito-Zerón et al., 2016). SS classification is divided into primary patients or patients without any potentially associated disease and polyautoimmune patients, where more than one autoimmune disease is found in addition to SS (Anaya et al., 2016). SS mainly affects the salivary glands and affects salivary flow.

Abbreviations: SS, Sjogren's Syndrome; pSS, primary Sjogren's syndrome; vWFA, von Willebrand Factor type A.

Microbiota–host interactions can introduce epigenetic changes and even changes in genetic functioning through methylations in DNA or microRNA, producing an immune reaction. This is the case of the molecular mimicry mechanism, by which some microorganisms carry antigens structurally like host cells that activate autoreactive B and T cells (de Paiva et al., 2016). Therefore, by regulating the dysbiosis of the host microorganism community and studying the pathophysiology pathogenesis involving the relationship of the oral microbiome and the generation of SS, early diagnosis and possible personalized treatment could be facilitated (Nikitakis et al., 2017).

The role of microorganisms in the development of autoimmune diseases is still unclear. Dysbiosis of the oral microbiome has been described in autoimmune diseases compared to controls. Some studies directly relate the decreased salivary flow to the community of microorganisms present in Sjogren's syndrome (van der Meulen et al., 2018). In the case of rheumatoid arthritis, there is a higher concentration of potentially proinflammatory bacteria *Porphyromonas gingivalis* and *Prevotella*, which often affects the periodontal status of patients suffering from it (Kroese et al., 2021; Scofield, 2014). However, it does not seem specific enough to establish a characteristic microbiome, given the diversity of clinical manifestations, variable rates of salivary secretion, and influential risk factors in patients with autoimmune diseases (van der Meulen et al., 2018). This highlights the need for longitudinal studies that relate the microbiota–host interaction in disease development.

In the oral cavity, different oral zones or niches can be distinguished with certain characteristics such as pH, oxygen, temperature, redox potential, hydration, and host immunological factors (Knight et al., 2017). In these areas, communities of microorganisms specifically adapted to these tissues can be found, forming an oral "biomap" (Mark Welch et al., 2020). Since oral tissues may be compromised in patients with SS, ecological niches located in the oral cavity could also affect oral microbial inhabitants. It should be noted that the decrease in secretion in turn affects the mucous membranes of the oral niche of the gingiva, buccal mucosa, or tongue. For example, the decrease in salivary flow in SS has an impact on the distribution of microorganisms in dental plaque (Proctor et al., 2018). However, the study of the SS microbiome beyond saliva or dental plaque has not been addressed. Therefore, the identification of a microbial community different from that found in healthy patients could identify biomarkers associated with the disease.

2 | AUTOIMMUNE DISEASES OF THE MOUTH: SJOGREN'S SYNDROME

Sjogren's syndrome is part of rheumatic autoimmune diseases characterized by oral and ocular dryness. It is considered the second most common autoimmune disease, preceded by rheumatoid arthritis (Bowman, 2018). It affects mainly women, in a 9:1 ratio with an estimated prevalence of 116.72 per 100,000 women and 5.53 per 100,000 men. It is diagnosed in women between the ages of 30 and 50, although it can appear at any age, affecting people over 60 years of age mainly and being rarer in children (Fernández Castro et al., 2016). SS can manifest in a healthy person with no other comorbidities, known as primary Sjogren's syndrome (pSS). However, the disease can be associated with other autoimmune diseases such as rheumatoid arthritis or lupus erythematosus, classified as polyautoimmunity (Anaya et al., 2016; Brito-Zerón et al., 2016).

The salivary and lacrimal glands are the main targets of this pathology, although they can affect any exocrine gland. Taking into account its intraoral manifestations, SS could cause decreased salivary secretion with symptoms of dry mouth. Other oral signs and symptoms in the patient with SS could include taste disturbance, difficulty swallowing, salivary gland edema, parotid and submandibular salivary gland enlargement, caries at the gingival margin, angular cheilitis, dry oral mucosa (Bowman, 2018; Serrano et al., 2020) (Figure 1a). Extraglandular systemic manifestations found in 20%–60% of individuals with SS that increase comorbidity and worsen the quality of life of these patients (Ramos-Casals et al., 2015). Furthermore, patients with SS have an increased risk of lymphoma associated with bronchial lymphoid tissue due to the present dysregulation of the immune system (Brito-Zerón et al., 2016; Yang et al., 2023).

Factors that contribute to the onset and development of the disease are related to the host and the environment, in addition to genetic factors (Bowman, 2018). Some epidemiological studies reveal that approximately 35% of SS patients have family members with the syndrome or another autoimmune disease (Agmon-Levin et al., 2017; Anaya, 2014). Genetic factors associated with SS are specific subsets of HLA-DR alleles and specific genetic polymorphisms, including STAT4, IL-12A, TNIP1, IRF5, BLK, and CXCR5 (Agmon-Levin et al., 2017). Genetic mutations found in these patients have been proposed to be responsible for modifying disease susceptibility genes by indirectly altering their expression, known as epigenetics (Bowman, 2018).

In SS, many epigenetic mechanisms are altered, such as DNA demethylation, abnormal microRNA expression, and abnormal positioning of chromatin related to antibody production (Agmon-Levin et al., 2017). In addition to genetic predisposition, several environmental factors such as pathogenic microorganism infections or smoking have been described (Vivino, 2017). SS patients have a more frequent colonization of the fungal species *Candida albicans* in the oral mucosa or the appearance of cervical caries due to overpopulation of the bacterial genus *Lactobacillus* on the surface of the tooth (Alam et al., 2020). In contrast, there is no high incidence of cases of patients with SS with periodontitis, suggesting that the proportions of microorganisms within the oral microbiota in patients with SS are determined and different from the physiological or that found in other pathologies (van der Meulen et al., 2016).

3 | THE ORAL MICROBIOME IN SJOGREN'S SYNDROME

In the oral cavity, more than 700 different bacterial species have been described. Furthermore, hundreds of fungi and viruses are present under healthy conditions (Ghannoum et al., 2010; Rusthen et al., 2019). In disease, a change in diversity and abundance produces an

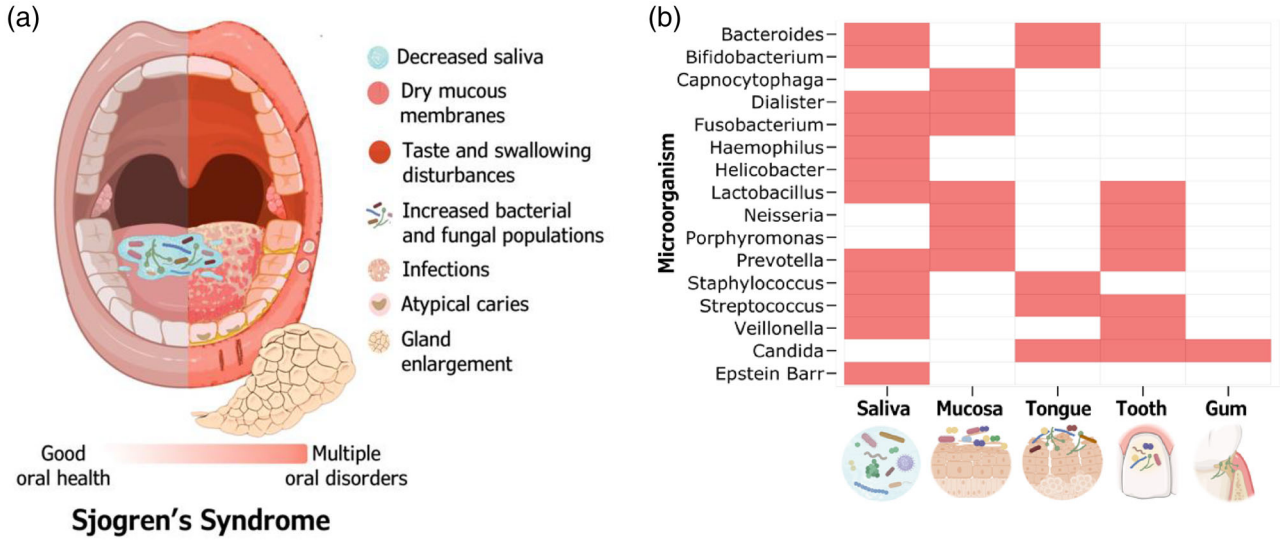

FIGURE 1 Oral clinical–microbial manifestations of Sjogren's syndrome. (a) Potential intraoral manifestations of Sjogren's syndrome with main oral disorders that might be found in patients. (b) Main microorganisms associated with oral niches in SS.

alteration in the community associated with physiological and histological changes in the host (Pride et al., 2012). The role of microorganisms in the development of autoimmune diseases, including SS, remains unclear. Dysbiosis, an imbalance in the oral microbiome, has been observed in SS-positive individuals compared to healthy controls. Some studies directly associate reduced salivary flow in SS with alterations in the oral microbial community (van der Meulen et al., 2018). However, other studies contradict this direct relationship, since they report similar microbial concentrations and populations in both SS and healthy individuals (Sembler-Møller et al., 2019).

Recent research has suggested that specific microorganisms can be associated with target tissues, with higher concentrations found in those tissues (de Paiva et al., 2016). The development of SS is not determined solely by the abundance or richness of the microbiota. The absence of certain bacteria, such as *H. parainfluenza*, has also been linked to disease (Tseng et al., 2021). Nevertheless, it appears that there is no characteristic microbiome specific to SS, considering the diverse clinical manifestations, variable salivary secretion rates, and influential risk factors among SS patients (van der Meulen et al., 2018). To better understand the interaction between the microbiota and the host in the development of SS, longitudinal studies are needed. These studies should investigate the microbiome–host relationship over time and consider the numerous factors that influence the disease. Such research efforts are crucial to understanding the complex interplay between microorganisms and SS and may contribute to better diagnosis and treatment strategies in the future.

In patients with Sjogren's syndrome, decreased salivary flow or xerostomia due to the involvement of salivary glandular tissue has various implications, including changes in buffer capacity, mineralization, and microbial clearance. The microbiome of SS patients shows an increased proportion of bacteria associated with dental caries, such as *S. mutans* and *Veillonella* (Siddiqui et al., 2016; Singh et al., 2021). Additionally, Veillonella, Actinomyces, and *S. aureus* bacteria are more prevalent in the oral microbiome of patients with SS. These microorganisms could acidify the oral environment through their metabolism, which can be problematic considering the reduced buffering capacity of saliva in SS (Siddiqui et al., 2016; Singh et al., 2021). These findings shed light on the oral symptomatology observed in SS. However, the exact relationship between these microorganisms and the development of SS is not yet clear, although they could potentially play a key role in the etiopathogenesis of the disease. More research is needed to fully understand the significance of these microorganisms in SS and their contribution to the disease process.

Controversial microbiome results have been associated with SS compared to controls. The most prevalent oral bacteria associated with SS have been included in the phyla *Bacillota*, *Actinomycetota*, *Bacteroidota*, *Fusobacteria*, and *Pseudomonadota*. Nonbacteria organisms associated with SS were fungi *Candida albicans* and Epstein–Barr virus (Yamazaki et al., 2005). The latter appears to participate in the activation and differentiation of autoreactive B cells, as was described in plasma cells of the ectopic lymphoid structures of the salivary glands (Talal et al., 1992). Furthermore, some peptides derived from the oral microbiota reported in the gum and mucosa suggest that they could induce an active immune response in autoreactive T Ro-60 cells related to the presence of *Prevotella disiens*, *Bacteroides intestinalis*, or *Bacteroides fragilis*, among others (De Luca & Shoenfeld, 2018; MacFarlane & Mason, 1974; Rusthen et al., 2019). However, the oral microbiome includes several niches; therefore, the microbial community can vary depending on the oral location disrupted by SS differently (Mark Welch et al., 2019).

Candida albicans, a fungus, is more abundant in the oral cavity of patients with SS. *C. albicans* is known as an opportunistic pathogen that can colonize oral surfaces in individuals with compromised immune systems (MacFarlane & Mason, 1974). In the case of SS, the involvement of the salivary glands, along with the dysfunction of the immune system, may create a favorable environment for colonization of this

TABLE 1 Microorganisms present with SS according to their location.

Microorganisms			
Genus	*Species*	Oral niche	References
Streptococcus	*mutans*	Tooth, tongue, saliva	(Proctor et al., 2018)
Staphylococcus		Tongue, saliva	(Alam et al., 2020; van der Meulen et al., 2018)
Lactobacillus	*salivarus*	Saliva, tooth, mucosa	(Alam et al., 2020; de Paiva et al., 2016; Proctor et al., 2018; van der Meulen et al., 2018)
Dialister		Saliva, mucosa	(Alam et al., 2020; Kuru et al., 2002; van der Meulen et al., 2018)
Prevotella	*melaninogenica* *histicola* *salivae*	Tooth, saliva, mucosa	(Alam et al., 2020; Kuru et al., 2002; Pride et al., 2012; Proctor et al., 2018; Rusthen et al., 2019; van der Meulen et al., 2018)
Neisseria		Tooth, mucosa	(Alam et al., 2020; de Paiva et al., 2016; Kuru et al., 2002; Proctor et al., 2018; van der Meulen et al., 2018)
Porphyromonas	*gingivalis*	Mucosa, tooth	(Kuru et al., 2002; Singh et al., 2021; Suárez et al., 2020)
Helicobacter	*pylori*	Saliva	(Hasni et al., 2011)
Veillonella	*rogosae* *parvula* *atypica*	Tooth, saliva	(Alam et al., 2020; Proctor et al., 2018; Siddiqui et al., 2016; Singh et al., 2021)
Bacteroides	*ceae*	Tongue, saliva	(de Paiva et al., 2016; Siddiqui et al., 2016)
Fusobacterium	*nucleatum*	Saliva, mucosa	(Kuru et al., 2002; Rusthen et al., 2019; Siddiqui et al., 2016)
Capnocytophaga		Mucosa	(Kuru et al., 2002; Li et al., 2016)
Bifidobacterium		Saliva, tongue	(Sharma et al., 2020)
Haemophilus	*parainfluenza*	Saliva	(Tseng et al., 2021)
Epstein Barr	–	Saliva	(Inoue et al., 2012; Talal et al., 1992)
Candida	*albicans*	Gum, tongue, tooth	(Baker et al., 2014; Leung et al., 2008)

fungus in the oral cavity. Maintaining good oral hygiene is crucial to prevent fungal colonization (Leung et al., 2008; Serrano et al., 2020). Furthermore, decreased salivary flow, combined with poor oral hygiene, has been associated with an increased prevalence of *C. albicans* in individuals with SS (Leung et al., 2007). To establish a clearer understanding of the relationship between *Candida* prevalence, oral hygiene, and colonization mechanisms in SS, longitudinal investigations with larger sample sizes would be necessary.

The microbial communities in the oral cavity can vary depending on the geographic location within the mouth. This suggests the existence of interdependencies, environmental factors, and functional relationships among microorganisms (Figure 1b, Table 1). For example, the presence of acidifying bacteria such as *Lactobacillus salivarius* in saliva may contribute to the colonization of *Streptococcus mutans* in the teeth and the development of cervical caries (Proctor & Shaalan, 2021). Therefore, interdependencies between microorganisms can affect the symptomatology experienced by people with SS. Understanding these microbial relationships could provide information on the onset and progression of autoimmune diseases and their associated symptoms. In particular, these interspecies relationships are that the subgingival microbial community, which occupies a distinct environment compared to other oral locations, remains unchanged in individuals with SS compared to healthy controls. This suggests that the observed codependent relationships among microorganisms may not significantly impact the microbial composition.

3.1 | Salivary microbiome

Saliva is an important factor in the development of oral biofilms, as decreased salivary flow results in a decrease in the availability of nutrients for the development of the microbiota, acidifying the medium, and losing its buffering capacity, which can lead to dysbiosis (Singh et al., 2021; van der Meulen et al., 2018). Because in Sjogren's syndrome salivary flow is diminished, certain case/control studies showed that the mean concentration of microbial DNA was lower in saliva from patients with SS compared to healthy patients (de Paiva et al., 2016). These results were highly variable, as SS is found in a wide range of saliva production, from moderately decreased salivary flow to no saliva at all, depending on salivary gland involvement (van der Meulen et al., 2018). Microbiological studies of saliva in SS with reduced salivary flow suggested that it could modify the composition of the microbiota, selecting acidogenic and aciduric species (Li et al., 2016). Other studies find relevant higher concentrations of *Veillonella* and *Streptococcus* in SS compared to non-SS patients (Kim et al., 2022; Siddiqui et al., 2016).

It is suggested that the appearance of *Veillonella*, *Streptococcus mutans*, *Lactobacillus lactobacilli* and the genus *Actinomyces* produces acidification of the oral cavity by their acidic metabolic products such as lactic and malic acid (Siddiqui et al., 2016; Singh et al., 2021). In addition, there could be co-dependence by the species *Veillonella parvula*, which is strictly anaerobic and cannot metabolize carbohydrates, so

its survival is linked to metabolic co-aggregation and complementation with *S. mutans*. The high concentrations of the latter species, in turn, could explain the high incidence of caries in pSS (Siddiqui et al., 2016; Singh et al., 2021). In contrast, a recent study found that dental caries and *Candida* carriage were independent of SS (Xing et al., 2023).

However, other factors associated with decreased salivary flow, such as medication, age, or genetics, could hide the direct association of the disease with dysbiosis. Most studies find a higher prevalence of the bacterial genera *Dialister*, *Lactobacillus*, *Fusobacterium*, *Helicobacter*, *Streptococcus*, *Veillonella*, *Pseudomonadota*, and *Neisseria* associated with SS patients (Alam et al., 2020; de Paiva et al., 2016; van der Meulen et al., 2018). Specifically, *Veillonella parvula* species showed higher concentration levels compared to healthy controls (Singh et al., 2021). The work of Tseng et al. (2021) showed that *Haemophilus parainfluenzae* in SS was significantly lower than in controls, and this absence was strongly associated with the development of autoimmune and chronic inflammatory diseases given the modulating capacity of the species in antigen-presenting cells. A notable feature of the salivary bacterial population associated with SS is the enrichment of Gram-positive species that suggests a higher survival of cell wall bacteria in this arid oral environment (Siddiqui et al., 2016).

On the contrary, other studies denied this direct relationship with the disease, since their studies showed similar microbial populations in Sjogren patients and control (Sembler-Møller et al., 2019; Tseng et al., 2021). The same edges present in previously studied articles were highlighted (De Luca & Shoenfeld, 2018; MacFarlane & Mason, 1974; Rusthen et al., 2019). The heterogeneity in the bacterial composition in patients with SS may be due to the variety of different salivary secretions and concentrations (Doaré et al., 2021). This suggests that, due to the reduced salivary secretion rate compared to healthy patients, a substantial part of the bacterial taxa is more abundant in the saliva of pSS than in controls. Therefore, the correlations between the bacterial phyla were weak or moderate, without excluding the influence of host-specific factors (medication, habits, and genetics) (van der Meulen et al., 2018). This correlation between salivary flow and bacterial abundance does not appear to occur with all phyla. Most studies find a higher prevalence of bacteria *Bacillota*, specifically the genera *Lactobacillus* and *Dialister*, and *Neisseria* (van der Meulen et al., 2018).

3.2 | Microbiome in supragingival and subgingival plaque

Salivary glands are one of the main target organs in SS. They are distributed in different areas of the oral cavity and their secretions directly impact the surface of the teeth. Therefore, drainage from the salivary glands of the parotid glands affects the upper surfaces of the vestibular molars, while the sublingual glands affect the inner surfaces of the lower incisors (Leung et al., 2008). This fact leads to a unique microenvironment in these teeth that is not found in the rest of the arches. Therefore, the decrease in saliva, along with the presence of dental caries described in patients with SS, suggests an impact on the microbiota of supragingival plaque. Different species of five main genera were found to be distributed nonrandomly according to the type of tooth (Proctor et al., 2018). The study determined the biogeography of the different dental groups (differentiated into molar and incisor groups) with respect to the microbiota of patients with SS, observing different taxonomic compositions (Proctor et al., 2018). The bacterial species *Streptococcus mutans* and *Lactobacillus salivarus* were found in high concentrations and would explain the high incidence of cervical caries and the decrease in the pH of the oral environment (Alam et al., 2020). Although there is a discrepancy between different studies regarding the abundance and phyla since this seems to be related to the oral hygiene factor of each patient (Leung et al., 2008).

The oral niches found on the tooth and gingiva corresponding to supragingival and subgingival plaque could vary in patients with SS depending on whether the environment is anaerobic or aerobic (Mark Welch et al., 2020). Singh et al. demonstrated a higher prevalence of *Veillonella parvula* species in supragingival samples in Sjogren patients versus healthy controls, but surprisingly, the subgingival flora was very similar, with low concentrations in *Tannerella forsythia*, *Porphyromonas gingivalis*, *Treponema denticola*, and *Treponema socranskii*. This may be because most of the bacteria present here are anaerobic and would not be influenced by the fluids and nutrients present in saliva, so the subgingival community may remain unchanged (Clancy et al., 2020; Singh et al., 2021).

In institutionalized elderly individuals with SS, who had poor oral hygiene and xerostomia in 19% of cases, it was shown that following a 3-month oral hygiene program, the prevalence of *Candida albicans* was significantly reduced. The findings demonstrated the relationship between patient habits and *Candida* prevalence and the possibility of reducing concentration in the mouth with good hygiene (Leung et al., 2008). However, the SS patients in the study by Leung et al. had good oral hygiene with up to 90% of the teeth without visible supragingival plaque, but the prevalence of *Candida* colonization was 50% of cases compared to 7% of controls with similar levels of oral hygiene (Leung et al., 2007). This suggests that other etiologic factors in addition to dental plaque can increase the risk of colonization with *C. albicans*, such as saliva volume and composition in patients with SS.

On the other hand, no increase in the abundance of *Porphyromonas gingivalis*, a bacterium associated with periodontal disease, has been found (Lugonja et al., 2016). This would explain the low risk of periodontitis in SS patients (Çelenligil et al., 1998; de Paiva et al., 2016). In several studies, lower levels of P*orphyromonas gingivalis* were found in pSS cases than in healthy controls (Siddiqui et al., 2016). In a study of the periodontium of pSS, periodontal pathogens such as *Aggregatibacter actinomycetemcomitans*, *Fusobacterium nucleatum*, *Prevotella intermedia*, *Treponema denticola*, *Porphyromonas gingivalis*, *Eikenella corrodens*, *Campylobacter rectus*, and *Bacteroides forsythus* were found, but at lower concentrations than in healthy controls (Kuru et al., 2002). The truth is that not many studies have been described addressing the microbiota in oral soft tissues in patients with SS, so the results are not clear.

3.3 | Buccal mucosa and tongue microbiomes

Patients with SS suffer from oral dryness, thinning, and epithelial desquamation of the oral mucosa, glandular edema, and acidification of oral pH that could affect microorganism colonization in oral tissues (van der Meulen et al., 2018). The diversity of microbial species has been described in patients with SS, which could be due to histological changes in these patients (Li et al., 2016). It may in turn be due to decreased salivary flow, making it an adverse and unpalatable environment for bacterial colonization.

The tongue contains a high diversity of microorganisms organized in intimate interaction with host tissue (Wilbert et al., 2020). However, the community present on the tongue could be influenced by the decrease in salivary flow and oral hygiene of SS (Serrano et al., 2020; Sharma et al., 2020). The appearance of *Candida albicans* is closely related to a decrease in pH due to decreased oral salivary flow and the destruction of the salivary glands. Oral hygiene products, such as toothpaste and rinses, can compensate for the pH drop, which does not occur in these patients with poor oral hygiene. Serrano et al. (2020) associated pH as a risk factor for the appearance of *Candida albicans* in patients with SS longitudinally. These findings suggest an impact on the integrity of tongue tissues due to the lack of saliva that could promote colonization of pathogenic opportunistic microorganisms such as *Candida albicans*.

However, the bacterial community on the tongue can also be disturbed in SS. The relationship between the autoimmune process of SS by autoreactive T cells with the Ro60 peptide (derived from the SSA autoantigen) could be observed in an in vitro assay in which activation with a bacterial von Willebrand factor type A (vWFA) domain protein was achieved, present in different oral and intestinal bacteria, and produced an Antiro-60 antibody response (Agmon-Levin et al., 2017). vWFA is present in oral microorganisms with *Capnocytophaga ochracea* and *Bacteroides intestinalis*, and the number of bacteria increased in patients with SS (van der Meulen et al., 2016). Another study showed that *Capnocytophaga ochracea*-produced vWFA was the most potent activator of SSA (Ro) and that reactive T cells in vitro were activated in its presence (Singh et al., 2021). With these results in mind, the association of the molecular mimicry mechanisms of microbial bacteria in the development of SS could be promoted.

4 | CONCLUSION

In conclusion, this study highlights the existence of a relationship between the oral microbiome and Sjogren's syndrome, as well as its role in the development of specific symptoms associated with the disease. Understanding the involvement of microorganisms and the mechanisms they employ for cellular invasion and disease progression is crucial to comprehending the etiopathogenesis of Sjogren's syndrome. However, the current body of evidence lacks sufficient clinical trials to establish a clear relationship between Sjogren's syndrome and the oral microbiome. Therefore, future studies in the field of microbiology are needed to further investigate this relatively understudied area and shed light on the clinical profile and niche-located microbiome in SS.

The association between oral microbial "biomap" in Sjogren's syndrome provides new insight into the potential role of commensal bacteria in the etiology of autoimmune diseases. Although more research is needed to understand the exact mechanisms involved, current evidence suggests that an individual's oral microbiome can strongly influence the development of autoimmune pathology. By better understanding the interaction between bacteria and host-associated risk factors, we can help identify those at risk of developing autoimmune diseases and develop effective treatments.

AUTHOR CONTRIBUTIONS

LB-L, AS-S: conception and design of the study. CS, MJR, AS-S: drafting of the manuscript. All authors critically reviewed the manuscript and gave their final approval of the version to be published.

ACKNOWLEDGMENTS

We thank the Spanish Association of Sjogren's Syndrome (AESS) and the Andalusian Association of Sjogren's Syndrome (AASS). Cartoons in figures were created with BioRender.com. This work was supported by the Spanish Ministry of Science and Innovation Grant ID PID2020-118557GA-I00 and the Plan Propio of the University of Seville Grant ID 2021/00001194.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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How to cite this article: Bustos-Lobato, L., Rus, M. J., Saúco, C., & Simon-Soro, A. (2023). Oral microbial *biomap* in the drought environment: Sjogren's syndrome. *Molecular Oral Microbiology*, *38*, 400–407. https://doi.org/10.1111/omi.12435