Dentistry: Advanced Research

Review Article

Denture Plaque Management of Denture-Related Stomatitis

Pierre Le Bars1*, Fabienne Jordana1, Alain Kouadio2

1Faculty of Dentistry, University of Nantes - University Hospital of Nantes, 1 Place Alexis Ricordeau, Nantes Cedex 1, France
2Faculty of Dentistry, University Félix Houphouët Boigny, Cote d’Ivoire; Faculty of Dentistry, University of Nantes, 1 Place Alexis Ricordeau, Nantes Cedex 1, France

*Corresponding author: Pierre Le Bars, Faculty of Dentistry, University of Nantes - University Hospital of Nantes, 1 Place Alexis Ricordeau, BP 84215 Nantes Cedex 1, France

Citation: Le Bars P, Jordana F, Kouadio A (2021) Denture Plaque Management of Denture-Related Stomatitis. Dent Adv Res 6: 173. DOI: 10.29011/2574-7347.100073

Received Date: 07 January, 2021; Accepted Date: 27 January, 2021; Published Date: 01 February, 2021

Abstract

The regular use of a removable prosthesis, in particular in fully edentulous aging subjects, constitutes, within the oral cavity, support for Denture Plaque (DP), promoting the growth of a candidosic population that often leads to Denture Stomatitis (DS).

This pathology is an infectious disease of multifactorial etiology. The purpose of this article is to review management approaches for DS at different levels: (1) lifestyle, (2) drug addiction, and (3) several general diseases such as diabetes mellitus, cardiovascular, respiratory and digestive diseases, and immune deficiencies that favor the development of DS.

Moreover, caries, periodontal diseases, a removable prosthesis that is poorly adapted or worn continuously, poor oral hygiene, and reduced salivary flow can promote Candida’s ability to colonize removable dentures. In the presence of a removable prosthesis, these parameters can influence homeostasis between the host and yeast, promoting the transition of Candida spp. from commensal to pathogenic.

The objectives of this review are, therefore, to analyze the influence of these different parameters on the balance of DP in oral ecology, and to explore the mechanisms and means for limiting its drift toward dysbiosis.

Keywords: Bacteria; Candida albicans; Dental plaque biofilm; Denture management; Denture hygiene; Denture-related stomatitis; Microbiome; Oral hygiene; Prevention; Systemic disorders

Introduction

A removable denture residing in the oral cavity being bathed in saliva is the ideal platform for dynamic Denture Plaque (DP) development [1-3]. The planktonic microbiota (bacteria, archaea, viruses and eukaryotic organisms) exposed to stress and flow can quickly create favorable conditions for DP growth [4-7] (Figure 1). This biofilm is defined as a community of over $10^{11}$ microorganisms per gram of dry weight [1,8,9] attached to the extrados and intrados of the denture surface and surrounded by an Extracellular Matrix (ECM) produced by the bacteria and Candida themselves [10,11]. This matrix is composed of macromolecules such as exopolysaccharides, proteins, and DNA [12], confers structural integrity to the biofilm, and constitutes a physical barrier that can be impenetrable to drugs [13-16].

Figure 1: Relationship between dentures and different environments and conditions.
Moreover, under the denture, the contact between soft living tissue and an inert material provides another favorable environment within the oral cavity for microbial colonization [5,17-19]. This reduced space beneath the dentures leads to a decrease in oxygenation, salivary flow, and pH that promotes the activity of Secreted Aspartyl Proteinases (SAP) from the matrix, which play a central role in Candida pathogenicity [20]. It seems that C. albicans biofilm maturation occurs in the same stages but more slowly (Figure 2) than bacterial biofilm (Figure 3). The presence of hyphae and pseudohyphae is the primary difference between the two biofilms. Recent targeted studies have explained the initial adhesion to the solid surface, secondly the subsequent development of mature biofilms [21], thirdly the formation of the extracellular matrix, and finally the mechanism of dispersion and identified numerous gene products required for each of these steps [9,22-24] (Figure 4).

**Figure 2:** Three chronological stages of biofilm formation by Candida albicans, highlighting the ability to exhibit different cell morphologies and the capacity to produce an extracellular matrix (271) [25]. Recent targeted studies have explained the initial adhesion to the solid surface, secondly the subsequent development of mature biofilms, thirdly the formation of the extracellular matrix and finally the mechanism of dispersion and identified numerous gene products required for each of these steps [26-28].

**Figure 3:** The four chronological stages of the onset of maturation and the decline of the microbial cycle of a biofilm on the abiotic surface of a removable prosthesis: stage I, attachment; stage II, growth; stage III, stationary; stage IV, death.

**Figure 4:** Biofilm envelops the dentures. Biofilm formation proceeds in distinct stages. First stage: adhesion; second stage: early development; third stage: mature biofilm; fourth stage: dispersal mechanisms. In the transition from the planktonic, free-floating state to the sessile state, attached microorganisms begin radically changing their gene and protein expression profiles [29].

The presence of a removable prosthesis in the oral cavity of 15% to 70% of patients is the cause of Denture Stomatitis (DS) [5]. This pathology is characterized by an imbalance of the microbial flora or dysbiosis resulting in, simultaneously:

1) The abundance of opportunistic pathogens like C. albicans [30,31];

2) The differential proliferation of certain bacterial species determined using Next-Generation Sequencing (NGS) [32-41] (Table 1). For example, natural water Pseudomonas fluorescens colonize the palatal mucosa of patients with DS with an abundance of 20% compared with only 2.3% in their non-DS counterparts [32];

3) A decrease in microbial diversity [33-35].
| Authors | Denture wearer with DS or NoDS | NGS : Platform of 16S r RNA gene pyrosequencing | Hypervariable regions of the 16S rRNA genes | Samples | Diversity and stability of microbiome | Genus level | Specie level |
|---------|--------------------------------|---------------------------------------------|------------------------------------------|---------|--------------------------------------|-------------|-------------|
| Shi B et al. 2016. [41] | 9 DS versus 10 NoDS. All with a complete removable denture and with a minimum of four remaining teeth. (China) | 454 FLX titanium platform. Detection of Candida via PCR. | V1-V3 26 genus (25 on the denture, and 24 on the teeth) 136 species / phylotypes | 2 samples - denture - teeth For the 2 groups with DS and No DS. | Data show, stability and similarity of the microbiome on the denture and on the teeth of the same person. Significantly difference exist between DS and NoDS at the species level. | Genus level the microbiota is similar on the denture and on the teeth with or without DS. | Fusobacterium nucleatum subp animalis is always present on the intrados of the denture with DS. |
| 0’ Donnell 2015 [33] | 123 denture wearers with partial or complete maxillary denture and at least one tooth. (Glasgow) 45 DS et 78 No DS | Illumina Miseq detection of Candida by culture 72% prevalence of Candida species on dentures. At the denture and mucosal microbiome, there was a positive correlation with the class Bacilli and a negative correlation with Fusobacteria at the denture. | V4 OTU, Genus uniquely | Samples on the mucosa, on the denture and the mucosa supragingival. | Patients with teeths shown microbiota diversity > Edentulous patients. No difference between microbiota of the denture and the teeth. Shannon index. | Precence of natural teeths with periodontal diseases can increase DS more than an totally edentulous patients. | Not specified |
| Campos et al. 2008 [19] | 10 healthy denture wearers/ 10 denture wearers with denture stomatitis (United States of America) | Culture- Cloning and sequencing And detection of Candida by ITS | Genus only | Samples on the denture - | 32 phylotypes on the denture | DS with the genera Atopobium (16%) and Prevotella (11%), both of which fall into the classes Actinobacteria and Bacteroidia, respectively - | Not specified |
Clinically, there are three main types of DS: (I) pinpoint erythema, (II) diffuse erythema, and (III) granular-type inflammatory papillary hyperplasia of the soft palate in close contact with the denture surface [36]. The severity of DS is linked to the mixed Candida species biofilm, which confers an approximate five-fold increased risk of severe disease (Newton’s type III DS) compared with denture wearers colonized solely by C. albicans (Newton’s type I DS) [37]. Notably, C. albicans is a frequent commensal colonizer of the oral mucosa, susceptible to highly adaptable microbial species and capable of causing infection at various anatomical sites [38]. In this way, mature natural and denture teeth biofilms have similar total numbers of bacteria but different proportions of species, especially Candida spp. [34]. Furthermore, the bacterial communities residing on the teeth and dentures of a single person are similar to each other independent of the surface material; therefore, denture health could impact the maintenance of the remaining teeth and vice-versa [6].

The epidemiology of the removable prosthesis wearers, while improving the health and quality of life of edentulous patients, corresponds primarily to elderly people; for example, 70% of UK adults older than 75 years wear dentures [39]. Under healthy conditions, these DPs are tolerated by patients [40]. However, in nearly three-quarters of patients, dentures are associated with stomatitis [31,36,41,42]. DS is considered a polymicrobial biofilm-mediated oral disease that is more common among elderly denture wearers and particularly among women [5,43]. A particularity of denture-induced stomatitis is the constant increase of C. albicans with the severity of the disease [44-51]. The etiology of DS is multifactorial, depending on age, lifestyle, and the use of tobacco and alcohol. It is reciprocally influenced by the presence of diabetes and cardiovascular, pulmonary, and digestive diseases. Locally, the lack of good hygiene practices and prosthetic trauma contribute to an environment favorable to the proliferation of C. albicans [52-55]. Generally speaking, wearers of prostheses suffering from candidiasis may present various asymptomatic signs such as a sensation perceived as burning, discomfort, difficulty swallowing, and a change in taste. However, elderly immunocompromised patients present Candida species in the oral cavity that can colonize the upper aero-digestive tract, causing sepsis, requiring hospitalization in 40 to 79% of cases [52,56]. Therefore, it is important for dental surgeons to be aware of the additional risk incurred by these patients and to promptly diagnose and treat oral thrush in the elderly with removable dentures. Although DP cannot be eradicated, it can be well-controlled by oral hygiene practices that include a daily regimen of brushing the mucosa and denture, followed by rinsing with an antiseptic mouthwash (MoW) [57-59]. Maintaining a healthy state is preferable for preventing the transition from harmless commensal to pathogenic. An effective oral hygiene regimen can help control DP biofilm formation and is a practical approach to preventing DS; in addition, it accrues benefits in certain systemic diseases [60]. The objective of this review is to understand, in the user of a removable prosthesis, the role that the host factors (general and local) played in maintaining a healthy oral ecology. Then grasp the current mechanisms and means that limit the drift of the oral microbiota toward dysbiosis. To do this, this article first presents the circumstances and mechanisms underlying the oral microbiota’s shift towards dysbiosis, followed by current approaches to limiting this pathological drift.

Table 1: Analysis of denture-related stomatitis microbiota with Next-Generation Sequencing (NGS); DS: Denture Stomatitis. PCR: Polymerase Chain Reaction. ITS: Internal Transcribed Space. OTU: Operational Taxonomic Unit.

| Morse D J. et al. 2019. [32] | 8 DS versus 11 NoDS Complete unimaxillary prosthesis (England) | Illumina Miseq + detection of Candida by culture and by PCR. | V1-V3 Genus and species | 3 samples by patient (tongue, palate, intradors of the denture) | Decrease of the OTU and the biodiversity of microbiota of the tongue with DS versus the healthy of palate mucosa. | At the level of the genus the microbiota is similarity on the palate mucosa and on the intradors of the denture with and without DS..(Shannon index) | Pseudomonas fluoressens is present (7%) on the intradors of the denture with DS (Pseudomonas fluoressens is most commonly found in natural water systems). |
|---|---|---|---|---|---|---|---|

Material and Methods

A literature search for articles published through August 31, 2020, was performed using the following keywords: “Denture plaque,” “Removable prosthesis,” “Influence factor,” “Removable denture,” “Interactions,” “Denture stomatitis,” “Denture biofilm,” “General pathologies,” “Microbiome,” “Denture management,” “Denture cleaning,” “Hygiene protocols,” “Treatment of denture-related stomatitis,” “Systemic disorders,” “Oral Hygiene,” “Prevention,” and “Microbiota.” Combinations of the keywords were also used. A total of 3700 articles were extracted from PubMed/MEDLINE. Article titles and abstracts were examined to exclude irrelevant and previously identified articles. Then, the abstracts of the selected articles were read to identify studies that met the inclusion criteria. Finally, additional articles were obtained.
by reviewing the references of the selected articles. Original articles written in English were included in this review if they met one of the following criteria:

**Inclusion criteria**

- Articles on the relationship of the denture with different environments and conditions
- Articles on the relationship between a removable denture and some species
- Articles on the relationship between a removable denture and some systemic disorders
- Articles on the biofilms under and above the removable denture
- Articles on the characterization the DP biofilms
- Articles on the denture wearer’s management

**Exclusion criteria**

- Clinical case reports
- Articles written in languages other than English
- Articles on the study of dentures other than the removable denture

The articles were divided into five thematic categories for this review.

The following themes were analyzed:

- Lifestyle
- General pathologies
- Local pathologies
- Maladaptive removable denture and DS
- Hygienic maintenance procedures
- Improvement of an effective solution against DP

The selection procedure for articles included in the literature review (Figure 5).

**Figure 5:** Selection procedure for articles included in the literature review.

**Results and Discussion**

**Denture plaque in oral environments**

**Remarks**

During the use of a removable denture, the DP is subjected to many mechanical and chemical constraints such as variations of the composition and flow of saliva, a variety of food toughness, temperature fluctuations, masticatory forces, and appliance loading [61,62]. Poor oral hygiene is frequently observed in denture wearers, which increases the microbial load of DP [63,34]. In this situation, the presence of the denture establishes an acidic salivary pH [65]; smoking, sugar consumption, commensal oral *Candida*, age of the denture, and choice of cleanliness influence DS [66].

Patients and denture hygiene control may present small variations of several microbial genera, which can initiate dysbiosis. Thus, depending on the site, *Scardovia* and *Lactobacillus* are found in significantly higher abundance on the dental surface, while *Fusobacterium* and *Schwartzia* are found on the surface of dental prostheses [67]. These same authors studied the influence of *Candida* on the biofilm of prostheses, mucous membranes, and teeth. They note that the candidosic load does not influence bacterial diversity, but can, on the contrary, impact their relative abundance. Thus, this incriminates the presence of *Candida* within the biofilm in terms of abundance rather than hygiene. An illustration of this phenomenon is provided by the relationship between the *Lactobacillus* species and the *Candida* load. Depending on the load of *Candida* and the location, there is a shift from antagonism (small load) to synergism (large load) between *Candida* and *Lactobacillus* [68,69]. The design of the biofilm brings together bacterial and fungal species, which illustrates the scaffolding role played by *Candida* [70] (Figure 6). The bacteria using this support for their colonization are more abundant [71] (Figure 7). Therefore, any hygienic strategy must be both antifungal and antibacterial. Clinically, good oral hygiene practices aim to stabilize and maintain a balance over time in the oral microbiome, without seeking to modify it. The development of an individualized strategy concerning biofilm formation based on the interactions between the fungal and bacterial domains must be considered.
Denture plaque management and treatment

Generalities

The prosthetic base acts as a support for the oral microbiota at the epithelial surface and is externally in contact with the planktonic flora. With time, some organisms can penetrate the resin [7,15,85]. The maxillary and mandibular mucosa, alternatively in contact with the removable prosthesis during the day and then released at night, represents an ecosystem. Under these conditions, the complexity of the denture-associated biofilm increases, with contact between the layer of epithelial cells and the intrados of the denture base (metallic or plastic biomaterials) (Figure 8). These two dynamic biofilms coexist for hours every day and are separated at night, following recommendations regarding the wearing and cleaning of removable prostheses [67,86-91]. The treatment differs between DP maintenance (essentially preventive) and DS treatment, in which an effective curative solution might be applied. The difficulty is relevant to ascertaining the differences between DP formed by commensal microorganisms and DS including pathogens that increase the pathogenesis. Common clinical examination and treatment based exclusively on Newton mucosal classification are inadequate [19]. Maintaining C. albicans at a tolerable level in denture wearers involves the interaction with commensal bacterial flora and host components [92]. Today, DS treatment and DP maintenance must be viewed from a holistic, global perspective and consider:

1- Lifestyle,
2- Tooth loss and general pathologies,
3- Local pathologies,
4- Removable dentures and DS, rehabilitation, and renewal,
5- Hygienic maintenance and medication,
6- Effective solutions for DP.

Figure 7: Schematic reproduction of a scanning electron micrograph of mixed C. albicans hyphal (green) and S. aureus (blue) cells.

Prolonged removable denture wearing day and night, with or without hygiene defects, contributes to DP development [72]. Additionally, at least 40% of elderly denture wearers do not adequately disinfect or remove their dentures at night (68). Under this condition, the risk of pneumonia [73,74] and cardiovascular diseases [75] doubles. Moreover, systemic factors are associated with the appearance of DS including deficient immune response [76], vitamin deficiency [77,78], diabetes [79], and the consumption of immunosuppressors [80], antibiotics [81], or addictive drugs [4,79,80,82-84]. However, locally, other parameters are involved in DP microbial composition, such as tooth extraction, sampling times during the day, oral health status, prosthetic restorations, dental and periodontal diseases, and dental treatment [61,62].

Figure 8: Schematic view of denture-induced stomatitis. Mature salivary conditioning film between biomaterial surfaces (denture base resin) is in contact with the mucosa. Bacterial species most frequently isolated with Candida albicans from these specific niches of the oral cavity include Streptococcus spp.: S. gordonii, S. mutans, and S. salivaris. Saliva, moisture, nutrient availability, hyphal Candida morphotype, and the presence of commensal bacteria influence the architecture and virulence characteristics of mucosal fungal biofilms.
1. Lifestyle and denture wearers in the world

The Horizon 2050 suggests that there will be two billion elderly people worldwide, which will likely correspond to a significant increase in the number of users of removable prostheses [93,94]. With great geographic diversity, this last segment of the population is often the victim of socio-economic deprivation, explaining the renunciation of an implant solution and the need to use a removable prosthesis [95]. Food habits vary around the world, but the use of removable dentures affects the choice of food and can influence a patient’s health [96]. In general, these patients tend to give up hard-to-chew foods such as stringy meats, vegetables, and raw fruits [97]. A high proportion of these people have DS [5,98] due mostly to poor oral and prosthetic hygiene [99]. The patient’s lifestyle, such as sleeping with their prostheses at night, impacts the oral microbiome. Patients who keep their removable prosthesis in at night have an abundantly unchanged dental microbiome but at the level of the mucous microbiome, one genus (Dialaster) is significantly increased. Moreover, concerning the prosthetic intradoss microbiome, several genera (Leptotrichia, Selenomonas, Moryella, and Prevotella) are significantly increased [67]. Only Prevotella increases in the presence of DS [41,99].

Around the world, the practice of prosthetic hygiene is often empirical. However, the treatment of prosthetic microbial plaque requires a hygienic methodology adapted to the complexity of the relationship between the microbiome and the oral mycobiome [29,41,66,67,100]. Much advice on the presence of prosthetic stomatitis is given to the patient. Smoking cessation in addition to the usual therapeutic measures is recommended [101-103]. Indeed, the adhesion of Streptococcus mutans and Candida albicans in the form of a biofilm to dental plaque is favored in cigarette smokers [104,105].

Drugs predisposing to oral candidiasis, such as broad-spectrum antibiotics and immunomodulatory and xerogenic drugs, must be identified. The strong interaction between bacteria and commensal yeasts confers resistance to the colonization of pathogenic microorganisms. The prescription of a broad-spectrum antibiotic in the long-term can disturb the relationship between the commensal bacterial and fungal flora [106,107]. The cessation of antibiotics promotes a return to typical levels of bacteria and can resolve candidiasis without further intervention [101,102,108]. Other aspects of a patient’s lifestyle and eating habits affect their defenses. For example, in the presence of high cholesterole content [109] or nutritional deficiencies (iron folate, vitamin C, vitamin B12, vitamin A), damage to the integrity of the oral mucosa can promote invasion and proliferation of Candidosic hyphae [8,101,110-114]. Another aspect of malnutrition is the increase in sugar consumption among prosthetic wearers. The consumption of sugar promotes the proliferation of fungi (Candida) on acrylic surfaces and the biofilm’s resistance to antifungals, thanks to the activity of phospholipase and increased production of the extracellular matrix [103]. Compared with glucose, fructose can inhibit C. albicans. Therefore, foods containing fructose can slow down the progression of candidiasis. The recommendation to consume fructose (xylitol) rather than glucose is particularly interesting for fitted patients with a biofilm containing Candida spp. [115].

2. Tooth loss and general pathologies

The quantitative and qualitative balance between microbes within the oral microbiota is fundamental to maintaining oral homeostasis; the presence of teeth influences this balance [116-118]. At the end of life, the oral microbiota of an elderly patient (> 65 y) who is toothless, fitted or not, is frequently associated with several general pathologies, such as ischemic heart disease [119], heart failure [119], stroke [120], peripheral vascular disease [121], and cancer [122,123]. Other characteristics, in the presence of a removable prosthesis, are the loss of teeth and the aging of patients, which accelerate the reduction of the function of Polymorphonuclear (PMN) neutrophils (oral and circulatory PMNs). This has the effect of promoting the appearance and maintenance of DS. Oral PMNs (salivary) are functionally impaired in edentulous participants; only the cPMN (circulatory) numbers were higher in the edentulous participants. This finding shows that this particular change of immunity in the oral cavities of edentulous patients leads to decreased resistance and, consequently, favors dysbiotic conditions like Candida infections [124]. Another characteristic site in the mouth is the surface of the mucous membrane of the tongue. It is the most populated microbial reservoir, carried by saliva; these microorganisms are capable of dispersing and colonizing other sites in the oral cavity [3,125].

Periodontal disease can spread microbes from periodontal infection via blood or saliva to target organs. Thus, a microbial relationship exists between several infectious diseases distant from the oral cavity and periodontal diseases, such as rheumatoid arthritis [126-128], cardiovascular disease [129,130], pneumonia, cystic fibrosis [131,132], hepatic or brain abscesses [133], endocarditis [134], and diseases of mental impairment [135]. Among these general diseases, some can interfere with the removable prosthesis if untreated for a long period.

21-General pathologies and DS

1-Systemic immune response on the DS:

Comparison of the profiles of lymphocytes and monocytes in the peripheral blood of two groups of elderly patients, one with DS (n = 20) associated with a fungal infection and the other free of DS (n = 24), does not show any significant difference. Only the number of CD 25+ T lymphocytes is significantly lower in DS patients. The researchers hypothesize that these aging patients with a hearing aid have a reduced potential to eliminate the infection [84]. Moreover, DS is notably more prevalent among HIV-infected female denture wearers compared to seronegative female denture wearers [136,137], and taking medication that can disrupt salivary secretion [138].
Immunosuppressive treatment in patients who have had a kidney transplant and removable dentures seems to increase the risk of *C. albicans* infection and, therefore, also of DS. *C. albicans* positive cultures of the oral mucosa were observed in 73.3% of transplant patients compared to 28% of the control group. Yeast was isolated from the surface of the denture in 83.3% of grafted patients compared to only 38% of the control group [139].

2-Cancer and DS:

A multivariate analysis of 390 sick patients, recruited from a hospice in England, identified the poor performance of the Eastern Cooperative Oncology Group. The presence of dentures, poor oral hygiene, and xerostomia is independently associated with oral thrush in community patients with advanced cancer [140]. Concerning patients with cancer receiving palliative care, the use of a removable denture in this context increases the risk of developing oral candidiasis. [136].

3-Cardiac risks influence endothelial function in older people with DS:

Comparing two groups of patients over the age of 64 years, one with DS and one without DS, revealed that the groups did not differ in ambulatory blood pressure, total cholesterol, and C-reactive protein. However, the dilation induced by blood flow was dramatically lower in patients with DS than in the control patients, whereas nitroglycerin-induced vaso-relaxation and their carotid intima-media thickness were similar. To summarize, DS is associated with endothelial dysfunction in terms of the aging of patients with removable dentures [109]. DS treatment can locally improve endothelial function assessed by flow-mediated vascular dilation [141-144].

4-Pulmonary infection, pneumonia, and dentures:

Lung infections are related to bacteria of oral origin by air [145,146]. Conversely, there are respiratory pathogens capable of colonizing the surface of removable prostheses [147]. Wearing prostheses at night promotes inflammation of the oral mucosa, increases the prosthetic microbial load, and can exacerbate pneumonia [73]. The establishment and maintenance of proper oral care in the elderly have been effective in preventing lung disease. In Japan, for two years, 163 elderly people living in institutions and benefiting from oral hygiene care and prosthetics, experienced a regression of pneumonia and consecutive deaths [148]. However, the assumption of the responsibility for oral care by the nursing staff is likely critical to effectively prevent pulmonary pathologies [149]. The removable prosthesis in the presence of poor oral hygiene can be colonized by microorganisms of periodontal disease and caries as well as those of respiratory origin [147]. Therefore, microbial sanitation of the oral cavity, especially in vulnerable patients, may decrease the risk of developing respiratory diseases [150]. Thus, the establishment of oral hygiene becomes necessary in patients at risk to ward off pulmonary infections [150,151]. Moreover, a study concerning fitted patients with pulmonary diseases with bacteria in the upper airways found that they had an increased frequency of chronic prosthetic stomatitis compared to healthy subjects [152].

5-The link between gastric *Helicobacter pylori* and denture-related inflammation:

Inflammation of the oral cavity caused by bacteria or fungi can be accompanied by gastric inflammation, particularly in the presence of DS [153,154]. *H. pylori* eradication from the oral cavity is more difficult than from the stomach. If the bacterium survives the antibacterial therapy in the oral cavity, it can re-infect the stomach in a few weeks. Oral health and hygiene practices seem unlikely to increase the efficacy of *H. pylori* eradication from the stomach. In contrast, long-term professional dental plaque control is associated with less gastric re-infection by *H. pylori*, suggesting that DP control may help to prevent *H. pylori*-induced gastric disease or re-infection. However, after antibiotic therapy, *H. pylori* in dental plaque may represent a risk factor for gastrointestinal re-infection and ulcer relapse [155-157].

6-The influence of glucosidic metabolic endocrine disorders (diabetes) on DS:

DS associated with *C. albicans* is notably more frequent and severe in denture wearers with insulin-dependent diabetes mellitus compared to denture wearers with balanced glucose metabolisms [158,159]. A large epidemiological study on oral health confirms that subjects with insulin-dependent diabetes mellitus and removable denture wearers are more likely to have clinical signs of DS than non-diabetic control subjects with removable dentures [160,161]. Diabetes mellitus is a major risk factor for DS [162-164]. In an elderly and diabetic patient, the presence of a removable denture in the oral cavity contributes to a favorable environment for candidiasis development [78].

7- Influence of drugs and medicines on DS:

The use of drugs with the presence of dentures greatly increases the likelihood that a person over 60 will have oral mucosal lesions [165]. Treatment with parenteral antibiotics has emerged as the overriding risk factor for oral candidiasis in the presence of a removable denture [81]. The use of oral antibiotics may be involved in the onset of DS [166]. When incorporated in a biofilm, *C. albicans* is more resistant to the antifungals used to treat DS (e.g., amphotericin B, nystatin, chlorhexidine, and fluconazole) [167] than when in its planktonic form.

8- Nutritional deficiency and prosthetic stomatitis:

Vitamins C, B12, and A deficiencies appear to reduce mucosal resistance to infection: Vitamin C deficiency in patients aged 82 years on average with removable denture increases their susceptibility to candidiasis [816]. The correlation between vitamin B12 deficiency and DS in the presence of strong *Candida* colonization is also established [77]. This vitamin B12 deficiency weakens the mucous membrane and promotes inflammation. A relationship has also been found between vitamin A deficiency and
the prevalence of DS [83]. In addition, this vitamin A deficiency is associated with a five-fold increased risk of having DS [168]. The restoration of these deficiencies is an integral part of the management of prosthetic stomatitis.

3. Local pathologies (caries, periodontal diseases) and DS

The level of candida appears to be a reliable indicator of microbial risk factors in caries [169,170]. This is the case for patients wearing overdentures or partial dentures, which increase the risk of caries [171]. Indeed, Candida albicans is highly acidogenic in vitro. Under this condition, DP-DS can cause the demineralization of the root or tooth1. A more recent study compares the microbiota of the fitting surface of dentures in edentulous subjects with healthy palates (n = 20) and the DP in patients with DS (n = 20). The proportions of S. mutans, Lactobacilli, Bifidobacteria, and yeast in the DP biofilm of subjects with DS were greater (p < 0.05) and similar to those found in carious lesions, indicating that this is a low-pH environment. The interdependence between prosthetic, dental and gingival biofilms via saliva implies overall oral hygiene. Controlling all of these biofilms can have beneficial consequences for both DS, caries and periodontal disease [41,172].

Periodontal diseases and denture wearers

Wearing overdentures, partial and complete dentures, is often associated with periodontal disease adjacent to the abutment teeth [173-175]. Naturally, C. albicans is an aerobic fungus that has been isolated in periodontal pockets in 15% to 17% of patients [176]. Moreover, anaerobic conditions within the periodontal pockets of patients with diabetes favor the phospholipase activity of C. albicans [177]. Furthermore, the amount of C. albicans is twenty-fold higher on the denture surfaces in DS versus non-DS (45). Under the denture, the decrease in pH caused by C. albicans relies on the production cytotoxic acetate, pyruvate, and propionate, which promote tissue damage [178,179]. Under these conditions, it is possible considering removable denture like a reservoir of micro-organisms capable with fluids (planktonic form) of diffusion in the oral cavity. The microbial communities residing on the denture and periodontal tissues of the same person are like each other, so denture health and periodontal diseases are subordinate [41].

4. Maladaptive removable denture and DS

Risk factors underlying DS must be identified and treated; accordingly, some prostheses need to be redone [180]. Clinical examination may reveal a defective removable prosthesis, which requires verifications of the vertical occlusion dimension [181], the level of the occlusal plane, and the position of the mandible relative to the base of the skull. All of these defects can cause prosthetic instability and worsen DS; in which case, prosthesis renewal is often necessary [182]. Another important factor is the age of the prosthesis. The accumulation of plaque increases with poor hygiene aggravated by the roughness of the surface of the prosthesis [110,114,183] moreover, this accumulation increases proportionally with the age of the prosthesis; beyond five years, 84% of patients have DS [184]. This justifies the need to regularly renew the removable prosthesis.

5. Procedure of maintenance for preventive strategies

First, inhibiting microbial adhesion, based on the fact that without initial adhesion we limit or slow down the development of DP. Three levels can be envisaged: 5.1-material optimization, 5.2-surface modifications, and 5.3-sequences for the maintenance of the denture.

5.1-Material optimization

Biofilm development on an acrylic denture increases the risk of DS five-fold compared with a metallic denture [185]. To prevent Candida infection in oral mucosa, some researchers have proposed using microbial molecules in the composition of denture materials [186-190]. Another domain of research involves nanoparticles. The effect of adding zirconia nanoparticles to cold-cured acrylic resin on C. albicans adhesion has been evaluated. Zirconium oxide nanoparticles possess antifungal properties against C. albicans and Aspergillus niger and could be used to prevent DS [191,192]. Among products of plant origin, incorporating neem powder (Azadirachta indica), which fights bacteria and fungi, into the acrylic base of the prostheses reduces the adhesion of C. albicans. So, this powder could be an effective means of preventing DS. While an in vitro assessment of the antifungal effects of neem powder added to polymethyl methacrylate denture base material has been published [193], these antimicrobial macromolecules require further in vivo testing before the products are brought to the market.

5.2-Surface modifications

Initially, to thwart the adherence of C. albicans, the dentures are polished and coated with hydrophilic materials to decrease the adhesion of C. albicans [5,194]. Candida and bacterial plaque adhesion to denture materials depend on both the surface roughness and contact angle. For the former, after polishing, the resin must be below 0.09 μm (clearly under the threshold of 0.2-μm surface roughness needed for plaque accumulation) [195,196]. The contact angle of a sample not coated in saliva is around 90°, while one with a salivary coating is reduced to 35° [197]. These two parameters indicate that a denture resin coated in saliva displays almost hydrophilic properties [198]. Recent research efforts consider these factors. For example, mannan, a hydrophilic polysaccharide coating on the acrylic surfaces of the denture base, inhibits the adhesion of C. albicans [199]. Specifically, overnight treatment with mannan (0.1 mg/mL) inhibits the adhesion of the hyphal form of C. albicans. Another polysaccharide chitosan with a similar process using, inhibit C. albicans adhesion, but also to slow down the formation and co-aggregation biofilm [199]. Otherwise, the salivary protein components in contact with the surface of different materials are directly transformed by adsorption, as seen in the pellicle. Therefore, it seems warranted to analyze the composition.
of the amount of salivary protein attached to each denture.

5.3- Sequences for the hygienic maintenance of the denture and the oral mucosa

The placement of the biofilm obeys different sequences that can affect the maintenance of the prosthesis. Effectively maintaining the oral health and hygiene of denture wearers requires a combination of mechanical and chemical measures [200,201].

Concerning the denture, the pathogen management process consists of meticulously brushing the prosthesis every day to reduce the pathogenic burden [202]. In concrete hygienic terms, the abiotic acrylic material acts as a reservoir for the *Candida* population near to the palatal tissue, with the consequence of inducing a local inflammatory response detectable clinically as erythema, edema, and hyperplasia [203,204]. The denture surface is colonized more by *C. albicans* than by the associated palatal mucosa; consequently, clinical treatment is essentially turned toward eliminating the biofilm on the denture. The goal is to avoid new colonization with relapse [91,205].

Hygienic oral preventive strategies are based on fundamental knowledge of the mechanisms involved in dental plaque formation concerning bacterial and fungal adherence. All denture wearers receive verbal and written instructions for effective hygiene protocols.

- First, the recommendation is to brush the palate region, dorsal tongue, and mucosa under the denture with a soft toothbrush and water for 2 min once per day.
- Second, the recommendation is to immerse the denture in a specific product once per day during the time proposed by the manufacturer and then brush for 2 min with a neutral soap three times per day. The use of antiseptics to inhibit or eliminate microorganisms and immersion in a chemical solution are recommended for 8 hours. Sodium hypochlorite, chlorhexidine digluconate, and alcohol disinfect or reduce the DP on prostheses [206, 207] without being cytotoxic [208].
- Third, the recommendation is to remove the denture at night and put it in a box.
- Fourth, the recommendation is to rinse the denture and put it back in the mouth every morning [86, 87, 143, 209, 210].

Curative strategy

In the presence of chronic prosthetic stomatitis, the practitioner may suspect a particular form of *Candida*, which is often confirmed by laboratory tests [102, 211]. Several methods are used to confirm the presence of *Candida* spp., such as a swab, imprint culture, whole saliva, and oral rinse [212]. The clinical diagnosis of chronic stomatitis supplemented by a culture swab (incubated for 48 hours with Sabouraud’s agar) was combined with a direct examination under the microscope [103]. Targeting the candidosic species is essential for rapid treatment [213]. The choice of antifungal drug depends on the patient’s general pathologies, the state of the oral cavity, and possible complications [102].

The three main classes of antifungals include polyenes (amphotericin B), azoles (fluconazole), and echinocandins (caspofungin) [214]. Amphotericin B is considered the “gold standard” of antifungal therapy but is toxic because there is no selectivity between fungal and mammalian cells [215]. However, fungal biofilms that mature on denture material become resistant to antifungals [216].

Treatment follows the chronology of *Candida* colonization

From the early stage of *C. albicans* biofilm formation, Chandra et al. showed in vitro that the minimum inhibitory concentrations (MICs) effectiveness of amphotericin B, fluconazole, nystatin, and chlorhexidine are 0.5, 1, 8, and 16 μg/mL, respectively, on polymethylmethacrylate strips [217]. These results suggest using these antifungal drugs during the early phase of biofilm formation related to adherence, instead of later [218].

Concerning the next stage, according to the co-aggregation of microorganisms, another mechanism is the involvement of antifungal drug resistance, linked to ECM surrounding the microorganisms in DS. In agreement, the extracellular β-1,3-glucan matrix attaches to amphotericin B, while its absence from ECM increases *C. albicans’* vulnerability to amphotericin B4, 15. Furthermore, chlorhexidine reduces the adhesion capacity of the *Candida* spp. on the denture surface [219].

Another strategy geared against the opportunistic *C. albicans* targets the two properties (filamentation and biofilm formation) of this pathogen [220]. Some small molecules (20,000 compounds from the NOV A Core library-quorum-sensing molecules) can modulate the switch from yeast to hypha form and biofilm formation of *C. albicans* [221]. Using a murine model *in vivo*, some of these compounds (SAP5, ECE1 [candidalysin], ALS3) have shown potential for efficacy with inhibitory activity against *C. albicans* biofilm formation, with no effect on overall growth [222, 223]. However, *in vivo*, the prosthetic biofilm exists only in a mixed “Bacteria-Candida” or mycofilm concept [70].

Treatment of mixed biofilms in DS

Other treatments of DS concern the mixed biofilms composed of multiple species, with different combinations of *Candida* and/or with *Candida* and bacteria, which interact as a community in synergistic and antagonistic relationships [224] (Tables 2 and 3). The pathogenicity of *C. albicans* increases in the presence of *S. mutans*, *S. sanguinis*, and *Actinomyces viscosus* [225], which justifies the use of antibacterial agents that could also serve to decrease fungal proliferation [189]. Thus, there is widespread use of recently developed therapies (probiotics) to prevent, disrupt, and otherwise render harmless the peculiar ability of *C. albicans* to form biofilms on almost any surface in the mouth.
**Table 2:** *C. albicans* interactions with oral *Streptococci* mutans Group, mitis Group (13 species), *Salivarius* Group, and *Staphylococcus aureus* in biofilm matrices *in vitro* and *in vivo.*
Interactions between…

| Porphyromonas Gingivalis (inhibe) | Actinomyces spp (favored) | Fusobacterium spp (commensalism) | Rothia dentocariosa (favored) | Aggregatibacter actinomycetemcomitans (inhibe) | Enterococcus faecalis (inhibe) |
|-----------------------------------|--------------------------|----------------------------------|-----------------------------|---------------------------------------------|----------------------------------|
| Candida spp: albicans, Kefyr, glabrata, dubliniensis, Tropicalis [238] | Detection of this periodontal pathogen in the edentulous subjects [3] | Biofilm growth on acrylic resin [243] | Co-aggregation between C. albicans and F. nucleatum [244] and with C. dubliniensis [245] | Biofilm formation on silicone rubber prostheses [246,247] | Detection of this periodontal pathogen, in the edentulous subjects [3] Aggregatibacter actinomycetemcomitans interacts with C. albicans [248] |

**Influenced by…**

| Hyphal-specific adhesion Als3 on the fungal surface [250] | Co-aggregation between C. albicans, A. viscosus, [241,192] A. (naeslundii) [225] A. odontyllicus, [251] (A. oris) [252,225] | Favored by arginine or mannose. [244] | Hyphal-specific adhesion Als3 play important role for the adhesion with Rothia dentocariosa [253] | A. actinomycetemcomitans produces a quorum-sensing molecule called autoinducer-2 (AI-2) significantly inhibited hypha formation of C. albicans [248] | E. faecalis can inhibit C. albicans filamentation, biofilm formation [254,255] |

**In multispecies**

| P. gingivalis can delay oral epithelium cell migration when interacting with C. kefyr and C. glabrata. [256] P. gingivalis in biofilm inhibit production C albicans hyphae [251] | Synergism in the triad mixed biofilms on denture materials (A. oris, S. oralis, C. albicans) [252] Increased C albicans hyphal production with Streptococcus sanguinis S gordoni Actinomyces , A viscosus biofilm [251] | Virulence of one-another (C. albicans and F. nucleatum) mutually may benefit from promoting long-term commensalism. [257] | R. dentocariosa, S. aureus, S. mitis, aided colonization of C. albicans and C. tropicalis on silicone rubber surface [247] | A. actinomycetemcomitans is able to inhibit biofilm formation by C. albicans in co-cultures; this effect is rescued using a luS mutant strain that cannot produce A1-2 [248] | Biofilms of C. albicans sap9Δ with S. oralis, S. sanguinis, S. parasanguinis, S. mutans and Enterococcus faecalis contained more matted hyphae and more bacteria bound to substratum compared to C. albicans wild type [258] |

Table 3: Interactions between Candida spp. and Porphyromonas gingivalis, Actinomyces spp., Fusobacterium spp., Rothia dentocariosa, Aggregatibacter actinomycetemcomitans, and Enterococcus.

**Probiotics**

Commercially available probiotics (Accuflora® and Culturelle®) that contain Lactobacillus species associated with mechanistic cleaning interfere with the in vitro ability of C. albicans to form biofilms on dentures [229,230]. Through the phenomenon of co-aggregation, the lactobacilli may secrete an adequate mass to be able to maintain a hostile micro-environment around Candida species through high concentrations of acids, H2O2, and bacteriocins, thereby possibly inhibiting the pathogen’s growth. Daily use of probiotic lozenges may reduce the prevalence of high oral Candida counts in elderly nursing home residents [231,259]. Recently, a probiotic, the bacterium Lactobacillus reuteri (DSM 17938 and ATCC PTA 5289) was tested against six oral Candida species (C. albicans, C. glabrata, C. krusei, C. tropicalis, C. dubliniensis, and C. parapsilosis) for the ability to co-aggregate and inhibit the growth of the yeasts. The Lactobacilli almost completely inhibited the growth of C. albicans and C. parapsilosis but did not affect C. krusei, which is known
to resist the acids produced by the *Lactobacilli* [63,260]. The effectiveness of another probiotic *Lactobacillus rhamnosus* SP1 requires careful oral hygiene. This remains essential to reduce the severity of DS among the elderly population [261].

Another interesting *in vivo* finding is the efficacy of methylene blue-mediated photodynamic inactivation of *C. albicans* on the oral mucosa and prostheses of patients with DS [232,262]. An *in vitro* experiment found that irradiation with 405-nm blue LED light causes the degradation of *C. albicans* and *C. glabrata* biofilms on the surface of polymethyl methacrylate resins. The effectiveness of 405-nm blue LED light on the degradation of *Candida* biofilms formed on PMMA denture base resin has recently been confirmed without resin degradation [263].

**Mouthwashes**

The second stage of antimicrobial therapy, including the use of MoWs, is intended to impede the transition of stage I biofilms (adhesion) to stage II by combatting the attachment and maturation of the biofilm [264]. Many recommended chemotherapeutic products and interventions are effective against planktonic oral bacteria; however, unfortunately, live intact biofilms can persist even after treatment with many products (e.g., sodium hypochlorite) [86]. Because of the complexity of the denture environment, an appropriate MoW can avoid or at least ameliorate oral infections, such as dental caries, gingivitis, periodontitis, and DS. Both prevention and/or treatment of bacteria (oral Streptococci) and fungi (*C. albicans*) may play a relevant role in the onset of these oral pathologies [59,192]. In *in vitro* studies, CHX MoWs free of alcohol (e.g., Curasept, Meridol, Dentosan, Parodontax) exhibit the strongest effects against *Candida* biofilm formation at different levels (adhesion, elicitation of pro-inflammatory responses, and avoiding phagocytosis), but *viridans* Streptococci can also form biofilms. Given the side effects of CHX (e.g., staining, altered taste and feeling) MoWs with Cetylperyridinium Chloride (CPC) does not present these same drawbacks, are an attractive alternative, all the more for impairing early biofilm formation by *S. salivarius* more effectively than of CHX. Other, MoWs, (Listerine) including essential oils after treated *S. salivarius* to be revealed susceptible to delays *Candida* biofilm formation. Moreover, MoWs such as Elmex that include fluorine molecules contribute to caries prevention [59] (Table 4). Finally, these *in vitro* studies show that MoWs containing CHX or CPC may be favorable for oral health in terms of microbial balance. However, these data must be confirmed by comparative in-depth *in vivo* studies.

| Effects of MoWs | Impair *C. albicans* adhesion/ epithel (5mn) | Impair *C. albicans* adhesion/ epithel (5mn) | Elicit Cytokines Secret by epithel | Capacity to develop *C. albicans* hyphal form | Capacity of 5 oral streptococci to produce biofilm | Mixed biofilm *S. salivarius* and *C. albicans* (pretreat with MoWs). |
|----------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| 1-Curasept 0.20% CHX | + | + | + | +( Could develop hyphal form) | Reduce biofilm production/ control (*inhibe s sanguinis*) | Impair *C. albicans* biofilm formation at early stage (24 hours) |
| 2-Dentosan Collutorio CHX | + | + | + | (no hyphal-form) | Reduce (*inhibe S. mitis/ oralis*) (*inhibe S. sanguinis*) | No effect |
| 3-Meridol collutorio 0.20% CHX | + | + | + | _ | Reduce (*inhibe S. mitis/ oralis*) (*inhibe S. sanguinis*) | No effect |
| 4-Parodontax 0.06% CHX | + | + | + | _ | Reduce (*inhibe S. mitis/ oralis*) | No effect |
| 5-Elmex sensitive professional Fluorine molecules | +/- (without blocking completely) | +/- (leaving fungus the ability to adhere to oral epithelial cells) | +/- fast | _ | No reduction of biofilm production/ control | Noeffect |
| 6-Listerine total care zero Essential oils | + | + | + | _ | Reduce (*lower effect*) [265-267] (*inhibe S. sanguinis*) | Impair *C. albicans* biofilm formation at early stage (24 hours, but not after 48h) |
Concerning denture cleaning tablets, the polarity of the resins, the concentrations of tablets, and the chemical content of the cleanser may directly affect concentrations of tablets, and the chemical content of the cleanser may vary depending on the resin used to make the prosthetic base. The use of microwave disinfection in combination with denture cleansers and brushing has also been shown to effectively disinfect dentures in vivo, though microwaves may also physically distort denture acrylics. There are several unconventional approaches to denture care, including soaking in vinegar, baking soda, sodium chloride, and liquid soaps. Though these practices aim to control the prosthetic biofilm, they may be insufficient. In-depth examinations are usually required to gain deeper understanding of the etiology and implementation of the most appropriate therapy.

In vitro, MoWs do not alter the way phagocytes perceive fungus in their surrounding environment, since their phagocytic activity remains almost unaffected.

- In MoWs Elmex and Curasept-pretreated *C. albicans*, the acidic phagolysosomes remain unchanged relative to a PBS control. This phenomenon can be ascribed to the inhibition of a fungal dimorphic transition. Fungi treated with MoWs including Dentosan, Meridol, Listerine, and Parodontax remain in yeast form.

- MoWs capable of impairing dimorphic transitions and in turn intracellular compartment acidification contain either CHX or essential oils in their formulations.

- The presence of an anti-discoloration system (ADS) in MoWs may partially interfere with its CHX-mediated anti-fungal effects.

- With MoWs 1 (Curasept), only *Candida*’s capacity to form a biofilm is impaired. Its susceptibility to phagocytes remains unchanged, while other virulence traits, such as adhesion and elicitation of proinflammatory cytokines, are affected.

- *S. salivarius* treated with MoWs Curacept and Listerine has delayed *Candida* biofilm formation, but these fail to stop it completely.

### 6. Improvement of an effective solution against DP

Although there is no consensus regarding how to best maintain prosthetic hygiene compatible with the patient’s state of health, the disadvantages of many procedures are thoroughly evidenced. Several habits should be avoided, such as rinsing with boiling water and prolonged maintenance in a dry atmosphere or water because these alter the qualities of some materials or promote microbial colonization. Both bleach and isopropyl alcohol (IPA) are highly antimicrobial, but bleach is incompatible with metal prosthetic components, and IPA MoWs damage polymethyl methacrylate. Concerning denture cleaning tablets, the polarity of the resins, the concentrations of tablets, and the chemical content of the cleanser may directly affect *C. albicans* biofilm formation and provide both anti-microbial efficacy and greater material compatibility. The dosage and prescription of disinfecting tablets can vary depending on the resin used to make the prosthetic base. The use of microwave disinfection in combination with denture cleansers and brushing has also been shown to effectively disinfect dentures in vivo, though microwaves may also physically distort denture acrylics. There are several unconventional approaches to denture care, including soaking in vinegar, baking soda, sodium chloride, and liquid soaps. Though these practices aim to control the prosthetic biofilm, they may be insufficient. In-depth examinations are usually required to gain deeper understanding of the etiology and implementation of the most appropriate therapy.

In summary, prosthetic oral hygiene requires daily control of the microbial plaque. The personalized implementation of the means at our disposal is informed by the general condition of the patient, the material composition of the prosthetic base, and the presence or absence of DS. *C. albicans*, a frequent commensal member of well-balanced oral microbiota, influences the treatment of the daily maintenance of prostheses has little influence on the microbiome but helps stabilize the microbial balance.

### Conclusion

The simple presence of a removable prosthesis in the oral cavity can upset the balance of the oral microbiota. The reciprocal relationship between the host and their prosthetic microbiome is both fragile and constantly dynamic. In addition, several general and local parameters contribute to this balance. These parameters can be specific to the host (heredity and general pathologies) but also dependent on their level of oral hygiene and their lifestyle, which makes it difficult to manage DS. DP biofilms cannot be eliminated, especially because they are invisible to the naked eye. In addition, it is essential to differentiate between tolerance of DP and persistent infections such as DS. Despite recent advances, we still lack a comprehensive understanding of many characteristics of DP biofilms. Their tolerance of existing antifungal drugs, their ability to evade components of the host immune system, their resistance to mechanical forces under the denture, and their capacity to seed
new infections make them the central targets of a more effective strategic plan in the fight against persistent oral infections such as DS.

References

1. Nikawa H, Hamada T, Yamamoto T (1998) Denture plaque–past and recent concerns. J Dent 26: 299-304.
2. Hao Y, Huang X, Zhou X, Li M, Ren B, et al. (2018) Influence of dental prosthesis and restorative materials interface on oral biofilms. Int J Mol Sci 19: E3157.
3. Sachdeo A, Haffajee AD, Socransky SS (2008) Biofilms in the edentulous oral cavity. J Prosthodont 17: 348 356.
4. Mitchell KF, Zarnowski R, Sanchez H, Edward JA, Reinicke EL, et al. (2015) Community participation in biofilm matrix assembly and function. Proc Natl Acad Sci USA 112: 4092 4097.
5. Gendreau L, Loewy ZG (2011) Epidemiology and etiology of denture stomatitis. J Prosthodont 20: 251 260.
6. Ercalkil-Yalcinkaya S, Ozcan M (2015) Association between oral mucosal lesions and hygiene habits in a population of removable prosthesis wearers. J Prosthodont 24: 271-278.
7. Latib YO, Olsen CP, Patel M (2018) Viability of candida albicans in denture base resin after disinfection: a preliminary study. J Int Prosthodont 31: 436 439.
8. Salerno C, Pascale M, Contaldo M, Esposito V, Busciolano M, et al. (2011) Candida-associated denture stomatitis. Med Oral Patol Oral Cir Buil 2011; 16: e139-143.
9. Cruz PC, Andrade IM, Peracini A, Souza-Gugelmin MCM, Silva-Lovato CH, et al. (2011) The effectiveness of chemical denture cleansers and ultrasonic device in biofilm formation from complete dentures. J Appl Oral Sci 19: 668-673.
10. Abaci Ö (2011) Investigation of extracellular phospholipase and proteinase activities of Candida species isolated from individuals denture wearers and genotypic distribution of Candida albicans strains. Curr Microbiol 62: 1308-1314.
11. Zarnowski R, Westler WM, Lacmbouh GA, Marita JM, Bothe JR, et al. (2014) Novel entries in a fungal biofilm matrix encyclopedia. mBio 5: e01333-01314.
12. Kavanagh JS, Flack CE, Lister J, RickerEB, Iberson CB, et al. (2019) Identification of extracellular DNA-binding proteins in the biofilm matrix. mBio 10: e01137-19.
13. Al-Fattani MA, Douglas LJ (2006) Biofilm matrix of Candida albicans and Candida tropicalis: chemical composition and role in drug resistance. J Med Microbiol 55: 999-1000.
14. Baille GS, Douglas LJ (2000) Matrix polymers of Candida biofilms and their possible role in biofilm resistance to antifungal agents. J Antimicrob Chemother 46: 397-403.
15. Nett J, Lincoln L, Marchillo K, Massey R, Holodyka K, et al. (2007) Putative role of beta-1,3 glucans in Candida albicans biofilm resistance. Antimicrob Agents Chemother 51: 510-20.
16. Nett JE, Sanchez H, Cain MT, Andes DR (2010) Genetic basis of Candida biofilm resistance due to drug-sequestering matrix glucan. J Infect Dis 202: 171-175.
17. Cahn LR (1936) The Denture sore mouth. Ann Dent 3: 33-36.
18. Koopmans AS, Kippuw N, de Graaff J (1988) Bacterial involvement in denture-induced stomatitis. J Dent Res 67: 1246-1250.
19. Campos MS, Marchini L, Bernardes LA, Paulino LC, Nobrega FG (2008) Biofilm microbial communities of denture stomatitis. Oral Microbiol Immunol 23: 419-424.
20. Schaller M, Borelli C, Korting HC, Hube B (2005) Hydrolytic enzymes as virulence factors of Candida albicans. Mycoses 48: 365-377.
21. Rickard AH, Gilbert P, High NJ, Kolenbrander PE, Handley PS (2003) Bacterial coaggregation: an integral process in the development of multi-species biofilms. Trends Microbiol 11: 94 100.
22. Ruhl S, Eidt A, Melzl H, Reischl U, Cisar JO (2014) Probing of microbial biofilm communities for coadhesion partners. Appl Environ Microbiol 80: 6583 6590.
23. Fox EP, Bui CK, Nett JE, Hartooni N, Mui MC, et al. (2015) An expanded regulatory network temporally controls Candida albicans biofilm formation. Mol Microbiol 96: 1226 1239.
24. Li P, Seneviratne CJ, Alpi E, Vizzaino JA, Jin L (2015) Delicate metabolic control and coordinated stress response critically determine antifungal tolerance of Candida albicans biofilm persisters. Antimicrob Agents Chemother 59: 6101 6112.
25. Chandra J, Mukherjee PK (2015) Candida biofilms: development, architecture, and resistance. Microbiol Spectr 3: 10.1128.
26. Fox EP, Bui CK, Nett JE, Hartooni N, Mui MC, et al. (2015) An expanded regulatory network temporally controls Candida albicans biofilm formation. Mol Microbiol 96: 1226-1239.
27. Zarnowski R, Westler WM, Lacmbouh GA, Marita JM, Bothe JR, et al. (2014) Novel entries in a fungal biofilm matrix encyclopedia. mBio 5: e01333-01314.
28. Li P, Seneviratne CJ, Alpi E, Vizzaino JA, Jin L (2015) Delicate Metabolic Control and Coordinated Stress Response Critically Determine Antifungal Tolerance of Candida albicans Biofilm Persisters. Antimicrob Agents Chemother 59: 6101-6112.
29. Ruhl S, Eidt A, Melzl H, Reischl U, Cisar JO (2014) Probing of Microbial Biofilm Communities for Coadhesion Partners. Spormann AM, éditeur. Appl Environ Microbiol 80: 6583-6590.
30. Santana IL, Gonçalves LM, de Vasconcellos AA, da Silva WJ, Cury JA, et al. (2013) Dietary carbohydrates modulate candida albicans biofilm development on the denture surface. PLoS One 8: e64645.
31. ArendorfTM, Walker DM (1980) The prevalence and intra-oral distribution of Candida albicans in man. Arch Oral Biol 25: 1-10.
32. Morse DJ, Smith A, Wilson MJ, Marsh L, White L, et al. (2019) Molecular Community Profiling of the Bacterial Microbiota Associated With Denture-Related Stomatitis. Sci Rep 9: 10228.
33. O’Donnell LE, Robertson D, Niel CJ, Cross LJ, Rickig M, et al. (2015) The oral microbiome of denture wearers is influenced by levels of natural dentition. PLoS One 10: e0137717.
34. Teles FR, Teles RP, Sachdeo A, Uzel NG, Song XQ, et al. (2012) The oral microbiome of denture wearers. J Periodontol 83: 1139-1148.
35. Murugesan S, Al Ahmad SF, Singh P, Saadaoui M, Kumar M, et al. (2020) Profiling the salivary microbiome of the qatari population. J Transl Med 18: 127.
36. Newton AV (1962) Denture sore mouth: A possible etiology. Br Dent J 112: 357-360.

37. Perić M, Živković R, Milić Lemić A, Radunović M, Miličić B, et al. (2018) The severity of denture stomatitis as related to risk factors and different Candida spp. Oral Surg Oral Med Oral Pathol Oral Radiol S212-4403: 30841.

38. Vila T, Sultan AS, Montelongo-Jauregui D, Jabra-Rizk MA (2020) Oral Candidiasis: a disease of opportunity. J Fungi (Basel) 6: E15.

39. Steele JG, Treasure ET, Fuller E, Morgan MZ (2011) Complexity and maintenance - a report from the adult dental health survey. In: O’Sullivan I, ed. Adult Dental Health Survey 2009 – Northern Ireland Key Findings. London: The health and social care information centre: 7-8.

40. Theilade E, Budtz-Jørgensen E, Theilade J (1983) Predominant cultivable microflora of plaque on removable dentures in patients with healthy oral mucosa. Arch Oral Biol 28: 675-880.

41. Shi B, Wu T, McLean J, Edlund A, Young Y, et al. (2016) The denture-associated oral microbiome in health and stomatitis. mSphere 1: e00215-16.

42. Yarborough A, Cooper L, Duquen I, Mendonça G, McGraw K, et al. (2016) Evidence regarding the treatment of denture stomatitis. J Prosthod 25: 289-301.

43. Gasparoto TH, Sipert CR, de Oliveira CE, Porto VC, Santos CF, et al. (2012) Salivary immunity in elderly individuals presented with Candida-related denture stomatitis. Gerodontology 29: e331-e339.

44. Budtz-Jørgensen E, Theilade E, Theilade J (1983) Quantitative relationship between yeast and bacteria in denture-induced stomatitis. Scand J Dent Res 91: 134-142.

45. CocoBJ, BaggJ, CrossLJ, JoseA, CrossJ, et al. (2008) Mixed Candida albicans and Candida glabrata populations associated with the pathogenesis of denture stomatitis. Oral Microbiol Immunol 23: 377-383.

46. Figueiral MH, Azul A, Pinto E, Fonseca PA, Branco FM, et al. (2007) Denture-related stomatitis: identification of aetiological and predisposing factors - a large cohort. J Oral Rehabil 34: 448-455.

47. Bilih N, Sulun T, Ekrose G, Kurt H, Erturan Z, et al. (2009) The role of Candida albicans hyphae and Lactobacillus in denture-related stomatitis. Clin Oral Invest 13: 363-368.

48. Gasparoto TH, Vieira NA, Porto VC, Campanelli AP, Lara VS (2009) Ageing Exacerbates Damage of Systemic and Salivary Neutrophils From Patients Presenting Candida-related Denture Stomatitis. Immun Ageing 6: 3.

49. Gasparoto TH, Dionisio TJ, de Oliveira CE, Porto VC, GelaniV, et al. (2009) Isolation of Candida dubliniensis from denture wearers. J Med Microbiol 58: 959-962.

50. Altarawneh S, Bencharit S, Mendoza L, Curran A, Barlow D, et al. (2013) Clinical and histological findings of denture stomatitis as related to intraoral colonization patterns of Candida albicans, salivary flow, and dry mouth. J Prosthodont 22: 13-22.

51. Pereira HM, Ferrier S, Walters M, Geller GN, Jongman RHG, et al. (2013) Essential Biodiversity Variables. Science 339: 277-278.

52. Akpan A, Morgan R (2002) Oral candidiasis. Postgrad Med J 78: 455–459.

53. Abaci O, Hallik-Uztan A (2011) Investigation of the susceptibility of Candida species isolated from denture wearers to different antifungal antibiotics. Afr J Microbiol Res 5: 1398–1403.

54. Wilson J (1998) The aetiology, diagnosis and management of denture stomatitis. Br Dent J 185: 380-384.

55. Webb BC, Thomas CJ, Willcox MD, Harty DW, Knox KW (1998) Candida-associated denture stomatitis: aetiology and management: a review: part I: factors influencing distribution of Candida species in the oral cavity. Aust Dent J 43: 45–50.

56. Fraser VJ, Jones M, Dunkel J, Storfer S, Medoff G, et al. (1992) Candidemia in a tertiary care hospital: epidemiology, risk factors, and predictors of mortality. Clin Infect Dis 15: 414–421.

57. Senpuku H, Sogame A, Inoshita E, Tsucha Y, Miyazaki H, et al. (2003) Systemic diseases in association with microbial species in oral biofilm from elderly requiring care. Gerontology 49: 301 309.

58. Yildirim-Bicer AZ, Peker I, Akca G, Celik I (2014) in vitro antifungal evaluation of seven different disinfectants on acrylic resins.Biomed Res Int: 519098.

59. Ardzizzone A, Pericolini E, Paulone S, Orsi CF, Castagnoli A, et al. (2018) In vitro effects of commercial mouthwashes on several virulence traits of Candida albicans, viridans streptococci and Enterococcus faecalis colonizing the oral cavity. PLoS ONE 13: e0207262.

60. LoF M, Janus M, Krom B (2017) Metabolic interactions between bacteria and fungi in commensal oral biofilms. J Fungi (Basel) 3: E40.

61. Marsh PD (2005) Dental plaque: biological significance of a biofilm and community life-style. J Clin Periodontol 32: 7 15.

62. Zheng W, Tsompana M, Ruscitto A, Sharma A, Genco R, et al. (2015) An accurate and efficient experimental approach for characterization of the complex oral microbiota. Microbiome 3: 48.

63. Radford DR, Challacombe SJ, Walter JD (1999) Dental plaque and adherence of candida albicans to denture base materials in vivo and in vitro. Crit rev Oral Biol Med 10: 99-116.

64. Baran I (2009) Self-reported denture hygiene habits and oral tissue conditions of complete denture wearers. Arch Gerontol Geriatr 49: 237-241.

65. Olsen I, Haanæs HR (1977) Experimental palatal candidosis and saliva flow in monkeys. Scand J Dent Res 85: 135-141.

66. Martori E, Ayuso-Montero R, Martinez-Gomis J, Viñas M, Peraire M (2014) Risk factors for denture-related oral mucosal lesions in a geriatric population. J Prosthet Dent 111: 273-279.

67. Delaney C, O’Donnell LE, Kean R, Sherry L, Brown JL, et al. (2019) Interkingdom Interactions on the Denture Surface: Implications for Oral Hygiene. Biofilm 1: 100002.

68. Alonsius CN, van den Broek MFL, De Boeck I, Kiekens S, OerlemansEFM, et al. (2017) Interplay between Lactobacillus rhamnosus GG and Candida and the involvement of exopolysaccharides. Microb Biotechnol 10: 1753-1763.

69. Matsuda Y, Cho O, Sugita T, Ogishima D, Takeda S. (2018) Culture supernatants of Lactobacillus gasseri and L. crispatus inhibit Candida albicans biofilm formation and adhesion to HeLa cells. Mycopathologia 183: 691-700.

70. Ryan KE, Ranjit Rajendran , Jennifer Haggarty, Eleanor M Townsend, Bryn Short , et al. (2017) Candida albicans mycofilms support Staphylococcus aureus colonization and enhances Miconazole resistance in dual-species interactions. Front Microbiol 8: 258.

71. Marleen M Janus, Hubertine M E Willems, Bastiaan P Krom (2016) Oral cavity. PLoS ONE 13: e0207262.
72. Thilakumara IP, Jayatihaie JAMS, Pallegama RW, Ellepola ANB (2017) Denture-induced stomatitis and associated factors in a group of patients attending a university dental hospital in Sri Lanka. J Investi Clin Dent 8: 1-2211.

73. Inuma T, Arai Y, Abe Y, Takayama M, Fukumoto M, Fukui Y, et al. (2015) Denture wearing during sleep doubles the risk of pneumonia in the very elderly. J Dent Res 94: 285-36S.

74. Jakubovics NS (2015) Intermicrobial interactions as a driver for community composition and stratification of oral biofilms. J Mol Biofilms 427: 3662-3675.

75. Zarco MF, Vess TJ, Ginsburg GS (2012) The oral microbiome in health and disease and the potential impact on personalized dental medicine. Oral Dis 18: 109-120.

76. SimsCR, Ostrosky-ZeichnerL, RexJH (2005) Invasive candidiasis in immune compromised hospitalized patient. Arch Med res 36: 660-671.

77. Dar-Odeh NS, Shehabi AA (1991) Oral candidosis in patients with removable dentures. Mycoses 46: 187-191.

78. Shulman JD, Rivera-Hidalgo F, Beach MM (2005) Risk factors associated with denture stomatitis in the United States. J Oral Pathol Med 34: 340-346.

79. Bianchi CM, Bianchi HA, Tadano T, Paula CR, Hoffmann-Santos HD, et al. (2016) Factors related to oral candidiasis in elderly users and non-users of removable dental prostheses. Rev Inst Med Trop Sao Paulo 58: 17.

80. López-Pintor RM, Hernández G, de Arriba L, de Andrés A (2013) Oral candidiasis in patients with renal transplants. Med Oral Patol Oral Cir Bucal 18: e381-e387.

81. Paillard E, Merlier I, Dupeyron C, Scherman E, Poupon J, et al. (2004) Oral candidiasis and nutritional deficiencies in elderly hospitalised patients. Br J Nutr 92: 861-867.

82. Al-Dwairi ZN (2008) Prevalence and risk factors associated with denture-related stomatitis in healthy subjects attending a dental teaching hospital in North Jordan. J Ir Dent Assoc 54: 80-83.

83. Lyon JP, de Resende MA (2006) Correlation between adhesion, enzyme production, and susceptibility to fluconazole in Candida albicans obtained from denture wearers. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 102: 632-638.

84. Maciag J, Mikolajczyk Y, Tatusik P, Nosalski R, Sagan A, et al. (2017) Systemic T cells and monocye characteristics in patients with denture stomatitis. J Prosthod 26: 19-28.

85. Rodríguez-Archilla A, Urquia M, Cutando A, Asencio R (1996) Denture stomatitis: Quantification of interleukin-2 production by mononuclear blood cells cultured with Candida albicans. J Prostheth Dent 75: 426-431.

86. Ramage G, O’Donnell L, Sherry L, Culshaw S, Bagg J, et al. (2019) Impact of frequency of denture cleaning on microbial and clinical parameters – a bench to chairside approach. J Oral Microbiol 11: 1538437.

87. Axe AS, Varghese R, Bosma M, Kiltson N, Bradshaw DJ (2016) Dental health professional recommendation and consumer habits in denture cleansing. J Prostheth Dent 115: 183-188.

88. Kiesow A, Sarembe S, Pizzey RL, Axe AS, Bradshaw DJ (2016) Material compatibility and antimicrobial activity of consumer products commonly used to clean dentures. J Prostheth Dent 115: 189-198.

89. Ramage G, Tomsett K, Wickes BL, López-Ribot JL, Redding SW (2004) Denture stomatitis: A role for Candida biofilms. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 98: 53-59.

90. Webb BC, Thomas CJ, Willcox MD, Harty DW, Knox KW (1998) Candida-associated denture stomatitis. Aetiology and management: A review. Part 3. Treatment of Oral Candidosis. Aust Dent J 43: 244-249.

91. Budz-Jörgensen E (1974) The significance of Candida albicans in denture stomatitis. Eur J Oral Sci 82: 151-190.

92. Dongari-Bagtzoglou A (2009) characterization of mucosal Candida albicans biofilms. Plos one 4: e7987.

93. Cunha-Cruz J, Hujopol PP, Nadanovsky P (2007) Secular trends in socio-economic disparities in edentulism: USA, 1972-2001. J Dent Res 86: 131-136.

94. United Nations, eds. World Population Aging Report. New York: United Nations, 2015.

95. Ramraj C, QuinonezCR (2013) Self-reported cost-prohibitive dental care needs among Canadians. Int J Dent Hyg 11: 115-120.

96. Cousson PY, Bessadet M, Nicolas E, Veyrune JL, Lesourd B, et al. (2012) Nutritional Status, Dietary Intake and Oral Quality of Life in Elderly Complete Denture Wearers. Gerodontontology 29: e685-e692.

97. KamenoffL (1987) Cumulative Index to Nursing and Allied Health Literature (CINAHL). Bull Med Lib Assoc 75: 268.

98. HannanVE, O’Donnell L, Robertson D, Ramage G (2017) Denture stomatitis: causes, cures and prevention. Prim Dent J 6: 46-51.

99. O’Donnell LE, Alalwan HKA, KeanR, CalvertG, NileCJ (2017) Candida albicans biofilm heterogeneity does not influence denture stomatitis but strongly influences denture cleansing capacity. J Med Microbiol 66: 54-60.

100. YitzhakiS, ReshefL, GophnaU, RosenbergM, StererN (2018) Microbiome associated with denture malodour. J Breath Res 12: 027103.

101. Williams DW, Kuriyama T, Silva S, Malic S, Lewis MAO (2011) Candida biofilms and oral candidosis: treatment and prevention. Periodontology 55: 250-65.

102. Farah CS, Lynch N, McCullough MJ (2010) Oral fungal infections: an update for the general practitioner. Aust Dent J 55: 48-54.

103. Darwazeh AMG, Darwazeh TA (2014) What makes oral Candidiasis recurrent infection? A clinical view. J Mycol: 758394.

104. Darwazeh AMG, Al-Dwairi ZN, Al-Zwairi AA (2010) The Relationship between tobacco smoking and oral colonization with Candida species. J Contemp Dent Pract 11: 17-24.

105. Semlali A, Killer K, Alanazi H, Chmielewski W, Rouabbia M (2014) Cigarette smoke condensate increases C. albicans adhesion, growth, biofilm formation, and EAP1, HWP1 and SAP2 geneexpression. BMC Microbiol 14: 61.

106. Brook I (1999) Bacterial Interference. Crit Rev Microbiol 25:155-172.

107. He X, McLean JS, Guo L, Lux R, Shi W (2014) The social structure of microbial community involved in colonization resistance. ISME J 8: 564-574.

108. Ryu M, Ueda T, Saito T, Yasui M, Ishihara K, et al. (2010) Oral environmental factors affecting number of microbes in saliva of complete denture wearers. J Oral Rehabil 37: 194-201.
109. Maciag J, Osmendg G, Nowakowski D, Wilk G, Maciag A, et al. (2014) Denture-related stomatitis is associated with endothelial dysfunction. Biomed Res Int 2014: 474016.

110. Naik AV, Pai RC (2011) A Study of factors contributing to denture stomatitis in a North Indian Community. Int J Dent 2011: 589064.

111. Hoshing C, Dixit S, Mootha A, Diwan N (2011) Role of Candida albicans in denture stomatitis. J Indian Acad Oral Med Radiol 23:617-619.

112. Chopde N, Jawale B, Pharande A, Chaudhari L, Hiremath V, et al. (2012) Microbial colonization and their relation with potential cofactors in patients with denture stomatitis. J Contemp Dent Pract 13: 456-459.

113. Danderkeri S, Prasad K, Shetty M, Hegde C, Sowmya MK, et al. (2013) Occurrence of streptococcus and Candida species and salivary ph in patients wearing complete denture. Int J Health Rehabil Sci 2: 198-203.

114. Dantas AFPM, Consani RLX, Sardi JCO, Mesquita MF, Silva MCVS, et al. (2014) Biofilm formation in denture base acrylic resins and disinfection method using microwave. J Res Pract Dent 2014: 112442.

115. Man A, Ciurea CN, Pasaroiu D, Savin Al, Toma F, et al. (2017) New perspectives on the nutritional factors influencing growth rate of Candida albicans in diabetics. An in vitro study. Mem Inst Oswaldo Cruz 112: 587-592.

116. DewhirstFE, ChenT, IzardJ, PasterBJ, Tanner, et al. (2010) The Human Oral Microbiome J Bacteriol 192: 5002-5017.

117. WadeWG (2013) The Oral microbiome in health and disease. Pharmacol Res 69: 137-143.

118. Aas JA, Paster BJ, Stokes LN, Olsen I, Dewhirst FE (2005) Defining the normal bacterial flora of the Oral cavity. J Clin Microbiol 43: 5721-5732.

119. Joshy G, Arora M, Korda RJ, Chalmers J, Banks E (2016) Poor oral health a risk marker for incident cardiovascular disease hospitalisation and all-cause mortality? Findings From 172 630 participants from the prospective 45 and up study. BMJ Open 6: e012386.

120. Iwasaki M, Sato M, Yoshihara A, Ansai T, Miyazaki H (2017) Association between tooth loss and medical costs related to stroke in healthy older adults aged over 75 years in Japan. Geriatr Gerontol Int 17: 202-210.

121. Joshipura K (2002) The relationship between oral conditions and ischemic stroke and peripheral vascular disease. J Am Dent Assoc 133: 23S-30S.

122. Wang Y, Peng J, Li Y, Luo H, Huang G, et al. (2016) Association between dental loss and risk of oesophageal cancer: A dose-response meta-analysis. Springerplus 5: 1020.

123. Yin XH, Wang YD, Luo H, Zhao K, Huang GL, et al. (2016) Association between tooth loss and gastric cancer: A meta-analysis of observational studies. PLoS One 11: e0149653.

124. Rijkskroeff P, Loos BG, Nicu EA (2017) Impaired polymorphonuclear neutrophils in the oral cavity of edentulous individuals. Eur J Oral Sci 125: 371-378.

125. Danser MM, Mantilla Gómez S, Van der Weijden GA (2003) Tongue coating and tongue brushing: A literature review. Int J Dent Hyg 1: 151-158.

126. Rutger Persson G (2012) Rheumatoid arthritis and periodontitis - inflammatory and infectious connections. Review of the literature. J Oral Microbiol 4.

127. Témoin S, Chakaki A, Askari A, El-Halaby A, Fitzgerald S, et al. (2012) Identification of Oral bacterial DNA in synovial fluid of patients with arthritis with native and failed prosthetic joints. J Clin Rheumatol 18: 117-121.

128. Kaur S, Bright R, Proudmann SM, Bartold PM (2014) Does periodontal treatment influence clinical and biochemical measures for rheumatoid arthritis? A systematic review and meta-analysis. Semin Arthritis Rheum 44: 113-122.

129. Beck JD, Offenbacher S (2005) Systemic effects of periodontitis: Epidemiology of periodontal disease and cardiovascular disease. J Periodontol 76: 2089-2100.

130. Serra e Silva Filho W, Casarin RC, Nicolela EL Jr, Passos HM, Sallum AW, et al. (2014) Microbial diversity similarities in periodontal pockets and atheromatous plaques of cardiovascular disease patients. PLoS One 9: e109761.

131. Heo SM, Haase EM, Lesse AJ, Gill SR, Scannapieco FA (2008) Genetic Relationships Between Respiratory Pathogens Isolated From Dental Plaque and Bronchoalveolar Lavage Fluid From Patients in the Intensive Care Unit Undergoing Mechanical Ventilation. Clin Infect Dis 47: 1562-1570.

132. Filkins LM, Hampton TH, Gifford AH, Gross MJ, Hogan DA, et al. (2012) Prevalence of Streptococci and increased polymicrobial diversity associated with cystic fibrosis patient stability. J Bacteriol 194: 4709-4717.

133. Antunes AA, de Santana Santos T, de Carvalho RW, Avelar RL, Pereira CU, et al. (2011) Brain Abscess of odontogenic origin. J Craniofac Surg 22: 2363-2365.

134. Pierce D, Calkins BC, Thornton K (2012) Infectious endocarditis: Diagnosis and treatment. Am Fam Physician 85: 981-988.

135. Gil-Montoya JA, Sanchez-Lara I, Carnero-Pardo C, Fornies F, Montes J, et al. (2015) Is Periodontitis a Risk Factor for Cognitive Impairment and Dementia? A Case-Control Study J Periodontol 86: 244-253.

136. MacPhail LA, Komaroff E, Alves ME, Navazesh M, Phelan JA, Redford M (2002) Differences in risk factors among clinical types of oral candidiasis in the Women's Interagency HIV Study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 93: 455-455.

137. Witzel AL, Piros M de F, de Carli ML, Rabelo GD, Nunes TB, et al. (2012) Candida albicans isolation from buccal mucosa of patients with hiv wearing removable dental prostheses. Int J Prosthodont 25: 127-131.

138. Lyenge Pedersen AM, Nauntofte B, Smidt D, Torpet LA (2015) Oral mucosal lesions in older people: relation to salivary secretion, systemic diseases and medications. Oral Dis 21: 721-729.

139. Golecka M, Oldakowska-Jedynak U, Mierzwińska-Nastalska E, Adamczyk-Sosińska E (2006) Candida-associated denture stomatitis in patients after immunosuppression therapy. Transplant Proc 38: 155-156.

140. Davies AN, Brailsford SR, Beighton D, Shorthose K, Stevens VC (2008) Oral candidosis in community-based patients with advanced cancer. J Pain Symptom Manage 35: 508-514.

141. Davies AN, Brailsford SR, Beighton D (2006) Oral candidosis in patients with advanced cancer. Oral Oncol 42: 698-702.

142. Osmenda G, Maciag J, Wilk G, Maciag A, Nowakowski D, et al. (2017) Treatment of denture-related stomatitis improves endothelial function assessed by flow-mediated vascular dilation. Arch Med Sci 13: 66-74.
143. Ribeiro AB, de Araújo CB, Silva LEV, Fazan-Junior R, Helio C Salgado, et al. (2019) Hygiene protocols for the treatment of denture-related stomatitis: local and systemic parameters analysis - a randomized, double-blind trial protocol. Trials 20: 661.

144. Peng J, Song J, Han J, Chen Z, Yin X, et al. (2019) The relationship between tooth loss and mortality from all causes, cardiovascular diseases, and coronary heart disease in the general population: systematic review and dose-response meta-analysis of prospective cohort studies. Biosci Rep 39: BSR20181773.

145. Green SL. (1979) Anaerobic pleuro-pulmonary infections. Postgrad Med 65: 62-66.

146. Martin BJ, Corlew MM, Wood H, Olson D, Golopol LA, et al. (1994) The association of swallowing dysfunction and aspiration pneumonia. Dysphagia 9: 1-6.

147. Sumi Y, Miura H, Sunakawa M, Michiwhaki Y, Sakagami N (2002) Colonization of denture plaque by respiratory pathogens in dependent elderly. Gerodontology 19: 25-29.

148. Yoneyama T, Yoshida M, Ohru T, Mukaiyama H, Okamoto H, et al. (2002) Oral Care Working Group. Oral care reduces pneumonia in older patients in nursing homes. J Am Geriatr Soc 50: 430-433.

149. El-Solh AA (2011) Association between pneumonia and oral care in nursing home residents. Lung 189: 173-180.

150. Scannapieco FA (2006) Pneumonia in non-ambulatory patients. The role of oral bacteria and oral hygiene. J Am Dent Assoc 137: 21S-25S.

151. Sumi Y, Ozawa N, Michiwhaki Y, Washimi Y, Toba K (2012) Oral conditions and oral management approaches in mild dementia patients. Nihon Ronen Igakkai Zasshi 49: 90-96.

152. Przybylowska D, Mierzynska-Nastalska E, Rubinsztajn R, Chazan, Rolski D, et al. (2015) The relationship between removable prosthesis and some systemic diseases, and coronary heart disease in the general population: systematic review and dose-response meta-analysis of prospective cohort studies. Biosci Rep 39: BSR20181773.

153. Le Bars P, Jordana F, Kouadio AA, N’goran JK, Badran Z, Soueidan A (2015) The association of swallowing dysfunction and aspiration pneumonia. Dysphagia 9: 1-6.

154. Peng J, Song J, Han J, Chen Z, Yin X, et al. (2019) The relationship between tooth loss and mortality from all causes, cardiovascular diseases, and coronary heart disease in the general population: systematic review and dose-response meta-analysis of prospective cohort studies. Biosci Rep 39: BSR20181773.

155. Green SL. (1979) Anaerobic pleuro-pulmonary infections. Postgrad Med 65: 62-66.

143. Ribeiro AB, de Araújo CB, Silva LEV, Fazan-Junior R, Helio C Salgado, et al. (2019) Hygiene protocols for the treatment of denture-related stomatitis: local and systemic parameters analysis - a randomized, double-blind trial protocol. Trials 20: 661.

144. Peng J, Song J, Han J, Chen Z, Yin X, et al. (2019) The relationship between tooth loss and mortality from all causes, cardiovascular diseases, and coronary heart disease in the general population: systematic review and dose-response meta-analysis of prospective cohort studies. Biosci Rep 39: BSR20181773.

145. Green SL. (1979) Anaerobic pleuro-pulmonary infections. Postgrad Med 65: 62-66.

146. Martin BJ, Corlew MM, Wood H, Olson D, Golopol LA, et al. (1994) The association of swallowing dysfunction and aspiration pneumonia. Dysphagia 9: 1-6.

147. Sumi Y, Miura H, Sunakawa M, Michiwhaki Y, Sakagami N (2002) Colonization of denture plaque by respiratory pathogens in dependent elderly. Gerodontology 19: 25-29.

148. Yoneyama T, Yoshida M, Ohru T, Mukaiyama H, Okamoto H, et al. (2002) Oral Care Working Group. Oral care reduces pneumonia in older patients in nursing homes. J Am Geriatr Soc 50: 430-433.

149. El-Solh AA (2011) Association between pneumonia and oral care in nursing home residents. Lung 189: 173-180.

150. Scannapieco FA (2006) Pneumonia in non-ambulatory patients. The role of oral bacteria and oral hygiene. J Am Dent Assoc 137: 21S-25S.

151. Sumi Y, Ozawa N, Michiwhaki Y, Washimi Y, Toba K (2012) Oral conditions and oral management approaches in mild dementia patients. Nihon Ronen Igakkai Zasshi 49: 90-96.

152. Przybylowska D, Mierzynska-Nastalska E, Rubinsztajn R, Chazan, Rolski D, et al. (2015) The relationship between removable prosthesis and some systemic diseases, and coronary heart disease in the general population: systematic review and dose-response meta-analysis of prospective cohort studies. Biosci Rep 39: BSR20181773.

153. Le Bars P, Jordana F, Kouadio AA, N’goran JK, Badran Z, Soueidan A (2015) The association of swallowing dysfunction and aspiration pneumonia. Dysphagia 9: 1-6.

154. Peng J, Song J, Han J, Chen Z, Yin X, et al. (2019) The relationship between tooth loss and mortality from all causes, cardiovascular diseases, and coronary heart disease in the general population: systematic review and dose-response meta-analysis of prospective cohort studies. Biosci Rep 39: BSR20181773.

155. Green SL. (1979) Anaerobic pleuro-pulmonary infections. Postgrad Med 65: 62-66.
Citation: Le Bars P, Jordana F, Kouadio A (2021) Denture Plaque Management of Denture-Related Stomatitis. Dent Adv Res 6: 173. DOI: 10.29011/2574-7347.100073

Samaranayake LP, Weetman DA, Geddes DAM, MacFarlane TW (1983) Carboxylic acids and pH of denture plaque in patients with denture stomatitis. J Oral Pathol Med 12: 84-89.

Samaranayake LP (1986) Nutritional factors and oral candidosis. J Oral Pathol Med 15: 61-65.

Kossioni AE (2011) The prevalence of denture stomatitis and its predisposing conditions in an older Greek population. Gerodontology 28: 85-90.

Sakar O, Sulun T, Bilhan H, Ispírılı E (2013) Does the presence of anterior mandibular teeth increase the incidence of denture stomatitis? J Prosthodont 22: 174-178.

Anastassiadiou V, Naka O, Heath MR, Kapari D (2002) Validation of indices for functional assessment of dentures. Gerodontology 19: 46-52.

Mima EG, Vergani CE, Machado AL, Massucco EMS, Colombo AL, et al. (2012) Comparison of photodynamictherapy versus conventional antifungal therapy for the treatment of denture stomatitis: a randomized clinical trial. Clin Microbiol Infect 18: 380-388.

Silva MM, Mima EGO, Colombo AL, Sanita PV, Jorge JH, et al. (2012) Comparison of denture microwave disinfection and conventional antifungal therapy in the treatment of denture stomatitis: a randomized clinical study. Oral Surg Oral Med Oral Pathol Oral Radiol 114: 469-479.

Scully C, Giovanni L (2020) Denture related stomatitis. European Association of Oral Medicine.

Arai T, Ueda T, Sugiyama T, Sakurai K (2009) Inhibiting microbial adhesion to denture base acrylic resin by titanium dioxide coating. J Oral Rehabil 36: 902-908.

Acosta-Torres LS, Mendieta I, Nuñez-Anita RE, Cajero-Juarez M, Castaño VM (2012) Cyto-compatible antifungal resin containing silver nanoparticles for dentures. Int J Nanomedicine 7: 4777-4786.

Sivakumar I, Arunachalam KS, Sajjan S, Ramaraju AV, Rao B, et al. (2014) Incorporation of antimicrobial macromolecules in acrylic denture base resins: a research composition and update. J Prosthodont 23: 284-290.

Zhang K, Ren B, Zhou X, Xu HHK, Chen Y, et al. (2016) Effect of Antimicrobial denture base resin on multi-species biofilm formation. Int J Mol Sci 17: 1033.

Chen H, Han Q, Zhou X, Zhang K, Wang S, et al. (2017) Heat-polymerized resin containing dimethylaminododecyl methacrylate inhibits Candida albicans biofilm. Materials (Basel) 10: 431.

Xu H, Sobue T, Bertolini M, Thompson A, Dongari-Bagtzoglou A (2016) Streptococcus oralis and Candida albicans synergistically activate μ-calpain to degrade e-cadherin from oral epithelial junctions. J Infect Dis 214: 925-934.

Cavalcanti YW, Morse DJ, Silva WJ da, Del-Bel-Cury AA, Wei X, et al. (2015) Virulence and pathogenicity of Candida albicans is enhanced in biofilms containing oral bacteria. Biofouling 31: 27-38.

Hamid SK, Al-Dubayan AH, Al-Awami H, Khan SQ, Gad MM (2019) In vitro assessment of the antifungal effects of neem powder added to poly(methyl methacrylate) denture base material. J Clin Exp Dent 11: 170-178.

Yoshijima Y, Murakami K, Kayama S, Liu D, Hirota K, et al. (2010) Effect of substrate surface hydrophobicity on the adherence of yeast and hyphal Candida. Mycoses 53: 221-226.

Bollen CM, Lambrechts P, Quirynen M (1997) Comparison of surface roughness of oral hard materials to the threshold surface roughness for bacterial plaque retention: a review of the literature. Dent Mater 13: 258-269.

Unushibara Y, Ohshima T, Sato M, Hayashi Y, Hayakawa T, et al. (2014) An analysis of the biofilms adhered to framework alloys using in vitro denture plaque models. Dent Mater J 33: 402-414.

Ohshima T, Ikawa S, Kitano K, Maeda N (2018) A proposal of remedies for oral diseases caused by Candida: A mini review. Front Microbiol 9: 1522.

Sato M, Ohshima T, Maeda N, Ohkubo C (2013) Inhibitory effect of coated mannan against the adhesion of Candida biofilms to denture base resin. Dent Mater J 32: 355-360.

Costa E, Silva S, Tavaria F, Pintado M (2014) Antimicrobial and antibiotic activity of chitosan on the oral pathogen Candida albicans. Pathogens 3: 908-919.

Radford DR, Sweet SP, Challacombe SJ, Walter JD (1998) Adherence of Candida albicans to denture-base materials with different surface finishes. J Dent 26: 577-583.

Nikawa H, Hamada T, Yamashiro H, Kumagai H (1999) A review of in vitro and in vivo methods to evaluate the efficacy of denture cleansers. Int J Prosthodont 12: 153-159.

Baena-Monroy T, Moreno-Maldonado V, Franco-Martínez F, Aldape-Barróns B, Quindós G, et al. (2005) Candida albicans, Staphylococcus aureus and Streptococcus mutans colonization in patients wearing dental prosthesis. Med Oral Patol Oral Cir Bucal 10: E27-39.

Jabra-Rizk MA, Kong EF, Tsui C, Nguyen MH, Clancy CJ, et al. (2016) Candida albicans Pathogenesis: Fitting within the Host-Microbe Damage Response Framework. Infect Immun 84: 2724-2739.

Lewis MAO, Williams DW (2017) Diagnosis and Management of Oral Candidosis. Br Dent J 223: 675-681.

Sultan AS, Rizk AM, Vila T, Ji Y, Masri R, et al. (2019) Digital design of a universal rat intraoral device for therapeutic evaluation of a topical formulation against candida-associated denture stomatitis. Infect Immun 87: e00617-19.

Bell JA, Brockmann SL, Feil P, Sackuvich DA (1989) The effectiveness of two disinfectants on denture base acrylic resin with an organic load. J Prostheth Dent 61: 580-583.

Smith K, Hunter IS (2008) Efficacy of common hospital biocides with biofilms of multi-drug resistant clinical isolates oliveira. J Med Microbiol 57: 966-973.

de Zoccoliotti J, Olga Tasso C, Amaya Arbeláez MI, Ferreira Malavolta I, da Silva Pereira EC, et al. (2018) Properties of an acrylic resin after immersion in antiseptic soaps: low-cost, easy-access procedure for the prevention of denture stomatitis. PLoS One 13: e0203187.

Jenkinson HF, Lappin-Scott HM (2001) Biofilms adhere to stay. Trends Microbiol 9: 9-10.

Kulak-Ozkan Y, Kazazoglu E, Arikam A (2002) Oral hygiene habits, denture cleanliness, presence of yeasts and stomatitis in elderly people. J Oral Rehabil 29: 300-304.
211. Abraham CM (2011) Advances and emerging techniques in the identification, diagnosis and treatment of oral candidiasis. Open Path J 5: 8-12.

212. Hoshi N (2011) Management of oral candidiasis in denture wearers. J prostho Dent Res 55: 48-52.

213. Sampaio-Maia B, Figueiral MH, Sousa-Rodrigues P, Fernandes MH, Scully C (2012) The effect of denture adhesives on Candida albicans growth in vitro. Gerodontontology 29: e348-e356.

214. Spampinato C, Leonard D (2013) Candida infections, causes, targets, and resistance mechanisms: traditional and alternative antifungal agents. Biomed Res Int 2013: 204237.

215. Pierce CG, Lopez-Ribot JL (2013) Candidiasis drug discovery and development: new approaches targeting virulence for discovering and identifying new drugs. Expert Opin Drug Discov 8: 1117-1126.

216. Hawser SP, Douglas LJ (1995) Resistance of Candida albicans biofilms to antifungal agents in vitro. Antimicrob Agents Chemother 39: 2128-2139.

217. Chandra J, Kuhn DM, Mukherjee PK, Hoyer LL, McCormick T, et al. (2001) Biofilm formation by the fungal pathogen Candida albicans: development, architecture, and drug resistance. J Bacteriol 183: 5385-5394.

218. Ramage G, VandeWalle K, López-Ribot JL, Wickes BL (2002) The filamentation pathway controlled by the Efg1 regulator protein is required for normal biofilm formation and development in Candida albicans. FEMS Microbiol Lett 214: 95-100.

219. McCourtie J, MacFarlane TW, Samaranayake LP (1985) Effect of Chlorhexidine gluconate on the adherence of candida species to denture acrylic. J Med Microbiol 20: 97-104.

220. Kuhn DM, George T, Chandra J, Mukherjee PK, Ghannoum MA (2002) Antifungal susceptibility of Candida biofilms: unique efficacy of amphotericin B lipid formulations and echinocandins. Antimicrob Agents Chemother 46: 1773-1780.

221. Pierce CG, Chaturvedi AK, LaZell AL, Powell AT, Saville SP, et al. (2015) A novel small molecule inhibitor of Candida albicans biofilm formation, filamentation and virulence with low potential for the development of resistance. NPJ Biofilms Microbiomes 1: 15012.

222. Romo JA, Pierce CG, Chaturvedi AK, LaZell AL, McHardy SF, et al. (2017) Development of anti-virulence approaches for candidiasis via a novel series of small-molecule inhibitors of Candida albicans filamentation. mBio 8: e01991-2017.

223. Romo JA, Zhang H, Cai H, Kadosh D, Koehler JR, et al. (2019) Global transcriptomic analysis of the Candida albicans response to treatment with a novel inhibitor of filamentation. mSphere 4: e00620-19.

224. Pathak AK, Sharma S, Shrivasstva P (2012) Multi-species Biofilm of Candida albicans and non-Candida albicans Candida species on acrylic substrate. J Appl Oral Sci 20: 70-75.

225. Cavalcanti IM, Del Bel Cury AA, Jenkinson HF, Nobbs AH (2017) Interactions between Streptococcus oralis, Actinomyces oris, and Candida albicans in the development of multispecies oral microbial biofilms on salivary pellicle. Mol. Oral Microbiol 32: 60-73.

226. Branting C, Sund ML, Linder LE (1989) The influence of Streptococcus mutans on adhesion of Candida albicans to acrylic surfaces in vitro. Arch Oral Biol 34: 347-353.

227. Pereira-Cenci T, Deng DM, Kranesveld EA, Manders EMM, Del Bel Cury AA, et al. (2008) The effect of Streptococcus mutans and Candida glabrata on Candida albicans biofilms formed on different surfaces. Arch Oral Biol 53: 755-764.

228. Yang C, Soffiefield J, Wu R, Deivanayagam C, Zou J, et al. (2018) Antigen I/II mediates interactions between Streptococcus mutans and Candida albicans. Mol Oral Microbiol 33: 283-291.

229. Montelongo-Jauregui D, Srinivasan A, Ramasubramanian A, Lopez-Ribot J (2018) An in vitro model for Candida albicans-Streptococcus gordonii biofilms on titanium surfaces. J Fungi (Basel) 4: E66.

230. Ujaonye S, Chandra J, Faddoul F, Chane M, Wang J, et al. (2014) In Vitro Effect of Over-the-Counter Probiotics on the Ability of Candida Albicans to Form Biofilm on Denture Strips. J Dent Hyg 88: 183-189.

231. Diaz PI, Xie Z, Sobue T, Thompson A, Blykoglu B, et al. (2012) Synergistic interaction between Candida albicans and commensal oral streptococci in a novel in vitro mucosal model. Infect Immun 80: 620-632.

232. Cavalcanti YW, Wilson M, Lewis M, Del-Bel-Cury AA, Silva WJ da, et al. (2016) Modulation of Candida albicans virulence by bacterial biofilms on titanium surfaces. Biofouling 32: 123-134.

233. Ishijima SA, Hayama K, Burton JP, Reid G, Okada M, et al. (2012) Effect of streptococcus salivarius K12 on the in vitro growth of candida albicans and its protective effect in an oral candidiasis model. Appl Environ Microbiol 78: 2190-2199.

234. Peters BM, Ovchinnikova ES, Krom BP, Schlecht LM, Zhou H, et al. (2012) Staphylococcus aureus adherence to Candida albicans hyphae is mediated by the hyphal adhesin Als3p. Microbiology 158: 2975-2986.

235. Kong EF, Tsui C, Kuchariková S, Andes D, Van Dijck P, et al. (2016) Commensal protection of staphylococcus aureus against antimicrobials by candida albicans biofilm matrix. mBio 7: e01365-01416.

236. Staat RH, Gawronski TH, Cresse D, Harris RS, Folke LEA (1975) Effects of dietary sucrose levels on the quantity and microbial composition of human dental plaque. J Dent Res 54: 872-880.

237. Xu H, Sobue T, Bertolini M, Thompson A, Vickerman M, et al. (2017) S. oralis activates the Efg1 filamentation pathway in C. albicans to promote cross-kingdom interactions and mucosal biofilms. Virulence 1: 8: 1602-1617.

238. Bertolini MM, Xu H, Sobue T, Nobile CJ, Del Bel Cury AA, et al. (2015) Candida-streptococcal mucosal biofilms display distinct structural and virulence characteristics depending on growth conditions and hyphal morphotypes. Mol Oral Microbiol 30: 307-322.

239. Garbaczka K, Jarzembowski T, Kwapisza E, Dacacand A, Witkowski J (2018) Do the oral Staphylococcus aureus strains from denture wearers have a greater pathogenicity potential?. Journal of oral microbiology 11: 1536193.

240. Krzyściak W, Kościelnia D, Papież F, Vyhouskaya P, Zagórska-Swieży K, et al. (2017) Effect of a Lactobacillus salivarius probiotic on multispecies oral streptococcal biofilms on titanium surfaces. J Fungi (Basel) 4: E66.

241. Diaz PI, Xie Z, Sobue T, Thompson A, Blykoglu B, et al. (2012) Synergistic interaction between Candida albicans and commensal oral streptococci in a novel in vitro mucosal model. Infect Immun 80: 620-632.

242. Cavalcanti YW, Wilson M, Lewis M, Del-Bel-Cury AA, Silva WJ da, et al. (2016) Modulation of Candida albicans virulence by bacterial biofilms on titanium surfaces. Biofouling 32: 123-134.

243. Ishijima SA, Hayama K, Burton JP, Reid G, Okada M, et al. (2012) Effect of streptococcus salivarius K12 on the in vitro growth of candida albicans and its protective effect in an oral candidiasis model. Appl Environ Microbiol 78: 2190-2199.

244. Peters BM, Ovchinnikova ES, Krom BP, Schlecht LM, Zhou H, et al. (2012) Staphylococcus aureus adherence to Candida albicans hyphae is mediated by the hyphal adhesin Als3p. Microbiology 158: 2975-2986.

245. Kong EF, Tsui C, Kuchariková S, Andes D, Van Dijck P, et al. (2016) Commensal protection of staphylococcus aureus against antimicrobials by candida albicans biofilm matrix. mBio 7: e01365-01416.

246. Staat RH, Gawronski TH, Cresse D, Harris RS, Folke LEA (1975) Effects of dietary sucrose levels on the quantity and microbial composition of human dental plaque. J Dent Res 54: 872-880.

247. Xu H, Sobue T, Bertolini M, Thompson A, Vickerman M, et al. (2017) S. oralis activates the Efg1 filamentation pathway in C. albicans to promote cross-kingdom interactions and mucosal biofilms. Virulence 1: 8: 1602-1617.

248. Bertolini MM, Xu H, Sobue T, Nobile CJ, Del Bel Cury AA, et al. (2015) Candida-streptococcal mucosal biofilms display distinct structural and virulence characteristics depending on growth conditions and hyphal morphotypes. Mol Oral Microbiol 30: 307-322.

249. Garbaczka K, Jarzembowski T, Kwapisza E, Dacacand A, Witkowski J (2018) Do the oral Staphylococcus aureus strains from denture wearers have a greater pathogenicity potential?. Journal of oral microbiology 11: 1536193.
Citation: Le Bars P, Jordana F, Kouadio A (2021) Denture Plaque Management of Denture-Related Stomatitis. Dent Adv Res 6: 173. DOI: 10.29011/2574-7347.100073

244. Wu T, Cen L, Kaplan C, Zhou X, Lux R, et al. (2015) Cellular Components Mediating Coadherence of Candida albicans and Fusobacterium nucleatum. J Dent Res 94: 1432-1438.

245. Jabra-Rizk MA, Falkler WA, Merz WG, Kelley JI (1999) Coaggregation of Candida dubliniensis with Fusobacterium nucleatum. J Clin Microbiol 37: 1464-1468.

246. Elving GJ, van Der Mei HC, Busscher HJ, van Weissenbruch R, Albers FW (2001) Air-flow resistances of silicone rubber voice prostheses after formation of bacterial and fungal biofilms. J Biomed Mater Res 58: 421-426.

247. van der Mei HC, Buisjes KJDA, van der Laan BFAM, Ovchinikova E, Geertsema-Doombusch GI, et al. (2014) Voice prosthetic biofilm formation and candida morphogenic conversions in absence and presence of different bacterial strains and species on silicone-rubber. PLoS One 9: e104508.

248. Bachtiar EW, Bachtiar BM, Jarosz LM, Amir LR, Sunarto H, et al. (2014) AI-2 of Aggregatibacter actinomycetemcomitans inhibits candida albicans biofilm formation. Front Cell Infect Microbiol 4: 94.

249. Dahlén G, Blomqvist S, Almståhl A, Carlén A (2012) Virulence factors and antibiotic susceptibility in enterococci isolated from oral mucosal and deep infections. J Oral Microbiol 4: 10.

250. Sztukowska MN, Dutton LC, Delaney C, Ramsdale M, Ramage G, et al. (2018) Community development between Porphyromonas gingivalis and Candida albicans Mediated by InlJ and Als3. mBio 9: e00202-18.

251. Morse DJ, Wilson MJ, Wei X, Bradshaw DJ, Lewis MAO, et al. (2019) Modulation of Candida albicans virulence in in vitro biofilms by oral bacteria. Lett Appl Microbiol 68: 337-343.

252. Cavalcanti IMG, Nobbs AH, Ricomini-Filho AP, Jenkinson HF, Del Bel Cury AA (2016) Interkingdom cooperation between Candida albicans, Streptococcus oralis and Actinomyces oris modulates early biofilm development on denture material. Pathog Dis 74: ftw002.

253. Uppuluri P, Busscher HJ, Chakladar J, van der Mei HC, Chaffin WL (2017) Transcriptional profiling of C. albicans in a two species biofilm with Rothia dentocariosa. Front Cell Infect Microbiol 7: 311.

254. Cruz MR, Graham CE, Gagliano BC, Lorenz MC, Garsin DA (2013) Enterococcus faecalis inhibits hyphal morphogenesis and virulence of Candida albicans. Infect Immun 81: 189-200.

255. Graham CE, Cruz MR, Garsin DA, Lorenz MC (2017) Enterococcus faecalis bacteriocin EntIV inhibits hyphal morphogenesis, biofilm formation, and virulence of Candida albicans. Proc Natl Acad Sci USA 114: 4507-4512.

256. Haverman TM, Laheij AMGA, de Soet JJ, de Lange J, Rozema FR (2017) Candida and Porphyromonas gingivalis: the effect on wound closure in vitro. J Oral Microbiol 9: 1328266.

257. Bor B, Cen L, Agnello M, Shi W, He X (2016) Morphological and physiological changes induced by contact-dependent interaction between Candida albicans and Fusobacterium nucleatum. Sci Rep 6: 27956.

258. Dutton LC, Jenkinson HF, Lamont RJ, Nobbs AH (2016) Role of Candida albicans secreted aspartyl protease Sap8 in interkingdom biofilm formation. Pathog Dis 74: ftw005.

259. Kraft-Bodi E, Jørgensen MR, Keller MK, Kragelund C, Twetman S (2015) Effect of Probiotic Bacteria on Oral Candida in Frail Elderly. J Dent Res 94: 181-186.

260. Jørgensen MR, Kragelund C, Jensen PØ, Keller MK, Twetman S (2017) Probiotic Lactobacillus reuteri has antifungal effects on oral Candida species in vitro. J Oral Microbiol 9: 1274582.

261. Lee X, Vergara C, Lozano CP (2019) Severity of Candida-associated denture stomatitis is improved in institutionalized elders who consume Lactobacillus rhamnosus SP1. Aust Dent J 64: 229-236.

262. de Senna AM, Vieira MMF, Machado-de-Sena RM, Bertolin AO, Núñez SC, et al. (2018) Photodynamic inactivation of Candida spp. on denture stomatitis. A clinical trial involving palatal mucosa and prophylaxis disinfection. Photodiagnosis Photodyn Ther 22: 212-216.

263. Tsutsumi-Arai C, Arai Y, Terada-Itö C, Takebe Y, Ide S, et al. (2019) In addition, mouthwashes remain essential for the treatment of stomatitis and for the maintenance of oral health. Lasers Med Sci doi: 10.1007/s10103-019-02751-2.

264. Koo H, Allan RN, Howlin RP, Stoodley P, Hall-Stoodley L (2017) Targeting microbial biofilms: current and prospective therapeutic strategies. Nat Rev Microbiol 15: 740-764.

265. Bowen WH, Koo H (2011) Biology of Streptococcus mutans derived glucosyltransferases: role in extracellular matrix formation of cariogenic biofilms. Caries Res 45: 69-86.

266. Nishimura J, Saito T, Yoneyama H, Bai LL, Okumura K, et al. (2012) Biofilm formation by Streptococcus mutans and related bacteria. Adv Microb 2: 208-215.

267. Koo H, Falsetta ML, Klein MI (2013) The exopolysaccharide matrix: a virulence determinant of cariogenic biofilm. J Dent Res 92: 1065-1073.

268. Rössing CK, Cavagni J, Gaio EJ, Muniz FWMG, Ranzan N, et al. (2017) Efficacy of two mouthwashes with cetylpyridinium chloride: a controlled randomized clinical trial. Braz Oral Res 31: e47.

269. Haydari M, Bardacki AG, Koldsland OC, Aass AM, Sandvik L, et al. (2017) Comparing the effect of 0.06%, 0.12% and 0.2% Chlorhexidine on plaque, bleeding and side effects in an experimental gingivitis model: a parallel group, double masked randomized clinical trial. BMC Oral Health 17: 118.

270. Paulone S, Malavasi G, Ardizzoni, Orsi CF, Peppoloni S, et al. (2017) Candida albicans survival, growth and biofilm formation are differently affected by mouthwashes: an in vitro study. New Microbiol 40: 45-52.

271. Hayran Y, Sarikaya I, Aydin A, Tekin YH (2018) Determination of the effective anticandidal concentration of denture cleanser tablets on some denture base resins. J Appl Oral Sci 26: e20170077.

272. Sesma N, Rocha AL, Lagana DC, Costa B, Morimoto S, et al. (2017) Efficacy of two mouthwashes with cetylpyridinium chloride: a controlled randomized clinical trial. Braz Oral Res 31: e47.

273. Wagner DA, Pipko DJ (2015) The effect of repeated microwave irradiation on the dimensional stability of a specific acrylic denture resin. J Prosthodont Off J Am Coll Prosthodont 24: 25-31.