Available online at www.ujpronline.com
Universal Journal of Pharmaceutical Research
An International Peer Reviewed Journal
ISSN: 2831-5235 (Print); 2456-8058 (Electronic)

RESEARCH ARTICLE

EFFECT OF REMOVABLE DENTURES ON COLONIZATION OF AEROBIC BACTERIA IN THE ORAL CAVITY AND ANTIBIOTIC PATTERN OF THE COMMON ISOLATED BACTERIA

Ibrahim Zaid Al-Shami[1], Yaser Ahmed Salem Alrubaidi[2], Khaled A AL-Haddad[3], Mohammed Mohammed Ali Al-Najhi[3,4], Hassan Abdulwahab Al-Shamahy[5,6], Taghreed Ahmed M Al-Kibsi[2], Abdulwahab Ismail Mohamed Al-Kholani

[1]Department of Conservative Dentistry and Oral Health, Faculty of Dentistry, Sana'a University, Republic of Yemen,
[2]Department of Oral and Maxillo-Facial Surgery, Faculty of Dentistry, Sana'a University, Republic of Yemen.
[3]Orthodontics, Pedodontics and Prevention Department Faculty of Dentistry, Sana'a University, Yemen.
[4]Orthodontics, Pedodontics and Prevention Department Faculty of Dentistry, Genius University for Sciences & Technology.
[5]Departement of Basic Sciences, Faculty of Dentistry, Sana'a University, Republic of Yemen.
[6]Medical Microbiology department, Faculty of Medicine, Genius University for Sciences & Technology, Dhamar city, Republic of Yemen.

Article Info:

Article History:
Received: 4 October 2022
Reviewed: 13 November 2022
Accepted: 28 December 2022
Published: 15 January 2023

Cite this article:
Al-Shami IZ, Alrubaidi YAS, AL-Haddad KA, Al-Najhi MMA, Al-Shamahy HA, Al-Kibsi TAM, Al-Kholani AIM. Effect of removable dentures on colonization of aerobic bacteria in the oral cavity and antibiotic pattern of the common isolated bacteria. Universal Journal of Pharmaceutical Research 2022; 7(6):1-8.
https://doi.org/10.22270/ujpr.v7i6.862

***Address for Correspondence:**
Dr. Hassan A. Al-Shamahy, Faculty of Medicine and Heath Sciences, Sana'a University. Faculty of Medicine, Genius University for Sciences and Technology Dhamar/Sana'a, Yemen; Tel: +967-1-239551.
E-mail: *shmahe@yemen.net.ye*

Abstract

Background and aims: Wearing a removable dental prosthesis causes a change in the micro flora of the mouth. The aim of this research was to verify the composition of aerobic bacterial in the oral cavity of patients with removable dentures and with normal teeth (without dentures), and antibiotics pattern for common isolates.

Methods: Bacteriological investigations were performed in 122 individuals (61 removable dentures: 61 normal teeth) attending dental clinics of Faculty of dentistry, Sana'a University, Yemen and private dental clinics. The culturing and antibiotic sensitivity were conducted in the Microbiology Department of the National Center of Public Health Laboratories (NCPHL) Sana'a, Yemen. Cultivation in microaerophilic (5% CO2) and oxygenic conditions were performed on solid selective and non-selective media in addition to media enriched with 5% blood.

Results: Regarding the prosthetic patients, the rate of bacterial isolates from the palate, back, tongue and dental plaque smears was higher potential pathogenic bacteria as *S. aureus* and *Enterobacteriaceae spp* in denture wearers, *as E. coli* (6.6% in dentures vs. 1.6% in the absence of dentures), *Klebsiella pneumoniae* (11.4% in dentures versus 1.6% in the normal teeth) and *Pseudomonas aeruginosa* (13.1% versus 0.0%).While in *Streptococcus viridians* including *Streptococcus mutans*, there was a lower colonization rate in denture patients (18% in palate verses, 73.8% in individuals without dentures).

Conclusion: The study demonstrated an elevated rate of bacterial isolates from palate, back, tongue and plaque swabs in denture patients of pathogenic bacteria such as *S. aureus*, *E. coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*; while in *Streptococcus viridians* including *S. mutans*, there was a lower colonization rate in denture patients verses a very high rate in individuals without dentures.

Keywords: Antibiotic susceptibility testing, micro flora of the mouth, normal teeth, oral cavity, removable dental prosthesis.

INTRODUCTION

Oral microbiology is the study of microorganisms in the oral cavity and their interfaces with other microorganisms in the oral cavity or with the host itself[1]. The environment in the human mouth is accurate for the growth of the distinctive microorganisms existing there since it affords a source of nutrients and water, in addition to a temperate temperature[2]. Oral-resident microbes adhere to the teeth and gums and in unison to counteract the mechanical flow from the mouth to the stomach where

acid-sensitive microbes are destroyed by hydrochloric acid[3]. Researchers found that oral bacteria have evolved mechanisms to influence their environment and avoid or modify the host's oral environment. Bacteria occupy the ecological niche provided by dental surfaces and mucosal epithelium[4,5]. A noteworthy factor that has been found to affect bacterial colonization in the oral cavity is the pH and oxygen concentration and availability on some surfaces of the mouth, which means that the loss of teeth and their replacement with dentures may lead to a change in their structure; As well as the mechanical forces acting on the surfaces of the mouth, the flow of saliva and fluids through the oral cavity, and ages of the host[5]. In spite of this, a highly effective innate host defense system constantly monitors bacterial colonization and prevents bacterial invasion of local tissues. There is a dynamic balance linking dental plaque bacteria and the host's innate defense system[4]. Of specific interest is the role of the oral micro biota in the two major dental diseases: periodontal diseases and dental caries[4]. Furthermore, research has connected deprived oral health and the resultant ability of oral bacteria to attack the body to affect heart health as well as cognitive function[6]. Wearing a removable dental prosthesis produces an alteration in oral bacteria[7,8]. For a number of individuals, this diverse environment is accountable for the development of a specific circumstance: denture-associated stomatitis and prosthetic stomatitis. Stomatitis is described by inflammation of the mucous membrane and redness under the dentures[3]. It is started by a microbial biofilm on the suitable surface of the denture from the surface of the mucosa, for example, the palate[9]. Denture-associated stomatitis (DAS) is one of the majority common irrefutable symptoms of oral candidiasis[10], and involves 24-60% of well-worn dentures[11]. Roughly 90% of cases are believed to be caused by yeast[9,12], typically *C. albicans*, even though lesions have also been connected with a diversity of further *Candida* species[10-13] over and above bacteria from numerous genera[3,10,14,15].

Nowadays, antimicrobial resistance (AMR) is the most important public health threat[16-20], and AMR bacteria in various hospital departments are increasing exponentially[21-23]. According to a published study, 700,000 deaths by reason of antimicrobial resistance are described per annum, and it has been estimated that if proper control and prevention measures are not taken, antimicrobial resistance will turn out to be one of the most important causes of death among non-hospitalized or hospitalized patients all over the world[24].

Oral bacteria such as *Staphylococcus aureus*, *Streptococcus viridians* group, and *Enterobacterace*a are also included as causative agent of systemic infections such as endocarditis, pneumonia, etc., so information on antibiotic profile is of importance in prescribing appropriate treatment in case of infection[25]. The aim of this study was to determine the aerobic bacterial composition of the oral cavity of patients with removable dentures and normal teeth individuals (without dentures), and to determine the antibiotic pattern of common isolates including *S. aureus* and *S. viridians* group.

MATERIALS AND METHODS

Bacteriological examinations were performed on 122 individuals (61 removable dentures: 61 natural teeth) in the dental clinics of the Faculty of Dentistry, Sana'a University, Yemen and private dental clinics (Al-Mortadda Dental Clinics, Al-Kahara Dental Clinics) in Sana'a, over a period of 3 months, which began in December 2021 and ended in February 2022.

Microbiological procedure

Cultivation and sensitivity to antibiotics were performed at the Microbiology Department of the National Center for Public Health Laboratories (NCPHL) Sana'a, Yemen. Swabs were collected from the mucous membrane of the palate and the tongue dorsa from dentures and natural teeth individuals, as well as swabs from the mucous part of the denture surfaces in prosthetic patients. Cultures were performed under oxygenated and microaerophilic conditions (5% CO_2) on selective and non-selective solid media as well as media enriched with 5% blood. Standard procedures for bacterial culture and identification[26] were applied.

Antibiogram: The antibiotic susceptibility profile was determined by disc diffusion method. The inoculums were adjusted to match the turbidity of 0.5 McFarland standards, and was swabbed on Brian heart infusion agar and allowed to dry for 10 min[27]. Then antibiogram profiling was performed to determine the susceptibility of 4 β-lactam antibiotics (Amoxicillin-Clavulanic Acid, Oxacillin (1 µg), Cloxacillin (2 µg), and Cefoxtine (30 µg) and 8 non β-Lactam antibiotics (erythromycin (15 µg), gentamicin (10 µg), amikacin (30 µg), ciprofloxacin (5 µg), clindamycin (2 µg) and vancomycin (30 µg)) (Oxide, USA) by disc diffusion method. Inhibition zone was measured after 24 h of aerobically incubation at 37°C. The experiments of each antibiotic were performed in triplicate. The results were interpreted according to Clinical and Laboratory Standards Institute (CLSI) methodology[28].

Ethical Consideration: Ethical approval for this study, No: 1771 dated September 11, 2021 was obtained from the Medical Ethics and Research Committee of the Faculty of Medicine and Health Sciences, Sana'a University. All procedures were according to the ethical guidelines of the review committee. A written informed consent was obtained from the selected participants.

RESULTS

There was an increased rate of colonization of *S. aureus* in denture patients (11.5% in the palate) versus 1.6% in individuals without dentures. While there was a decrease in the rate of Coagulase-negative colonization in denture patients (16.4% in the tongue) and a higher incidence of Coagulase (47.5%) in people without dentures. In viridians (apathy) *Streptococcus* including *S. mutans*, there was a lower colonization rate in denture patients equal to 18% in palate verses, a

very high rate (73.8%) in individuals without dentures. Also, potentially pathogenic *Enterobacteriaceae spp* bacteria were more colonized in denture patients than in individuals without dentures: eg *E. coli* (6.6% in dentures vs. 1.6% in the absence of dentures), *K. pneumoniae* (11.4% in dentures versus 1.6% in the absence of dentures) and *P. aeruginosa* (13.1% in the dentures versus 0.0% in the absence of dentures).

Table 1: Isolation frequency (%) of bacteria in hard palate and tongue dorsa of denture wearers and non-denture wearing patients.

Bacteria	Denture n=61			No-denture n=61		Total isolates
	Palate N (%)	Tongue N (%)	Denture plague	Palate	Tongue	
S. aureus	7 (11.5%)	9 (14.8%)	8 (13.1%)	1 (1.6%)	5 (8.2%)	30
Coagulase-negative	9 (14.8%)	10 (16.4%)	12 (19.7%)	13 (21.3%)	29 (47.5%)	73
Streptococci						
S. pyogens	2 (3.3%)	0 (0.0%)	0 (0.0%)	1 (1.6%)	0 (0.0%)	3
S. mitior	9 (14.8%)	13 (21.3%)	2 (3.3%)	16 (26.2%)	8 (13.1%)	47
S. sanguis	10 (16.4%)	15 (24.6%)	3 (4.9%)	13 (21.3%)	8 (13.1%)	49
S. mutans	11 (18%)	13 (21.3%)	2 (3.3%)	45 (73.8%)	41 (68.9%)	112
S. alivarius	15 (24.6%)	12 (19.7%)	3 (4.9%)	15 (24.6%)	8 (13.1%)	53
S. milleri	1 (%)	1 (1.6%)	0 (0%)	2 (3.3%)	7 (11.5%)	9
Neisseria species	41 (67.2%)	45 (73.8%)	9 (14.8%)	40 (65.6%)	48 (78.7%)	183
Haemophilus influenza	1 (1.6%)	1 (1.6%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2
H. parainfluenzae	5 (8.2%)	7 (11.5%)	8 (13.1%)	12 (19.7%)	9 (14.8%)	41
Enterobacteriaceae spp.						
E. coli	3 (4.9%)	4 (6.6%)	2 (3.3%)	1 (1.6%)	1 (1.6%)	11
K. pneumoniae	7 (11.4%)	6 (9.8%)	3 (4.9%)	1 (1.6%)	1 (1.6%)	18
M. morganii	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)	2 (3.3%)	2
E. cloacae	2 (3.3%)	2 (3.3%)	0 (0 %)	0 (0.0%)	0 (0%)	4
C. freundii	1 (1.6 %)	1 (1.6%)	0 (0 %)	0 (0%)	0 (0 %)	2
P. aeruginosa	8 (13.1%)	7 (11.4%)	10 (16.4%)	0 (0.0%)	0 (0.0%)	25

Table 2: Antibiotic patterns of *S. aureus* isolated from hard palate and tongue dorsa of denture wearers and non-denture wearing patients (n=30 isolates).

Antibiotic name	Antibiotics/classes	Resistance N (%)	Sensitive N (%)
Tetracycline	Tetracycline	25 (83.3)	5 (16.7)
Erythromycin	Macroloides	21 (73.3)	9 (26.7)
Co-trimoxazole	sulfonamides	12 (40)	18 (60)
Amoxicillin-Clavulanic Acid	β-lactamase inhibitor combinations	11 (36.7)	19 (62.3)
Gentamicin	Aminogylcosides	11 (36.7)	19 (62.3)
Oxacillin	Penicillin's	6 (20)	24 (80)
Ciprofloxacin	Fluoroquinolones	4 (13.3)	26 (86.7)
Cloxacillin	Penicillin -stable penicillin	3 (10)	27 (90)
Cefoxtine	2nd Cephalosporins β–lactam	2 (6.7)	28 (93.3)
Amikacin	Aminogylcosides	2 (6.7)	28 (93.3)
Clindamycin	Lincosamides	1 (3.3)	29 (96.7)
Vancomycin	Glycopeptides	1 (3.3)	29 (96.7)

S. aureus showed a high rate of resistant to tetracycline (83.3%), erythromycin (73.3%) and co-trimoxazole (40%), while isolates showed high frequency of sensitive to vancomycin (96.7%), clindamycin (96.7%), amikacin (93.3%), cefoxtine (93.3%) and cloxacillin (90%). CoNS showed a high rate of resistance to tetracycline (86.3%), erythromycin (80.8%) and co-trimoxazole (46.6%), while the isolates showed a high frequency of sensitivity to vancomycin (94.5%), clindamycin (94.5%), cloxacillin (93.2%), ciprofloxacin (87.7%), amikacin (86.3%), gentamicin (869.7%), and cefoxetine (79.5%). *S. mutans* showed a moderate rate of resistance to tetracycline (56.3%), and low rate of resistance to erythromycin (12.5%), co-trimoxazole (13.4%) and oxacillin (11.6%), while the isolates showed a high frequency of sensitivity to vancomycin (97.3%), clindamycin (99.1%), cloxacillin (98.2%), ciprofloxacin (97.3%), amikacin (98.2%), gentamicin (94.6%), and cefoxetine (95.5%). *S. mitior* showed a moderate rate of resistance to tetracycline (44.7%), and low rate of resistance to erythromycin (17%), and co-trimoxazole (10.6%), while the isolates showed a high frequency of sensitivity to vancomycin (97.9%), clindamycin (100%), cloxacillin (97.9%), ciprofloxacin (97.9%), amikacin (97.9%), gentamicin (97.9%), and cefoxetine (97.9%). *S. sanguis* showed a moderate rate of resistance to tetracycline (40.8%), and low rate of resistance to erythromycin (18.3%), and co-trimoxazole (14.3%), while the isolates showed a high frequency of sensitivity to vancomycin (98%), clindamycin (100%), cloxacillin (100%), ciprofloxacin (98%), amikacin (98%), gentamicin (95.9%), and cefoxetine (95.9%). *S. alivarius* showed a moderate rate of resistance to tetracycline (58.5%), and low rate

of resistance to erythromycin (22.6%), and co-trimoxazole (20.8%), while the isolates showed a high frequency of sensitivity to vancomycin (98.1%), clindamycin (100%), cloxacillin (100%), ciprofloxacin (98.1%), amikacin (98.1%), gentamicin (96.2%), and cefoxetine (96.2%).

Table 3: Antibiotic patterns of Coagulase-negative staphylococcus isolated from hard palate and tongue dorsa of denture wearers and non-denture wearing patients (n=73 isolates).

Antibiotic name	Antibiotics/classes	Resistance N (%)	Sensitive N (%)
Tetracycline (30 µg)	Tetracycline	63 (86.3)	10 (13.7)
Erythromycin 15 µg	Macroloides	59 (80.8)	14 (19.2)
Co-trimoxazole (23.75 µg)	sulfonamides	34 (46.6)	39 (53.4)
Amoxicillin-Clavulanic Acid	β -lactamase inhibitor combinations	29 (39.7)	42 (60.3)
Oxacillin (1 µg)	Penicillin's	24 (32.9)	49 (67.1)
Cefoxtine (30 µg)	2nd Cephalosporins β–lactam	15 (20.5)	58 (79.5)
Gentamicin (10 µg)	Aminogylcosides	10 (13.7)	63 (86.3)
Amikacin (30 µg)	Aminogylcosides	10 (13.7)	63 (86.3)
Ciprofloxacin (5 µg)	Fluoroquinolones	9 (12.3)	62 (87.7)
Cloxacillin (2 µg)	Penicillin -stable penicillin	5 (6.8)	68 (93.2)
Clindamycin (2 µg)	Lincosamides	4 (5.5)	69 (94.5)
Vancomycin (30 µg)	Glycopeptides	4 (5.5)	69 (94.5)

Table 4: Antibiotic patterns of *S. mutans* isolated from hard palate and tongue dorsa of denture wearers and non-denture wearing patients (n=112 isolates).

Antibiotic name	Antibiotics /classes	Resistance N (%)	Sensitive N (%)
Tetracycline	Tetracycline	63 (56.3)	49 (43.8)
Co-trimoxazole	sulfonamides	15 (13.4)	97 (86.6)
Erythromycin	Macroloides	14 (12.5)	98 (87.5)
Oxacillin	Penicillin's	13 (11.6)	99 (88.4)
Amoxicillin-Clavulanic Acid	β -lactamase inhibitor combinations	7 (6.3)	105 (93.7)
Gentamicin	Aminogylcosides	6 (5.4)	106 (94.6)
Cefoxtine	2nd Cephalosporins β –lactam	5 (4.5)	107 (95.5)
Ciprofloxacin	Fluoroquinolones	3 (2.7)	109 (97.3)
Vancomycin	Glycopeptides	3 (2.7)	109 (97.3)
Amikacin	Aminogylcosides	2 (1.8)	110 (98.2)
Cloxacillin	Penicillin -stable penicillin	2 (1.8)	110(98.2)
Clindamycin	Lincosamides	1 (0.9)	111 (99.1)

DISCUSSION

A large amount of the adult population wears full or partial dentures. Reasons connected with tooth loss – dental caries, loss of periodontal support, tooth-alveolar trauma, and history of dental care are additive over time, and thus wearing dentures is more associated with older age although it can sometimes be recorded at earlier ages[29]. Oral conditions especially connected with the wearing of dentures are denture-associated stomatitis (DAS)[9-15,30] of bacterial or *Candida* origins. In the current study there was an increased rate of *S. aureus* colonization in dentures patients (11.5% in the palate) versus 1.6% in individuals without dentures. While there was a lower incidence of Coagulase-negative colonization in dentures patients (16.4% in the tongue) and a higher incidence of Coagulase-negative (47.5%) in subjects without dentures. In *viridians* (apathy) *Streptococcus* including *S. mutans*, there was an 18% lower colonization rate in dentures in the verses of the palate, which is very high (73.8%) in individuals without dentures. The results of the current study are similar to those reported in previous studies in that there are many similarities in the microbial composition, and there were some significant differences between the compositions in the adult population wearing full or partial dentures and adults with intact teeth[29]. There are relatively few studies on dental microbiology, and the factors affecting their quantity and types at the present time, although most of them were conducted in the eighties of last century[29,31]. Recent studies have discussed dentures in the adult population wearing full or partial dentures and adults with normal teeth and factors effected oral microbia[8,29,32] and many publications have focused on *Candida* only[3,7,29,30]. Thus other groups of organisms may be overlooked in the mouth. This is especially true of obligate anaerobes, which are important if bad breath is the focus of study[29,32,33].

In the current study, potentially pathogenic *Enterobacteriaceae* spp bacteria were more colonized in denture patients than in individuals without dentures: eg *E. coli* (6.6% in dentures versus 1.6% in the absence of dentures), *K. pneumoniae* (11.4% in dentures versus 1.6% in the absence of dentures) and *P. aeruginosa* (13.1% in the dentures versus 0.0% in the absence of dentures). Also respiratory pathogens such as *S.*

aureus, *S. pneumoniae*, *Haemophilus influenzae*, and *H. parainfluenzae* were isolated in denture patients more than in normal individuals. Obtained result is similar to that reported by Tyrrell *et al*.,[33], Sumi *et al*.,[34], Goldberg *et al*.,[35] and Senpuku *et al*.,[36] where some uncommon microorganisms are found in oral microbiota but have been isolated from dentures and include respiratory pathogens such as *S. aureus*, *S. pneumoniae*, *H. influenzae*, *H. para-influenzae*, *E. coli*, *K. pneumoniae*, and *P. mirabilis*, *Enterobacter cloacae* and *P. aeruginosa*[33-36].

Table 5: Antibiotic patterns of *S. mitior* isolated from hard palate and tongue dorsa of denture wearers and non-denture wearing patients (n=47 isolates).

Antibiotic name	Antibiotics /classes	Resistance N (%)	Sensitive N (%)
Tetracycline	Tetracycline	21 (44.7)	28 (55.3)
Co-trimoxazole	sulfonamides	5 (10.6)	42 (89.4)
Erythromycin	Macroloides	8 (17)	39 (83)
Oxacillin	Penicillin's	5 (10.6)	42 (89.4)
Amoxicillin-Clavulanic Acid	β -lactamase inhibitor combinations	1 (2.1)	46 (97.9)
Gentamicin	Aminogylcosides	1 (2.1)	46 (97.9)
Cefoxtine	2nd Cephalosporins β – lactam	1 (2.1)	46 (97.9)
Ciprofloxacin	Fluoroquinolones	1 (2.1)	46 (97.9)
Vancomycin	Glycopeptides	1 (2.1)	46 (97.9)
Amikacin	Aminogylcosides	1 (2.1)	46 (97.9)
Cloxacillin	Penicillin -stable penicillin	1 (2.1)	46 (97.9)
Clindamycin	Lincosamides	0 (0)	47 (100)

Table 6: Antibiotic patterns of *S. sanguis* isolated from hard palate and tongue dorsa of denture wearers and non-denture wearing patients (n=49 isolates).

Antibiotic name	Antibiotics/classes	Resistance N (%)	Sensitive N (%)
Tetracycline	Tetracycline	20 (40.8)	29 (59.2)
Co-trimoxazole	sulfonamides	7 (14.3)	42 (85.7)
Erythromycin	Macroloides	9 (18.3)	40 (81.7)
Oxacillin	Penicillin's	7 (14.3)	42 (85.7)
Amoxicillin-Clavulanic Acid	β-lactamase inhibitor combinations	2 (4.1)	47 (95.9)
Gentamicin	Aminogylcosides	2 (4.1)	47 (95.9)
Cefoxtine	2nd Cephalosporins β–lactam	2 (4.1)	47 (95.9)
Ciprofloxacin	Fluoroquinolones	1 (2)	48 (98)
Vancomycin	Glycopeptides	1 (2)	48 (98)
Amikacin	Aminogylcosides	1 (2)	48 (98)
Cloxacillin	Penicillin-stable penicillin	0 (0)	49 (100)
Clindamycin	Lincosamides	0 (0)	49(100)

In a number of studies, 48% of sampled dentures harbored members of *Enterobacteriaceae*[35,36]. Inhalation pneumonia is a widespread cause of death among the debilitated elderly, and thus the role of dentures in harboring such potential pathogens may be important. A variety of potential respiratory pathogens had colonized the dentures (denture palague) of the examined patients, the predominant one being *Staphylococcus* spp. (33%), among them *S. aureus* contributes to 13.1%. The other putative respiratory pathogens were as follows: *H. parainfluenzae* (13.1%), *K. pneumoniae* (4.9%), and *P. aeruginosa* (16.4%) (Table 1). Dental plaque and tongue dorsa can serve as reservoirs for potential respiratory pathogens. Sumi *et al*.,[37,38] concluded that denture plaque can act as a reservoir for potential pathogens to facilitate colonization in the oropharynx, and suggests that denture hygiene condition is an important factor in encouraging oropharyngeal bacterial colonization.

It has been suggested that the surface of the tongue may also represent an additional, and probably more constant, reservoir of respiratory pathogens[37,38]. The majority of the antibiotics used in this study were usually prescribed by dentists[39,40]. The number of *streptococci* resistant to oral mutant is larger in people commonly exposed to antibiotics, even though resistant bacteria can also be established in healthy people who have not been in recent times treated with antibiotics[39]. β-Lactam antibiotics are the mainly commonly used chemo preventive agent's in general dental practice. However, penicillin resistance among oral *streptococci* is increasing[41].

The number of resistant oral *streptococci* is greater in people frequently exposed to antibiotics[42], although these bacteria may also be found in healthy subjects who have not been recently treated with an antimicrobial[43]. Bacterial resistance to antibiotics such as penicillin and other *β*-Lactam is a health issue in numerous parts of the world. In current study it was observed that a significant level of oxacillin resistance (11.6%) in *S. mutans* isolates.

Table 7: Antibiotic patterns of *S. alivarius* isolated from hard palate and tongue dorsa of denture wearers and non-denture wearing patients (n=53 isolates).

Antibiotic name	Antibiotics/classes	Resistance N (%)	Sensitive N (%)
Tetracycline	Tetracycline	31 (58.5)	22 (41.5)
Co-trimoxazole	sulfonamides	11 (20.8)	42 (79.2)
Erythromycin	Macroloides	12 (22.6)	41 (77.4)
Oxacillin	Penicillin's	9 (17)	44 (83)
Amoxicillin-Clavulanic Acid	β -lactamase inhibitor combinations	2 (3.8)	51 (96.2)
Gentamicin	Aminogylcosides	2 (3.8)	51 (96.2)
Cefoxtine	2nd Cephalosporins β–lactam	2 (3.8)	51 (96.2)
Ciprofloxacin	Fluoroquinolones	1 (1.9)	52 (98.1)
Vancomycin	Glycopeptides	1 (1.9)	52 (98.1)
Amikacin	Aminogylcosides	1 (1.9)	52 (98.1)
Cloxacillin	Penicillin-stable penicillin	0 (0)	53 (100)
Clindamycin	Lincosamides	0 (0)	53 (100)

The high prevalence of resistance to penicillin group in *S. mutans* in current study is like that previously observed in Yemen (14.9%)[16], South Africa and Spain in oral *S. viridans*[44,45]. Several in-vitro studies have demonstrated the capability to transfer penicillin resistance determinants among related species[46]. These mechanisms, together with selective antibiotic pressure, may play an important role in the emergence and spread of penicillin resistance in oral *streptococci*. Also, the significant level of penicillin group resistance (11.6%) in *S. mutans* clinical isolates in current study is similar to Pasquantonio *et al*.,[47] study that reported a significant level of penicillin resistance: 13.4% of 550 oral *streptococcal* clinical isolates, out of 50 isolates of *S. mutans* 14% were resistant to penicillin[47]. However, obtained result is lower than the rate of a study conducted in 2014 by Dhamodhar *et al*.,[48] in which 38% isolates of *S. mutans* showed a complete resistance to penicillin and ampicillin. One-hour prior dental procedure, the American Heart Association suggests antimicrobial prophylaxis for high-risk cardiovascular patients, such as amoxicillin (2g) as first choice and clindamycin (600mg) as a second choice[49]. Production of *β-lactamase* is, however, unusual for most of *streptococci*, where resistance is happening by slightly altered of penicillin binding proteins[50-52]. However, in current study we observed a significant level of tetracycline resistance (56.3%) in the isolates of *S. mutans;* and 13.4% for co-trimoxazole, and erythromycin (12.5%) and only 0.9% for clindamycin in the isolates. Thus, in this condition first choice should be going to clindamycin or cephalosporin's in which resistance to cephalosporins is less than 4.5% (Table 4). Ultimately, the resistant developed by *S. mutans* is obscure. Updated information on antibiotic susceptibility testing such as reported in the present study helps to notify pharmaceutical makers to design new strategies for effective prophylaxis against dental infections. This result also gives an ideal choice to the dentist to prescribe a suitable antibiotic in Yemen.

Limitations of the study

Verifying the composition of aerobic bacteria in the oral cavity of patients who have removable dentures and comparing them with those who have natural teeth in the world and Yemen has not been adequately studied. Conducting a prospective study to include more numbers of patients, studying anaerobic species, and testing other more numbers of antibiotics for common isolates.

CONCLUSIONS

The study demonstrated an elevated rate of bacterial isolates from palate, back, tongue and plaque swabs in denture patients of pathogenic bacteria such as *S. aureus* and *Enterobacteriaceae* spp such as *E. coli*, *K. pneumoniae*, and *P. aeruginosa*; while in *S. viridians* including *S. mutans*, there was a lower colonization rate in denture patients verses a very high rate in individuals without dentures. Also, the study demonstrates significant levels of antibiotics resistance in *S. aureus*, CoNs and *S. viridians* oral isolates in dental patients. Further study is required to know the minimum inhibitory concentration of *β*-Lactam and non *β*-Lactam antibiotics. In conclusion, denture hygiene is the obvious way to ensure that dentures stay clean. There are many oral hygiene products accessible for use by denture wearers.

ACKNOWLEDGEMENTS

The authors thank the Faculty of Dentistry, Sana'a University, Sana'a, and Yemen for their generous support.

AUTHOR'S CONTRIBUTIONS

Al-Shami IZ: writing original draft, methodology. **Alrubaidi YAS:** research design, data collection. **AL-Haddad KA:** statistical analysis, conceptualization. **Al-Shamahy HA:** editing, methodology, supervision. **Al-Najhi MMA:** methodology, investigation. **Al-Kibsi TAM:** formal analysis, conceptualization. **Al-Kholani AIM:** research design, data collection. Final manuscript was read and approved by all authors.

DATA AVAILABILITY

The datasets generated during this study are available from the corresponding author upon reasonable request.

CONFLICT OF INTEREST

None to declare.

REFERENCES

1. Schwiertz A. Microbiota of the Human Body: Implications in health and disease. Preface. Adv Exp Med Biol 2016; 902:v. PMID: 27532080.
2. Sherwood L, Willey J, Woolverton C. Prescott's Microbiology (9th ed.). New York: McGraw Hill 2013. 713-721. ISBN 9780073402406. OCLC 886600661.
3. Spratt D. 4.1. Dental plaque and bacterial colonization. In: Medical biofilms. Jass J, Surman S, Walker J, editors, John Wiley and Sons Ltd, 2003; 175-98.
4. Rogers AH (editor). Molecular Oral Microbiology, 2008. Caister Academic Press. ISBN 978-1-904455-24-0.
5. Richard J. Lamont, George Hajishengallis, Howard F. Jenkinson. Oral microbiology and immunology (2nd ed.). Washington, DC: ASM Press. 2014. ISBN 978-1-55581-673-5. OCLC 840878148.
6. Noble JM, Scarmeas N, Papapanou PN. Poor oral health as a chronic, potentially modifiable dementia risk factor: review of the literature. Current Neurol Neurosci Rep 2013; 13 (10): 384. *https://orcid.org/10.1007/s11910-013-0384-x*
7. Al-Kebsi AM, Othman AM, Al-Shamahy HA, *et al*. Oral C. albicans colonization and non-candida albicans candida colonization among University students, Yemen. Universal J Pharm Res 2017; 2(5): 5-9. *https://orcid.org/10.22270/ujpr.v2i5.R2*
8. Alhasani AH, Al-Akwa AAY, Al-Shamahy HA, *et al*. Biofilm formation and antifungal susceptibility of Candida isolates from oral cavity after the introduction of fixed orthodontic appliances. Universal J Pharm Res 2020; 5(4):1-8. *https://orcid.org/10.22270/ujpr.v5i4.435*
9. Al-deen HMS, Obeyah AA, Al-Shamahy HA, *et al*. Oral *Candida albicans* colonization rate in fixed orthodontics patients. Universal J Pharm Res 2020;5(2):1-6. *https://orcid.org/10.22270/ujpr.v5i2.380*
10. Lamfon H, Al-Karaawi Z, McCullough M, Porter SR, Pratten J. Composition of *in vitro* denture plaque biofilms and susceptibility to antifungals. FEMS Microbiol Lett 2005 Jan 15; 242(2):345-51. *https://orcid.org/10.1016/j.femsle.2004.11.032*
11. Al-Kebsi AM, Othman AM, Al-Shamahy HA, *et al*. Oral *C. albicans* colonization and non-candida albicans candida colonization among university students, Yemen. Universal J Pharm Res 2017; 2(5):1-5. *https://orcid.org/10.22270/ujpr.v2i5.R2*
12. Al-Sanabani NF, Al-Kebsi AM, Al-Shamahy HA, Abbas MA. Etiology and risk factors of stomatitis among Yemeni denture wearers. Universal J Pharm Res 2018; 3(1):1-5. *https://orcid.org/10.22270/ujpr.v3i1.R9*
13. Al-Haddad KA, Al-dossary OAE, Al-Shamahy HA. Prevalence and associated factors of oral non-candida albicans candida carriage in denture wearers in Sana'a city-Yemen. Universal J Pharm Res 2018;3(4):1-6. *https://orcid.org/10.22270/ujpr.v3i4.176*
14. Palmqvist S, Unell L, Lindquist B. Denture stomatitis in nursing home patients. Swedish Dent J, 1984; 8: 73-80.
15. Alhasani AH, Ishag RA, Al-Akwa AAY, Al-Shamahy HA, Al-labani MAA. Association between the *Streptococcus mutans* biofilm formation and dental caries experience and antibiotics resistance in adult females. Universal J Pharm Res 2021; 5(6):1-6. *https://orcid.org/10.22270/ujpr.v5i6.507*
16. Al-Shami HZ, Al-Haimi MA, Al-Shamahy HA, *et al*. Patterns of antimicrobial resistance among major bacterial pathogens isolated from clinical samples in two tertiary's hospitals, in Sana'a, Yemen. Universal J Pharm Res 2021; 6(5):60-67. *https://doi.org/10.22270/ujpr.v6i5.674*
17. WHO. Antimicrobial resistance: global report on surveillance 2014. WHO. WHO. Archived from the original on 15 May 2015. Retrieved 19 September 2022.
18. Dadgostar P. Antimicrobial Resistance: Implications and Costs. Infect Drug Resis 2019; 12: 3903–3910. *https://doi.org/10.2147/IDR.S234610*
19. CDC. The biggest antibiotic-resistant threats in the U.S. Centers for Disease Control and Prevention. 6 November 2019. Retrieved 19 September 2022.
20. Samuel S. Our antibiotics are becoming useless. Vox 2019. Retrieved 29 September 2022.
21. O'Brien TF, Clark A, Peters R, Stelling J. Why surveillance of antimicrobial resistance needs to be automated and comprehensive. J Glob Antimicrob Resist 2019 Jun; 17:8-15. *https://doi.org/10.1016/j.jgar.2018.10.011*
22. Pormohammad A, Nasiri MJ, Azimi T. Prevalence of antibiotic resistance in *Escherichia coli* strains simultaneo-usly isolated from humans, animals, food, and the environment: A systematic review and meta-analysis. Infect Drug Resis 2019; 12:1181. *https://doi.org/10.2147/IDR.S201324*
23. Tian L, Sun Z, Zhang Z. Antimicrobial resistance of pathogens causing nosocomial bloodstream infection in Hubei Province, China, from 2014 to 2016: A multicenter retrospective study. BMC Public Health 2018;18(1):1121. *https://doi.org/10.1186/s12889-018-6013-5*
24. Alhasani AH, Ishag RA, Al Shamahy HA, *et al*. Association between the *Streptococcus mutans* biofilm formation and dental caries experience and antibiotics resistance in adult females. Universal J Pharm Res 2020; 5(6):1-3. *https://doi.org/10.22270/ujpr.v5i5.478*
25. DeMoor CE, DeStoppelaar JD, Van Houte J. The occurrence of *S. mutans* and *S. sanguis* in the blood of endocarditis patients. Caries Res 1972; 6:73.
26. Cheesbrough M. District laboratory practice in tropical countries. Cambridge: Cambridge University Press; 2010. *https://doi.org/10.1017/CBO9780511581304*
27. Jebashree HS, Kingsley SJ, Sathish ES, Devapriya D. Antimicrobial activity of few medicinal plants against clinically isolated human cariogenic pathogens- An *in vitro* study. ISRN Dent 2011. *https://doi.org/10.5402/2011/541421*
28. CLSI. Performance Standards for Antimicrobial Disc Susceptibility Tests. (11th edn.), Approved standard M02-A11– Publication of Clinical and Laboratory Standards Institute [CLSI), 2012; USA, 32.
29. Verran J. Malodour in denture wearers: An ill-defined problem. Oral Dis 2005; 11 Suppl 1:24-8. *https://doi.org/10.1111/j.1601-0825.2005.01083.x*
30. Nikawa H, Hamada T, Yamamoto T. Denture plaque--Past and recent concerns. J Dent 1998; 26(4):299-304. *https://doi.org/10.1016/s0300-5712(97)00026-2*
31. Budtz-Jorgensen E, Theilade J, Zander HA. Method for studying the development, structure and microflora of denture plaque. Scand J Dent Res 1980; 89: 149-56.
32. Jass J, Surman S, Walker J, editors. Medical biofilms. John Wiley and Sons Ltd, 2003.
33. Tyrrell KL, Citron DM, Warren YA, Nachnani S, Goldstein EJ. Anaerobic bacteria cultured from the tongue dorsum of subjects with oral malodor. Anaerobe 2003; 9(5):243-6. *https://doi.org/10.1016/S1075-9964(03)00109-4*
34. Sumi Y, Miura H, Sunakawa M, Michiwaki Y, Sakagami N. Colonization of denture plaque by respiratory pathogens in dependent elderly. Gerodontol 2002 Jul;19(1):25-9. *https://doi.org/10.1111/j.1741-2358.2002.00025.x*
35. Goldberg S, Cardash H, Browning H 3rd, Sahly H, Rosenberg M. Isolation of Enterobacteriaceae from the mouth and potential association with malodor. J Dent Res 1997 Nov;76(11):1770-5.

https://doi.org/10.1177/00220345970760110801

36. Senpuku H, Sogame A, Inoshita E, Tsuha Y, Miyazaki H, Hanada N. Systemic diseases in association with microbial species in oral biofilm from elderly requiring care. Gerontol 2003 Sep-Oct;49(5):301-9. *https://doi.org/10.1159/000071711*
37. Sumi Y, Kagami H, Ohtsuka Y, Kakinoki Y, Haruguchi Y, Miyamoto H. High correlation between the bacterial species in denture plaque and pharyngeal microflora. Gerodontol 2003 Dec; 20(2):84-7. *https://doi.org/10.1111/j.1741-2358.2003.00084.x*
38. Sumi Y, Miura H, Nagaya M, Michiwaki Y, Uematsu H. Colonisation on the tongue surface by respiratory pathogens in residents of a nursing home--A pilot study. Gerodontol 2006 Mar;23(1):55-9. *https://doi.org/10.1111/j.1741-2358.2006.00093.x*
39. Salman HA, Senthikumar R. Identification and antibiogram profile of *Streptococcus mutans* and *Streptococcus sobrinus* from dental caries subjects. Contemp Clin Dent 2017; 5(06): 054-057. *https://doi.org/10.7324/JAPS.2015.50608*
40. Sweeney LC, Dave J, Chambers PA, Heritage J. Antibiotic resistance in general dental practice--A cause for concern? J Antimicrob Chemother 2004 Apr;53(4):567-76. *https://doi.org/10.1093/jac/dkh137*
41. European Committee on Antimicrobial Susceptibility Testing [EUCAST) Clinical breakpoints, 2010.
42. Escribano E, Linares J, Alcaide F, *et al*. Increasing antimicrobial resistance among blood isolates of viridans" *Streptococcus*. American Society for Microbiology 1990; Washington DC, USA.
43. Tozer RA, Boutflower S, Gillespie WA. Antibiotics for the prevention of bacterial endocarditis during dental treatment. Lancet 1966; 1: 686-688. *https://doi.org/10.1016/S0140-6736(66)91629-1*
44. Working Party of the British Society for the Antimicrobial Chemotherapy. The antibiotic prophylaxis of infective endocarditis. Lancet 1982; 2: 1323-1326.
45. Potgieter E, Chalkley LJ. Reciprocal transfer of penicillin resistance genes between *Streptococcus pneumoniae*, *Streptococcus mitior* and *Streptococcus sanguis*. J Antimicrob Chemother. 1991 Sep;28(3):463-5. *https://doi.org/10.1093/jac/28.3.463*
46. Sanchez R, Munoz P, Rodriguez-Creixems M, *et al*. Susceptibility pattern of *Streptococcus viridans* group isolated from blood cultures. 1994; American Society for Microbiology, Washington DC, USA.
47. Pasquantonio G, Condò S, Cerroni L, Bikiqu L, Nicoletti M, Prenna M, Ripa S. Antibacterial activity of various antibiotics against oral streptococci isolated in the oral cavity. Int J Immunopathol Pharmacol 2012 Jul-Sep;25(3):805-9. *https://doi.org/10.1177/039463201202500331*
48. Dhamodhar P, Sreenivasa Murthy, Channarayappa, Shanthakumar SS, Indiresha HN. Prevalence, characterization and heterogeneity studies on *Streptococcus mutans* isolated from Bangalore urban population. Int J Pharm Bio Sci 2014; 5(3): 122-128.
49. Dajani AS, Taubert KA, Wilson W. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. J Am Dent Assoc 1997; 96: 358-366. *https://doi.org/10.1161/01.CIR.96.1.358*
50. Chambers HF. Penicillin-binding protein-mediated resistance in pneumococci and staphylococci. J Infect Dis. 1999 Mar;179 Suppl 2:S353-9. *https://doi.org/10.1086/513854*
51. Cvitkovitch DG. Genetic competence and transformation in oral streptococci. Crit Rev Oral Biol Med. 2001; 12(3):217-43. *https://doi.org/10.1177/10454411010120030201*
52. Hakenbeck R. Mosaic genes and their role in penicillin-resistant *Streptococcus pneumoniae*. Electrophoresis. 1998 Apr; 19(4):597-601.*https://doi.org/10.1002/elps.1150190423*