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## RESEARCH ARTICLE

## THE EFFECT OF DENTAL IMPLANTS ON AEROBIC BACTERIA COLONIZATION IN THE ORAL CAVITY AND THE ANTIBIOTIC PROFILE OF COMMON ISOLATED AEROBIC BACTERIA

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## ABSTRACT

**Background and aims:** The mouth's microflora may alter as a result of dental implants. The purpose of this study was to examine the composition of aerobic bacteria in patients with dental implants and those who had natural teeth (without implants) as well as the response of those bacteria to antibiotic treatment.

**Methods:** Bacteriological tests were performed on 72 patients (36 dental implants and 36 natural teeth) who visited dental clinics run by Sana'a University's Faculty of Dentistry and private dental clinics. Antibiotic susceptibility tests and culture trials were carried out at the National Center for Public Health Laboratories (NCPHL) in Sana'a, Yemen. Swabs were taken from the mucous membrane of the palate and the dorsum of the tongue from both groups, and cultured on selective and non-selective solid medium, in addition to culturing the swabs on medium enriched with 5% blood, then all cultured plates were incubated to microaerophilic conditions (5% CO<sub>2</sub>) and oxygenated in 37°C for 48 hours. Then bacterial growth was identified by standard methods.

**Results:** In implant patients, the rate of bacterial isolates from the palate and tongue was slightly higher for potentially harmful bacteria such as *E. coli* (8.3% in tongue implant patients vs. 2.8% in non-implant patients) and *Pseudomonas aeruginosa* (5.6% versus 0%). While in *viridians Streptococcus* including *S. mutans*, there was a higher colonization rate in implants patients (83.3% in the palate versus, 75% in the palate of individuals without implants). A low level of oxacillin resistance (5.1%) in *S. mutans* isolates but *S. mutans* had a substantial level of tetracycline resistance (55.93%), 11.9% for co-trimoxazole, 10.2% for erythromycin, and just 1.7% for clindamycin.

**Conclusion:** The study found that pathogenic bacteria like *E. coli* and *Pseudomonas aeruginosa* were isolated from the palate and the back of the tongue swabs at a slightly elevated rate in implant patients; also colonization rates of *Streptococcus viridians*, including *S. mutans*, were higher in implant patients compared to those without implants. There was a significant levels of antibiotics resistance in *S. aureus*, CoNs and *S. viridians* oral isolates in both groups of tested individuals.

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

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## INTRODUCTION

Oral microbiology is primarily concerned with the study of oral bacteria, as well as their interactions with other oral microbes and the host. The environment in

the human mouth is optimal for the growth of the specific bacteria that live there since it provides a source of nutrients and water as well as a temperate temperature. The mechanical movement from the mouth to the stomach, where hydrochloric acid kills

acid-sensitive microorganisms, is resisted by oral-resident microbes adhering to the teeth and gums<sup>1,2</sup>. Researchers have discovered that oral bacteria have evolved strategies to control their surroundings and avoid or alter the oral environment of their hosts. The mucosal epithelium and dental surfaces fill the ecological niche created by bacteria<sup>1-3</sup>. The pH and oxygen content and availability on some mouth surfaces, as well as mechanical forces acting on those surfaces, the movement of saliva and fluids through the oral cavity, and the host's age, have all been shown to influence bacterial colonization in the oral cavity. This means that the loss of teeth and their replacement with dental implants or dentures may change the structure of those surfaces<sup>1-3,6</sup>. Despite this, a strong intrinsic host defense system constantly checks bacterial colonization and prevents bacterial penetration of local tissues. A dynamic balance exists between dental plaque bacteria and the host's inherent defense system<sup>4</sup>. The function of the oral microbiota in the two major dental illnesses, periodontal disease and dental caries, is of particular interest<sup>1</sup>. Furthermore, research has linked poor oral health to the potential of oral bacteria to assault oral tissues and the body, affecting teeth, heart health, and cognitive function<sup>5</sup>. Today, dental implants are a significant therapy option for individuals who need oral rehabilitation due to tooth loss. The routine use of dental implants may result in an increase in peri-implant illnesses and implant problems. As a result, implant dentistry clinicians need to understand the etiology, impact on normal flora colonization, causes, categorization, and treatment protocol of peri-implant disorders. When there is a negative balance between bone creation and resorption within the basic multicellular unit, bone loss and skeletal injury result<sup>6</sup>. Implant success has been discovered to be influenced by a number of internal (host) and external (surgery or implant) variables, including oral colonization of pathogenic microorganisms. Bone loss occurs during the postoperative recovery phase for a variety of reasons, including severe surgery, bacterial infiltration, and the host's latent healing capacity<sup>6-8</sup>. Even though pathogenic bacterial adhesion may be one of the major causes of cortical bone loss, exposure to a rough surface on the implant due to other factors such as surgical or occlusal trauma has been shown to stimulate bacterial adhesion to the implant and biofilm formation that may lead to inflammation of the soft tissue and bone infection (peri-implantitis)<sup>6,9</sup>. Peri-implantitis is frequently defined as an inflammation of soft tissue accompanied by bone loss of more than 0.5 mm<sup>6,9</sup>. However, there is disagreement over the definition of mucositis, with some authors suggesting that it only involves a soft tissue lesion and others suggesting that it also involves bone loss of less than 0.5 mm, comparable to peri-implantitis<sup>9</sup>. Antimicrobial resistance (AMR) is currently the biggest public health problem, and the number of AMR bacteria in various hospital departments is rising dramatically<sup>1,10-16</sup>. Oral bacteria such as *Staphylococcus aureus*, the *Streptococcus viridans* group, and *Enterobacteraceae* are also known to cause oral infections as well as systemic illnesses such as

endocarditis, pneumonia, and others<sup>1,6</sup>. The goal of this study was to look at the composition of aerobic bacteria in individuals who had dental implants vs those who had natural teeth (no implants), as well as how those bacteria responded to antibiotic therapy.

## MATERIALS AND METHODS

Bacterial examinations were performed on 72 people (36 dental implant patients: 36 natural teeth) over the course of three months, beginning in December 2022 and ending in February 2023, at the Faculty of Dentistry, Sana'a University, Yemen, and private dental clinics (Al-Mortadda Dental Clinics, Al-Kahara Dental Clinics) in Sana'a, Yemen.

### Microbiological procedure

Cultivation and antibiotic sensitivity testing were carried out at the National Center for Public Health Laboratories (NCPHL) in Sana'a, Yemen. Swabs were collected from the mucous membranes of the palate and tongue dorsa of both implant patients and people with natural teeth. Under oxygenated and microaerophilic (5% CO<sub>2</sub>) conditions, cultures were carried out on selective and non-selective solid medium, as well as media enriched with 5% blood. Standard bacterial identification and culture procedures<sup>17</sup> were utilized.

**Antibiogram:** The disc diffusion method was used to determine the antibiotic susceptibility profile. The inoculums were modified to correspond to 0.5 McFarland standards of turbidity, then swabbed onto Brian heart infusion agar and left to dry for 10 minutes<sup>17-19</sup>. The susceptibility to eight non-lactam antibiotics, including erythromycin (15 mg), gentamicin (10 mg), amikacin (30 mg), ciprofloxacin (5 mg), clindamycin (2 mg), and vancomycin (30 mg), was then assessed using antibiogram profiling (Oxide, USA). After 24 hours of aerobic incubation at 37°C, the inhibition zone was determined. Each antibiotic's experiments were carried out three times. Clinical and Laboratory Standards Institute (CLSI) methods was used to analyze the results<sup>18,19</sup>.

**Ethical Consideration:** The Faculty of Medicine and Health Sciences at Sana'a University granted approval for this study under the number 2781, dated October 13, 2022, to its Medical Ethics and Research Committee. All procedures adhered to the review committee's ethical criteria. The subjects who were chosen provided written informed permission.

**Statistical Analysis:** Epi-info Statistics version 7 was used for data analysis. The rates of bacteria isolated were expressed as the percentage that was compared between cases and controls. Sensitivity to antibiotics was also expressed as (%) and antibiotic resistance for tested antibiotics was compared between cases and controls.

## RESULTS

The study included 36 dental implant patients, with a 61.1% male to 38.9% female ratio and ages ranging from 37 to 62, with a mean age and standard deviation of 49.5 and 6.8 respectively. Ages 46 to 55 made up

the majority of the participants (52.8%) (Table 1). Regarding the quantity of implants, 3.3 0.91 implants are the mean and standard deviation. 41.7 percent of individuals had three implants, and 30.6% had four. Most patients (61.1%) had implants for a period of time ranging from 13 to 24 months, with a mean and standard deviation of 17.8 and 6.5 months, respectively.

**Table 1: General characteristics of implant patients participate in the study.**

Characters	Number (%)
Sex	
Male	22 (61.1)
Female	14 (38.9)
Ages	
≤45	10 (27.8)
46-55	19 (52.8)
≥56	7 (19.4)
Mean age	49.5Years
SD	6.8 Years
Mode	51 Years
Median	51 Years
Min-Max	37-62 Years
Number of implants	
2 implant	7 (19.4)
3 implants	15 (41.7)
≥5 implants	3 (8.4)
Mean ± SD	3.3±0.91 implants
Duration of implants	
≤ 12 months	8 (22.2)
13-24 months	22 (61.1)
≥25 months	6 (16.7)
Mean ± SD	17.8±6.5 months

In the current study *S. aureus*, Coagulase-negative, *H. influenza*, *H. parainfluenzae*, *E. coli*, *K. pneumoniae*, *Citrobacter freundii*, and *P. aeruginosa* were coloni-

zed the oral cavity of implant patients. *S. aureus* colonization was higher in implant patients (5.6% in the palate) compared to 2.8% in those without implants in the palate. While implant patients had a lower rate of Coagulase-negative colonization (8.3% in the palate), persons without implants had a greater frequency of Coagulase (22.2%). There was a higher colonization rate in implant patients equivalent to 83.3% in palate versus, a lower rate (75%) in individuals without implants in viridians (apathy) Streptococcus, including *S. mutans*. Additionally, potentially harmful Enterobacteriaceae spp bacteria colonized implant patients slightly more than non-implant patients (Table 2). In current study it was observed that a low level of oxacillin resistance (5.1%) in *S. mutans* isolates. However, in current investigation, we found that the isolates of *S. mutans* had a substantial level of tetracycline resistance (55.93%), 11.9% for co-trimoxazole, 10.2% for erythromycin, and just 1.7% for clindamycin. The antibiotic sensitivity results are presented in Table 3 to Table 8.

## DISCUSSION

Both main and secondary colonizers of the microbiota can have an impact on the peri-implant's health. Early bacterial colonization in a healthy implant site takes about 6 months to reach a stage where it resembles the microbiome of the remaining natural dentition in the arch and varies depending on the condition of the normal dentition<sup>6,9</sup>. Additionally, according to the literature, when the sulcus depth was less than 4 mm from an implant-abutment junction, the peri-implant sulci's microbiota resembled that of tooth sulci<sup>9</sup>. The microbiota surrounding problematic implants, however, varied depending on the infection stage (mucositis, peri-implantitis, and failing implant)<sup>20-22</sup>.

**Table 2: Isolation frequency (%) of bacteria in hard palate and tongue dorsa of dental implant patients and health normal subjects.**

Bacteria	Implants patients N=36		Healthy subject N=36		Total isolates
	Palate N (%)	Tongue N (%)	Palate	Tongue	
<i>S. aureus</i>	2 (5.6)	2 (5.6)	1 (2.8)	3 (8.3)	8
Coagulase-negative	3 (8.3)	4 (11.1)	8 (22.2)	17 (47.2)	32
<i>Streptococci</i>					
<i>S. pyogenes</i>	1 (2.8)	0 (0.0)	1 (2.8)	0 (0.0)	2
<i>S. mitior</i>	2 (5.6)	7 (19.4)	9 (25)	5 (13.9)	23
<i>S. sanguis</i>	5 (13.9)	8 (22.2)	8 (22.2)	5 (13.9)	26
<i>S. mutans</i>	30 (83.3)	29 (80.6)	27(75)	25(69.4)	111
<i>S. alivarius</i>	9 (25)	8 (22.2)	9 (25)	5(13.9)	31
<i>S. milleri</i>	1 (2.8)	1 (2.8)	1(2.8)	4 (11.1)	7
<i>Neisseria</i> spp.	19 (52.8)	21 (58.3)	24(66.7)	28(77.8)	92
<i>Haemophilus influenza</i>	1 (2.8)	0 (0.0)	0 (0.0)	0 (0.0)	1
<i>H. parainfluenzae</i>	2 (5.6)	2 (5.6)	7 (19.4)	6(16.7)	17
<i>Enterobacteriaceae</i> spp.					
<i>Escherichia coli</i>	2 (5.6)	3 (8.3)	1 (2.8)	1 (2.8)	7
<i>Klebsiella pneumoniae</i>	1 (2.8)	2 (5.6)	1 (2.8)	1 (2.8)	5
<i>Morganella morganii</i>	0 (0)	0 (0)	0 (0)	1 (2.8)	1
<i>Citrobacter freundii</i>	1 (2.8)	1 (2.8)	0 (0)	0 (0)	2
<i>Pseudomonas aeruginosa</i>	1 (2.8)	2 (5.6)	0 (0.0)	0 (0.0)	3

**Table 3: Antibiotic patterns of *S. aureus* isolated from hard palate and tongue dorsa of implant and normal healthy subjects, N=8 isolates.**

Antibiotic name	Dental implant N=4		Normal healthy subjects N=4	
	Resistance N (%)	Sensitive N (%)	Resistance N (%)	Sensitive N (%)
Tetracycline	3 (75)	1 (25)	2 (50)	2 (50)
Erythromycin	3 (75)	1 (25)	2 (50)	2 (50)
Co-trimoxazole	2 (50)	2 (50)	1 (25)	3 (75)
Amoxicillin-Clavulanic acid	1 (25)	3 (75)	1 (25)	3 (75)
Gentamicin	1 (25)	3 (75)	0 (0.0)	4 (100)
Oxacillin	1 (25)	3 (75)	1 (25)	3 (75)
Ciprofloxacin	0 (0.0)	4 (100)	0 (0.0)	4 (100)
Cloxacillin	1 (25)	3 (75)	1 (25)	3 (75)
Cefoxitine	0 (0.0)	4 (100)	0 (0.0)	4 (100)
Amikacin	0 (0.0)	4 (100)	0 (0.0)	4 (100)
Clindamycin	0 (0.0)	4 (100)	0 (0.0)	4 (100)
Vancomycin	0 (0.0)	4 (100)	0 (0.0)	4 (100)

In the current study *S. aureus*, *Coagulase-negative*, *Haemophilus influenza*, *H.parainfluenzae*, *E. coli* *K. pneumoniae*, *Citrobacter freundii*, and *P. aeruginosa* were colonized the oral cavity of implant patients making biofilm. Adhesion, growth, maturation, and dispersion are the stages in the formation of biofilm by the colonization of microorganisms<sup>9,23</sup>. Electrostatic attraction leads to the first adherence of bacteria to a surface, which is followed by the production of extracellular polymers<sup>24</sup>. A mechanical adhesion of the bacteria is also encouraged by the host's rough surface through mechanical retention<sup>6,9</sup>.

Specific signaling molecules are required for the co-aggregation of bacteria, which determines which bacterial species may adhere in the colony-forming unit. Through the production of a biomarker that either facilitates or hinders the creation of biofilms, bacterial aggregation causes an inflammatory response in the host tissue. However, the biofilm becomes resistant to antimicrobial therapy as it matures and produces extracellular polymers<sup>25,26</sup>. The development of biofilms and peri-implant infection are aided by the interaction between bacteria and the inflammatory response of the host.

**Table 4: Antibiotic patterns of coagulase-negative staphylococcus isolated from hard palate and tongue dorsa of implant and normal healthy subjects, N=32 isolates.**

Antibiotic name	Dental implant N=7		Normal healthy subjects N=25	
	Resistance N (%)	Sensitive N (%)	Resistance N (%)	Sensitive N (%)
Tetracycline	6 (85.7)	1 (14.3)	15 (60)	10 (40)
Erythromycin	5 (71.4)	2 (28.6)	7 (28)	18 (72)
Co-trimoxazole	3 (42.9)	4 (57.1)	6 (24)	19 (76)
Amoxicillin-Clavulanic acid	2 (28.6)	5 (71.4)	9 (36)	16 (64)
Gentamicin	2 (28.6)	5 (71.4)	3 (12)	22 (88)
Oxacillin	1 (14.3)	6 (85.7)	4 (16)	21 (84)
Ciprofloxacin	1 (14.3)	6 (85.7)	2 (8)	23 (92)
Cloxacillin	1 (14.3)	6 (85.7)	1 (4)	24 (96)
Cefoxitine	2 (28.6)	5 (71.4)	1(4)	24 (96)
Amikacin	0(0.0)	7 (100)	0(0.0)	25 (100)
Clindamycin	0(0.0)	7 (100)	0(0.0)	25 (100)
Vancomycin	0 (0.0)	7 (100)	0(0.0)	25 (100)

Understanding the cellular mechanisms behind biofilm formation will help to block the bacterial adhesion and proliferation that precede biofilm formation. In the current study, implanted patients had a low rate of *S. aureus* colonization (5.6% in the palate) compared to 2.8% in non-implanted persons. The current study results differ from those of Al-Shamiet al.<sup>1,11</sup>, who found that the rate of bacterial isolates from the palate, back, tongue, and dental plaque smears was higher in denture wearers than in natural teeth patients (11.5% versus 1.6% in the palate). While there was a lower incidence of Coagulase-negative colonization in implanted patients (11.1% in the tongue) and a higher

incidence of Coagulase-negative (47.2%) in subjects without implanted teeth in the current study, Al-Shami et al.,<sup>1,11</sup> found a lower incidence of Coagulase-negative colonization in denture patients (16.4% in the tongue) and a higher incidence of Coagulase-negative (47.5%) in subjects without dentures.

There was a greater colonization rate of *viridians* (apathy) *Streptococcus*, including *S. mutans*, in the implanted group in the palate, compared to people with healthy teeth (69.4%). The findings of the current study are consistent with those of earlier studies in that there are many microbial compositional similarities between the populations of adults wearing full or partial

dentures and adults with intact teeth, as well as some notable compositional differences<sup>1,11,27</sup>. Recent research have studied dentures in the adult population wearing full or partial dentures and individuals with natural teeth as well as factors affecting oral microbia in Yemen<sup>1,11,27,28</sup>. Numerous articles have solely addressed *Candida*<sup>27-31</sup>. However, researchers of

current study were unaware of any published study that addresses the colonization of potentially harmful aerobic bacteria in implanted individuals. Currently, there aren't many studies on dental microbiology and the factors influencing their variety and quantity, though most of them focused on anaerobic bacteria<sup>9</sup>.

**Table 5: Antibiotic patterns of *S. mutans* isolated from hard palate and tongue dorsa of implant and normal healthy subjects, N=111 isolates.**

Antibiotic name	Dental implant N=59		Normal healthy subjects N=52	
	Resistance N (%)	Sensitive N (%)	Resistance N (%)	Sensitive N (%)
Tetracycline	33 (55.9)	26 (44.1)	23 (44.2)	29 (55.8)
Erythromycin	6 (10.2)	53 (89.8)	3 (5.7)	49 (94.2)
Co-trimoxazole	7 (11.9)	52 (88.1)	7 (13.5)	98 (86.5)
Amoxicillin-Clavulanic acid	6 (10.2)	53 (89.8)	5 (9.6)	47 (90.4)
Gentamicin	4 (6.8)	55 (93.2)	5 (9.6)	47 (90.4)
Oxacillin	3 (5.1)	56 (94.9)	2 (3.8)	50 (96.2)
Ciprofloxacin	2 (3.4)	107 (96.9)	5 (9.6)	47 (90.4)
Cloxacillin	1 (1.7)	58 (98.3)	3 (5.7)	49 (94.2)
Cefoxitine	2 (3.4)	107 (96.9)	3 (5.7)	49 (94.2)
Amikacin	0 (0.0)	59 (100)	0 (0.0)	52 (100)
Clindamycin	1 (1.7)	58 (98.3)	0 (0.0)	52 (100)
Vancomycin	0 (0.0)	59 (100)	0 (0.0)	52 (100)

In one such study, the analysis of the microbiota in failing or failed implants revealed significantly elevated levels of *Prevotella intermedia*, *Capnocytophaga* spp, *Porphyromonas gingivalis*, *Peptostreptococcus micros*, *Fusobacterium* spp, *Campylobacter rectus*, *Actinomycetemcomitans*, *Treponema denticola* and *Candida albicans*<sup>9,32</sup>.

In the current study, potentially pathogenic *Enterobacteriaceae* spp bacteria were more colonized in implanted patients than in individuals with normal teeth: for example, *E. coli* (8.3% in implanted versus 2.8% in the absence of implants) and *P. aeruginosa* (5.6% in implanted versus 0.0% in the absence of implants).

**Table 6: Antibiotic patterns of *S. mitior* isolated from hard palate and tongue dorsa of implant and normal healthy subjects, N=23 isolates.**

Antibiotic name	Dental implant N=9		Normal healthy subjects N=14	
	Resistance N (%)	Sensitive N (%)	Resistance N (%)	Sensitive N (%)
Tetracycline	5 (55.6)	4 (44.4)	6 (42.9)	8 (57.1)
Erythromycin	1 (11.1)	8 (88.9)	1 (7.1)	13 (92.9)
Co-trimoxazole	2 (22.2)	7 (77.8)	2 (14.3)	12 (85.7)
Amoxicillin-Clavulanic acid	1 (11.1)	8 (88.9)	5 (10.6)	42 (89.4)
Gentamicin	1 (11.1)	8 (88.9)	1 (7.1)	13 (92.9)
Oxacillin	0 (0.0)	9 (100)	0 (0.0)	14 (100)
Ciprofloxacin	0 (0.0)	9 (100)	0 (0.0)	14 (100)
Cloxacillin	0 (0.0)	9 (100)	0 (0.0)	14 (100)
Cefoxitine	0 (0.0)	9 (100)	0 (0.0)	14 (100)
Amikacin	0 (0.0)	9 (100)	0 (0.0)	14 (100)
Clindamycin	0 (0.0)	9 (100)	0 (0.0)	14 (100)
Vancomycin	0 (0.0)	9 (100)	0 (0.0)	14 (100)

The implants (implant palague) of current investigated patients had been colonized by a variety of possible respiratory infections, with *S. aureus* accounting for 5.6% of these. Table 2 lists the remaining probable respiratory pathogens as follows: *P. aeruginosa* (5.6%), *K. pneumoniae* (5.6%), *H. influenzae* (2.8%), and *H. parainfluenzae* (5.6%). Reservoirs for possible respiratory infections can be found in dental plaque and the tongue dorsa. According to Sumi et al., dental prosthesis plaque may act as a reservoir for potential

pathogens to help colonization in the oropharynx, and poor dental prosthesis hygiene is a significant factor in increasing oropharyngeal bacterial colonization. It has been proposed that the surface of the tongue may serve as an additional and likely more consistent, reservoir of respiratory pathogens<sup>6,9,33</sup>. In both implanted patients and healthy people, respiratory pathogens such as *S. aureus*, *Haemophilus influenzae*, *H. parainfluenzae*, and *Neisseria* species were isolated. Obtained result is similar to that reported by Tyrrell et al., Sumi et al.,

Goldberg *et al.*, and Senpuku *et al.*, where some uncommon microorganisms are found in oral microbiota<sup>34-36</sup>. Dental professionals often prescribe the majority of the antibiotics utilized in this study<sup>1,9</sup>. Although resistant bacteria can also develop in healthy individuals who have not recently received antibiotic treatment, the frequency of *streptococci* resistant to oral medication is higher in those who are frequently exposed to antibiotics<sup>13</sup>.

In regular dentistry practice,  $\beta$ -lactam antibiotics are the most frequently utilized chemo preventive agents. However, oral *streptococci* are becoming more resistant to penicillin<sup>1,10,11</sup>. Although these bacteria can be found in healthy patients who have not recently received antimicrobial treatment, the prevalence of resistant oral *streptococci* is higher in those who get antibiotics on a regular basis<sup>1</sup>.

**Table 7: Antibiotic patterns of *S. sanguis* isolated from hard palate and tongue dorsa of implant and normal healthy subjects, N=26 isolates.**

Antibiotic name	Dental implant N=13		Normal healthy subjects N=13	
	Resistance N (%)	Sensitive N (%)	Resistance N (%)	Sensitive N (%)
Tetracycline	6 (46.2)	7 (53.8)	5 (38.5)	8(61.5)
Erythromycin	1 (7.7)	12 (92.3)	2 (15.4)	11 (84.6)
Co-trimoxazole	2 (15.4)	11 (84.6)	2 (15.4)	11 (84.6)
Amoxicillin-Clavulanic acid	1 (7.7)	12 (92.3)	1 (7.7)	12 (92.3)
Gentamicin	0 (0.0)	13 (100)	0 (0.0)	13 (100)
Oxacillin	0 (0.0)	13 (100)	0 (0.0)	13 (100)
Ciprofloxacin	0 (0.0)	13 (100)	0 (0.0)	13 (100)
Cloxacillin	1 (7.7)	12 (92.3)	1 (7.7)	12 (92.3)
Cefoxitine	1 (7.7)	12 (92.3)	0 (0.0)	13 (100)
Amikacin	1 (7.7)	12 (92.3)	0 (0.0)	13 (100)
Clindamycin	0 (0.0)	13 (100)	0 (0.0)	13 (100)
Vancomycin	0 (0.0)	13 (100)	0 (0.0)	13 (100)

**Table 8: Antibiotic patterns of *S. alivarius* isolated from hard palate and tongue dorsa of implant and normal healthy subjects, N=31 isolates.**

Antibiotic name	Dental implant N=17		Normal healthy subjects N=14	
	Resistance N (%)	Sensitive N (%)	Resistance N (%)	Sensitive N (%)
Tetracycline	9 (52.9)	8 (47.1)	8 (57.1)	6 (42.9)
Erythromycin	3 (17.6)	14 (82.4)	3 (21.4)	11 (78.6)
Co-trimoxazole	3 (17.6)	14 (82.4)	3 (21.4)	11 (78.6)
Amoxicillin-Clavulanic acid	2 (11.8)	44 (83)	1 (7.1)	13 (92.9)
Gentamicin	1 (5.9)	16 (94.1)	1 (7.1)	13 (92.9)
Oxacillin	1 (5.9)	16 (94.1)	1 (7.1)	13 (92.9)
Ciprofloxacin	0 (0.0)	17 (100)	0 (0.0)	17 (100)
Cloxacillin	0 (0.0)	17 (100)	0 (0.0)	17 (100)
Cefoxitine	0 (0.0)	17 (100)	0 (0.0)	17 (100)
Amikacin	0 (0.0)	17 (100)	0 (0.0)	17 (100)
Clindamycin	0 (0.0)	17 (100)	0 (0.0)	17 (100)
Vancomycin	0 (0.0)	17 (100)	0 (0.0)	17 (100)

Bacterial resistance to antibiotics such as penicillin and other  $\beta$ -lactams is a major public health concern in many parts of the world. In current study it was observed a low level of oxacillin resistance (5.1%) in *S. mutans* isolates. The low prevalence of resistance to penicillin group in *S. mutans* in current study is different from that previously observed in Yemen (14.9%)<sup>10</sup> and (11.6%)<sup>11</sup> resistance, South Africa and Spain in oral *S. viridians*<sup>1,9</sup>. Penicillin resistance genes can be transmitted between related species, according to a number of *in-vitro* investigations<sup>1,9</sup>. The development and spread of penicillin resistance in oral *streptococci* may be significantly influenced by these mechanisms as well as selective antibiotic pressure. In addition, a considerable level of penicillin resistance was found by Pasquantonio *et al.*,<sup>37</sup>.

Out of 50 isolates of *S. mutans*, 14% were penicillin-resistant, representing 13.4% of 550 oral streptococcal clinical isolates. However, the rate of full resistance to penicillin and ampicillin among *S. mutans* isolates in a research by Dhamodhar *et al.*,<sup>38</sup> done in 2014 was higher than current obtained data. The American Heart Association recommends antimicrobial prophylaxis one hour before to dental surgery for high-risk cardiovascular patients, such as amoxicillin (2g) as first choice and clindamycin (600mg) as a second choice<sup>1,11</sup>. However, production of  $\beta$ -lactamase is infrequent for most *streptococci*, where resistance is caused by slightly changed penicillin binding proteins<sup>1,9,10</sup>. However, in current investigation, it was found that the isolates of *S. mutans* had a substantial level of tetracycline resistance (55.93%), 11.9% for co-

trimoxazole, 10.2% for erythromycin, and just 1.7% for clindamycin. As a result, the first drug of choice in this case should be clindamycin, whose resistance rate is 1.7%. In the end, *S. mutans*' evolved resistance is unknown. The present study's updated information on antibiotic susceptibility testing aids in informing pharmaceutical companies to develop new methods for efficient prophylaxis against dental infections. This outcome also provides the dentist in Yemen with the best option for prescribing a suitable antibiotic.

#### Limitations of the study

In the world and Yemen, no sufficient study has been undertaken to verify the composition of aerobic bacteria in the oral cavity of implant patients and compare them to those with natural teeth. A prospective study with a larger number of patients, anaerobic species, and other antibiotics tested for common isolates is required to investigate the effect of implants on bacterial colonization of the oral cavity as well as its effect on antibiotic susceptibility patterns.

#### CONCLUSION

The study found an increased rate of pathogenic bacteria such as *S. aureus* and *Enterobacteriaceae* spp such as *E. coli*, *K. pneumoniae*, and *P. aeruginosa* in implant patients, while there was a higher colonization rate of *S. viridians* including *S. mutans* in implant patients compared to a moderate rate in individuals with normal teeth.

Also, the study demonstrates significant levels of antibiotics resistance in *S. aureus*, CoNs and *S. viridians* oral isolates in both groups (implant group and normal group). Further study is required to include more penicillin group and cephalosporin group and know the minimum inhibitory concentration of  $\beta$ -lactam and non  $\beta$ -lactam antibiotics towards *S. viridians* group.

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#### CONFLICT OF INTEREST

There are no competing interests involved in this work.

#### AUTHOR'S CONTRIBUTIONS

The researcher that oversaw the research for this study, Hassan Al-Shamahy, came up with the idea, wrote the first draft of the publication, and cooperated with other authors to review the data.

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