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Original article

Appraisal and characterization of candida load isolated from the oral cavity of smokers

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ABSTRACT

Cigarette smoking is regarded as a major global health risk, therefore the aim of this work was to investigate the association of oral *Candida* spp. as one of the etiological agents of denture stomatitis with smokers of cigarette, hookah (shisha), and electronic smoking, also a dose–response relationship between the duration of smoking and the probability of denture stomatitis between volunteers. Oral rinse samples were collected from 47 male volunteers including 34 smokers and 13 non-smokers, also data of volunteers were collected via a questionnaire forum. Patterns of smoking were shown that smokers using tobacco cigarettes 17 (36.2 %), electronic cigarettes 16 (34.04 %), and hookah smokers 8 (17.02 %). A comparison of smokers and non-smokers regarding effects on oral health showed significantly finding ($P < 0.05$) indicating that smoking affects oral health in all evaluated parameters (an oral mucosal abnormality, mouth ulcers, bad breath, and feeling of dry mouth). Out of 19 Candida isolates, 18 (94.7 %) were identified as Candida albicans and 1 (5.3 %) as Candida tropicalis. Among the volunteers who presented with oral Candida (19 volunteers), 17 (89.5%) were smokers, while non-smoker volunteers were 2 (10.5 %), so it can be concluded that smoking was a significant positive correlation to the presence of Candida in the oral cavity. Five volunteers suffered from chronic diseases; 4 (8.5%) diabetes mellitus and 1 (2.1%) anemia as a systemic predisposing factor for oropharyngeal infection. Amphotericin and Nystatin had varying degrees of activity against isolated Candida isolates.

1. Introduction

According to estimates, smoking has a 2 trillion-dollar economic impact, with healthcare costs and treating ailments connected to smoking making up about 30% of that (Frazer et al., 2022). Smoking is seen as a major hazard to world health due to its widespread exposure and legal and societal acceptance in many countries (Ohlrogge et al., 2022). Worldwide smoking rates are increasing despite repeated warnings from several health organizations (Dai et al., 2022).

Massive health problems are brought on by tobacco smoke, which affects not just smokers but also others who have been exposed to it. Smoking contains a variety of biologically active chemicals, such as genotoxic, oxidizing, and immune-stimulating agents (Dalrymple et al., 2016). While smoking cigarettes increases the risk of many infectious diseases and can have a major negative

Abbreviations: SDA, Sabouraud dextrose agar; CFU, Colony-forming unit; µg, Micrograms; ml, Millilitre; AMB, Amphotericin B; FY, Flucytosine.

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impact on human health by causing atherosclerosis, carcinogenesis, and chronic lung ailments such as chronic obstructive pulmonary disease (Dahdah et al., 2022; Luca et al., 2023).

Yet, the mouth cavity is a special place with a variety of microscopic microbial homes, including the tongue, soft and hard palates, teeth, and buccal mucosa. These habitats collectively form a rich, multispecies ecological system (Kilian, 2018). Bacteria, viruses, and fungi are just a few of the microorganisms that live in the mouth. Actinomycetes, Proteobacteria, Bacillus, and Firmicutes are among the bacteria that predominate in the mouth's ecosystem (Segata et al., 2012; Mark Welch et al., 2016). Streptococci form the largest group of resident oral microflora (Alghamdi, 2022). *Candida* species create microbial biofilm with *Streptococcus* to engage in a harmful activity (Wang et al., 2012).

Moreover, the mouth cavity is one of the key openings of the human body where plenty of germs enter through food, water, and air. Some of them settle inside the cavity, develop, and multiply, forming unique microbial communities. The physiochemical environmental condition in the cavity affects these microbial growths and the choice of habitat (Sarmah et al., 2021). The host's molecules and the oral microbial members all contribute to the environment. The microbial communities in the cavity may alter if this cavity's molecular composition changes. As a result, many diseases and altered eating patterns may encourage changes in the microbial presence in the mouth cavity. So, the presence of particular microbial together or in greater quantity in the mouth cavity may be a useful sign of disease prediction (Sarmah et al., 2021).

Candida species is a good indicator of oral cavity health because it is a widespread opportunistic microbe that is a component of the natural microbial flora found on mucosal surfaces, including those of the oral cavity, and the main source of mucosal and invasive fungal infections (Singh et al., 2014; Pathakumari et al., 2020). There are many risk factors for oral candidiasis that may be symptomatic or asymptomatic including age, dental implants, removable dental prostheses use, immunosuppressive therapy, and salivary pH (Hato et al., 2022). Some other risk factors until now have not been confirmed, but many factors may be enhanced by smoking some studies have not proven the relationship between smoking and oral fungus, while some other studies have shown that smoking has a clear effect on oral fungus (Kadir et al., 2002; Soysa and Ellepola, 2005; Darwazeh et al., 2010; Haghighi et al., 2022).

Consequently, the purpose of this article is to investigate *Candida* colonization in smokers' and non-smokers' oral cavities. This study looked at the relationship between oral *Candida* spp. and denture stomatitis among volunteers who smoked cigarettes, hookah (shisha), and electronic cigarettes as well as the dose–response relationship between the length of smoking and risk of developing denture stomatitis. Moreover, some antifungal activity against isolated *Candida* isolates was assessed.

2. Materials and methods

2.1. Study design and sample size

This cross-sectional study included 47 male volunteers (18 to 50 years) who were divided into two groups (smokers and non-smokers). During March and April 2022, the oral rinse samples as well as questionnaire data (Google form questionnaire to achieve confidentiality and accuracy) were collected from students and employees person male of the College of Applied Medical Sciences, Prince Sattam bin Abdulaziz University at Al-Kharj, Saudi Arabia. Processing of samples was done at the Mycology and Parasitology lab of the Medical Laboratory Sciences Department. All sampling was performed by one person between 8 Am and 5 PM. None of the volunteers had taken any drinks or food, use any practicing oral hygiene (not using a toothbrush or Miswak), or smoked for a minimum of 1 h before the sampling.

2.2. Exclusion criteria

Persons who took any medicine recognized to affect oral candidiasis, including corticosteroids, antibacterial or antifungal drugs, or using antiseptic mouthwash for the previous three months. Volunteers wearing removable dental prostheses were excluded from this study.

2.3. Sample collection

2.3.1. Concentrated oral rinse

Each participant was given 10 ml of sterile saline (0.9 % NaCl) in a wide-mouth sterilized container and instructed to thoroughly rinse the mouth of volunteers for 15 s before the rinse was expectorated into the container. Oral sample containers were coded for further processing tests, then processed immediately or stored in a thermocol ice box (containing ice) within 30 min until they were processed. For optimal microbial desegregation, the rinse container sample was centrifuged (3500 rpm) at 20 °C (refrigerated centrifuge, Universal 320 R, Hettich) for 10 min, discard the supernatant, and the pellets were diluted with 2 ml sterile saline (0.9 % NaCl) and mixed by vortex for 20 s to mix and suspended concentrated oral rinse. This study was approved by the ethical approval committee at the medical laboratory science, college of applied medical sciences, and was conducted in accordance with guidelines.

2.3.2. Microbial isolation, identification, and counting

For *Candida* spp. isolation; 50, 100, and 500 µl of the suspended concentrated oral rinse (2 ml) were inoculated on suitably prepared agar plates. Sabouraud dextrose agar (SDA, Scharlau, Barcelona, Spain) supplemented with Cefotaxime (128 µg/ml to inhibit bacterial growth) medium plates were used for the isolation of yeast. The sterile loop was used to spreading the suspended microbial suspension on the surface of SDA plates to achieve separated colonies. Yeast colonies on each plate were counted by Manual Colony Counter (aCOLade Colony Counter, Synbiosis) and calculated to count colony-forming units/ 10 ml (CFU/ 10 ml).

2.3.3. Identification of Candida

Colony characteristics and color on the modified SDA of isolated counted yeast were subjected to further methods of identification (Chromogenic agar and germ tube formation) for the presumptive identification of different common *Candida* spp.

2.3.4. Chromogenic agar

Presumptive identification of isolated *Candida* species by a chromogenic agar medium (CHROM agar *Candida* Becton Dickinson/BBL). The basic scientific identification of this medium according to the differential release of chromogenic breakdown products (indicated by color change) from several specific substrates due to the enzymatic activities produced by different yeast isolates.

2.3.5. Germ tube test

One ml of sterile serum was inoculated using a clean Pasteur tip-making yeast suspension adjusted to be equivalent to 0.5 McFarland (approximately 1.5×10^8 cell/ml) turbidity., then incubate at 37 °C for 2–2.30 h, then one drop of the yeast-serum mixture was placed on a slide with a coverslip and examined microscopically.

2.4. Antibiotic susceptibility testing

2.4.1. Antifungal test

Antifungal susceptibility testing was performed on Sabouraud-dextrose agar (SDA) medium (Scharlau, Barcelona, Spain) by disk diffusion method for two available antifungal drugs; Amphotericin B (AMB 20 μg) Flucytosine (FY 1 μg) (MASTDISCS CO) and Nystatin (BBL CO) disks. After *Candida albicans* identification, the yeast suspension was adjusted to 0.5 McFarland standard. *Candida* suspension streaked on SDA plates and antifungal drugs were placed, the plates were incubated at 37 °C for 48 h, and the diameters of the inhibition zone were measured by millimeters.

2.4.2. Statistical analysis

Data were analyzed using SPSS (Statistical Package for the Social Sciences) version 23.0, and differences between smokers and nonsmokers were calculated using the chi-squared test. The chi-squared test was used to compare the proportions of *Candida* carriers and noncarriers. Significant differences were examined at *p*-value < 0.05was considered.

3. Results

Table 1 revealed that 75.6% of the volunteers were smokers, 95.7% were between the ages of 18 and 25, and 44.1% had smoked continuously for five years or more. As shown in Table 2, there was no correlation between the frequency of tooth brushing among smokers and non-smokers. Moreover, there was no statistically significant relationship between smoking and bad oral hygiene ($p = 0.074$).

According to Table 3, tobacco cigarettes accounted for the highest percentage (36.2%) of volunteers' smoking patterns, followed by electronic cigarettes (34.04%). The results also showed that 58.8% of smokers smoked 20 or more cigarettes per day, while 14.8% of smokers reported using more than one type of tobacco. In terms of smoking electronic cigarettes, the findings revealed that 75% of hookah (Shisha) smokers or e-cigarette consumers consume 1–6 times weekly as the highest ratio, with 43.7% using an electronic hookah (Shisha) or e-cigarettes 3–4 h per day. Smoking negatively impacts oral health in all of the tested criteria, including the sensation of dry mouth at night or upon waking, having bad breath, having an abnormality of the oral mucosa, and developing mouth ulcers, according to a comparison of smokers and non-smokers (Table 4).

Candida prevalence in the mouth between the two groups that were being studied. After purification of yeast-like structure colonies, they were inspected under a microscope and put through the identification processes of *Candida* spp. using chromogenic media and germ tube formation (Fig. 1 and 2). The prevalence of *Candida* was assessed, and the number of candidal colonies was counted by a manual colony counter. One *Candida tropicalis* isolate (5.3%) and 18 *Candida albicans* isolates (94.7%) were found among the 19 *Candida* isolates (Table 5).

Smoking was found to have a significant positive correlation with the presence of *Candida* in the oral cavity ($p = 0.031$), as evidenced by the fact that 17 (89.5%) of the participants who presented with oral *Candida* (19 volunteers) were smokers and 2 (10.5%) were non-smokers. However, there was no statistically significant relationship between smoking and the total number of *Candida* found in an oral rinse (less than or equal to 1000) ($p = 0.087$). According to our calculations, there are 7 (36.44%) volunteers who have<1000 CFU of *Candida* spp. growing on them, 6 (85.7%) of them are smokers, and 1 (14.3%) of them are not (Table 5).

Table 6 showed a comparison of smokers using one or two types of smoking regarding the presence of *Candida* had significant finding ($p = 0.013$). Besides, there is no significant relationship between chronic disease and the presence of *Candida* spp.

The antifungal sensitivity pattern for candidal isolates was obtained from this study (Table 7 & Fig. 3). The three available antifungal sensitivity drugs; Amphotericin B, Nystatin, and Flucytosine showed that; all isolated strains of *Candida* showed resistance to Flucytosine (1 μg) while Amphotericin (20 μg) and Nystatin (IU) had varying degrees of activity against isolated *Candida* spp.

4. Discussion

There are conflicting findings on the relationship between oral *Candida* and smoking, according to certain literature research (Kadir et al., 2002; Darwazeh et al., 2010). A possible connection between oral *Candida* and smoking or nicotine products was also noted in various articles (El-Sakhawy et al., 2023). Hence, the purpose of this study is to support or refute any of these hypotheses. Oral *Candida* colonization and smoking or nicotine products have been linked, according to some studies. So, the duration and amount of smoking's effects on the candidal number were assessed as well as oral *Candida* and oral health in our study to determine whether there is a connection between smoking and oral *Candida*.

The selection of only male volunteers in the present study for excluding the different influences of hormonal effects which may be affected by using contraceptives or during periods of the menstrual cycle and pregnancy (Aminzadeh et al., 2016; Fujiwara et al., 2017). Persons who took any medicine recognized to affect oral candidiasis, including corticosteroids, antibacterial or antifungal drugs, or using antiseptic mouthwash for the previous three months were excluded (Krishnan et al., 2020). Also, volunteers wearing removable dental prostheses were excluded from this study. Wearing a removable prosthesis and other factors alter the oral microbiome and increase the presence of *Candida* colonization, which is achieved by decreasing salivary flow or the nature of prosthesis materials (Diaz and Dongari-Bagtzoglou 2021; Kinkela et al., 2021).

No significant association between tooth brushing habits was found between smoking and nonsmokers. Al-Qurashi et al., 2016 reported that approximately 64.5% of the respondents used tooth brushing and 62.5% used Miswak and in the same study, regarding

Table 1
Distribution of age of studded groups and distribution of smoking duration by years.

Age distribution of studded groups					**Distribution of smokers according to smoking duration by years**	
Age (Years)	**Number (%)**	**Non-smoker**	**Smoker**	***P* value**	**Smoking duration by years**	**Number (%)**
18–25	45 (95.7)	11 (24.4)	34 (75.6)	0.221	**1–2 years**	9 (26.5)
26–33	1 (2.1)	1(100)	0(0.00)		**3–4 years**	10 (29.4)
33–41	1 (2.1)	1 (100)	0(0.00)		**5 years and more**	15 (44.1)
Total	47 (100)	13 (27.7)	34 (72.3)		**Total**	34 (100)

*Significant ($P < 0.05$).

Table 2
Tooth brushing habits of volunteers (smokers and nonsmokers) on the basis of using a tooth-brush or Miswak and frequency.

The number of times uses a toothbrush	Number (%)	Non-smoker	Smoker	*P* value
Once daily	18 (38.3)	2(11.1)	16(88.9)	
Twice daily	17 (36.2)	7(41.2)	10(58.8)	0.074
After meals	2 (4.30)	2(100)	0(0.00)	
Use Miswak sometimes	4 (8.50)	1(25.0)	3(75.0)	
Use Miswak regularly	2 (4.30)	0(0.00)	2(100)	
Don't use a tooth-brush or Miswak	4 (8.50)	1(25.0)	3(75.0)	
Total	47 (100)			

*Significant ($P < 0.05$).

Table 3
Patterns of smoking among smoking volunteers.

Kind and patterns of smoking	Number of Smokers (%)
Non-smokers	13 (27.7)
Volunteers using electronic hookah or e-cigarettes daily / hour	
Total no.	16 (34.04)
1–2 h per day	6 (37.5)
3–4 h per day	7 (43.7)
5–6 h per day	3 (18.8)
Volunteers using tobacco cigarettes /day	
Total no.	17 (36.2)
1:9 cigarettes daily	3 (17.7)
10:19 cigarettes daily	4 (23.5)
20 and more cigarettes daily	10 (58.8)
Volunteers using a hookah consumed /week	
Total no.	8 (17.02)
1–6 times weekly	6 (75.0)
7–12 times weekly	0(0.00)
More than 12 times weekly	2 (25.0)
Total no. of smokers	34 (72.3)
Smokers used more than one kind of smoking	7 (14.8)

oral hygiene habits, no discernible differences between smokers and non-smokers were found. Furthermore, Santos et al (2015) concluded that there were no statistically significant differences between smokers and nonsmokers regarding the use of mouthwashes.

Results of the current study showed that there is no significant relationship between chronic disease and the presence of *Candida* spp. Besides, Patterns of smoking among smoking volunteers showed that the highest kind is tobacco cigarette smokers followed by electronic cigarette smokers. According to (Guggenheimer et al., 2000), there is growing evidence that smoking tobacco increases a diabetic patient's risk of developing oral candidiasis. Besides, (Kulak-Ozkan et al., 2002) stated that, smoking, mobile prosthetics, and systemic disease were found as predisposing factors for the development of oral candidiasis. In addition, Nishimaki et al (2019) revealed a close connection between the amount of oral *Candida* and the host's underlying health issues. In immunosuppressed hosts, oral *Candida* growth may increase.

A comparison of smokers and non-smokers regarding effects on oral health showed significantly finding indicating that smoking affects oral health in all evaluated parameters between smokers and nonsmokers. Al-Qurashi et al., 2016 prove that dry mouth was more prevalent in smokers compared to non-smokers, and the difference was statistically significant. In addition, smokers had a higher prevalence of mouth and tooth sensitivity than non-smokers did. On the other hand, Dosunmu, Lawal, & Akinyemi (2015) elaborated that even while smokers had less frequent gingival bleeding and a lower gingival index, there were no appreciable differences between how well non-surgical periodontal therapy worked for them compared to non-smokers.

Many research papers approved that several parameters or conditions increase *Candida* colonization and vice versa; pro-candidal factors found in tobacco (Rath and Haller, 2022), the relationship between saliva acidity, which is partly brought by smoking, and *Candida* carriage (Vila et al., 2020), Cigarette smoking might lead to localized epithelial alterations allowing candidal colonization (Manfredi et al., 2018). Smoking affects the prevalence of *Candida* species in the mouth. Both have detrimental effects on oral health (Muzurovic et al., 2013).

The results of the present study elaborated that, the presence of *Candida* was evaluated and the number of *Candida* colonies was counted by Manual Colony Counter between smokers and non-

Table 4
Comparison of smokers and non-smokers responses groups regarding effects on oral health.

Effects on oral health	Number (%)	Non-smoker	Smoker	*P* value
The feeling of dry mouth at night or when waking up.				
Yes	30 (63.8)	4(13.3)	26(86.7)	0.005*
No	17 (36.2)	9(52.9)	8(47.1)	
Total Number	47 (100)			
Suffering from bad breath				
No	18 (38.3)	9(50.0)	9(50.0)	0.020*
May be	24 (51.1)	4(16.7)	20(83.3)	
Always	5 (10.6)	0	5(100)	
Total Number	47 (100)			
Suffering from any oral mucosal abnormality (gingivitis, oral pathology, dental plaque deposition periodontitis, and prominent)				
Rarely	26 (55.3)	11(42.3)	15(57.7)	0.039*
Some time	17 (36.2)	2(11.8)	15(88.2)	
Frequently	4 (8.5)	0(0.00)	4(100)	
Total Number	47 (100)			
Suffering from mouth ulcers from time to time frequently				
Rarely	39 (82.9)	11(28.2)	28(71.8)	0.023*
Some time	6 (12.8)	0(0.00)	6(100)	
Frequently	2 (4.3)	2(100)	0	
Total Number	47 (100)			

*Significant ($P < 0.05$).

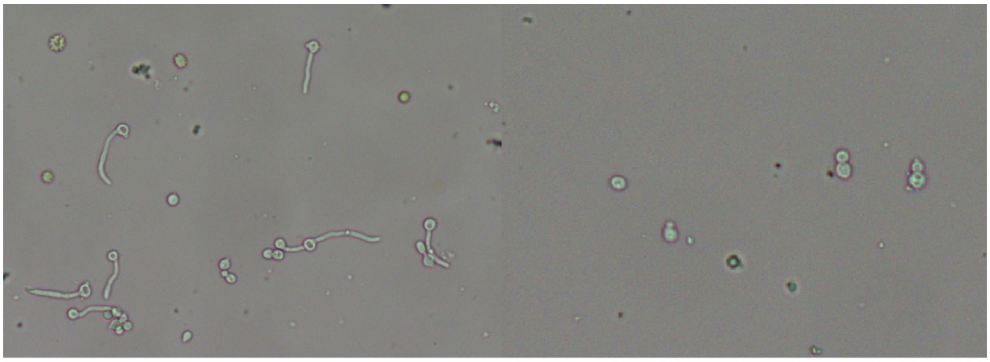

Fig. 1. Micrograph of Germ tube formation in *Candida* spp. (a); *Candida albicans* (slender tube structure without constrictions, germ tube positive). (b); Sample no. RO 39 *Candida tropicalis*, (yeast cells without pseudohyphae/tube structure, Negative results) (×40).

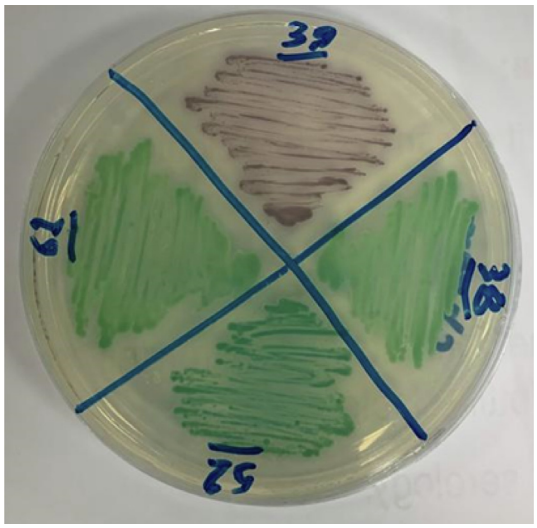

Fig. 2. Photograph of different *Candida* spp. on Chromogenic agar medium (CHROMagar *Candida*). RO 38, 52, and 61 samples, different strains of Candia albicans showed light green colored colonies while RO 39 metallic blue after 48 h of aerobic incubation at 37 °C.

Table 5
Relation between the presence of *Candida* spp. and its number between smokers and non-smokers.

Presence of *Candida* spp.	Number (%)		*P* value
	Smoker	Non-smoker	
Yes	17 (89.5)	2 (10.5)	0.031*
No	17 (60.7)	11 (39.3)	
Total *Candida* spp.	19 (100)		
Candida albicans	17 (89.5)	1 (5.6)	
Total of *Candida albicans*	18 (100)		
Candida tropicalis	0(0.00)	1 (5.26)	
Total of *Candida tropicalis*	1 (100)		
The number of *Candida* spp.			
<1000	6 (85.7)	1 (14.3)	0.087
1000 and more	11 (91.7)	1 (8.3)	
Mean ± SD	648.35 ± 1837.63	220.46 ± 492.5	0.415
Total	19 (100)		

*Significant ($P < 0.05$).

smokers. Out of 19 *Candida* isolates, 18 (94.7 %) were identified as *Candida albicans* and 1 (5.3 %) as *Candida tropicalis*. The most often isolated species from the oral cavity in people with oral candidiasis and other health conditions is *Candida albicans* (Akpan and Morgan, 2002). Moreover, *Candida albicans* was considered the species that was the most commonly human-fungal isolates (Muzurovic et al., 2013).

Table 6
Correlation between smokers using one or two types of smoking and between volunteers suffering from the chronic disease with the presence of *Candida* spp.

Presence of *Candida* spp.	Number (%)		*P* value
	Smokers using more than one type (n = 7)	Smokers using only one type (n = 27)	
Yes	2 (10.5)	17 (89.5)	0.013*
No	5 (33.3)	10 (66.7)	
Presence of *Candida* spp.	**Volunteers who suffer from chronic disease (n = 5)**	**Volunteers who don't suffer from chronic disease (n = 42)**	***P* value**
Yes	3(15.8)	16 (84.2)	0.555
No	2 (7.1)	26 (92.9)	

*Significant ($P < 0.05$).

Among the volunteers who presented with oral *Candida,* 89.5% were smokers, while non-smoker volunteers,10.5 %, so it can be concluded that smoking was a significant positive correlation to the presence of *Candida* in the oral cavity. Volunteers with the presence of oral *Candida* were smokers in 82.5% of cases, while patients without *Candida* were smokers in 44% of cases (Muzurovic et al., 2013). But the correlation between smoking and the number of *Candida* per total oral rinse was not statically significant. We figured out that the volunteers who showed growth of<1000 CFU of *Candida* are 7(36.84%), the total of smokers is 6 (85.7%) while the non-smokers 1(14.3%). This finding was matched with Darwazeh, Al-Dwairi, & Al-Zwairi (2010) concluded that in healthy patients, smoking tobacco did not seem to lead to an increase in oral *Candida* species colonization. While, Muzurović, et al (2013) stated that, patients who had oral *Candida* were smokers in 33 (82.5%) cases, compared to 44 (44%) patients who did not have oral *Candida*. Smoking affects the prevalence of *Candida* species in the mouth, both are detrimental to dental health. The present study showed that a comparison of smokers using one or two types of smoking regarding the presence of *Candida* showed a significant finding.

According to our results, the proposed hypothesis of a possible link between smoking and oral *Candida* carriage was confirmed. This may be due to several causes that have been covered in previous studies on smoking or nicotine products (*in vivo* and *in vitro* studies): *Candida* colonization is influenced by mucosal changes (Ye et al., 2021), nicotine affects the growth of *C. parapsilosis* as well as *C. albicans* biofilms (Gunasegar and Himratul-Aznita,

Table 7
Antifungal susceptibility of different antifungal antibiotics against different isolates of *Candida* spp.

Sample Code	Ro 36	Ro 39	Ro 52	Ro 37	Ro 7	Ro 61	Ro 38	Ro 4	Ro 25	Ro 30	Ro 8	Ro 53	Ro 6	Ro 60	Ro 26	Ro 29	Ro 31	Ro 50	Ro 39
Antifungal drug	Mean zone of inhibition (mm)																		
NY	25	27	25	27	20	22	21	21	30	28	27	27	22	21	24	25	25	25	26
FY	00	00	00	00	00	00	00	00	00	12	00	00	00	00	00	00	00	00	00
AMB	15	15	18	19	14	16	13	15	15	24	17	20	18	12	18	20	20	20	16

AMB; Amphotericin B (20 μg), FY; Flucytosine (1 μg), NY; Nystatin (IU), Diameter of the inhibition zone measured by millimeters (mm).

Fig. 3. Photograph of Antifungal susceptibility testing of Amphotericin B (AMB 20 μg), Flucytosine (FY 1 μg), and Nystatin (NY IU) showing zone of inhibition on Sabouraud's dextrose agar (SDA after 48 h of aerobic incubation at 37 °C.

2019). Smoking may affect the risk factors of oral candidiasis, one of them acidity of saliva correlated to the state of candidal carriage (Hato et al., 2022). Changes to the epithelium may be caused by smoking which allows candidal colonization. *C. albicans* may potentially receive sustenance from cigarette smoke (Navabi et al 2021). Tobacco is an effective inducer of the yeast-to-hyphal form transition, which may increase *C. albicans*' pathogenicity in humans (Ali and Karuppayil, 2018). Furthermore, cigarette smoke encouraged *C. albicans* to change from blastospore to pseudohyphal form enhancing human pathogenicity. The pretreatment of cigarette smoke condensates high amounts of chitin gene expression, measuring two-fold to eight-fold under hyphal conditions, was seen in *C. albicans* (Alanazi et al., 2014; Ali and Karuppayil, 2018). Smoking causes oral mucosa immunosuppression, which increases oral mucosal sensitivity to *C. albicans*, this is due in part to the negative regulatory action of nuclear factor erythrocyte 2-related factor 2 (Nrf2). Smoking and *C. albicans*-related oral illnesses including oral leucoplakia are thought to have Nrf2 as a critical regulator and a possible therapeutic target due to its connection to oxidative stress and inflammation (Ye et al., 2021).

5. Conclusion

Our study indicated that; smoking was a significant positive correlation to the presence and number of *Candida* in the oral cavity this means smoking is an important predisposing factor for yeast in the oral cavity, and the majority of this yeast is *Candida albicans*, approximately 95% of cases. There are no discernible differences in the sensitivity of isolated *Candida* to antifungals. Smoking has an impact on oral health across all examined parameters (an oral mucosal abnormality, mouth ulcers, bad breath, and feeling of dry mouth). While there was no difference between smokers and nonsmokers in their tooth-brushing practices.

Data availability

No data were used to support this study.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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