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Prevalence of Candida Species in regard to the type of Cancer Patients Undergoing Chemotherapy in Erbil City, Iraq

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***Abstract*— Candida species, particularly *Candida albicans*, cause oral candidiasis, which is an infection of the oral mucosa. A total of 100 oral swab samples were obtained from patients with oral lesions as well as patients with cancer receiving chemotherapy, radiation therapy in in Erbilto study the relationship of Candida species and cancer. On CHROM agar, they were identified depending on the color of the colony, including: *Candida albicans* (light green), *C. dubliniensis* (blue to dark green), *C. tropicalis* (dark green, often with a halo), *C. parapsilosis* (white or creamy), C. *lusitaniae* (Creamy to pale pink), *C. kefyr* (pink to purple), and *C. glabrata* (Pink to creamy). Method Congo Red Agar (CRA) was used for the detection of biofilm formation, while only a few Candida species had biofilm production. The results of CHROM agar and Vitek 2 compact assays were identical to some extent with the molecular method. The antifungal susceptibility of Candida species was evaluated using the drug agar well diffusion method against five antifungal agents: Fluconazole, Griseofulvin, Itraconazole, Ketoconazole, and Nystatin. Most isolates demonstrated resistance to Fluconazole, Griseofulvin, Itraconazole, and Ketoconazole, whereas Nystatin showed sensitivity against most tested Candida species. In this research, each ethanol Golden berry extract (EGB), aquatic Golden berry extracts (AGB) were used. In the agar well diffusion method, most Candida showed low susceptibility values against (EGB) extracts, while all (AGB) extracts were resistant. In conclusion, *Candida albicans* was the predominant species isolated from the oral cavity of cancer patients. This study provides the prevalence, antifungal resistance patterns, and molecular identification of oral Candida species.**

***Keywords*— Oral Candidiasis, CHROM agar Candida sp. Vitek YBC, antifungal sensitivity test.**

I. Introduction

Candida species are a type of fungus that can lead to opportunistic infections, especially in people with weakened immune systems, though they may also be present in healthy individuals in areas like the mouth, digestive system, and vagina[1-2]. Non-Candida albicans Candida species (NCACs) have increased recently; however, *Candida albicans* still is the most prevalent *Candida* species reported in candidiasis[3-4]. In patients with cancer undergoing chemotherapy or radiation therapy, oral candidiasis is a frequent side effect. Depending on the patient and treatment strategy, its reported prevalence might range from 30% to 60%[5]. Recent studies suggest a significant association between Candida infections and increased cancer risk. Candida species have been linked to both short- and long-term development of cancers, including those of the oral cavity, throat, stomach, pancreas, colon, liver, breast, and skin[6-8]. *Candida albicans* may be involved in oral carcinogenesis through immunological regulation, carcinogenic metabolite synthesis, and chronic inflammation, according to recent data. The development of oral squamous cell carcinoma (OSCC) has been linked to persistent oral colonization by *Candida albicans* [9]. The pathogenesis of Candida depends on numbers of elements, including various morphological forms, phenotypic transition, biofilm formation, tissue-damaging enzymes released by fungi, and environmental pH variation [10]. Candida identification employs phenotypic, chromogenic, automated, rapid, and molecular techniques. Traditional methods relied on phenotypic traits such as morphotyping, biotyping, serotyping, and chemical resistance, but molecular approaches are increasingly favored for their high sensitivity, specificity, and accuracy. Classical Polymerase Chain Reaction (PCR) is a widely used molecular method that amplifies variable fungal DNA regions, such as the Internal Transcribed Spacer (ITS) or 18S rRNA gene, using species-specific primers. This enables precise differentiation of Candida species, including *C. albicans*, *C. glabrata*, and *C. parapsilosis*. Although less advanced than real-time PCR or sequencing, classical PCR remains a rapid, reliable, and cost-effective tool for routine identification [9-12]. Golden berry (*Physalis peruviana*), a member of the Solanaceae family, is a medicinal plant traditionally used to treat various ailments. It can be cultivated as a perennial or annual crop and is widely grown in subtropical regions. Rich in phytochemicals, including vitamins, minerals, and antioxidants, golden berries exhibit notable nutritional value along with anti-inflammatory and antibacterial properties [13-15]. This study provides data on the prevalence, antifungal resistance patterns, and molecular characterization of oral

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Candida isolates among cancer patients in Erbil**,** and uniquely explores the antifungal potential of golden berry extract against these clinical isolates. The aims of the study were the isolation and identification of fungi from the oral cavity in cancer patients, by using microscopic examination, CHROM agar medium, and molecular genetic analysis. Then, we determined the antifungal resistance pattern in the isolates, and examined the effectiveness of plant extracts in inhibiting or reducing the development of the isolates.

II. Materials and Methods

A. Collection and Isolation of Samples

One hundred oral swab samples were collected from cancer patients receiving chemotherapy and/ or radiation therapy at various hospitals in Erbil city. The patient visited an oncology clinic with symptoms suggestive of oral candidiasis, such as whitish plaque, halitosis, discomfort, dry mouth, erythematous lesions, ulcerative lesions, and impaired taste perception. The samples were taken from both sexes randomly, ages between 4 to 85 years, from September to November 2024. The ethics committee of the Science College at Salahaddin University approved the protocol. Demographic and clinical data (cancer type, age, sex, location of patients, number of cancer therapy doses, and duration of the disease) were documented. The swabs of patients were cultured on Sabouraud Dextrose Agar (SDA)plates. All samples were moved to the mycological laboratory in the department of Biology, College of Science. Then the swabs were cultivated on SDA with chloramphenicol, and incubated aerobically for 48 hours at 37 °C [16].

B. Fungal identification.

1- Phenotypic Identification.

Following a 48-hour incubation period, cultures were classified as positive if yeast growth was present and negative if no yeast growth was detected. A tiny portion of the colony was transferred onto a clean glass slide and stained with a drop of lactophenol cotton blue. After covering the plate with a cover slide, the colony was inspected macroscopically to ensure that all cultures were positive. The slide was examined with a 40X light microscope. This makes it possible to see blossoming oval yeast cells and pseudo hyphae, which are characteristics of several *Candida* species [17]. Additionally, Hi Crome *Candida* differential agar (Hi media Mumbai, India) was used to speciate *C. albicans* and NCAC. This confirms that their species-level identified by acting as a differential and selective medium. On Candida differential agar (CDA), *Candida albicans* produced green colonies, *Candida kefyr* produced pink to purple colonies, *Candida tropicalis* produced dark blue colonies often with a halo, and *Candida glabrata* produced pink to creamy colonies.

2- Congo Red Agar

To assess biofilm formation, Congo red agar was prepared using a combination of brain heart infusion broth, glucose, and Congo red dye. *Candida species* were inoculated onto the plates using a streaking method and incubated at 37°C for 2 to 3 days Saxena et al. [18].

3- Vitek 2

The VITEK 2 Compact system (manufactured by bioMérieux, France) is a fully automated diagnostic tool commonly employed in clinical microbiology laboratories to identify both bacteria and yeast isolates. It operates by detecting color and fluorescence changes resulting from biochemical reactions, enabling fast and accurate microorganism identification.

For the identification of yeasts, particularly Candida species, the system utilizes YST ID cards, which include a series of biochemical tests specifically designed to differentiate among clinically significant yeasts and yeast-like organisms. The procedure involved preparing a yeast suspension in sterile saline adjusted to a specific turbidity (generally between 1.8 and 2.2 McFarland units), after which the suspension was loaded into the YST card and placed into the VITEK2 machine for incubation. Identification results were typically generated within 18 to 48 hours**,** depending on the organism's growth characteristics [19- 20].

4- Molecular identification

The fungal DNA's 500-550 bp region in the rDNA fragments was effectively amplified using universal primers created by [12]. A PCR master mix with a total volume of 25 µL was prepared, including 3 µL of genomic DNA, 12.5 µL of 2XGoTaq Green Master Mix (Promega, USA), and 1 µL each of the ITS1 forward and ITS4 reverse primer (see Table 1) for sequences: ITS1 – 5′-TCC GTA GGT GAA CCT GCG G-3′ and ITS4 – 5′-TCC TCC GCT TAT TGA TAT GC-3′). The remaining volume was adjusted by adding 7.5 µL of nuclease-free water. PCR amplification was carried out using a Techne thermocycler (UK) with the following thermal profile: initial denaturation at 95 °C for 5 minutes, followed by 35 cycles of denaturation at 94 °C for 1 minute, primer annealing at 55 °C for 1 minute, and extension at 72 °C for 1 minute. A final extension step was performed at 72 °C for 7 minutes. The amplified PCR products were checked by electrophoresis on a 2% agarose gel using 1X TBE buffer. The DNA target sequence analysis was done using MEGA11 and alignment to NCBI- BLAST (http://www.ncbi.nlm.nih.gov/BLAST/). The PCR products were sequenced on an3500 Genetic analyzer (Applied Biosystems). Candida isolates' PCR products were forwarded to Macrogen in South Korea for sequencing.

TABLE 1. Sequence of Universal primers

Gene	Sequence of primer
ITS1	′5-TCC GTA GGT GAA CCT GCG G-′3
ITS4	5′ TCC TCC GCT TAT TGA TAT GC-′3

5- Antifungal Sensitivity Test

Both agar well and disc diffusion methods were used to ensure accurate antifungal testing. Agar well diffusion

suits liquid samples or plant extracts, allowing direct placement in wells for better diffusion. Disc diffusion is standardized for pure antifungal drugs using impregnated paper discs.

5.1. Agar Well Diffusion Method

Antifungal drugs such as Itraconazole, Griseofulvin, Nystatin, fluconazole, and ketoconazole were tested using *Candida* isolates shown in Table 2. For every medication, a stock solution was prepared, except for itraconazole, which was dissolved in dimethyl sulfoxide (DMSO). To make stock solutions, the other medications were dissolved in SDW. The cultures were reduced to $1x10^6$/ml using a bright-line hemocytometer. The *C. albicans* inoculum was made from forty-eight-hour-old colonies that were grown on SAB 2%.In Pennsylvania's Horsham, Hausser Scientific[21]. Briefly, a 0.1 mL (100 µL) suspension of each isolate was uniformly spread onto SDA agar plates. Using a sterile corkborer, wells measuring 6 mm in diameter were punched into the agar. Each well was then loaded with 100 µL of the corresponding antifungal agents. The plates were incubated at 37 °C for 24 hours, after which the inhibition zone diameters were measured in millimeters with a ruler for each drug tested [22- 23].

TABLE 2. Antifungal susceptibility test drugs

No	Antifungals	Batch No.	Potency	Conc.	Company
1	Fluconazole	20951	150mg/ Cap.	15mg/m l	D.Ph./Germ any
2	Griseofulvin	288	500mg/ Tab.	50mg/m l	Brusseis,Be lglum
3	Itraconazole	KA334 92	100mg/ Cap.	10mg/m l	CIPLA LTD. India
4	Ketoconazole	122026 0	200mg/T ablet	20mg/m l	Amman-Jordan
5	Nystatin	121002 86	500000I U/Tab.	50000IU /ml	Lasi,Roma nia,EU

5.2. Disc diffusion method

The National Committee for Clinical Laboratory Standards currently recommends this methodology as the reference method (NCCLS). The isolates were tested for their susceptibility to antifungal drugs in vitro using the Kirby-Bauer disk diffusion technique. Utilizing Biorex Diagnostics Microbiology Sensitivity Discs, the test included 50mg of Econazole, 50 mg of Ketoconazole, 50mg of Miconazole, and 100mg of Nystatin, shown in Table 3. The United Kingdom is where these CDs were produced. For the experiment, SDA agar enhanced with 8% glucose was utilized. This methodology, currently considered the reference standard, was recommended by the National Committee for Clinical Laboratory Standards (NCCLS) [24].

TABLE 3. Antifungal susceptibility test discs

No.	Antifungals	Code	Disc potency (Unit/disc)	Company
1	Econazole	42-FOO-012	50mg	Biorex/UK
2	Ketoconazol	42-FOO-015	50mg	Biorex/UK
3	Miconazol	42-FOO-014	50mg	Biorex/UK
4	Nystatin	42-FOO-009	100IU	Biorex/UK

5.3 Plant extraction and preparation

Forty grams of peel powder were weighed and combined with 160ml of sterile distilled water (SDW) to create an aqueous extract. After weighing twenty grams of materials, 200 milliliters of ethanol (95%) were added. Then, the extract was shaken carefully in a shaker for 1 hr in (shaking incubator-4045/Gallen Kamp-9 B/England) and kept at 4 °C for twenty-four hours. The extract was filtered by using Whatman paper, dried in a Petri dish, and then the powder was placed in vials and kept in a refrigerator. A stock solution was prepared for each extract by mixing 1g of ethanol plant extract with Dimethyl Sulfoxide (Riedel-DeHaen AG, Germany) and 1g of aqueous plant extract with SDW (5 ml). Millipore filters were then used to sterilize the mixture(0.2µm) [25-26]. Each sample was replicated three times using SDW for the aquatic extract and DMSO for the alcoholic extract as a control.

III. Statistical Analysis

Statistical analysis in this study was performed using GraphPad Prism version 9.0. Categorical variables were evaluated using the Pearson Chi-square (χ^2) test, while quantitative data were expressed as the mean ± standard error (SE). Differences between group means were determined through analysis of variance (ANOVA).

IV. Results and Discussion

In the current study, the prevalence rate of oral candidiasis was determined in the investigation among 100 cancer patients, Males (42%) and females (58%). The negative cultures were 24 (24%), while the positive cultures were 76 (76%), which includes: non-Candida species (3), 3.94 %, oral candidiasis 73 (73%). Phenotypically summarized the isolates in Table 4 and Figure 1. Each of *Candida albicans* (58.90%), *C. lusitaniae* (2.73%), *C. dubliniensis* (13.69%), *C. glabrata* (2.73%), *C. kefyr* (9.58%), *C. parapsilosis* (4.10%), and *C. tropicalis* (8.21%) was identified phenotypically; the number of *C. albicans* is higher than non-albicans species. They were identified phenotypically, based on macroscopic and microscopic examination, colony color, and germ tube production.

TABLE 4. Phenotypic identification of the isolates

Age	Isolates	NO	%	CT	RT	Ma. E	Mi. E	CT&RT	CT&BT	CT, RT&BT
20-71	No growth	24	(24)	13	3	-	-	2	6	0
61-76	Non-*Candida* spp.	3	(3)	2	0	-	-	0	1	0
4-85	*Candida* spp.	73	(73)	44	8	+	+	4	16	1

Note: CT (chemotherapy), RT (radiotherapy), BT (biological therapy), Ma.Macroscopic examination, Mi. E (microscopic examination).

In the biofilm test, the results showed biofilm; only 2 samples had (++) moderate against Candida species, while 14 samples were (+) weak positive, and 57 samples had no biofilm production phenotypic identification on Chromogenic agar (Hi media, India), based on colony color, Germ tube, and Biofilm formation. Illustrated in Table 5.

TABLE 5. phenotypic identification of *Candida* species

No	***Candida* isolate**	**Chromagar**	**Germ tube**	**Biofilm on Congo red agar**
1	*C. albicans*	light green	+	+
2	*C. dubliniensis*	Blue (dark green)	-	-
3	*C. parapsilosis*	White or creamy	-	-
4	*C. tropicalis*	Dark green often with a halo	-	-
5	*C. keyfr*	pink to purple	-	-
6	*C. lusitaniae*	Creamy to pale pink	-	-
7	*C. glabrata*	Pink to creamy	-	-

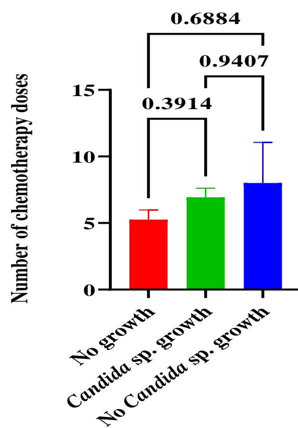

Fig 1: Chemotherapy doses and possibility of fungal infection.

Illustrates the prevalence of various *Candida* species in cancer patients receiving chemotherapy shows the average chemotherapy doses among patients. *C. albicans* had the Highest mean dose, followed by *C. kefyr and C. lusitaniae,* while *C. glabrata* had the lowest. Statistical analysis found no significant differences between species. Suggesting chemotherapy dose doesn't substantially influence the type of Candida infection. P-value>0.05 (ns) shown in Fig. 2.

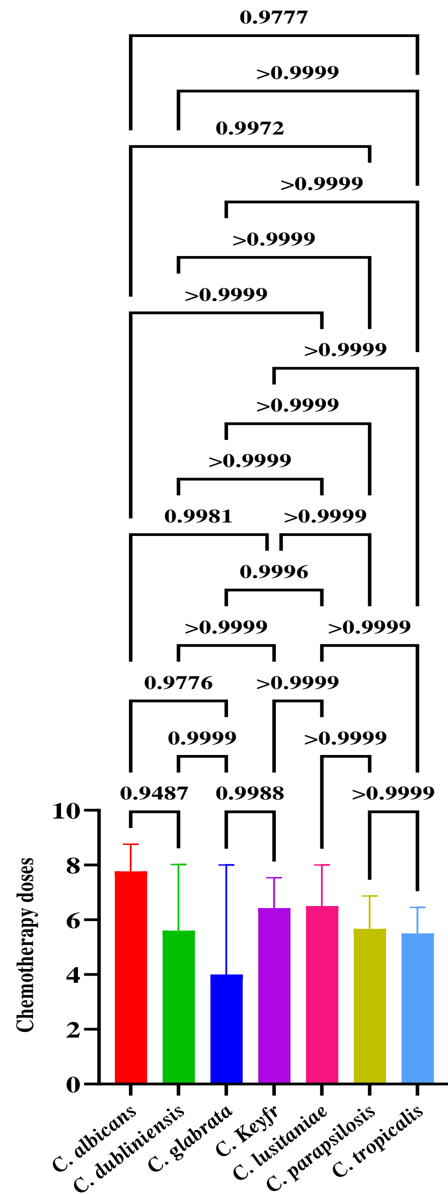

Fig 2: Chemotherapy doses and possibility of *Candida* sp. infection.

The prevalence of various *Candida* species in cancer patients receiving chemotherapy, classified by cancer type. Fig. 3.

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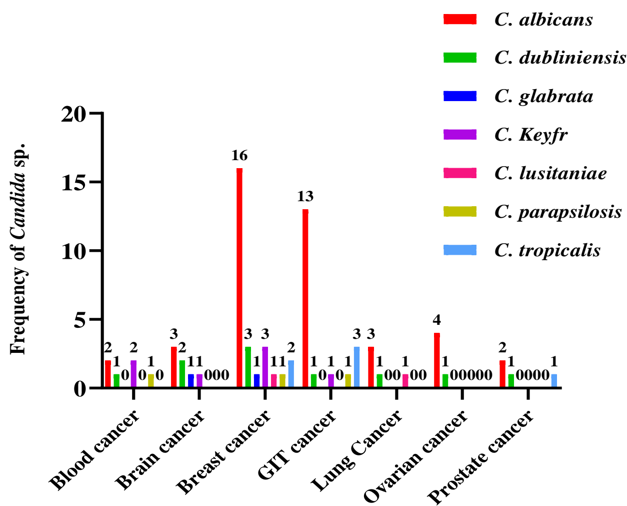

Fig 3: Prevalence of *Candida* sp. regarding the type of cancer Patients Undergoing Chemotherapy

Susceptibility testing of *Candida* species against antifungal discs: Clotrimazole, Ketoconazole, Miconazole, and Nystatin, treating C. dubliniesis using the disc diffusion method. All dics sensitive against Candida is shown in Fig. 4.

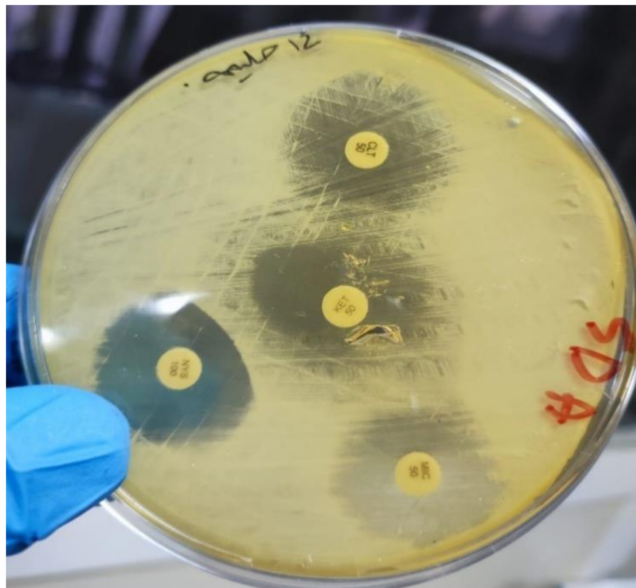

Fig. 4: Susceptibility testing of antifungal discs against *Candida dubliniensi*.

The sensitivity test of the antifungals showed that Candida species were resistant to Itraconazole, Fluconazole, Ketoconazole, and Griseofulvin as shown in Figures 5, 7, 8, and 9, while most of the isolates of Candida species were sensitive to Nystatin in Fig. 6.

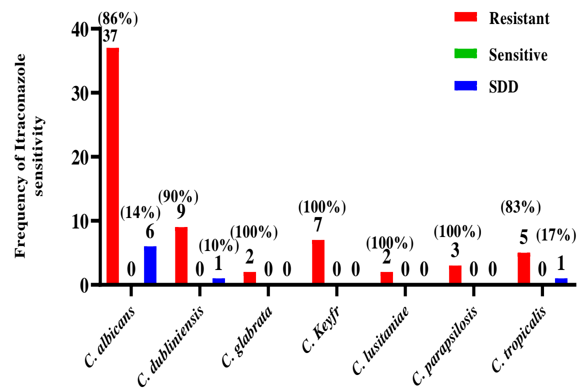

Fig 5: Sensitivity test of Itraconazole antifungal agent according to the *Candida sp.* Most of them are isolating c. sp resistance to

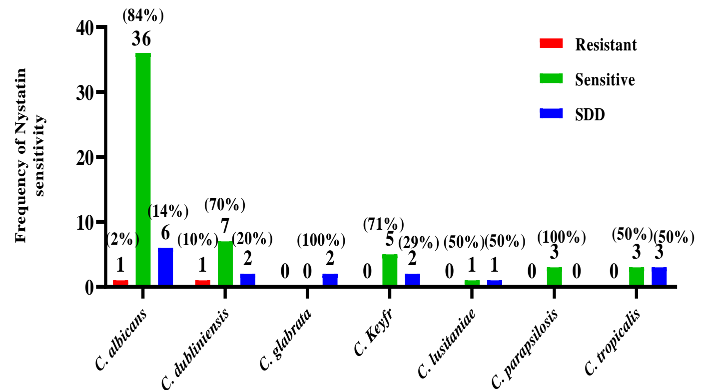

Fig. 6: Sensitivity test of Nystatin antifungal agent according to the *Candida sp.*

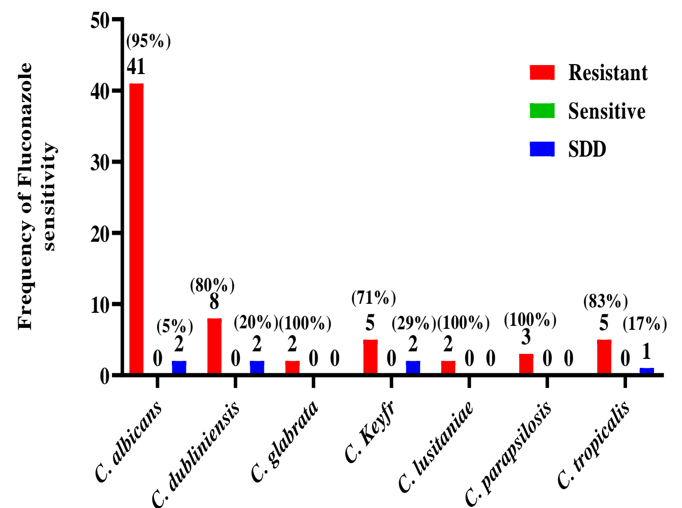

Fig 7: Sensitivity test of Fluconazole antifungal agent according to the *Candida sp.*

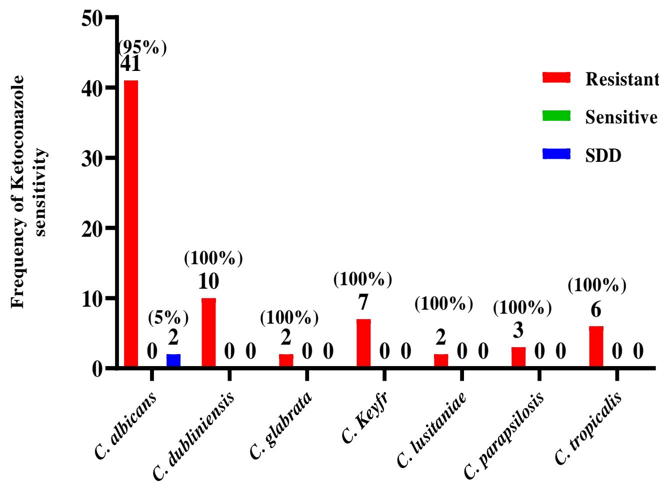

Fig. 8: Sensitivity test of the Ketoconazole antifungal agent according to the *Candida sp.*

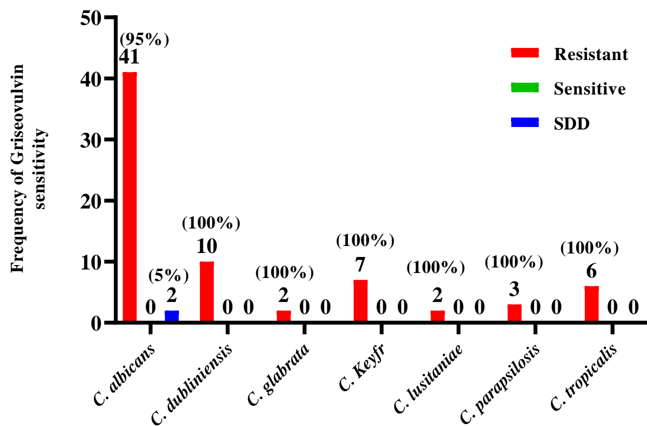

Fig 9: Sensitivity test of Griseoulvin antifungal agent according to the *Candida sp*.

Susceptibility testing of golden berry extract against an isolate of *Candida albicans*. The results Showed that the growth did not impact by the aquatic extract, but it was by the alcoholic extract. In which (6 mm) was the lower susceptibility value against ethanol golden berry extracts (GBE), which had a low effect shown in Table 6. Negative less than 6.

TABLE 6. Susceptibility testing of *Candida* species against Plant extracts

***Candida* isolates**	**Plant extracts**	
	Diameter of inhibition zone(mm)	
	GB	
	GBE	**GBA**
C. albicans	6	0
C. tropicalis	6	0
C. dubliniensis	5	0
C. parapsilosis	2	0
C. keyfr	4	0
C. lusitaniae	2	0
C. glabrata	5	0

Sequencing analysis of multiple amplification products demonstrated 99-100% sequence similarity with Candida strains. The nucleotide sequences of the isolates were submitted to GenBank and assigned specific accession numbers. Additionally, the isolates were deposited in GenBank under these accession numbers. A BLAST search using the NCBI database identified the isolates as *Candida albicans*, *Candida tropicalis*, and *Candida dubliniensis*. is shown in Table 7

TABLE 7. GenBank accession number for Candida isolates

Candida isolates	**GenBank accession no.**
Kluyveromyces marxianus (*C. kefyr*)	PV690258
C.albicans	PV690260
C.albicans	PV690261
C. albicans	PV690262
Kluyveromyces marxianus (*C. kefyr*)	PV690263
Candida tropicalis	PV690264
C. dubliniensis	PV690265
Candida albicans	PV690266
Clavispora lusitaniae	PV690267

ITS1 and ITS4 gene amplification were used for molecular identification, and the results were in agreement with the phenotypic study. The molecular identification of isolated Candida was done using universal primer gene amplification-PCR. Many specimens of molecular information from ITS nucleotide sequences allowed for precise isolate identification and characterization illustrated in Figure 10.

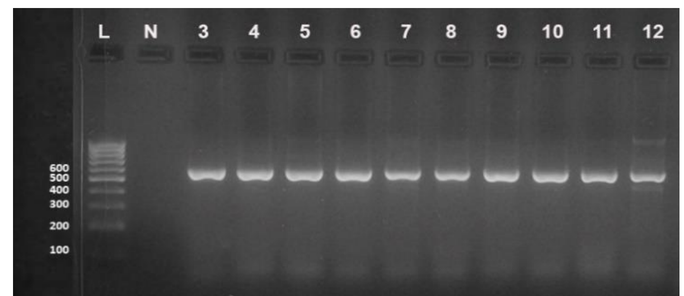

Fig. 10: PCR amplification of an adjacent segment of the nuclear small subunit rRNA gene. Lane L: DNA ladder 100bp, Lane N: Negative control, Lane 3-12: Fungal ITS region amplification (550bp).

Phylogenetic tree for internal transcribed spacer gene sequences forms 6 fungal isolates using primers (ITS1, ITS4). The numbers at the nodes indicate the level of bootstrap support (%) based on Maximum Likelihood analysis of 100 re-sample datasets. The scale bar indicates the phylogenetic distance corresponding to 0.010 changes per 100 bases.

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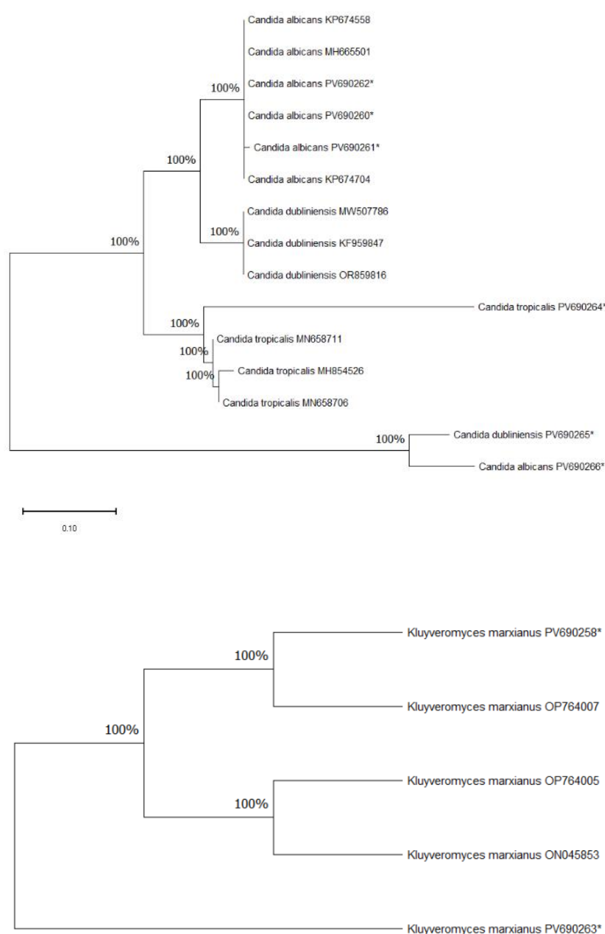

Fig 11: Phylogenetic tree for internal transcribed spacer gene sequences form 6 fungal isolates using primers (ITS1, ITS4).

The overgrowth of *Candida albicans* in the oral cavity is the main cause of oral candidiasis, sometimes referred to as oral thrush, an opportunistic fungal illness. Additionally, it becomes more frequently linked to non-albicans Candida species, especially in patients who are medically weakened and previously had several azole antifungal therapies [27-29].

Opportunistic fungal infections, particularly oral candidiasis, are common in patients receiving chemotherapy or radiotherapy. Radiation therapy for malignancy alters the oral environment, creating conditions that favor the colonization of the oral mucosa by yeast species, most often Candida.

Patients undergoing chemotherapy have a weak immune system, making them highly susceptible to infections. The oral cavity provides an ideal environment for the growth of microorganisms and fungi, which can lead to life-threatening complications in these individuals [30-31].

This study's findings align with Mohsin and Ali (2021), who used Sabouraud Dextrose Agar (SDA) and microscopic examination to analyze and classify each fungal sample[32]. Microscopic techniques were utilized by Hussain *et al.*,(2020) to detect and differentiate Candida strains [33]. Ozcan *et al.*(2010). Lewis and Williams (2017) further verified that *Candida albicans* was the most common pathogen found in both health and disease, present in 80% of samples, and isolated from human oral cavity specimens [34].

Numerous studies have shown that antifungal resistance and biofilm development are important factors in the pathogenesis of the disease [35- 36]. According to a 2010 study, about 70% of persons have *Candida albicans*, and several additional studies have discovered that the prevalence of *C. albicans* is higher than that of NCACs[37, 38]. The results agree with those of Faqe Abdulla, who studied a test for yeast about the production of biofilms using Congo red agar (CRA) with no biofilm was observed [39]. However, the findings disagree with those of Faqe Abdulla and Ismael, who used phenotypic tests such as Congo red agar (CRA) to identify the development of biofilms by *Candida albicans* [40]. The Congo Red Agar (CRA) method demonstrated an increasing number of positive *Candida* biofilm cases with prolonged incubation time, consistent with the findings of Oliveira et al [41]. This variation is attributed to differences in biofilm-forming capacity, as not all isolates produce strong biofilms; some exhibit moderate or weak biofilm formation [42]. reported that Black colonies with a dry crystalline consistency were identified as strong biofilm producers, and pink colonies as weak biofilm producers. On the other hand, white or light pink colonies were identified as non-biofilm producers.

Candida strains were classified as sensitive (Zone diameter ≥19mm), Susceptible dose-dependent (15-18mm), and resistant (≤14mm) in a disk diffusion approach that assessed the samples' drug susceptibility [43]. Nystatin was shown to be more effective than Clotrimazole, Fluconazole, and Ketoconazole in the antifungal susceptibility pattern of Candida isolates, according to Gandhi et. al [44]

Additionally, Burgess et al. evaluated fluconazole with isolates of Candida species classified as resistant and susceptible-dose dependent (SDD) [45].

Because Golden berry has few effects against Candida species in the treatment of oral candidiasis, while Garlic (*Allium sativum*) has demonstrated significant antifungal properties, primarily attributed to its active compound allicin. Studies have shown that garlic extract exhibits strong antifungal activity against *Candida albicans*, the primary causative agent of oral candidiasis. For instance, a study found that garlic extract at a 75% concentration exhibited the highest antifungal activity against *C. albicans* growth[46]. Antifungal resistance has significant clinical implications in the management of oral candidiasis. While Nystatin, a polyene antifungal, is generally effective against *Candida albicans* with low resistance rates, azoles such as fluconazole are increasingly facing resistance, particularly in immunocompromised patients. This resistance can lead to treatment failure, prolonged infections, and the need for alternative or combination therapies. Therefore, understanding the resistance profiles of Candida species is

crucial for selecting appropriate therapy and ensuring effective clinical outcomes.

Sequencing examination of a few amplification products revealed 99% to 100% sequence similarity with the Candida strains. Isolate sequencing data yielded a nucleotide sequence GenBank accession number. The isolates of Candida were added to a gene bank assigned accession numbers [47].

In the present investigation, PCR amplification and sequencing were applied to detect Candida species and to validate their phenotypic traits, demonstrating the advantage of combining molecular assays with phenotypic characterization for reliable species identification [48].

Overall, our findings revealed that all Candida species isolates exhibited significant differences in their sensitivity to each of the tested antifungal discs and drugs.

This result close to A Ghojoghi. Our study found that *C. albicans* was the most commonly isolated yeast species, followed by *C. dubliniensis* and *C. glabrata*. Although *C. albicans* continues to be the predominant species, the prevalence of non-albicans Candida (NAC) species has increased notably over the past two decades[49].

The close morphological and physiological resemblance between *C. albicans* and *C. dubliniensis* often results in Misidentification. While molecular-based methods are the most dependable for distinguishing between them, there is still a need for accurate, rapid, and cost-effective tests for use in many laboratories[50].

The increased occurrence of breast and GIT cancers could be attributed to conditions such as damage to the mucosal barrier, disruptions in the normal microbial balance (dysbiosis), and the immune suppression caused by chemotherapy, all of which can promote Candida proliferation[51].

Based on the phylogenetic tree, the isolates are categorized into two groups or one clade. It was shown that there is a close association between *Candida albicans* and other species.

V. Conclusions

In this study, Candida species were isolated from oral lesions in cancer patients, with *Candida albicans* was the most prevalent species. Molecular methods provided more accurate and sensitive detection. Accurate species identification and analysis of antifungal resistance profiles are crucial for optimizing treatment and preventing misuse of antifungal drugs. While topical Nystatin is commonly used for treatment of oral candidiasis, the increasing frequency of infections in patients undergoing chemotherapy and radiotherapy, antifungal critically important in cancer patients because they are highly susceptible to invasive fungal infections due to immunosuppression. Ensures that antifungal drugs are used appropriately, reducing the risk of resistance development, minimizing drug toxicity, and improving clinical outcomes.

Conflict of Interest

Authors declare that they have no conflict of interest.

References

[1]N. Valand, O. Gazioglu, H. Yesilkaya, M. Shivkumar, N. Horley, R. Arroo, R. Wallis, U. Kishore, and U. Venkatraman Girija, "Interactions of Candida tropicalis pH-related antigen 1 with complement proteins C3, C3b, factor-H, C4BP and complement evasion," *Immunobiology,* vol. 228, no. 1, pp. 152303, 2023.

[2]D. Launder, J. Dillon, L. Wuescher, T. Glanz, N. Abdul-Aziz, E. Yi, J. Naglik, R. Worth, and H. Conti, "Immunity to pathogenic mucosal C. albicans infections mediated by oral megakaryocytes activated by IL-17 and candidalysin," *Mucosal Immunol,* vol. 17, no. 2, pp. 182-200, 2024.

[3]C. Logan, I. Martin-Loeches, and T. Bicanic, "Invasive candidiasis in critical care: challenges and future directions," *Intensive Care Med,* vol. 46, no. 11, pp. 2001-2014, 2020.

[4]L. Černáková, M. Roudbary, S. Brás, S. Tafaj, and C. Rodrigues, "Candida auris: A Quick Review on Identification, Current Treatments, and Challenges," *Int J Mol Sci,* vol. 22, no. 9, 2021.

[5]R. Lalla, M. Latortue, C. Hong, A. Ariyawardana, S. D'Amato-Palumbo, D. Fischer, A. Martof, O. Nicolatou-Galitis, L. Patton, and L. Elting, "A systematic review of oral fungal infections in patients receiving cancer therapy," *Supportive care in cancer,* vol. 18, no. 8, pp. 985-992, 2010.

[6]M. Nørgaard, R. Thomsen, D. Farkas, M. Mogensen, and H. Sørensen, "Candida infection and cancer risk: a Danish nationwide cohort study," *European journal of internal medicine,* vol. 24, no. 5, pp. 451-455, 2013.

[7]L. Narunsky-Haziza, G. Sepich-Poore, I. Livyatan, O. Asraf, C. Martino, D. Nejman, N. Gavert, J. Stajich, G. Amit, and A. González, "Pan-cancer analyses reveal cancer-type-specific fungal ecologies and bacteriome interactions," *Cell,* vol. 185, no. 20, pp. 3789-3806. e17, 2022.

[8]A. Dohlman, J. Klug, M. Mesko, I. Gao, S. Lipkin, X. Shen, and I. Iliev, "A pan-cancer mycobiome analysis reveals fungal involvement in gastrointestinal and lung tumors," *Cell,* vol. 185, no. 20, pp. 3807-3822. e12, 2022.

[9]S. Ahmad, and Z. Khan, "Invasive candidiasis: a review of nonculture-based laboratory diagnostic methods," *Indian journal of medical microbiology,* vol. 30, no. 3, pp. 264-269, 2012.

[10]I. Mba, and E. Nweze, "Mechanism of Candida pathogenesis: revisiting the vital drivers," *Eur J Clin Microbiol Infect Dis,* vol. 39, no. 10, pp. 1797-1819, 2020.

[11]S. Singh, “Molecular-genetic approaches for identification and typing of pathogenic Candida yeasts: a review,” *International Journal of Innovative Research in Science, Engineering and Technology,* vol. 3, no. 9, pp. 199-211, 2014.

[12]T. J.White, T. Bruns, S. Lee, and J. Taylor, “Amplification and direct sequencing of fungal ribosomal RNA genes for phylogenetics,” *PCR protocols: a guide to methods and applications,* M. A. Innis, D. H. Gelfand, J. J. Sninsky, and T. J. White, Eds., San Diego, CA, USA: Academic Press, 1990, ch. 38, pp. 315–322

[13]O. Rop, J. Mlcek, T. Jurikova, and M. Valsikova, “Bioactive content and antioxidant capacity of Cape gooseberry fruit,” *Central European Journal of Biology,* vol. 7, pp. 672-679, 2012.

[14]Ö. Çakir, M. Pekmez, E. Çepni, B. Candar, and K. Fidan, “Evaluation of biological activities of Physalis peruviana ethanol extracts and expression of Bcl-2 genes in HeLa cells,” *Food Science and Technology,* vol. 34, pp. 422-430, 2014.

[15]A. Corrales-Bernal, A. Vergara, B. Rojano, E. Yahia, and M. Maldonado, “Características nutricionales y antioxidantes de la uchuva colombiana (Physalys peruviana L.) en tres estadios de su maduración,” *Archivos Latinoamericanos de Nutrición (ALAN),* vol. 65, no. 4, pp. 254-262, 2015.

[16] R. Yonis, K. Luti, and G. Aziz, “Statistical optimization of chitin bioconversion to produce an effective chitosan in solid state fermentation by Asperigellus flavus,” *Iraqi Journal of Agricultural Sciences,* vol. 50, no. 3, 2019.

[17]M. Babić, and M. Hukić, “Candida albicans and non-albicans species as etiological agent of vaginitis in pregnant and nonpregnant women,” *Bosnian journal of basic medical sciences,* vol. 10, no. 1, pp. 89, 2010.

[18]S. Abdulla, B. Dheeb, S. AL-Qaysi, M. Farhan, M. Massadeh, and A. Hamid, “Isolation and Diagnosis of Biofilms of Klebsiella pneumoniae Bacteria and Candida albicans Yeast, and Studying the Sensitivity of The Pathogens to Antibiotics and Antifungals,” *Egyptian Academic Journal of Biological Sciences, G. Microbiology,* vol. 15, no. 2, pp. 73-88, 2023.

[19]D. H. Larone, *Medically important fungi: a guide to identification*, 3rd ed. New York, NY, USA: Elsevier,1993.

[20]B. Posteraro, E. De Carolis, A. Vella, and M. Sanguinetti, “MALDI-TOF mass spectrometry in the clinical mycology laboratory: identification of fungi and beyond,” *Expert review of proteomics,* vol. 10, no. 2, pp. 151-164, 2013.

[21]E. Aboualigalehdari, N. Sadeghifard, M. Taherikalani, Z. Zargoush, Z. Tahmasebi, B. Badakhsh, A. Rostamzad, S. Ghafourian, and I. Pakzad, “Anti-biofilm Properties of Peganum harmala against Candida albicans,” *Osong public health and research perspectives,* vol. 7, no. 2, pp. 116-118, 2016.

[22]D. Srinivasan, S. Nathan, T. Suresh, and P. Perumalsamy, “Antimicrobial activity of certain Indian medicinal plants used in folkloric medicine,” *Journal of ethnopharmacology,* vol. 74, no. 3, pp. 217-220, 2001.

[23]S. Abdulateef, O. Atalla, M. L-Ani, T. Mohammed, F. Abdulateef, and O. Abdulmajeed, “Impact of the electric shock on the embryonic development and physiological traits in chicks embryo,” *Indian Journal of Animal Sciences,* vol. 90, no. 11, pp. 1541-1545, 2021.

[24]K. Othman, S. Abdullah, B. Ali, and M. Majid, “Isolation and Identification Candida spp from Urine and Antifungal Susceptibility Test,” *Iraqi Journal of Science,* vol. 59, no. 4B, pp. 1981-1988, 2018.

[25]J. Rios, M. Recio, and A. Villar, “Antimicrobial activity of selected plants employed in the Spanish Mediterranean area,” *Journal of ethnopharmacology,* vol. 21, no. 2, pp. 139-152, 1987.

[26]L.-Y. Cai, F.-X. Shi, and X. Gao, “Preliminary phytochemical analysis of Acanthopanan trifoliatus (L.) Merr,” *Journal of Medicinal Plants Research,* vol. 5, no. 17, pp. 4059-4064, 2011.

[27]U. Kibwana, J. Manyahi, D. Kamori, M. Mushi, A. Mwandigha, and M. Majigo, “Predominance of non-Candida albicans species oral colonisation among patients on anticancer therapy: findings from a cross-sectional study in Tanzania,” *BMJ Open,* vol. 13, no. 4, pp. e070003, 2023.

[28]B. Nguyen, H. Nguyen, T. Vo, M. Le, V. Tran-Nguyen, T. Vu, and P. Nguyen, “Prevalence and drug susceptibility of clinical Candida species in nasopharyngeal cancer patients in Vietnam,” *One Health,* vol. 18, pp. 100659, 2024.

[29]A. Alfaifi, T. Wang, P. Perez, A. Sultan, T. Meiller, P. Rock, D. Kleiner, D. Chertow, S. Hewitt, B. Gasmi, S. Stein, S. Ramelli, D. Martin, B. Warner, and M. Jabra-Rizk, “SARS-CoV-2 infection of salivary glands compromises the production of a secreted antifungal peptide with potential implications for development of oral candidiasis,” *PLoS Pathog,* vol. 20, no. 12, pp. e1012375, 2024.

[30]H. Wong, “Oral complications and management strategies for patients undergoing cancer therapy,” *ScientificWorldJournal,* vol. 2014, pp. 581795, 2014.

[31]A. Mirabile, G. Numico, E. Russi, P. Bossi, F. Crippa, A. Bacigalupo, V. De Sanctis, S. Musso, A. Merlotti, M. Ghi, M. Merlano, L. Licitra, F. Moretto, N. Denaro, O. Caspiani, M. Buglione, S. Pergolizzi, A. Cascio, J. Bernier, J. Raber-Durlacher, J. Vermorken, B. Murphy, M. Ranieri, and R. Dellinger, “Sepsis in head and neck cancer patients treated with chemotherapy and radiation: Literature review and consensus,” *Crit Rev Oncol Hematol,* vol. 95, no. 2, pp. 191-213, 2015.

[32]Z. Mohsin, and W. Ali, “Antagonistic Activity of Bacteriocin-producing Lactobacillus Against Candida spp,” *Iraqi Journal of Science,* vol. Vol. 62, no. No. 7, pp. pp: 2153-2162, 2021.

[33]N. Hussain, I. Latif, and H. Obaid, “Antimicrobial Activity of Non-bond Colicin on Candida albicans Biofilm,” *Iraqi Journal of Science*, pp. 2831-2837, 2020.

[34]M. Lewis, and D. Williams, “Diagnosis and management of oral candidosis,” *British dental journal,* vol. 223, no. 9, pp. 675-681, 2017.

[35]Z. Malinovská, E. Čonková, and P. Váczi, “Biofilm Formation in Medically Important Candida Species,” *J Fungi (Basel),* vol. 9, no. 10, Sep 22, 2023.

[36]O. Ajetunmobi, H. Badali, J. Romo, G. Ramage, and J. Lopez-Ribot, “Antifungal therapy of Candida biofilms: Past, present and future,” *Biofilm,* vol. 5, pp. 100126, 2023.

[37]P. Dharmik, A. Gomashe, and V. Upadhyay, “Susceptibility pattern of various azoles against Candida species causing vulvovaginal candidiasis,” *The Journal of Obstetrics and Gynecology of India,* vol. 63, pp. 135-137, 2013.

[38]L. Krishnasamy, D. Rubini, J. Senthilganesh, C. Saikumar, G. Kumaramanickavel, A. W. Aruni, and P. Nithyanand, “Phylogenetic characterization of biofilm forming multidrug resistant Candida albicans and non albicans Candida causing vulvovaginal candidiasis,” *Gene Reports,* vol. 19, pp. 100644, 2020.

[39]N. Faqeabdulla, “The First Isolation and Diagnosis of Cytobasidium slooffiae from Women Suffering from Vulvovaginitis in the City of Erbil, Kurdistan Region, Iraq,” *Rafidain Journal of Science,* vol. Vol. 34,, no. No. 1,, pp. pp. 1-11,2025.

[40]N. Faqe Abdulla, and H. Ismael, “The Efficacy of Antifungal Medications and Plant Extracts Against *Candida albicans* Isolated from Vulvovaginitis Women ” *Iraqi Journal of Science,* vol. 64, no. 2, pp. 560-572 2023.

[41]M. Oliveira, S. Nunes, C. Carneiro, R. Bexiga, F. Bernardo, and C. Vilela, “Time course of biofilm formation by Staphylococcus aureus and Staphylococcus epidermidis mastitis isolates,” *Veterinary microbiology,* vol. 124, no. 1-2, pp. 187-191, 2007.

[42]F. Eftekhar, and T. Dadaei, “Biofilm formation and detection of icaAB genes in clinical isolates of methicillin resistant Staphylococcus aureus,” *Iranian Journal of Basic Medical Sciences*, vol. 14, no. 2, pp. 132–136, 2011 2011.

[43]S. Hosseini, H. Joshaghani, T. Shokohi, A. Ahmadi, and Z. Mehrbakhsh, “Antifungal Activity of ZnO Nanoparticles and Nystatin and Downregulation of SAP1-3 Genes Expression in Fluconazole-Resistant Candida albicans Isolates from Vulvovaginal Candidiasis,” *Infect Drug Resist,* vol. 13, pp. 385-394, 2020.

[44]T. Gandhi, M. Patel, and M. Jain, “Antifungal susceptibility of Candida against six antifungal drugs by disk diffusion method isolated from vulvovaginal candidiasis,” *International Journal of Current Research and Review,* vol. 7, no. 11, pp. 20, 2015.

[45]D. Burgess, R. Hastings, K. Summers, T. Hardin, and M. Rinaldi, “Pharmacodynamics of fluconazole, itraconazole, and amphotericin B against Candida albicans,” *Diagnostic microbiology and infectious disease,* vol. 36, no. 1, pp. 13-18, 2000.

[46]T. Agustantina, and R. Soekartono, “Antifungal activity from garlic extract (Allium sativum) against Candida albicans growth,” *Indonesian J Dent Med,* vol. 4, pp. 60-2, 2021.

[47]N. Q. FaqeAbdulla, “The Prevalence of Cervical-Vulvovaginal Candidiasis in Erbil Province and the Antibiofilm Activity of Some Plant Extract against Isolated Species,” Ph.D. dissertation, Dept. of Biology, Salahaddin Univ., Erbil, Iraq, 2022.

[48]G. Rodríguez-Leguizamón, A. Fiori, L. López, B. Gómez, C. Parra-Giraldo, A. Gómez-López, C. Suárez, A. Ceballos, P. Van Dijck, and M. Patarroyo, “Characterising atypical Candida albicans clinical isolates from six third-level hospitals in Bogotá, Colombia,” *BMC microbiology,* vol. 15, no. 1, pp. 199, 2015.

[49]A. Ghojoghi, M. Erfaninejad, E. Ahmadpour, E. Nazar, A. Barac, and M. Fatahinia, “Non-albicans candidemia in cancer patients as an increasing health problem: A comprehensive review and meta-analysis,” *Asian Pacific Journal of Tropical Medicine,* vol. 15, no. 9, pp. 387-399, 2022.

[50]S. Gamarra, E. Mancilla, C. Dudiuk, and G. Garcia-Effron, “Candida dubliniensis and Candida albicans differentiation by colony morphotype in Sabouraud-triphenyltetrazolium agar,” *Rev Iberoam Micol,* vol. 32, no. 2, pp. 126-8, 2015.

[51]M. Netea, G. Brown, B. Kullberg, and N. Gow, “An integrated model of the recognition of Candida albicans by the innate immune system,” *Nat Rev Microbiol,* vol. 6, no. 1, pp. 67-78, 2008.