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Emergence of multi-drug resistance Candida auris in Saudi Arabia

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Title: Emergence of multi-drug resistance *Candida auris* in Saudi Arabia

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Abstract:

Background: *Candida auris* is an emerging multi-drug resistant pathogen with high mortality rate, several cases and nosocomial infections have been reported worldwide causing a major challenge for clinicians and microbiological laboratories.

Objectives: The study aim to, describe new cases of *C. auris* from different sites of infection confirmed by MALDI TOF MS and ribosomal sequencing, develop the phylogenetic analysis of these isolates, detect resistant strains of *C. auris* to both azole and echinocandin by amplification of ERG11 and FKS1 genes.

Methods: A total of six specimens were collected from blood, urine, ear swab and groin screening sample during the period from November 2018 to March 2019. Isolates were incubated for 48 hours on Sabroud agar at 42°C, then confirmed by MALDI-TOF MS. Sequences of 18S rRNA gene from isolates and phylogenetic analysis were performed. Finally, molecular analysis of resistance genes was performed to detect the efficacy of treatment.

Results: Clinical isolates were identified by the growth on Sabroud agar at 42°C and confirmed by MALDI-TOF MS analysis. Evolutionary analyses were conducted in MEGA7 and sequences of 18S rRNA gene were submitted to GenBank. All samples were positive for both ERG11 and FKS1 which confer azole- and echinocandin-resistant strains.

26 **Conclusions:** This study shed light on a public health threat of an emerging pathogen.
27 Therefore, the hospital implemented strict contact screening and infection control precautions
28 to prevent *C. auris* infection. Finally, there is a critical need to monitor the antifungal
29 resistance in different geographical areas and implementation of efficient guidelines for
30 treatment.

31 **Keywords:**

32 *Candida auris*, nosocomial infections, emerging pathogen, resistance genes, Saudi Arabia.

33 **Introduction:**

34 *Candida auris* is an emerging pathogen that had been reported in the past decade as a rising
35 threat and a challenging nosocomial infection (1). *C. auris* tends to transmit rapidly from
36 person to person and persist on medical devices and on the surfaces of hospital areas (2,3).

37 It has emerged in Japan from a culture of external ear canal in 2009 and within the last decade,
38 *C. auris* is frequently isolated from the bloodstream, urinary and respiratory tract and it has
39 been reported from 16 countries in Africa, Asia, Europe, America and the Middle East with
40 significant fatality rate (4).

41 The detection of *C. auris* is still a challenging dilemma for clinical laboratories because it is
42 closely related to other *Candida species* such as, *C. haemulonii*, *C. duobushaemulonii,* and *C.*
43 *lusitaniae* (5,6). *C. auris* grows well on Sabouraud and chromogenic agar at 37°C and 42°C
44 and there are different methods to identify *C. auris*, however, molecular sequencing and
45 MALDI-TOF MS are the most accurate techniques and remain the most efficient diagnostic
46 tools (7–9).

47 Azoles, echinocandins, and amphotericin B are the three main classes of antifungal agents, the
48 increasing fluconazole resistance could be explained by mutations in *ERG11* gene that
49 encoding lanosterol 14-alpha-demethylase which has an essential role in the ergosterol

50 synthesis pathway (10). In contrast, echinocandin resistance remains quite low in most
51 *Candida species* except *Candida glabrata* and it is linked to mutations of *FKS*, which is the
52 gene encoding the catalytic subunits of the enzyme β-1,3-d-glucan synthase that target the drug
53 (11). Antifungal sensitivity of *C. auris* can be evaluated by using either microdilution or disk
54 diffusion test. However, genotyping of *ERG11* and *FKS1* is the most valuable method to detect
55 azole and echinocandin resistance and the efficacy of antifungal therapy (12)

56 In 2018, the first two cases from Saudi Arabia were reported in 2018, followed by five cases
57 of *C. auris* infection in two different cities (13,14). The aim of this study is to describe new
58 cases of *C. auris* infection and to detect antifungal resistance genes namely,
59 *ERG11* and *FKS1* in isolates of this global emerging pathogen.

60 **Materials and Methods:**

61 **Samples collection and microbial identification:**

62 A total of six specimens were collected from five patients who were admitted to a tertiary care
63 hospital in Al Khobar, Saudi Arabia. Specimens were collected from sterile and non-sterile
64 sites including; from blood, urine, ear swab and groin screening sample during the period from
65 November 2018 to March 2019. Clinical microbiology testing was performed according to IPP
66 in the hospital using different culture media. All clinical isolates were incubated for 48 hours
67 on Sabroud agar at 42°C then confirmed by MALDI-TOF MS, the identification procedure was
68 performed according to the manufacturer's protocol and guidelines of yeast identification (15).

69 **DNA extraction and sequencing of *18S rRNA* gene**

70 Total genomic DNA from isolates, CA1, CA3, C4, CA5, CA7 and CA8 were extracted as per
71 the manufacturer's instruction using Qiagen's Yeast/Bact. Kit (Gentra Puregene Yeast/Bact.
72 Kit, Qiagen, Germany). The *18S rRNA* gene (1655 bp) of all the strains was amplified using

73 18SrRNAF (5'-GCTTAATTTGACTCAACACGGGA-3') and 18SrRNAR (5'-
74 AGCTATCAATCTGTCAATCCTGTC-3') primers, (MoleQule-On, New Zealand) at
75 annealing temperature at 61.8 °C using absolute master mix (MoleQule-On, New Zealand) in
76 T-Professional thermocycler (Biometra, Germany) for 35 cycles.

77 The PCR amplicons were purified using QIAquick PCR Purification Kit (Qiagen, Germany)
78 after visualized the product using 2% agarose gel. The purified amplicons were sequenced
79 using 3500 genetic analysers (Applied Biosystems, United States) with the forward reverse
80 primers used for the amplification using BigDye® Terminator v3.1 Cycle Sequencing Kit
81 (Applied Biosystems, United States).

82 **Molecular identification**

83 The 18S rRNA gene sequence from isolates, CA1, CA3, C4, CA5, CA7 and CA8 were aligned
84 and analysed using Basic Local Alignment Search Tool, PHYMYCO-DB, and FungiDB
85 (16,17). An evolutionary relationship of samples was constructed using Maximum Likelihood
86 method based on the Tamura-Nei model in MEGA7 software package with bootstrap
87 consensus tree from 500 replicate (18–20).

88 **Detection of resistance genes**

89 Primers for the *FKS1* and *ERG11* genes were designed and used for the study. *FKS1* gene was
90 amplified using forward primer (FKS1aF 5'ATGTCTTACGATAACAATCACAACTAC-
91 3'), reverse primer (FKS1aR 5'-AGTAAGATTCGGCCAACTTAGCAG-3') (MoleQule-On,
92 New Zealand) with the 1900 bp amplicon. *ERG11* gene was amplified using forward primer
93 (ERG11aF 5'- ATGGCCTTGAAGGACTGCATCGT -3'), reverse primer (ERG11aR 5'-
94 TTAGTAAACACAAGTCTCTCTTTTCTCCCA -3') (MoleQule-On, New Zealand) with the
95 1575 bp amplicon. The *FKS1* and *ERG11* genes were amplified at 59.4°C and 62.2°C annealing
96 temperature respectively using absolute master mix (MoleQule-On, New Zealand) in T-

97 Professional thermocycler (Biometra, Germany) for 35 cycles. Amplicons were visualized
98 using 2% agarose gel and documented.

99 **Results:**

100 All specimens were collected from Saudi male patients who were diabetic and had different
101 comorbidity diseases. Unfortunately, three patients died almost one month after detection of *C.*
102 *auris* (Table 1). All clinical samples were isolated on culture media then, confirmed by
103 MALDI-TOF MS analysis.

104 Sequences of 18S rRNA gene were submitted to GenBank, accession number: CA1:
105 MN658527.1; CA3: MN658529.1; CA4: MN658530.1; CA5: MN658531.1; CA7:
106 MN658533.1 and CA8: MN658534.1. The evolutionary analysis was concluded with bootstrap
107 consensus tree from 500 replicates (18–20). The analysis included 58 nucleotide sequences,
108 there were an overall of 333 positions in the final dataset and the tree with the highest log
109 likelihood (-1643.31) was demonstrated, Fig.1.
110 All samples were positive for both *ERG11 and FKS1* genes which confer azole- and
111 echinocandin-resistant, amplification of *ERG11* (~1575bp) and *FKS1* (~1900 bp), Fig.2.

112 **Discussion:**

113 *Candida auris* is the first fungal considered as a public health threat, it can spread easily among
114 patients in hospitals, and may cause serious diseases (8). High risk factors associated to *C.*
115 *auris* infections including using broad spectrum antibiotics, catheters, ICU admission(6,21),
116 diabetic patients and mostly infections arise two to seven weeks after admission. *C. auris*
117 infection is correlated with higher resistance to fluconazole and moderately low resistance to
118 echinocandins, therefore echinocandin is recommended as an empirical therapy before
119 antifungal susceptibility testing of collected strains (4).

120 According to a study that reported the first hospital outbreak of *C. auris* in a European hospital,
121 most of clinical manifestations of *C. auris* included colonization followed by candidemia,
122 wound infections, urinary catheter infection and others (22)

123 Guidelines of CDC and recent publications stated that invasive infections may develop after
124 colonization and prophylactic antifungal treatment should be considered in case of a patient
125 colonized with *C. auris* subsequently deteriorates (23–25). In the present study, 90% of the
126 patients were diabetics and using broad spectrum antibiotics, with age range from 62 to 85
127 except one patient, who was 26 years old. Caspofungin was prescribe and the hospital instituted
128 strict contact screening and infection control precautions according to CDC guidelines (4,26).

129 In Saudi Arabia, the first two cases of *C. auris* were detected almost from one to three months
130 after hospital admission and echinocandins had been used for successful treatment (13). Four
131 more cases were reported, where 90% of the isolates were misidentified as *C. haemulonii* and
132 in the same year, one case was confirmed via MALDI-TOF MS (14,27). Recently, a study
133 reported high mortality rate of seven patients with *C. auris* infection (28).

134 Present results concur with a study in Kuwait reported multidrug resistance and high mortality
135 rate among cases of *C. auris* infections(29). Also, two studies in Oman reported of *C.*
136 *auris* occurrence at different hospitals, isolates were highly resistant to fluconazole and the
137 onset of infection after hospitalization ranged from one to two months (30,31)

138 A multicentre study in India showed that 41% of isolates were resistant to two classes of
139 antifungal agents and numerous ERG11 mutations have been detected in *C. auris* isolates from
140 several geographic areas (32). MALDI-TOF MS and gene sequencing are advanced and
141 expensive techniques. Thus, there is a critical necessity to develop cost-effective technique for
142 the detection of this emerging pathogen in underdeveloped countries (33).

Three salient features of this study, descripting new cases of *C. auris* from different sites confirmed by MALDI TOF MS, ribosomal sequencing and developing the phylogenetic analysis of the isolates, and detection of *ERG11* and *FKS1* genes among *C. auris* isolates which related to both azole and echinocandin resistance.

Further studies are crucial to analyse ITS and sequence of resistance genes in addition to the Whole Genome Sequence (WGS) of *C. auris* isolates to discover mechanism of resistance and potential effective treatment for this emerging pathogen.

Abbreviations:

C.auris: Candida auris; MALDI TOF MS: Matrix-Assisted Laser Desorption/Ionization-Time of Flight mass spectrometry; PCR: Polymerase Chain Reaction; IPP: Infection Prevention Program; CDC: Centers for Disease Control and prevention; ITS: Internal transcribed spacer; WGS: Whole Genome Shotgun Sequencing.

Authors Contributions

The study was conceived by Reem Aljindan, Doaa M AlEraky and J. Francis Borgio participated in the design of this study, Reem Aljindan, Nehal M. Hosin coordinate the collection of specimens, maintenance of clinical data and microbial identification, Sayed AbdulAzeez, J. Francis Borgio performed genes identification and sequencing, Doaa M AlEraky, Sayed AbdulAzeez, and J. Francis Borgio wrote the manuscript. All authors reviewed and approved the final manuscript.

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Availability of data and materials:

Data and materials have been provided in the main manuscript, where necessary additional information of the study can be made available from the corresponding author on reasonable request.

173 **Ethics approval and consent to participate:**

174 The study was conducted according to the ethical guidelines and approved by the Microbiology
175 Department of College of Medicine at Imam Abdulrahman bin Faisal University. There were
176 no experiments on human participants and the processing of personal data was anonymized.

177 **Consent for publication:**

178 Not applicable

179 **Competing interests:**

180 The authors declare that they have no competing of interests

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281

282 **Legend**

283 **Table 1:** Five patients with comorbidity diseases indicated positive *C. auris*.

284 **Figure 1: Molecular Phylogenetic analysis of the 6 isolates (CA1, CA3, CA4, CA5, CA7
285 and CA8) of *Candida auris* by Maximum Likelihood method.** The evolutionary history was
286 inferred by using the Maximum Likelihood method based on the Tamura-Nei model (19). The
287 bootstrap consensus tree inferred from 500 replicates is taken to represent the evolutionary
288 history of the taxa analysed(18). Branches corresponding to partitions reproduced in less than
289 50% bootstrap replicates are collapsed. Initial tree(s) for the heuristic search were obtained
290 automatically by applying Neighbor-Join and BioNJ algorithms to a matrix of pairwise
291 distances estimated using the Maximum Composite Likelihood (MCL) approach, and then
292 selecting the topology with superior log likelihood value. The analysis involved 58 nucleotide
293 sequences. Codon positions included were 1st+2nd+3rd+Noncoding. All positions containing
294 gaps and missing data were eliminated. There was a total of 333 positions in the final dataset.
295 Evolutionary analyses were conducted in MEGA7(20).

296 **Figure 2:** The *ERG11* (Lanosterol 14-alpha demethylase) and *FKS1* (1,3-beta-glucan synthase
297 component) gene PCR amplification from *Candida auris*. L: 100 bp ladder; CA1, CA3, CA4,
298 CA5, CA7 and CA8: Isolates of *Candida auris.* CA2 (GenBank: MK910118.1) and CA6
299 (GenBank: MK910117.1): Internal positive controls of *Candida auris*.

Figures

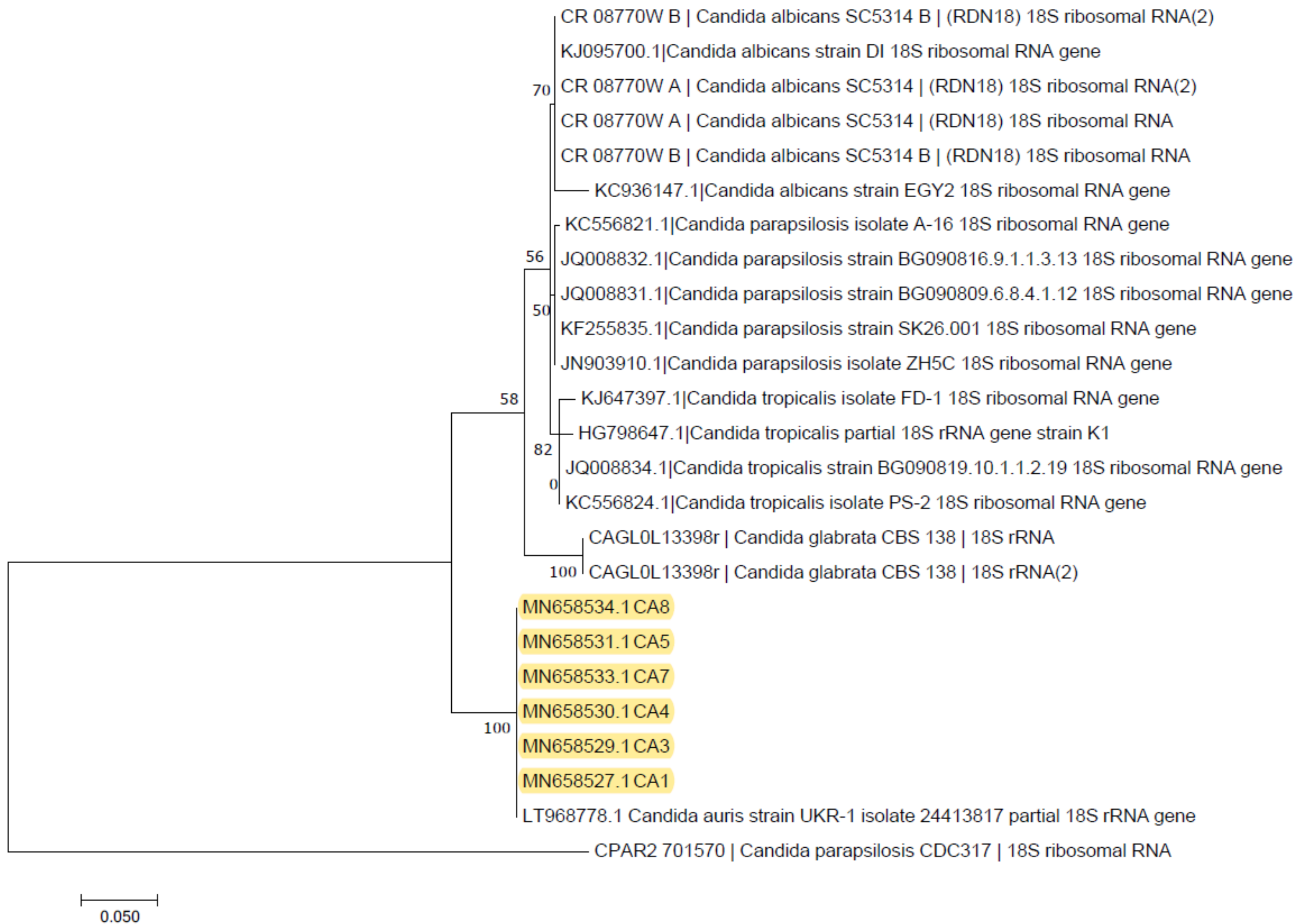

Figure 1

Molecular Phylogenetic analysis of the 6 isolates (CA1, CA3, CA4, CA5, CA7 and CA8) of Candida auris by Maximum Likelihood method. The evolutionary history was inferred by using the Maximum Likelihood method based on the Tamura-Nei model (19). The bootstrap consensus tree inferred from 500 replicates is taken to represent the evolutionary history of the taxa analysed(18). Branches corresponding to partitions reproduced in less than 50% bootstrap replicates are collapsed. Initial tree(s) for the heuristic search were obtained automatically by applying Neighbor-Join and BioNJ algorithms to a matrix of pairwise distances estimated using the Maximum Composite Likelihood (MCL) approach, and then selecting the topology with superior log likelihood value. The analysis involved 58 nucleotide sequences. Codon positions included were 1st+2nd+3rd+Noncoding. All positions containing gaps and missing data were eliminated. There was a total of 333 positions in the final dataset. Evolutionary analyses were conducted in MEGA7(20).

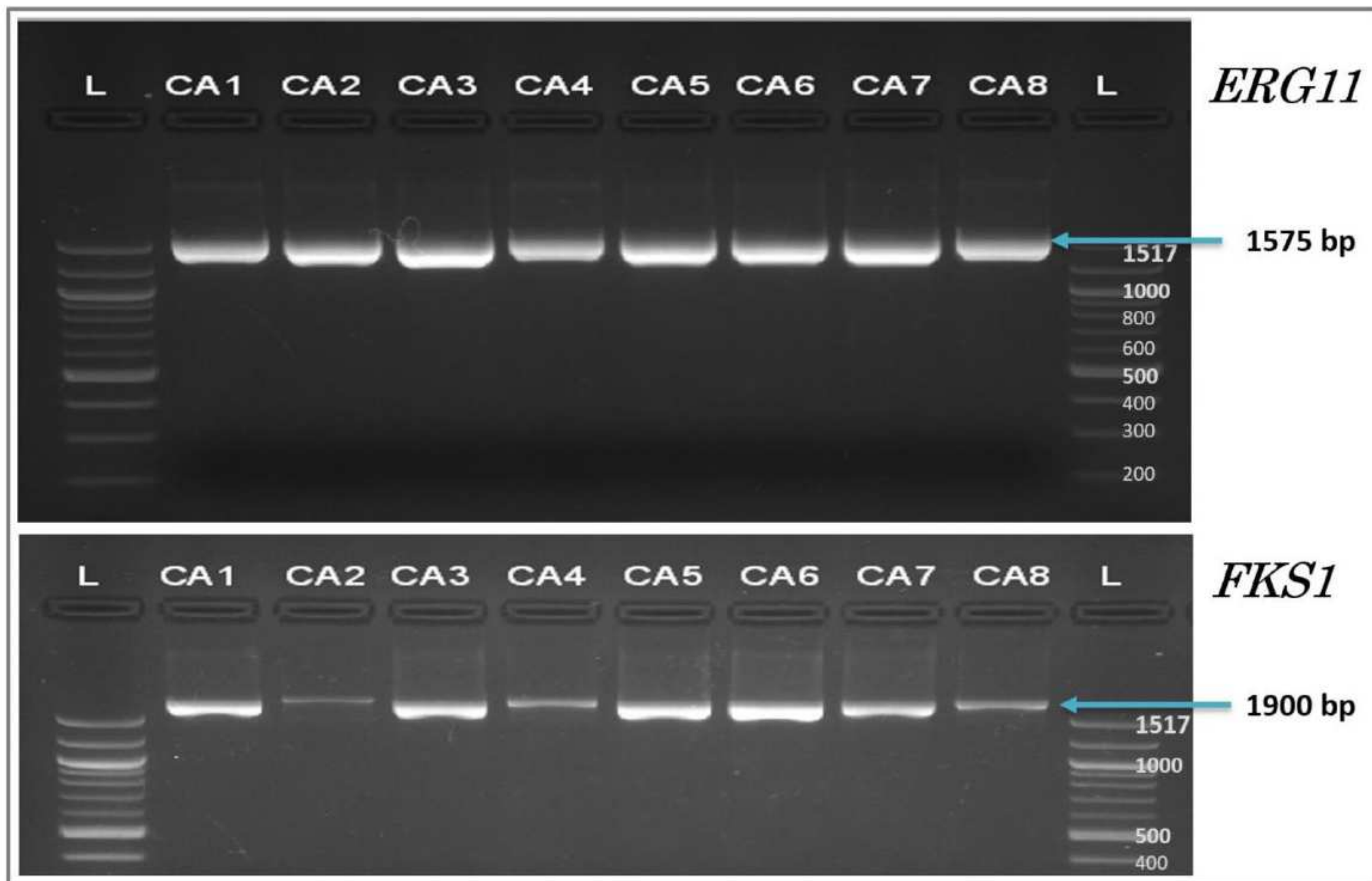

Figure 2

The ERG11 (Lanosterol 14-alpha demethylase) and FKS1 (1,3-beta-glucan synthase component) gene PCR amplification from Candida auris. L: 100 bp ladder; CA1, CA3, CA4, 297 CA5, CA7 and CA8: Isolates of Candida auris. CA2 (GenBank: MK910118.1) and CA6 (GenBank: MK910117.1): Internal positive controls of Candida auris.

Supplementary Files

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- Table1.pdf