# Isolation and molecular identification of *Candida albicans* yeast from women with recurrent vulvovaginal candidiasis and correlation with *MBL-2* gene polymorphism

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Ethics approval statement: this research study has received ethical approval from the Scientific Committee (University of Mosul, College of Environmental Sciences) under approval number 2892 (10/1/2024). All procedures conducted in this study adhere to the ethical guidelines and regulation.

Informed consent statement: this study was conducted in accordance with ethical standards, and informed consent was obtained from all participants. Before participation, individuals were provided with comprehensive information about the study's purpose, procedures, and benefits involved. Participants were informed that their involvement was entirely voluntary, and they had the right to withdraw at any time without any repercussions. Each participant was required to sign a written consent form, which confirmed that they understood the information provided and agreed to participate in the study. Confidentiality of all data collected was assured, and participants were informed that their identities would remain anonymous in any published results.

Availability of data and materials: this published article contains all of the data created or examined during this study

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## Abstract

The abnormal growth of *Candida albicans*, resulting from the imbalance between yeast and bacteria that exists in the mucous membrane of the female reproductive system, leads to the infection with Recurrent Vulvovaginal Candidosis (RVVC). Eighty samples of blood and vaginal swabs were collected from 60 women with RVVC and 20 healthy women. *C. albicans* isolates were identified morphologically and by Polymerase Chain Reaction (PCR) technology. Also, Tetra-primer Amplification Refractory Mutation System-Polymerase Chain Reaction (Tetra-ARMS-PCR) technique was used to identify the single nucleotide polymorphism of *MBL-2* gene in the women blood samples using 4 primers. Results indicate that all women who suffered from RVVC were infected with *C. albicans* and that there is a relationship between the subjects who suffer from RVVC and the genetic variation of *MBL-2*. The results indicate that there are 3 genotypes: normal homozygous CC, hyterozygous CT, and mutant homozygous TT. Additionally, the natural allele repetition C had a value of 71% compared to the mutant allele T, which was 29%, while the Odds Ratio (OR) of the mutant allele was 1.18, which is considered as a serious factor of the case complication. The Hardy-Weinberg Equilibrium Test showed that there is a variation within the recorded and expected genetic patterns and this is due to the effect of factors like antibiotics (Tetracyclines, Quinolones, Broad-spectrum penicillins), pregnancy and hormonal changes, uncontrolled diabetes, impaired immune system.

## Introduction

Recurrent Vulvovaginal Candidosis (RVVC), resulting from the overgrowth of *Candida albicans,* is considered a significant problem all over the world. Studies indicate that 75% of women suffer from RVVC at least once throughout their lives, and about half of them suffer from at least one repetition.[1,2] Although *C. albicans* is considered to be a component of normal flora for many women, the infection takes place due to low immunity or is due to taking contraceptives or the random use of antibiotics,[3] and this results in certain symptoms like itching and irritation and white secretions.[4] Although the human immune system plays a vital role in identifying the receptors that exist on the cell wall of *C. albicans* and causing phagocytosis and inhibition of invading fungi, the incidence of several genetic changes affects the innate immunity, and this increases the host's susceptibility to infection.[5] The most important of these genes is *MBL-2*,[6-8] which is located on the 10q 21-1 chromosome and is considered one of the first defense line against *C. albicans,* because it provides instructions to manufacture the mannose binding protein, which is a member of the Collectin family and is pro-

duced in the liver in response to the infection, targeting the epidemic agent that must be swallowed and broken down by certain immune cells.[9,10] Mannose binding protein participates in the immune defense through its bonding with Mannan (mannose polymers) fraction that exists on *C. albicans* surface and then this association is disengaged by means of activating the complement system (Lectin pathway), which is a set of proteins of the immune system that acts, side by side, to destroy the cause of RVVC.[11,12] The study aimed to explore the relationship between women with RVVC infection and *MBL-2* gene polymorphism compared to healthy women, using tetra Amplification Refractory Mutation System Polymerase Chain Reaction (ARMS-PCR) technique.

## Materials and Methods

The samples were collected from women suffering from RVVC by taking vaginal swabs as well as blood samples; the total samples were 80; 60 of them were women with RVVC and 20 were healthy women. The ages of the women ranged between 20 and 50 years, the mean age of the sample individuals was 27.14 and the standard deviation was ±7.2. Samples were collected at Al-Khansa Hospital in Mosul, Iraq in the period January-July 2024 by a women gynecologist after participants filled a research consent form and the study was approved by the Research Protocol Ethics Committee in Iraq. All experiments were conducted at the College of Environmental Sciences, University of Mosul. The vaginal swabs were put on Potato Dextrose Agar medium and incubated in petri dishes at 28°C for 48 h (fungi Growth Incubator, Indian Scientific systems Pvt. Ltd., Jogeshwari West, Mumbai, Maharashtra, India). *C. albicans* were identified by the color and the general morphology using the simple microscope model TC/XSP-13A (Ticare®, Xiamen, China). As for blood samples, 2 mL of venous blood were taken and placed in sealed tubes containing Ethylene Diamine Tetraacetic Acid (EDTA) anticoagulant and then stored in the refrigerator to be used later on for DNA extraction process using PCR technique.[13]

### Molecular identification of fungi

After *C. albicans* grew in the plate (60 samples), the mycelia were transferred to Potato dextrose broth medium in 250 mL flasks and they were allowed to grow in a shaking incubator at 150 rpm at 28°C for 7 days. The fungi were then collected to extract the DNA using Wizard Genomic (Promega, Madison, Wisconsin, USA) DNA Purification Kit according to the instructions of the manufacturing company; the DNA was amplified by PCR technique using the following primers.

Forward: 5TGAATCACGACTCTTTGAACGC3
Reverse: 5TTTCTTTCCTCCGCTTATTGATAT3

The steps of PCR were denaturation at 94°C for 4 minutes and annealing at 54°C for 1 minute, and finally at 74°C for 1 minute. The reaction was repeated for 30 cycles with a final extension at 74°C for 7 minutes. At the end of the reaction, 2% agarose gel was run to detect the amplification bands.[14,15]

### Extraction of DNA from blood samples

The DNA was extracted from blood samples to search for the gene *MBL-2* and investigate the variation in genotypes and their relationship with the vaginal fungi infection using the method of Iranpur and Esmailizadeh; then, the results were analyzed by means of conducting five molecular tests: i) Tetra arms PCR results, ii) determining the rate of allele frequency and the observation level, iii) Hardy-Weinberg Equilibrium (HWE), iv) verification that the inheritance of this variation in the study population is recessive, v) verification that the inheritance of this variation in the study groups is dominant.[16,17]

### Determination of the genetic variation of *MBL2*(C/T) by using Tetra-ARMS-PCR

A mutation C→ T was identified for the gene *MBL-2* by using allele-specific primers of the gene *MBL-2*, as shown in Table 1.

The PCR condition was an initial denaturation at 95°C for 5 minutes, a denaturation at 95°C for 1 minute, and then annealing at 61°C for 1 minute, extension at 72°C for 1 minute, and then final extension at 72°C for 7 minutes.[18,19]

### Identifying the nucleotides sequence of the amplified pieces

The amplified DNA of *C. albicans* species and *MBL-2* were sequenced and the result was analyzed using BLAST in NCBI (an abbreviation for Basic Local Alignment Search Tool) that compares DNA sequences with those in biological databases in order to find similarities between them. The National Center for Biotechnology Information (www.ncbi.nlm.nih.gov) offers BLAST in both standalone and web versions. The online version allows users to search the entire genomes of numerous organisms, including humans.[20]

### Statistics

The statistical analysis was conducted using T test program (SPSS IBM Corporation, Armonk and New York, USA) that was used to compare genotypes and alleles between women with RVVC and healthy women; the accepted significance level was $p \leq 0.05$. Confidence Interval (CI), p value and Odds Ratio (OR) were calculated. Additionally, the likelihood level (possibility of RVVC) and Hardy's balance were calculated (Hardy–Weinberg equilibrium shows that the allele and genotype frequencies in women will remain constant from generation to generation in the absence of other evolutionary influences).

**Table 1.** Primers used in determining the genetic variation in *MBL-2* gene using the Polymerase Chain Reaction (PCR) technique.

| Primer | Sequence | Band size | Annealing |
|---|---|---|---|
| F-outer<br>R-outer | 5-CTGGTTCCCCCTTTTCTCCCTTGGGGT-3<br>5- CCAGGCAAAGATGGGCGTGAGGG-3 | 445 bp | 61 |
| F-inner | 5- AAAATTCATCCTGTGTCCCACGGGATGG-3 | 297 bp | |
| R-inner | 5- GGTGAGGACCATGTCCCTGTTTCCATCA-3 | 198 bp | |

## Results

The results of the morphological diagnosis showed that *C. albicans* is the main cause of RVVC, as shown in Figure 1. The results of the molecular diagnosis using PCR confirmed that all women suffering from RVVC were infected with *C. albicans* and this because *C. albicans* is characterized by virulence factors and specific tactics that enable it to cause the illness and confront the host's defense mechanisms. These tactics include polymorphism, biofilm formation, the release of exogenous enzymes and mycotoxins. All these tactics enable the development of the illness, the pathogens' survival and the circumvention of the host's defenses.[21,22]

### Analysis of the genetic variation of *MBL-2* and determination of its relationship with the incidence of RVVC

The results of the genetic variation of *MBL-2* were analyzed by submitting them to five molecular tests.

#### *Tetra arms PCR results*

The results of Tetra arms-PCR reaction (Figure 2) show that there is a relationship between the women who suffer from RVVC and the genetic variation of *MBL-2*. The genetic variation of *MBL-2* was manifested by 3 separate genetic alleles which are:

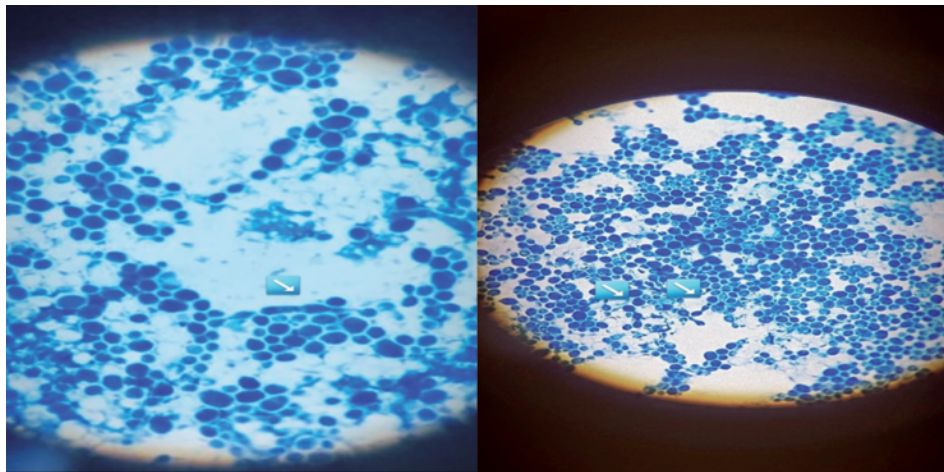

**Figure 1.** Morphological diagnosis of *C. albicans*. The arrows indicate the transformation to a filamentous form.

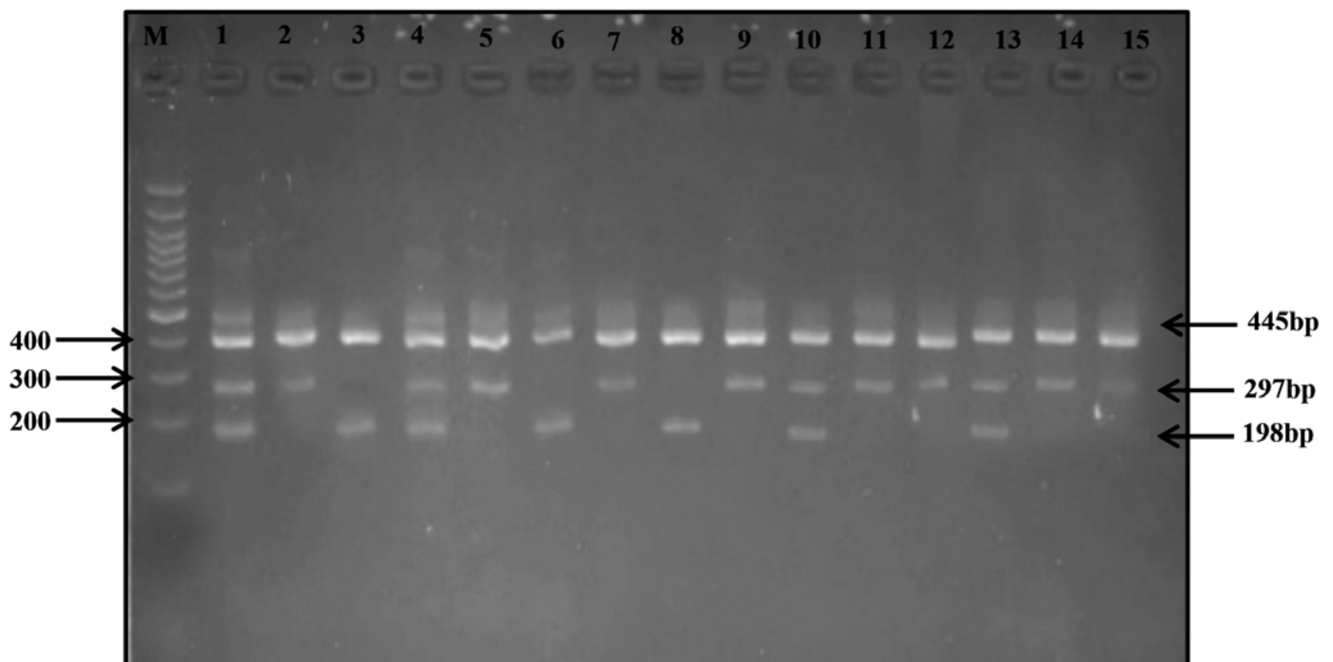


**Figure 2.** Pattern of DNA bands under ultraviolet (UV) light.

CT, CC, and TT, as illustrated in Figure 2, with varying sizes. They were clearly visible in the PCR findings; only fifteen samples were selected for the purposes of clarifying the band and genotypes.

The result of the reaction produced three bands; the first band (size 445 bp) for the normal alleles, the second band (size 297 bp) for the heteroallele, the third band (size 198 bp) for the mutant alleles and the ladder Mix (it is a standard ready mixture containing a sequence of known DNA length for estimating unknown DNA lengths).

Table 2 shows the different genetic patterns of *MBL-2* gene in the patients who suffer from RVVC and the percentages of the CC, TT, and CT genotypes (60, 17, 23%). As for the control group, the percentages of the genotypes were 60, 10, and 30%. Also, from Table 2, it is clear that the OR of the mutant genotype was 1.8; so, as it is higher than 1.0 at a likelihood level of $p<0.05$, it is regarded as a risk factor for the development of the disease.

### *Determining the rate of allele frequency and the observation level*

Results show that the frequency of the wild allele (C) was high (71%) compared with the mutant allele (T), which was 29% and this is considered one of the main reasons behind losing the biological function of the mannose binding protein due to a malfunction in the *MBL-2* gene expression,[19] as shown in Table 3.

The OR of the mutant allele was 1.18 and so it is higher than 1.0 at a likelihood level of $p=0.6828$ which is considered as a virulence factor for the development of the disease.

### *Testing the results of the genetic variation by Hardy-Weinberg Equilibrium (HWE) in* MBL-2 *gene polymorphism*

This is considered the third analysis of the studied variation to identify its effect on the disease case according to the equation used for this purpose.

$$P^2 + 2Pq + q^2 = 1$$

Where:

$P^2$ is the Predicted frequency of CC
$2pq$ is the Predicted frequency of CT
$q^2$ is Predicted frequency of TT

When comparing the results of this test as shown in Table 4, the HWE test yielded and the predicted observation rate, the p value was=0.0009; this indicates that there is a variation between the two groups of the study and these results are submitted to the equilibrium law (Khan Academy). There is also a variation in the percentages of the recorded and the expected genetic pattern (30.8 CC, 24.4 CT, 4.8 TT) and this indicates the effect of the environmental factor and the disappearance of the effect of the genetic factor on this variation in the disease case in question; this is regarded as one of the risk factors for the development of the disease.

The inheritance of this variation in the study population was verified to be recessive, which means that both alleles must be carriers of the mutation. Inheriting this variation in the study groups was ver-

**Table 2.** Distribution of the percentage of allelic observations and genotype of the *MBL-2* gene among the group of healthy people and patients with vaginal yeast infections. The C allele is a normal allele and the T allele is a mutant allele.

| Genotypes | RVVC | | Control | | p-Value | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | | | |
| CC | 36 | 60 | 12 | 60 | 1.0000 | 1.0000 | 0.3559 to 2.8095 |
| CT | 14 | 23 | 6 | 30 | 0.5520 | 0.7101 | 0.229 to 2.1942 |
| TT | 10 | 17 | 2 | 10 | 0.4745 | 1.8000 | 0.3594 to 9.0138 |

RVVC, recurrent vulvovaginal candidosis; OR, odds ratio; CC, CT, TT, genotypes considered; CI, confidence interval.

**Table 3.** Frequency of alleles in the samples.

| Alleles | RVVC | | Control | | p-Value | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | | | |
| C | 86 | 71 | 30 | 75 | 0.6828 | 1.1860 | 0.5232 to 2.6888 |
| T | 34 | 29 | 10 | 25 | | | |

RVVC, recurrent vulvovaginal candidosis; OR, odds ratio; CI, confidence interval.

**Table 4.** Results of the Hardy-Weinberg Equilibrium (HWE) test.

| Genotype | CC | CT | TT |
|---|---|---|---|
| Observed genotype | 36 | 14 | 10 |
| Expected genotype | 30.8 | 24.4 | 4.8 |
| | p-value = 0.0009 | Chi squared value $X^2$=10.86 | |

CC, CT, TT, genotypes considered.

ified to be dominant, and this means that one allele that carries the mutation is sufficient to promote the infection, as shown in Table 5.

When comparing the difference in the percentage of the genotypes for the patients and the control group individuals within the test of recessive distribution, it was observed that the value was p=0.4745, but when comparing the results within the dominance distribution, it was noticed that the value is p=1.000 and so it is higher than $p < 0.05$, *i.e.* the mutant genotype has a recessive impact on the case; it has no dominant effect and the mutant genotype TT should be considered a risk factor.

## Determining the matching of *MBL-2* nucleotides in the study samples with the gene sequencing in NCBI location

The purpose of conducting the test of nucleotides sequence is to ensure that the primers used in this study are related to the gene *MBL-2*. The test also identifies new variations that might be directly or indirectly important for the gene effectiveness; in addition, these variations might be an important reason for the development of the disease because the results of the chain amplification test of *MBL-2* showed that there are unrecorded differences in the sequence of some nucleotides (Table 6).

# Discussion

The aim of the current study was to investigate the role of *C. albicans* as the main cause of RVVC. *C. albicans* exists in three biological stages: budding yeast, mycelial hyphae and pseudohyphae.[4,21] In addition, the transformation of *C. albicans* from budding yeast to hyphae might assist the fungus to evade phagocytosis by macrophages and this leads to increasing possibility of invading the tissues of the women reproductive system and causing RVVC.[23] The results of the research are in conformity with the results of Donders *et al.*,[22] who stated that RVVC is a hard to treat chronic vaginal infection and the main cause is the aggressive strategy by *C. albicans* that subverts the host response and can get the nutrients. Therefore, it can reproduce equally in rich or poor nutrient conditions. Also, the results are in line with the study of Sarpong *et al*.,[24] who showed that *C. albicans* develop resistant biofilms against the antibiotics on the vagina epithelium and this is considered a virulence factor causing RVVC.

In addition, the results indicate that there is a relationship between *MBL-2* polymorphism and RVVC because *MBL-2* gene plays an important role in the innate immune defense against *C. albicans*.[25] These results are similar to those by Hammad *et al.*,[19] who concluded that mutations and polymorphism of *MBL-2* play a vital role in RVVC and found that there is a relationship between RVVC and *MBL-2* polymorphism exon1 codon54. The results are also in agreement with those of Nedovic *et al.*,[10] who found that there is a correlation between *MBL-2* polymorphism and RVVC in women because many common mutations in the gene lead to a case called mannose-binding lectin, and women who are infected suffer from low level of mannose-binding lectin and thus they are susceptible to RVVC. Several mutations that are related to RVVC take place in the exon1 area of the gene *MBL-2* and these mutations change the single amino acids in the mannose-binding lectin. A change in one amino acid in the mannose-binding lectin unit leads to terminate the ability of the gene to aggregate in the functional mannose-binding lectin.[25]

**Table 5.** Distribution of *MBL-2* polymorphism in the study group under recessive model and dominant model.

| Genotype | RVVC | Control | OR | CI | p value |
|---|---|---|---|---|---|
| CC + CT<br>TT | 46 (73%)<br>14 (27%) | 19 (90%)<br>1 (5%) | 0.5556 | 0.1109 to 2.7820 | 0.4745 |
| CC<br>CT + TT | 24 (42%)<br>36 (58%) | 18 (85%)<br>2 (15%) | 1.0000 | 0.3559 to 2.8095 | 0.0100 |

RVVC, recurrent vulvovaginal candidosis; OR, odds ratio; CC, CT, TT, genotypes considered; CI, confidence interval.

**Table 6.** Results of matching with the nucleotide sequence of *MBL-2* gene RefSeqGene (LRG-154) on chromosome 10 sequence ID:NG-008196.1 compared with the sequences of the original gene at the NCBI site.

| Nucleotide | Location | Mutation type |
|---|---|---|
| C → T | 5226 | Transition |
| A → | 5067 | Deletion |
| G → | 5064 | Deletion |
| A → | 5440 | Deletion |
| C → T | 5226 | Transition |
| C → T | 5362 | Transition |
| G → C | 5109 | Transversion |
| A → C | 5112 | Transversion |
| A → | 5440 | Deletion |
| G → A | 5219 | Transversion |
| A → C | 5109 | Transversion |
| G → C | 5112 | Transversion |
| A → | 5067 | Deletion |

Also, the mutations in the promoter region in the gene *MBL-2* result in decreasing the production of the mannose-binding lectin and this, in turn, leads to a decrease in functional protein quantities and in the amount of the branch units available for protein aggregation. With the decrease of the levels of mannose-binding lectin the immune system will not be able to identify *C. albicans* and to fight it efficiently, so that eventually the RVVC occurs.[26]

## Conclusions

RVVC is a common fungal infection that affects the women reproductive system. *Candida albicans* is the main causative agent of RVVC due to its virulence factors such as the hyphal form and the ability to morphologically transform from budding yeast cell to hyphae and to form biofilms. It has also been found that there are 3 genotypes: normal homozygous CC, hyterozygous CT, and mutant homozygous TT in the *MBL-2* gene, which is one of the innate immune genes against *C. albicans*. The research results also indicate a strong need for further studies on the phenotypic and genotypic variations within *C. albicans*, as well as on the genes associated with the innate immunity, to detect the emergence of more dangerous strains that cause RVVC, which occurs during the disruption of the balance between beneficial bacteria and *C. albicans*, leading to increased growth of *C. albicans*, thus causing RVVC.

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