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In silico metabolic pathways analysis of *Candida albicans* for potential drug targets and their interactions with drugs

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Abstract

*Candida albicans*is a fungus or yeast that normally grows in the mouth and digestive tract which cause Candidiasis. *C. albicans* can convert from a benign commensal into a disease-causing pathogen, causing infections in the oral, gastrointestinal and genital tracts. Recurrent vaginal thrush is caused by a deficiency in local immunity to candidiasis, which may be caused by excessive prostaglandin E2 (PGE2) synthesis. There are variances in the pathogenicity of *C. albicans* strains, suggesting that virulence factors specific to the strain may play a role in disease severity. *In silico* comparative analysis of metabolic pathways of the host *Homo sapiens* and the pathogen *Candida albicans* was performed. The blastp e-value threshold cut-off was set to 0.005. A total of, 118 enzyme sequences out of 281 enzymes, are non homologous to *Homo sapiens* protein sequences, and among them, 24 enzymes are found to be essential for the survival of the *Candida albicans* according to the DEG database. CELLO v.2.5:subCELlulor Localization predictor Results showed that about 57% of enzymes are found to be in the cytoplasm, 15% of enzymes are found to be in mitochondria, 12% enzymes are plasma membrane protein, and 6% enzymes are found to be in nuclear,5% enzymes are found to be in chloroplast and peroxisomal. The identified potential drug targets form a platform for further investigation in the discovery of novel therapeutic compounds.

Keywords: pathogen, Enzymes, non homologous and metabolic pathways

Introduction

Candida albicans is an opportunistic fungus infection that causes a significant percentage of mortality in immunocompromised people around the world, mostly patients with diabetes, cancer, AIDS, organ transplantation, and other immunosuppressive diseases[Barchiesi*e t al.*, 2016]. It is the most prevalent species implicated in both superficial and systemic infections, and it is the third most common bloodstream pathogen recovered in hospitalised patients [Talapko*et al.*, 2021]. These fungal diseases can infect and colonise humans by forming biofilms on surfaces. *Candida* biofilms can easily formed on catheters, denture acrylic strips, voice prosthesis, contact lenses, and other implantable prosthetic devices, and can have serious consequences. *C. albicans* is a major cause of hospital-acquired infections, and biofilm formation is the most common cause of antifungal resistance in patients [Junqueira*et al.*, 2012].

Fungi are eukaryotic organisms with approximately 300 000 different species. Of these, about 200 are potential parasites, with only a few of these affecting humans. Fungal diseases of mammals, mycoses, range from the common mild cutaneous or subcutaneous skin infections, such as athletes foot, to the potentially lethal acute or chronic infection of deep tissues that are typically caused by *Candida* species. Of the *Candida* species afflicting humans, *Candida albicans* is by far the most common.[Pfaller MA*et al.*,2005]*Candida albicans* belongs to the class Ascomycetes and the family, Saccharomycetaceae. This yeast can live as harmless commensal in many different body locations, and is carried in almost half of the population. However, in response to a change in the host environment, *C. albicans* can convert from a benign commensal into a disease-causing pathogen, causing infections in the oral, gastrointestinal and genital tracts. The infection caused by *C. albicans* can be defined in two broad categories, superficial mucocutaneous and systematic invasive, which involves the spread of *C. albicans* to the blood stream (candidemia) and to the major organs[Mayer, F. L*et al.*,2013]. Systemic candidemia is often fatal. Superficial infections affect the various mucous membrane surfaces of the body such as oral and vaginal thrush[Tsui C *et al.*, 2016].

The incidence of vulvovaginal candidiasis (thrush) has increased approximately 2 fold in the last decade. Approximately 75 % of all women experience a clinically significant episode of vulvovaginal candidiasis (VVC) at least once during the reproductive period. VVC is a relatively benign condition that responds well to anti-fungal treatment. It is proposed that the infection is due to minor changes in epithelial conditions, such as pH, altered glucose/glycogen concentration or changes in epithelial integrity. During pregnancy the risk of vaginal thrush increases, possibly due to changes in hormone production, leading to increased glycogen content in the vagina[Jacob L . *et al.*, 2018]

The pathogenesis of recurrent vaginal thrush involves a defect in the local immunity to candidiasis, possibly through inappropriate prostaglandin E2 (PGE2) production. The role of prostaglandins during the infection is not very clear, however, it has been demonstrated that mononuclear cells from the patients suffering from recurrent vaginal candidiasis produce higher levels of PGE2 as compared with cells from control women, indicating the important role of PGE2 during infection. [Tan TG *et al.*,2019] Recurrent vaginal candidiasis is also common in female patients with acquired immune deficiency syndrome (AIDS), suggesting a role for depressed cell-mediated immunity in candidiasis. Factors responsible for recurrent vaginal candidiasis may 2 originate from both the microorganism and the host cells, tissues and organs. Therefore, the severity of *Candida* infection often depends upon the status of the host's immune system[Rosati D . *et al.*, 2020]

However, there are differences in the pathogenicity of *C. albicans* strains which suggests that strain related virulence factors may

play a role in disease severity. Numerous virulence factors have been attributed to the pathogenicity of *C. albicans*. These include dimorphism, phenotypic switching and immune interference.

Materials and Methods

Metabolic pathway analysis(KEGG database):

The metabolic pathways for the host Homo sapiens and the pathogen *Candida albicans*were extracted using the KEGG [Kanehisa M *et al.*, 2002] pathway database. The pathogen-specific pathways were then manually compared to find those that were unique to the pathogen. The pathways were chosen based on whether they were not present in the host, but were present in the pathogen and specific to *Candida albicans*. The database yielded the identification numbers for all metabolic pathways in both organisms. The names of each unique pathogen route were then compared to the pathways of the host, H. sapiens, in a manual comparison. Pathways that were nonexistent in humans but appeared in mice, according to the KEGG database annotations. Pathways that were absent in humans but were in the pathogen were considered unique to *Candida albicans*, according to the KEGG database annotations. The enzymes implicated in these unique pathways were also extracted from the KEGG database, and sequencewas retrieved from NCBI database. in FASTA format.

Finding the non-homologous enzymes(BLASTp):

Using NCBI-BLASTP [Sudbery PE. 2011], these important genes were compared to proteins from the human RefSeq protein database for non-homology. Non-host proteins were chosen based on their E-value threshold of 0.005.

Finding the essential targets(DEG database):

Essential genes are those that are required for an organism's survival, and their functions are thus considered foundational to life. DEG (Database of Essential Genes) [Kanehisa M *et al.*, 2000] was used to assess the dataset for essentiality, with a cutoff of e-value 10^{-10} and bit score>100. The necessary proteins were checked for non-homologous to human proteins using NCBI BLASTP[Altschul*et al.*,1997], with a threshold expectation value of $>10^{-3}$ and a bit score of 100.

Identification of Drug targets(DRUGBANK):

Using the DRUGBANK database to find drug targets. Both approved and investigational medication targets are available. Each DrugCard record has about 150 data fields, with half of them devoted to drug/chemical information and the other half to drug target or protein information [Yang H *et al.*, 2016].

Finding of biological significance of the targets:

CELLO v.2.5:subCELlulor Localization predictor was used to investigate the biological importance and distribution of these critical targets. This is necessary in order to identify surface membrane proteins that could be potential vaccination targets[Yu NY *et al.*, 2010]

Docking Analysis

The most common and freely available programme, ArgusLab 4.0.1, was utilized for docking analysis. The "Argus dock" docking engine was used to geometrically optimize the inhibitor and target protein. The calculation mode was set to "dock," while the ligand was set to "flexible." 0.40 was chosen as the grid resolution. The lowest energy signified the ligand and receptor's ease of binding [Forli S *et al.*,2016].

Results and Discussion

Essential enzymes for survival of *Candida albicans,* (identified from DEG analysis)

A total of 181 distinct enzymes were discovered throughout the metabolic pathway study. DEG analysis revealed that 113 of the 181 unique enzymes are non homologous enzymes, and 24 of the 113 enzymes are essential enzymes were shown in Table 1.

Table 1: List of metabolic pathways unique to *candida albicans*

S.NO	METABOLIC PATHWAY	TARGET ENZYMES
1	Starch and Sucrose metabolism	trehalose 6-phosphate synthase/phosphatase [EC:2.4.1.15,3.1.3.12]
2	Sulfer metabolism	phosphoadenosinephosphosulfatereductase [EC:1.8.4.8]
3	Sulfer metabolism	homoserine O-acetyltransferase [EC:2.3.1.31]
4	Fatty acid biosynthesis	fatty acid synthase subunit alpha, fungi type FAS2[EC:2.3.1.86]
5	Purine metabolism	urate oxidase [EC:1.7.3.3]
6	Glycine,Serine,Threonine metabolism and Phenylalanine,Tyrosine and Tryptophan biosynthesis	tryptophan synthase [EC:4.2.1.20]
7	Phenylalanine,Tyrosine and Tryptophan biosynthesis	chorismate synthase [EC:4.2.3.5]
8	Cysteine and Methionine metabolism	homoserine O-acetyltransferase [EC:2.3.1.31]
9	Valine, Leucine and Isoleucine biosynthesis	ketol-acid reductoisomerase [EC:1.1.1.86]
10	Valine, Leucine and Isoleucine biosynthesis	dihydroxy-acid dehydratase [EC:4.2.1.9]
11	Histidine metabolism	imidazoleglycerol-phosphate dehydratase [EC:4.2.1.19]
12	Riboflavin metabolism	3,4-dihydroxy 2-butanone 4-phosphate synthase [EC:4.1.99.12]
13	Riboflavin metabolism	2,5-diamino-6-(ribosylamino)-4(3H)-pyrimidinone 5'-phosphate reductase [EC:1.1.1.302]
14	Riboflavin metabolism	riboflavin synthase [EC:2.5.1.9]
15	Vitamin B6 metabolism	glutamineamidotransferaseYaaE [EC:2.6.-.-]
16	Pantothenate and CoA biosynthesis	ketol-acid reductoisomerase [EC:1.1.1.86]
17	Pantothenate and CoA biosynthesis	dihydroxy-acid dehydratase [EC:4.2.1.9]
18	Biotin metabolism	biotin synthase [EC:2.8.1.6]
19	Folate biosynthesis	dihydroneopterinaldolase / 2-amino-4-hydroxy-6-hydroxymethyldihydropteridine diphosphokinase / dihydropteroate synthase [EC:4.1.2.252.7.6.32.5.1.15]
20	Methane metabolism	fructose-bisphosphate aldolase, class II [EC:4.1.2.13]
21	MPAK signaling pathway -yeast	transcription factor STE12
22	MPAK signaling pathway -yeast	osomolarity two-component system, phosphorelay intermediate protein YPD1
23	MPAK signaling pathway -yeast	osomolarity two-component system, sensor histidine kinase SLN1 [EC:2.7.13.3]
24	MPAK signaling pathway -yeast	osomolarity two-component system, response regulator SSK1

Approved drug target identified by the drug bank server

The approved targets from the drug bank were shown in the Table 2

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Table 2: Approved drug targets from DRUG BANK

S.NO	ENZYME NAME	DRUG BANK TARGET	NO.OF DRUGS	BIT SCORE
1	fatty acid synthase subunit alpha, fungi type FAS2	probable fatty acid synthase fas (fatty acid synthetase) 3-oxoacyl-[acyl-carrier-protein] synthase 1 3-oxoacyl-[acyl-carrier-protein] synthase 2	1 1 1	239.965 53.1434 46.595
2	riboflavin synthase	Riboflavin synthase alpha chain	1	122.865
3	dihydroneopterinaldolase / 2-amino-4-hydroxy-6-hydroxymethyldihydropteridine diphosphokinase / dihydropteroate synthase	Dihydropteroate synthase Dihydropteroate synthase 1 Dihydropteroate synthase 2 Dihydropteroate synthetase Dihydropterate synthase	9 1 1 6 1	160.614 137.502 95.5153 93.9745 68.9366

Approved drugs from drug bank:

There are 20 drugs were obtained from the drug bank server for *candida albicans* were shown in the Table 3.

Table 3:List of approved drugs from drug bank

S.NO	TARGET ENZYME	DRUG NAME	DRUG BANK ID	DRUG TYPE	DRUG GROUPS
1	**probable fatty acid synthase fas (fatty acid synthetase)**	Pyrazinamide	DB00339	Small molecule	Approved
2	**3-oxoacyl-[acyl-carrier-protein] synthase 1&2**	Cerulenin	DB01034	Small molecule	Approved
3	**Riboflavin synthase alpha chain**	Riboflavin	DB00140	Small molecule	Approved

4	**Dihydropteroate synthase**	Sulfacetamide Sulfacytine Sulfamerazine Sulfamethazine Sulfamethizole Sulfamethoxazole Sulfanilamide Sulfaphenazole Sulfisoxazole	DB00634 DB01298 DB01581 DB01582 DB00576 DB01015 DB00259 DB06729 DB00263	Small molecule Small molecule Small molecule Small molecule Small molecule Small molecule Small molecule Small molecule Small molecule	Approved Approved Approved Approved Approved Approved Approved Approved Approved
5	**Dihydropteroate synthase 1&2**	Dapsone	DB00250	Small molecule	Approved, investigational
6	**Dihydropteroate synthetase**	Sulfadiazine Sulfadoxine Sulfametopyrazine Sulfamoxole Sulfathiazole Sulfoxone	DB00359 DB01299 DB00664 DB08798 DB06147 DB01145	Small molecule Small molecule Small molecule Small molecule Small molecule Small molecule	Approved Approved Approved, withdrawn Approved Approved Approved
7	**Dihydropterate synthase**	Sulfapyridine	DB00891	Small molecule	Approved

Experimental drug target evaluated by the drug bank server:

The experimental targets obtained from the drug bank server were shown in the Table 4.

Table 4: List of Experimental drug targets

S.NO	ENZYME NAME	DRUG BANK TARGET	NO.OF TARGETS	BIT SCORE
1	urate oxidase	**Uricase**	1	71.633
2	tryptophan synthase	**Tryptophan synthase beta chain** **Tryptophan synthase alpha chain**	12 2	426.402 154.451
3	fatty acid synthase subunit alpha, fungi type FAS2	**Transferase** **3-oxoacyl-[acyl-carrier-protein] synthase 1** **3-oxoacyl-[acyl-carrier-protein] synthase 2**	1 6 2	66.2402 53.1434 46.595
4	chorismate synthase	**Chorismate synthase** **Chorismate synthase**	1 2	178.333 95.5153
5	biotin synthase	**Biotin synthase**	2	300.827

6	dihydroneopterinaldolase / 2-amino-4-hydroxy-6-hydroxymethyldihydropteridine diphosphokinase / dihydropteroate synthase	**Dihydropteroate synthase** **Dihydropteroate synthase 1** **2-amino-4-hydroxy-6-hydroxymethyldihydropteridine pyrophosphokinase** **2-amino-4-hydroxy-6-hydroxymethyldihydropteridine pyrophosphokinase**	4 1 1 7	179.104 129.028 65.4698 65.4698
7	trehalose 6-phosphate synthase/phosphatase	**Alpha,alpha-trehalose-phosphate synthase [UDP-forming]**	4	196.052
8	fructose-bisphosphate aldolase, class II	**Fructose-bisphosphate aldolase class 2** **Tagatose-1,6-bisphosphate aldolaseagaY**	1 1	373.629 71.2478
9	osomolarity two-component system, sensor histidine kinase SLN1	**Adenylate cyclase** **PPH** **Osmolarity sensor protein envZ** **Chemotaxis protein cheY**	3 1 1 3	54.299 61.2326 54.6842 59.6918

Sub cellular localization prediction:

Sub cellular localization of proteins could be used to obtain information about their potential functions. Sub cellular localization of the drug targets were carried, and the results obtained were further validated with CELLO v2.5 . The enzymes are present in following localizations were shown in the Table 5.

S.No	Sequence id	Protein names	Subcellular localization
1	CaO19.2114 URO99	Urate Oxidase; K00365 urate oxidase	Peroxisomal 1.453 * Nuclear 1.284 *
2	CaO19.4718 TRP5	tryptophan synthetase alpha chain similar to *S. cerevisiae* TRP5 (YGL026C)	Cytoplasmic 3.044 *
3	CaO19.13370	fatty acid synthase subunit alpha, fungi type	Cytoplasmic 2.741 *
4	CaO19.1986 ARO2	Chorismate synthase	Chloroplast 1.363 * Nuclear 1.282 * Mitochondrial 1.074 * Cytoplasmic 1.047 *

5	CaO19.11507 RIB5	riboflavin synthase	PlasmaMembrane 1.585 * Cytoplasmic 1.134 *
6	CaO19.579 FOL1	dihydropteroate synthase	Nuclear 1.860 * Cytoplasmic 1.658 *
7	CaO19.10556	K16055 trehalose 6-phosphate	Cytoplasmic 2.581 *

Docking results for drug targets:

The Docking interactions were obtained for the 7 enzymes and their compounds and their binding energy were calculated and their interactions were shown in Table 6.The best interaction were shown in the Figure 1.

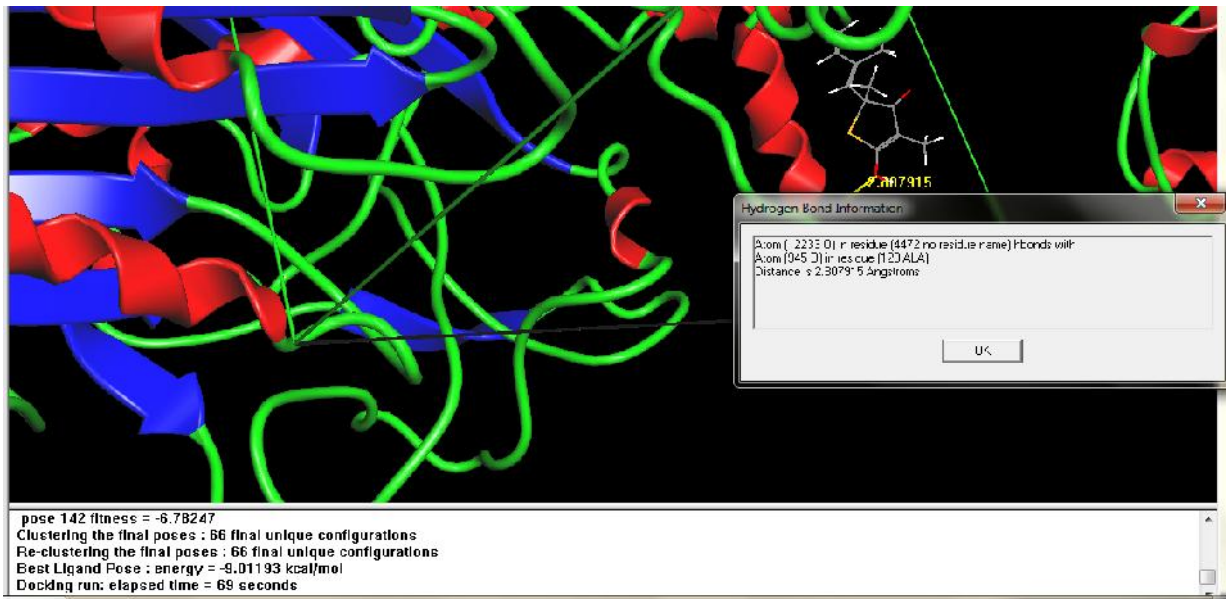
Table 6: Molecular Docking interactions between the enzymes and drugs

S.NO	ENZYME	DRUG NAME	PUBCHEM ID	ENERGY VALUE (KCAL/MOL)	BINDING INFORMATION	
					BOND TYPE (D-H…A)	DISTANCE (Å)
1	Tryptophan synthase alpha chain	Citric acid	311	-6.90348	172TYR (O……N)	2.581561
					172TYR (O…….O)	2.259145
		Indole-3-propanol phosphate	3713	-7.73709	172TYR (O……O)	2.259279
					172TYR (O…....O)	2.581963
					230SER (O…….O)	2.446065
					230SER (O…….O)	2.580954
					230SER (N…….O)	2.739449
					99TYR (O…….O)	2.588875
					99TYR (O…….O)	2.899344
					59GLY (N…….O)	2.483661

2	chorismate synthase	Riboflavin Monophosphate	5702760	-8.03633	17HIS (O…….O)	2.647971
3	Biotin synthase	D-Dethiobiotin	445027	-8.06821	222ASN (N…….O) 293THR (N…….O)	2.999379 2.775232
		Tris(Hydroxymethyl)Aminomethane	3777159	-7.43852	153ASN (N……..O) 153ASN (O……..O) 153ASN (O……..O) 173ARG (N……...O) 168ARG (N……O)	2.931985 2.550994 2.490197 2.929370 2.665171
4	Chemotaxis protein cheY	3-Aminosuccinimide	32017976	-5.10568	104SER (N……O)	2.701874
		S-Methyl Phosphocysteine	192579	-6.62707	126LYS (N…….O) 99ALA (O…….O) 119LYS (N……O) 105GLY (N……O)	2.687374 2.621809 2.999901 2.765227

5	3-oxoacyl-[acyl-carrier-protein] synthase 1	(5R)-4-HYDROXY-3,5-DIMETHYL-5-[(1E,3E)-2-METHYLPENTA-1,3-DIENYL]THIOPHEN-2(5H)-ONE	5494446	-8.72612	128ALA (O…….O)	2.712795
		2-PHENYLAMINO-4-METHYL-5-ACETYL THIAZOLE	735838	-7.90335	38GLU (N……O) 37GLN (N…….O)	2.998980 2.752553
		4-Hydroxy-3,5-Dimethyl-5-(2-Methyl-Buta-1,3-Dienyl)-5h-Thiophen-2-One	445629	-9.01193	128ALA (O……O)	2.807915
6	Adenylate cyclase	Adenosine-5'-Rp-Alpha-Thio-Triphosphate	46936362	-4.67818	1150ARG (N……O)	2.998334
		Alpha,Beta-Methyleneadenosine-5'-Triphosphate	6323221	-8.0521	1150ARG (N……O) 1061ASP (O…….O) 1017ASP (O……..O)	2.375475 2.442045 2.672896
7	3-oxoacyl-[acyl-carrier-protein] synthase 2	3-({3-[(1S,4aS,6S,7S,9S,9aR)-1,6-dimethyl-2-oxodecahydro-6,9-epoxy-4a,7-methanobenzo[7]annulen-1-yl]propanoyl}amino)-2,4-dihydroxybenzoic acid	16086836	-8.0521	205ALA (O……O)	2.786742
		PLATENSIMYCIN	6857724	-7.66937	205ALA (O…….O)	2.760732

Figure1: 4-Hydroxy-3,5-Dimethyl-5-(2-Methyl-Buta-1,3-Dienyl)-5h-Thiophen-2-One drug target shows best (least) binding energy. It shows -9. 01193 Kcal/mol binding energy.



Conclusion

These identified putative targets may be exploiting further for developing drugs against *Candida albicans.* It is quite obvious that increase of drug resistance properties requires more potential targets and by this*In silico* approaches reduces the effort of wet lab and also increases the probability of success. By this present study we have tried to evaluate the targets could be better target for rational drug designing.The drug targets from the unique pathways would also be extended as common targets for designing inhibitors against fungal diseases. This approach enables rapid potential drug target identification and thereby greatly facilitating the search for new drugs.

Conflict of interest:

The authors declare they have no competing interests.

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