UNIVERSITI PUTRA MALAYSIA

PHENOTYPIC AND MOLECULAR GENETIC PROFILES OF FLUCONAZOLE AND VORICONAZOLE SENSITIVE VERSUS RESISTANT *CANDIDA* SPP

PRIYA MADHAVAN

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By

PRIYA MADHAVAN

Thesis Submitted to the School of Graduate Studies, Universiti Putra Malaysia, in Fulfilment of the Requirements for the Degree of Doctor of Philosophy

June 2014

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Abstract of thesis presented to the Senate of Universiti Putra Malaysia in fulfillment of the requirement for the degree of Doctor of Philosophy.

PHENOTYPIC AND MOLECULAR GENETIC PROFILES OF FLUCONAZOLE AND VORICONAZOLE SENSITIVE VERSUS RESISTANT *CANDIDA* SPP

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June 2014

Chairman: Professor Farida Jamal, MRCPath.
Faculty: Medicine and Health Sciences

Frequent use of azole group of antifungal drugs for prophylaxis and treatment of *Candida* infections has contributed to the emergence of resistant strains, especially among non-albicans *Candida* species. The present study was done to identify genetic variations and changes in cellular morphology among non-albicans *Candida* isolates resistant to fluconazole and voriconazole. *Candida* isolates obtained from two local hospitals were identified using CHROMagar Candida™ and commercial biochemical test kits. Among these 41 isolates, the most predominant species was *C. tropicalis* (n=10), followed by *C. albicans* (n=7), *C. parapsilosis* (n=6), *C. krusei* (n=6), *C. rugosa* (n=6), *C. dubliniensis* (n=3) and *C. glabrata* (n=3). Resistance breakpoints of fluconazole and voriconazole were determined for these 41 *Candida* isolates using the E-test method. *C. glabrata* and *C. parapsilosis* strains that were susceptible and resistant towards the two azoles were selected for further studies as they were commonly isolated pathogens in patients with candidiasis in various parts of the world. A less commonly studied species, *C. rugosa* was also selected. The variations of genes in the resistant and susceptible strains of *Candida* species were investigated using Random Amplification of Polymorphic DNA-PCR (RAPD-PCR). The isolates were genotyped and grouped into 3 major groups according to their species using composite DNA type (based on three primers) comprising *C. glabrata*, *C. parapsilosis* and C. *rugosa*. Although some of the strains within the same group were highly similar, they were not clones, as indicated by variations in their genotypic profiles. The morphological differences between the drug-resistant and drug-susceptible strains treated with fluconazole and voriconazole were observed with scanning and transmission electron microscopy. A scoring system developed in this study revealed pronounced damage on the cell membrane for cells treated with 10X MIC of fluconazole and MIC of voriconazole. Biofilm formation was studied in these three species, followed by the effect of fluconazole and voriconazole on the pre-formed biofilms using the XTT metabolic assay. The biofilm cells exhibited between 2 and 64 folds higher MIC_{50} and MIC_{80} for both the azoles compared to the planktonic cells. Coating the wells with the azole drugs reduced the MIC of the

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biofilms for all clinical strains. Expression of candidate genes was compared between the drug-resistant and drug-susceptible strains using semi-quantitative reverse transcription-PCR method in *C. glabrata*. Candidate genes selected were based on their involvement in ergosterol biosynthesis (*ERG11*), efflux of drugs (*CDR1*) and biofilm formation (*EPA1*, *EPA6* and *EPA7*). The expression level of the selected genes of the *Candida* isolates was normalized to beta actin gene of *Candida* and was reported as a ratio. Upregulations were observed in all genes except for *EPA7* gene in the resistant strain compared to the ATCC strain. In the susceptible strain, upregulations were observed only in *EPA7* and *CDR1* genes treated with fluconazole, and in all except *EPA7* gene in the voriconazole treated cells. The results obtained in this research contribute to the knowledge on the morphological and genetic characteristics of clinical strains of *C. glabrata*, *C. parapsilosis* and *C. rugosa* sensitive and resistant to fluconazole and voriconazole.

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PROFIL GENETIK, FENOTIP DAN MOLEKUL KERENTANAN SPESIS *KANDIDA* TERHADAP FLUKONAZOL DAN VORIKONAZOL

Oleh

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Pengerusi: Profesor Farida Jamal, MRCPath.
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Kekerapan penggunaan antifungus dari kumpulan azola sebagai profilaksis dan rawatan jangkitan *Candida* telah menyumbang kepada kemunculan kerentanannya, terutamanya spesis *Candida* selain *C. albicans*. Penyelidikan ini dilakukan untuk mengenalpasti variasi genetik dan morfologi sel di kalangan *Candida* bukan-*albicans* yang rentan dan resistan terhadap flukonazol dan vorikonazol. Pemencilan *Candida* adalah dari dua buah hospital tempatan. Spesis *Candida* dikenalpasti dengan menggunakan CHROMagar Candida™ dan kit ujian biokimia komersial. Antara 41 pemencilan, spesis yang paling banyak adalah *C. tropicalis* (n=10), diikuti dengan *C. albicans* (n=7), *C. parapsilosis* (n=6), *C. krusei* (n=6), *C. rugosa* (n=6), *C. dubliniensis* (n=3) dan *C. glabrata* (n=3). Tahap kerentanan terhadap flukonazol dan vorikonazol ditentukan bagi 41 *Candida* dengan menggunakan jalur ujian-E. *C. glabrata dan C. parapsilosis* yang rentan dan resistan terhadap kedua-dua antifungus telah dipilih bagi kajian seterusnya kerana kedua-dua spesis ini didapati dipemencilkan daripada pesakit dengan kandidiasis dari pelbagai bahagian dunia. Satu spesis yang jarang dikaji, *C. rugosa* juga dipilih kerana bilangannya yang tinggi dipemencilkan dalam kajian ini. Variasi gen di kalangan spesis *Candida* telah disiasat menggunakan RAPD-PCR. Semua strain yang dikaji dikumpulkan ke dalam 3 kumpulan utama mengikut spesis menggunakan DNA komposit (berdasarkan tiga primer), iaitu *C. glabrata*, *C. parapsilosis* dan *C. rugosa*. Walaupun beberapa strain dalam kumpulan yang sama adalah serupa, tetapi mereka bukannya klon. Ini menunjukkan adanya variasi dalam genotip mereka. Perbezaan morfologi antara strain rentan dan resistan terhadap flukonazol dan vorikonazol diamati dengan pengimbasan mikroskop elektron. Kerosakan pada membrane sel didapati lebih signifikan untuk sel-sel yang dirawat dengan 10X MIC flukonazol dan MIC vorikonazol dengan menggunakan system penskoran morfologi yang direka dalam penyelidikan ini. Pembentukan biofilm dikaji di antara ketiga-tiga spesies, diikuti dengan kesan flukonazol dan vorikonazol terhadap pembentukan biofilm menggunakan assay metabolic XTT. Terdapat antara 2 dan 64 kali ganda MIC_{50} dan MIC_{80} bagi biofilm berbanding dengan sel-sel biasa. Pelapisan plat 96-well dengan

kedua-dua antifungus telah mengurangkan MIC biofilm untuk semua strain klinikal. Ekspresi gen calon antara strain rentan dan resistan telah dikaji dengan menggunakan kaedah semi kuantitatif transkripsi berbalik-PCR untuk strain-strain *C. glabrata*. Gen calon terpilih adalah berdasarkan penglibatan mereka dalam sintesis ergosterol (*ERG11*), efluks antifungus (*CDR1*) dan pembentukan biofilm (*EPA1*, *EPA6* dan *EPA7*). Tahap ekspresi gen dipilih daripada strain *C. glabrata* telah dibandingkan dengan gen beta actinnya yang dilaporkan sebagai nisbah. Ekspresi berlebihan diperhatikan dalam semua gen kecuali gen *EPA7* dalam strain resistan-flukonazol. Dalam strain rentan-flukonazol, ekspresi berlebihan diperhatikan hanya dalam gen-gen *EPA7* dan *CDR1* selepas dirawat dengan flukonazol, dan dalam semua gen kecuali gen *EPA7* selepas dirawat dengan voriconazol. Keputusan yang diperolehi dalam kajian ini menyumbang kepada pengetahuan tentang ciri-ciri *C. glabrata*, *C. parapsilosis* dan *C. rugosa* rentan dan resistan terhadap flukonazol dan vorikonazol.

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APPROVAL

I certify that a Thesis Examination Committee has met on 18 June 2014 to conduct the final examination of Priya a/p Madhavan on her thesis entitled "Phenotypic and Molecular Genetic Profiles of Fluconazole and Voriconazole Sensitive Versus Resistant *Candida* spp." in accordance with the Universities and University Colleges Act 1971 and the Constitution of the Universiti Putra Malaysia [P.U.(A) 106] 15 March 1998. The Committee recommends that the student be awarded the Doctor of Philosophy.

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This thesis was submitted to the senate of Universiti Putra Malaysia and has been accepted as fulfilment of the requirement for the degree of Doctor of Philosophy.

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LIST OF ABBREVIATIONS

ABC	ATP-binding cassette
ALS	Agglutinin-like sequence
ANOVA	One way analysis of variance
ATCC	American type culture collection
ATP	Adenosine triphosphate
BEC	Buccal epithelial cells
$BLAST_n$	Basic local alignment search tool for nucleotide
$BLAST_x$	Basic local alignment search tool for translated nucleotide
BNA	Biosynthesis of nicotinic acid
cDNA	Complementary deoxyribonucleic acid
CDR	*Candida* drug resistance
CFU	Colony forming unit
CGD	Candida Genome Database
CLSI	Clinical Laboratory Standard Institute
CYP	Cytochrome P450
DEPC	Diethylpyrocarbonate
DMSO	Dimethyl sulfoxide
DNA	Deoxyribonucleic acid
EBI	European bioinformatics institute
EFG	Enhanced filamentous growth
EPA	Epithelial adhesin
EPS	Extracellular polymeric substances
ERG	Ergosterol
GIMC	Gleaneagles Intan Medical Centre
GPI	Glycosylphosphatidylinositol
Hep	Hepatocellular carcinoma
HUVEC	Human umbilical vein endothelial cells
HVS	High vaginal swab
HWP	Hyphal wall protein
ITS	Internal transcribed spacer
MDR	Multi drug resistant
MFS	Major Facilitator Superfamily
MIC	Minimal inhibitory concentration
MIMS	Monthly index of medical specialities
MLST	Multi locus sequence typing
MLT	Mating type locus
M-MuLV	Moloney murine leukemia virus
MOPS	Morpholinophosphonylsulfate
mRNA	Messenger ribonucleic acid
NA	Nicotinic acid
NaMN	Nicotinic acid mononucleotide
NCBI	National Center for Biotechnology Information
NRE	Negative regulatory element

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OPA	Operon set A
PBS	Phosphate buffer saline
PDR	Pleiotropic drug resistance
PFGE	Pulsed field gel electrophoresis
PLB	Phospholipases
RAPD-PCR	Random amplified polymorphic DNA-polymerase chain reaction
RNA	Ribonucleic acid
RPMI	Royal Park Memorial Institute
SAP	Secreted aspartyl proteases
SDA	Sabouraud dextrose agar
SDB	Sabouraud dextrose broth
SDD	Susceptible dose dependent
SEM	Scanning electron microscopy
SIR	Silent information regulator
SNQ	Sensitivity to 4-NitroQuinoline-N-oxide
sqRT-PCR	Semi quantitative reverse transcription-polymerase chain reaction
TBE	Tris borate EDTA buffer
TEM	Transmission electron microscopy
TPN	Total parenteral nutrition
Tup	Transcriptional repressor protein
UMMC	University Malaya Medical Centre
UPGMA	Unweighted pair-group method using arithmetic averages
WHI	White phase specific gene
XTT	2,3-bis-(2-methoxy-4-nitro-5-sulfophenyl)-2H-tetrazolium-5-carboxanilide
YDR	Yeast dubious open reading frame

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CHAPTER 1

INTRODUCTION

During the past few decades, increase in the incidence of opportunistic fungal infections has been reported worldwide. Many of these infections are caused by commensal fungi. The diagnosis of such infections is difficult due to their unequivocal signs and symptoms, leading to misdiagnosis. Early diagnosis and identification of the causative agent is required for appropriate treatment, preventing recurrences and reducing the rate of antifungal resistance. Most opportunistic fungal infectionsare caused by the genus *Candida*, which includes superficial infections e.g. vaginal and oral infections, and systemic infections of the bloodstream and internal organs. Predisposing factors of candidiasis include impaired cellular immunity, prolonged antibiotic therapy, metabolic and hormonal disorders, glucocorticosteroids therapy and presence of intravascular devices (Ichhpujani and Bhatia, 2007; Rodrigues and Wolff, 1974). *C. albicans* is the most common species involved in these infections. It can be differentiated from other species of *Candida* by sugar fermentation and sugar assimilation tests, formation of germ tube in serum and growth on corn-meal agar culture which produces large, spherical chlamydospores (Arora, 2004).

Apart from *C. albicans*, other species that are reported to cause infections in humans include *C. tropicalis, C. parapsilosis, C. krusei, C. guilliermondii, C. glabrata, C. kefyr, C. lusitaniae, C. rugosa, C. viswanathii* and *C. stellatoidea* (Basetti *et al.*, 2007 and Arora, 2004). Currently, some species are increasingly being reported as causative agents of invasive candidiasis especially among the immunocompromised patients. In a study conducted at a teaching hospital in Malaysia, *Candida albicans* was reported as the main causative agent of vaginal candidiasis, followed by *C. glabrata, C. lusitaniae, C. famata, C. krusei* and *C. parapsilosis*. Re-infections in patients were not only caused by identical strains but different strains of species of *Candida* (Chong *et al*., 2003). Increase in the number of disseminated candidiasis among acute leukaemia patients following chemotherapy was reported by Cantu (2005). Oral colonisation by *Candida* species in 90% cases of acute leukaemia patients could be a precipitating factor (Rodu *et al*., 2006). Treatment with ketoconazole for five days among these patients resulted in complete eradication only in 9 out of 20 patients. Related studies have documented an increase in disseminated *Candida krusei* infection among bone marrow transplant and neutropenic patients (Wingard *et al*., 1991). There are also reports of candidaemia among burn patients in France, mainly caused by *C. albicans*, *C. parapsilosis* and *C. tropicalis* (Ha *et al*., 2011).

The choice of antifungal therapy depends on the physician's knowledge, availability of antifungal agent, severity of the illness, concomitant medications and cost (Gallagher *et al*., 2005). Antifungal agents, mainly those comprising the azole

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groups have been recommended for the treatment of initial and subsequent *Candida* infections. Prophylactic and empirical administration of antifungal drugs were adopted by physicians to avoid recurrences (de Pauw, 2011). Failure of antifungal therapy in patients is due to several reasons, including the genetic resistance to azole drugs among yeasts and acquired resistance following the overuse of the azole drugs. In the past, systemic *Candida* infections were mostly caused by *C. albicans* and treated with amphotericin B. Currently, azole group is recommended as it is less toxic (Wilson and Gisvold, 1998). However, fluconazole was found to be ineffective over time with many *Candida krusei* infections, a species known to be resistant to many azole drugs. In some cases, a combination of the azole drugs was used for better efficacy (Buchner *et al*., 2002). Comparative studies between the use of amphotericin B and caspofungin, an echinocandin agent was done by a group of researchers from Central America (Mora-Duarte *et al*., 2002). These antifungal agents were used in patients with invasive candidiasis. Caspofungin was found to be as effective or better in some cases compared to amphotericin B. Treatment with caspofungin was also reported to be successful in an acute lymphoblastic leukaemia patient with *Candida krusei* fungemia (McGee and Tereso, 2003). The incidence of candidemia in one year was 13% among hospitalised patients in two teaching hospitals in Italy (Basetti *et al*., 2007). Due to the causative strain's resistance towards amphotericin B, some of these patients were given fluconazole, caspofungin or voriconazole for re-infections. Extensive use of antifungal drugs such as fluconazole has contributed to the resistance of *Candida* spp towards it. Resistance towards newer drugs like voriconazole was also found in some cases (Perfect *et. al*., 2003).

The increasing resistance in *Candida* species towards azoles involves multiple mechanisms. Studies have shown that the main mechanism of resistance is a mutation of a previously susceptible strain, leading to alteration of the drug target enzyme and over expression of a gene responsible for ergosterol synthesis, an important component of the fungal cell wall (White *et. al*., 1998). This leads to change in the cell's characteristics, including its morphology. This also influences the ability of the cells to form biofilms, which play an important role in pathogenesis of *Candida* infections. The problem identified from past literature is that *Candida albicans* has been the focus of basic research for several decades. However, less information is available on antifungal resistance among newly emerging species.

Therefore, the present research was aimed at addressing these issues. Genetic mutations and the resultant changes in cellular morphology among *Candida* species resistant to fluconazole and voriconazole were studied. The overall experimental design that addresses the stated objectives below is shown in Figure 1.1.

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The specific objectives were:

1. To determine the resistance breakpoints of fluconazole and voriconazole among clinical isolates of *Candida* species.

2. To identify genetic variations in the azole resistant and susceptible strains of *Candida* species.

3. To observe morphological differences between the azole resistant and susceptible strains of *Candida* species.

4. To investigate the effect of fluconazole and voriconazole on the biofilm formation by *Candida* species.

5. To evaluate the expression of candidate genes between the azole resistant and susceptible strains of *Candida* species.

The results obtained in this research would contribute to the knowledge on the characteristics of *C. glabrata*, *C. parapsilosis* and *C. rugosa* strains that are resistant and sensitive to fluconazole and voriconazole, respectively. The findings of this study could also be used to develop diagnostic tools based on the novel genes identified for drug resistant strains.

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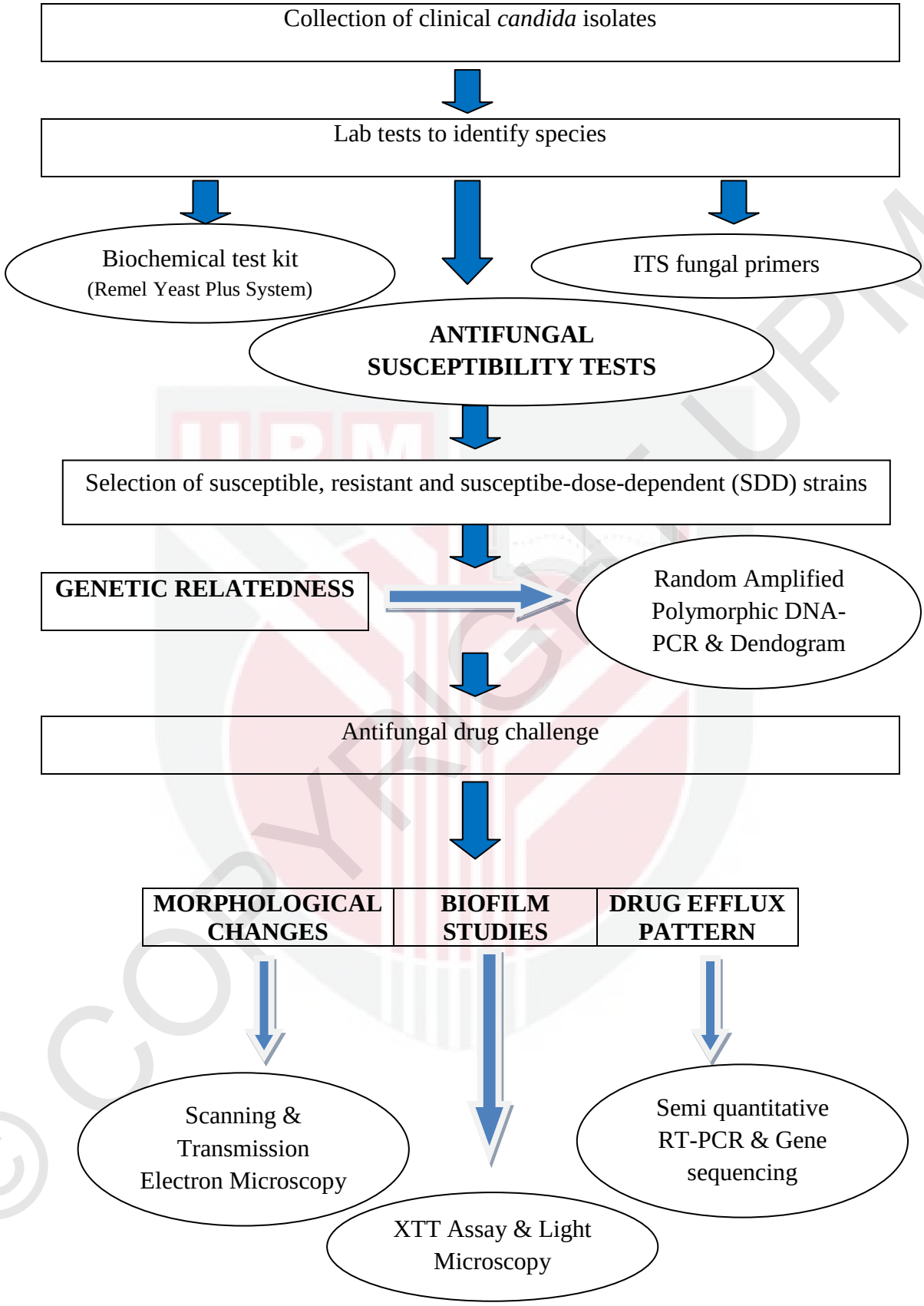

Figure 1.1 Experimental design

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