Investigating Host and Environmental Influences of *Fusarium solani* Using a Novel Monoclonal Antibody

Submitted by

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I certify that all the material in this thesis which is not my own work has been identified and that no material has been previously submitted and approved for the award of a degree by this or any other University.

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May 2016

Abstract

Human fungal infections among severely immunocompromised individuals have increased dramatically over the last 30 years and that coincidence with an expanding patient numbers of bone marrow and solid organ transplantation and those receiving aggressive cytotoxic chemotherapy for neoplastic diseases or immunosuppressive drugs. In recent years, many of opportunistic fungi have emerged as serious human pathogens and causing life-threatening infections of humans such as *Fusarium* species. Due to lack of a highly accurate diagnostic test for tracking the pathogenic *Fusarium* species, fusariosis is frequently misdiagnosed as aspergillosis. Delays in identification and differentiation of *Fusarium* spp. from other causative agents of hyalohyphomycetes associated with high morbidity and mortality rate among immunocompromised patients. This research aimed to develop a highly specific monoclonal antibody (mAb) using hybridoma technology to produce a highly genus-specific murine mAb ED7 that could be used for tracking and early detecting circulating *Fusarium* species antigens from other opportunistic pathogens. At present, a very little is known about the pathogenicity and interaction of human pathogenic *F. solani* and cells of the innate immune system like alveolar macrophages (AMØ), the residential innate immune cells of alveoli. For this reason, *F. solani* was genetically transformed with GFP gene and a model of immunoassay was developed to investigate the interactions of *F*. *solani* with AMØ that would allow studying the fungal pathogenicity, visualising and quantification of the pathogen during the process of macrophage phagocytosis. In addition, this model can be used to evaluate the effect of a mAb on fungal uptake by AMØ. Habitates providing direct human

exposure to infectious propargules are largely unknown, but there is growing evidence that plumbing systems are sources of human pathogenic strains in the *F. solani* and *F. oxysporum* species complexes, the most common groups infecting humans. Using mAb ED7 specific to the *Fusarium* species, this work demonstrates how the mAb can be used as a powerful immunodiagnostic tool for accurately tracking the *Fusarium* species antigens in sink drain biofilms and water system samples containing mixed populations of human opportunistic filamentous and yeasts pathogenic fungi across a University campus and a tertiary care hospital. Specificity of the ELISA was confirmed by sequencing of the internally transcribed spacer 1 (ITS1)-5.8S-ITS2 rRNA-encoding regions of culturable yeasts and moulds that were recovered using mycological culture, while translation elongation factor (TEF)-1α analysis of *Fusarium* isolates included FSSC 1-a, FOSC 33 and FDSC ET-gr, the most common clinical pathotypes in each group. The mAb ED7 is, therefore, suitable to be carried forward for use in diagnostic assays, such as the lateral flow device.

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Abbreviations

mAb	Monoclonal Antibody
ITS	Internal Transcribed Spacer
rRNA	ribosomal Ribonucleic Acid
rDNA	ribosomal Deoxyribonucleic Acid
GM	Galactomannan
AIDS	Acquired Immune Deficiency Syndrome
PAMPs	Pathogen Associated Molecular Patterns
PRRs	Pattern Recognition Receptors
M∅	Macrophage
TLRs	Toll-like receptors
ELISA	Enzyme-Linked Immunosorbent Assay
PCR	Polymerase Chain Reaction
TEF-1α	Translation Elongation Factor-1α
BDG	1,3-β-D-Glucan
PDA	Potato Dextrose Agar
PDB	Potato Dextrose Broth
SDB	Sabouraud Dextrose Broth
MQ-H_2O	Milli-Q H_2O
SMQ-H_2O	Sterile Milli-Q- H_2O
PBS	Phosphate Buffered Saline
BSA	Bovine Serum Albumin
TCS	Tissue Culture Supernatant
TMB	TetraMethylBenzidine
TCM	Tissue Culture Medium
FBS	Fetal Bovine Serum
PBSA	Phosphate Buffered Saline containing Bovine Serum Albumin
AMØ	Alveolar Macrophage

TNF	Tumour Necrosis Factor
IL	Interleukins
MET	Macrophage Extracellular Traps
GFP	Green Fluorescent Protein
FS-GFP	*Fusarium solani*-Green Fluorescent Protein
ΔFBS	Heat-inactivated Fetal Bovine Serum
P%	Phagocytosis Percentage
Pi	Phagocytosis Index
IFI	Invasive Fungal Infections

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Chapter 1. General Introduction

1.0 Overview

The human disease invasive fusariosis, caused by species in the fungal genus *Fusarium*, has emerged as a serious life-threatening disease with high rates of mortality and morbidity in immunocompromised patients. Specific and sensitive diagnostic tests for this infectious fungal pathogen are currently lacking, and early detection of *Fusarium* infections is vital for the prompt and appropriate management of disease (Jain *et al*., 2011; Thornton, 2011; van Diepeningen *et al.*, 2015). Very little is known about the interaction of emerging human pathogenic fungi such as *Fusarium* and the immune system (Nucci & Anaissie, 2007). Furthermore, the response of *Fusarium* to antifungal drugs is poorly defined (Al-Hatmi *et al.*, 2015; Azor *et al.,* 2009). Arguably, one of the main reasons for this lack of knowledge is the insufficiency of techniques that allow accurate detection of the fungus *in vivo* and for tracking the fungus in epidemiological and immune cell interaction studies (Muhammed *et al.,* 2011).

This chapter will introduce the Kingdom Fungi, and will discuss the importance of *Fusarium* species as agents of disease in crops, animals and humans. The pivotal role of innate and adaptive immune system also will be highlighted, as will the immune response to fungal infections. This overview will encompass diagnosis of the *Fusarium* genus using conventional methods such as mycological culture and direct microscopic examination, in addition to non-cultural-base methods including nucleic acid-based techniques and antigen detection using mAbs. The concept of water-borne fungal disease and nosocomial infections, fungal dispersal, and the potential role of biofilms as

a source of infectious propagules in domestic and hospital settings will also be discussed.

1.1 Kingdom Fungi

Fungi are heterotrophic eukaryotic microorganisms that occur ubiquitously in almost all environments encompassing terrestrial, aquatic, polar, temperate and tropical ecosystems (Moore *et al.*, 2011; Poole *et al.*, 2013; Timling *et al*., 2014) They constitute a single kingdom, Kingdom Fungi, comprising seven phyla, Chytridiomycota, Blastocladiomycota, Glomeromycota, Microsporidia, Zygomycota, Ascomycota and Basidiomycota (**Figure 1.1**) (Berbee, 2001; Bonfante *et al.*, 1996; Das *et al*., 2007; Mohanta & Bae, 2015; Moore *et al*., 2011; Pöggeler & Wöstemeyer, 2011; Rossman, 2008). Since fungi can produce a broad range of enzymes such as *α*-amylase, cellulase, lipase, protease, and xylanase (Green & Beezhold, 2011) they are the main decomposers and recyclers of organic substrates in terrestrial ecosystems, with saprotrophism (or saprotrophic) enabling colonisation and utilisation of organic matter for nutrition. Since fungi are unable to fix carbon, they obtain nutrients from different organic sources to provide the energy required for growth and reproduction and so are called heterotrophic organisms. Fungi produce extracellular enzymes to degrade complex organic compounds into simpler metabolisable units such as glucose or amino acids and absorb these into the cell through the cell wall and plasma membrane. Most fungi have a saprotrophic phase in their life cycles, obtaining their nutrients from decaying organic matter of plants or animals (Jung *et al*., 2013). Pathogenic fungi are also able to derive

nutrients from parasitized hosts either biotrophically or via necrotrophism (Lowe & Howlett, 2012).

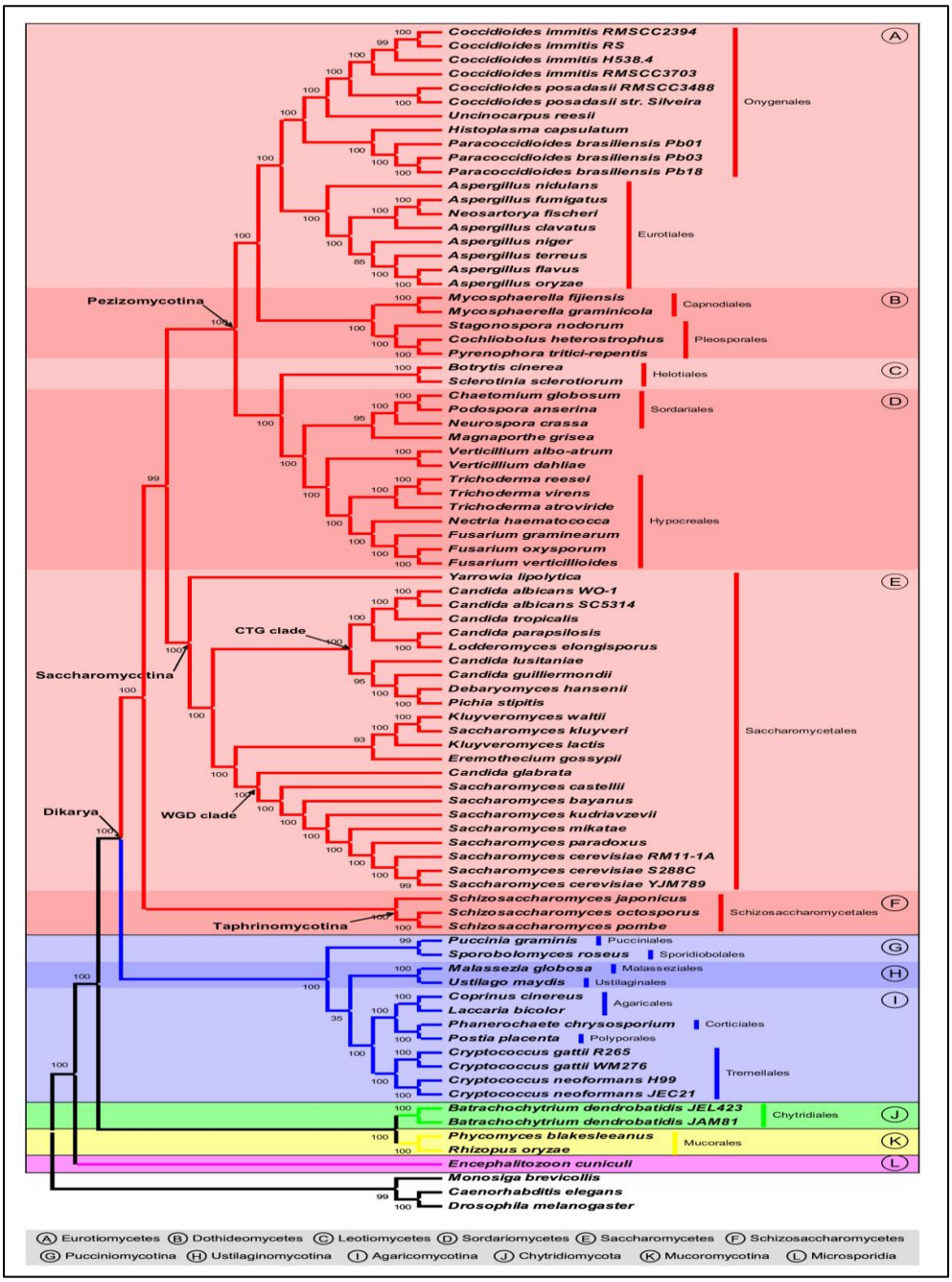


Figure 1.1. Phylogeny tree of the Kingdom Fungi. Strain names are given only when more than one organism in that species appeared in our dataset. Blocks coloured in red, blue, green, yellow and purple correspond to the Ascomycota, Basidiomycota, Chytridiomycota, Mucoromycotina and Microsporidia, respectively. The majority of fungi are related to the Ascomycota and Basidiomycota are distinguished by alternate red and blue colours, respectively. (Wang *et al*., 2009).

The structure and composition of the fungal cell wall is a defining characteristic of these eukaryotes and may differ in architecture and dynamic function. The cell wall plays a vital role in in several biological processes including the fungal cell morphogenesis, growth, cell-cell interaction, and it provides a protective barrier from environmental conditions as it acts as a sensor and response to surrounding environment change such as osmotic pressure (Adams, 2004; Klis, 2001). For the pathogenic fungi, the cell wall components involve in virulence and pathogenicity through mediating fungal cell adhesion, colonization and biofilm developing (Bowman & Free, 2006; Free, 2013). The fungal cell wall is a complex structure composed of glycoproteins embedded within a polysaccharide matrix including of chitin and glucan, and there is evidence of extensive cross-linking between these components together to generate a three-dimensional structure with the cell membrane that contains a fungal unique lipid, ergosterol (**Figure 1.2**) (Bowman & Free, 2006; Latgé, 2010; Masuoka, 2004; Parks & Casey, 1995; Pasanen *et. al*., 1999). For this reason carbohydrates is the first component to contact host tissue (Hall *et al.*, 2013; Masuoka, 2004). The inner layer of cell wall consists of chitin which is synthesized by chitin synthases, and glucan which is a homopolymer of glucose that include β-1,3-glucan, mixed β-1,3- with β-1,4-, β-1,6-, and α-1,3-glucan. The glucan–chitin complex is covalently bound to other polysaccharides, and that varies in the pathogenic fungi. For example, *Aspergillus fumigatus* polysaccharides are composed of galactomannan and β-1,3 and β–1,4-glucan, whereas in *Candida albicans* β-1,6-glucan is the main contain (Aimanianda *et al*., 2009; Fontaine *et al*., 2000). Glycoproteins including galactomannnan and/or mannan are the main composition of the outer layer of the cell wall. Once the glucan braches, chitin, and glycoproteins are released into the cell

wall space, the components covalently cross-linked to form a rigid cell wall (Barreto-Bergter & Figueiredo, 2014; Free, 2013; Latgé, 2010; Masuoka, 2004)

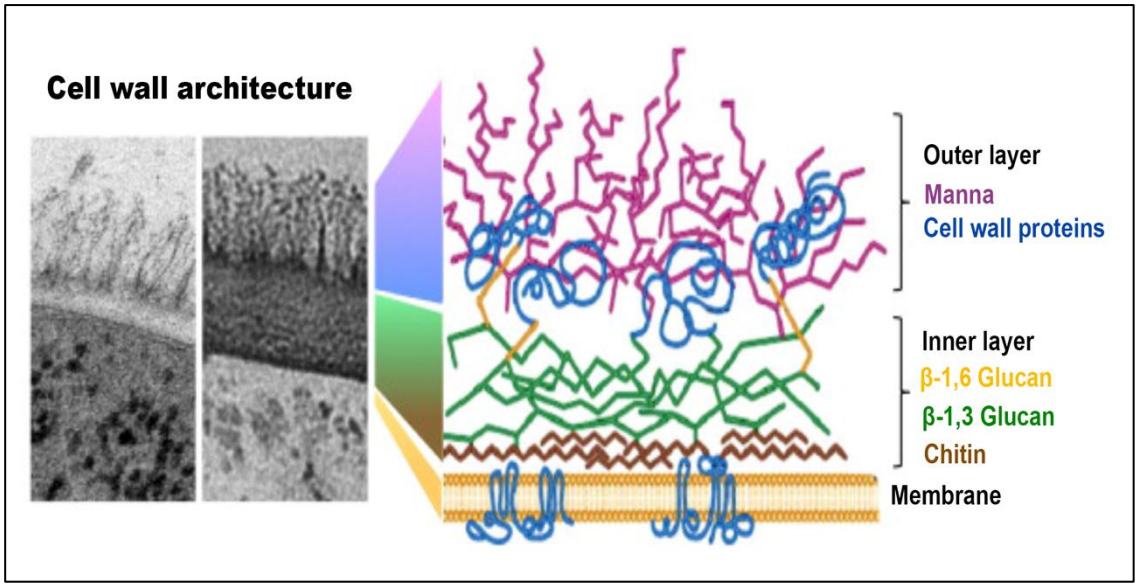


Figure 1.2. Fungal cell wall architecture. Transmission electron micrographs of the *Candida albicans* cell walls are shown on the left. The right illustrates the structure of the *C. albicans* cell wall. (Brown *et. al.,* 2014).

Fungi are a major source of beneficial bioactive compounds used in medicine and agriculture, such as antibiotics (Bérdy, 2005) and plant-growth-promoting compounds (Naznin *et al*., 2013), but also produce highly toxic secondary metabolites and allergens deleterious to human health (Brase *et al*., 2009; Desjardins & Proctor, 2007). As opportunists, fungi can cause serious life-threating infections both in immunocompetent and immunocompromised individuals, particularly those with haematological malignancies or bone marrow tansplants (Perfect, 2012).

In 2001, it was estimated that one and a half million species of fungi exist on the planet (Hawksworth, 2001). However, more recent molecular studies using

analysis of the ITS rRNA region suggests that more than 5 million fungal species exist (Blackwell, 2011; O'Brien *et al*., 2005).

The majority of fungal species that have been discovered belong to the phyla Ascomycota and Basidiomycota, and so they are combined into a separate fungal sub-kingdom called Dikarya (Hibbett *et al.,* 2007). Pathogenic fungi are often found in the phyla Basidiomycota, Ascomycota, and Zygomycota (Galagan *et al.*, 2005).

The Basidiomycota includes important human pathogens such as the yeast *Cryptococcus* spp. (causative agent of the disseminated human disease cryptococcosis), *Trichosporon* spp. (disseminated trichosporonosis) and *Malassezia* spp. that causes superficial skin diseases (Anaissie *et al.*, 2009; Gaitanis *et al*., 2012). Species of *Rhizopus*, *Mucor* and *Lichtheimia* are the main pathogenic agents of mucormycosis (Ribes *et al*., 2000; Skiada *et al*., 2011).

1.1.1 Phylum Ascomycota

Ascomycota is the largest fungal phylum and contains, at present, 64,000 species (Weete *et al*., 2010). The main characteristic feature of this phylum is the formation of a sac-like structure (ascus) containing ascospores (Carlile *et al*., 2001; Webster & Weber, 2007). Many ascomycetes reproduce asexually by producing copious airborne conidia or spores generated from mycelia. Alternatively, they can reproduce sexually, indicated by a teleomorphic phase (Hogg, 2005). For example, *Nectria haematococca* is the teleomorph state of *Fusarium solani* (the anamorph or asexual stage).

Ascomycetes include filamentous (hyphal)-organisms and yeasts. Unlike zygomycetes, ascomycete hyphae are septate with cross walls called septa. These fungi are ubiquitous and associated with food spoilage and are the main producers of mycotoxins. Examples include the filamentous fungi *Aspergillus* and *Fusarium* species (Pitt & Hocking, 2009). Many ascomycetes produce carcinogenic mycotoxins, such as aflatoxins and trichothecenes that can cause serious harm to animals and humans (Nelson *et al*., 1993; Yazar & Omurtag, 2008; Yli-Mattila, 2010). Nevertheless, ascomycetes can be beneficial, and are amenable biotechnologically for the production of antibiotics such as penicillin, immunomodulatory compounds such as cyclosporins and edible mycoproteins. However, many ascomycete species are recognizable as aggressive plant pathogens, causing destructive crop diseases, such as Panama disease of banana caused by *F. oxysporum* f.sp. *cubense* race 4 (Dean *et al*., 2005; De Ascensao & Dubery, 2000).

The clinical importance of opportunistic ascomycete fungi, such as *Aspergillus* (the cause of invasive pulmonary aspergillosis), *Fusarium* (fusariosis), and *Candida* (candidiasis) is increasing amongst the ever expanding populations of immunocompromised patients such as those with neutropenia, haematological malignancies and recipients of bone marrow transplants. Opportunistic ascomycetes are emerging as the most significant source of mortality and morbidity in these high-risk patient groups (Hanson, 2008; Kauffman *et al.*, 2011; Moore *et al*., 2011).

1.1.1.1 The fungus *Fusarium*

The genus *Fusarium* is one of the most ubiquitous groups of filamentous fungi and contains several economically important plant pathogens and producers of mycotoxins such as fumonisins and trichothecenes. This heterogeneous genus is also an important producer of bioactive metabolites, including antibiotics and anti-cancer agents (Baker & Tatum, 1989; Chakravarthi *et al*., 2008; Gerber & Ammar, 1979; Mohamed, 2012; Qureshi *et al*., 2003; Tayung *et al*., 2011).

A saprotroph and general opportunist, it is increasingly recognised as a serious human pathogen (Leslie & Summerell, 2006). This genus has been found in virtually all ecosystems on the planet and more than 1000 species of the genus have been described (Rios & Ortega, 2012; Summerell *et al.*, 2003).

Fusarium was initially described based on the presence of fusiform spores in 1809 by Link and validated by Fries in 1821 (Booth, 1971). The anamorphic (asexual) stage of *Fusarium* species is dominant and often produces both microconidia and macroconidia. The microconidia are small in size and oval to ovoid in shape and grow from the aerial mycelium on phialides. The macroconidia are distinctive, large, and septate, with curved to straight appearances, and are initiated from cushion-like structures called sporodochia (Leslie & Summerell, 2006; Toussoun & Nelson, 1975) as shown in **Figure 1.3.**

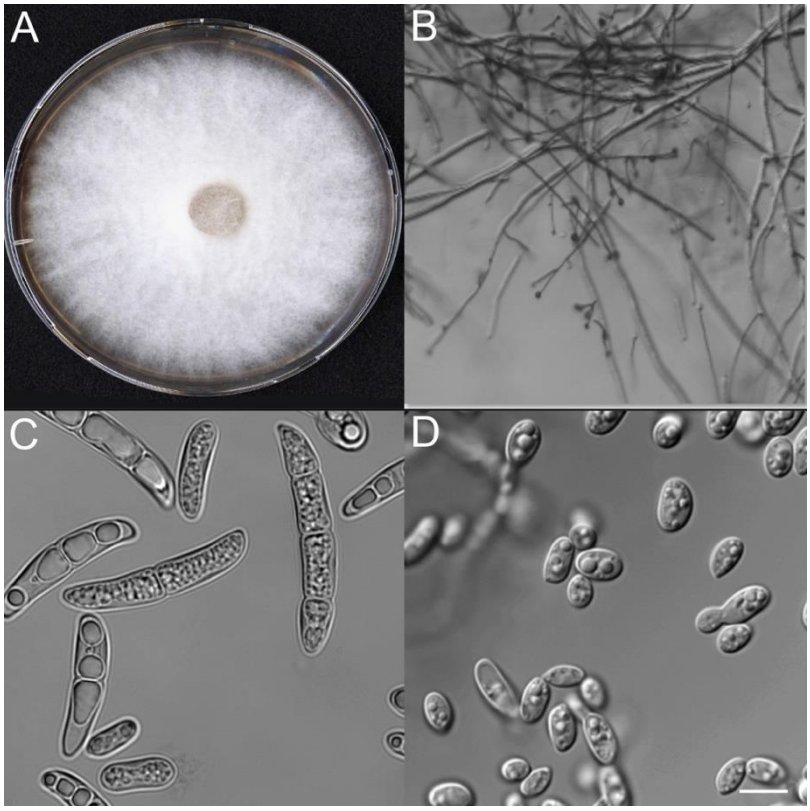


Figure 1.3. Gross morphology of *F. solani* (CBS224.34) in axenic culture on potato dextrose agar (A), with mycelia (B), macroconidia (C) and microconidia (D). Scales bars = 20 μm. Images of Marwan Al-Maqtoofi.

1.1.1.2 Classification of the genus *Fusarium*

The first *Fusarium* taxonomical system was established in 1935 by Wollenweber and Reinking and in the 1940s by Snyder and Hansen (Nelson, 1975; Nelson, 1991). Since then, many species have been added or removed from the *Fusarium* genus due to morphological similarity (Rossman & Palm-Hernández, 2008). Plant pathogenic species within morphological groupings were further divided based on host specificity. As a consequence, varieties (var.) and formae speciales (f. sp.) are used to describe host specificities of *Fusarium* species (Agrios, 2005; Ma *et al*.,2010). For example, *F. oxysporum* f.

sp. *lycopersici* and *F. solani* f. sp. *pisi* are narrow in host infection, infecting the only tomato and pea plants, respectively (Lievens *et al*., 2009; Namiki *et al*., 1994; Suga *et al*., 2002), while *F. graminearum* or *F. verticillioides* are selective pathogens for cereal crops (Shim *et al*., 2006). However, some species, such as *F. oxysporum,* infect a broad range of hosts. As such, *F. oxysporum* is currently ranked as the fifth most important plant pathogen (Dean *et al*., 2012; Ma *et al*., 2010).

The genus *Fusarium* is currently classified as follows:

Phylum: Ascomycota

Subphylum: Pezizomycotina

Class: Sordariomycetes

Sub-class: Hypocreomycetidae

Order: Hypocreales

Family: Nectriaceae (Lumbsch & Huhndorf, 2007; Rossman *et al*., 2013).

Genus: *Fusarium*

1.2 Etiology and Pathogenesis

Recently, the incidence of fungal infections has increased dramatically concomitant with the ever-expanding population of immunocompromised humans. This population is highly susceptible to fungal infections due to impaired host immune responses. Many health factors have been associated with suppression of host immune defence, including AIDS, malignant diseases, especially patients those receiving toxic chemotherapy agents like corticosteroids and intensive broad-spectrum antibiotic therapy, and solid organ transplantation (SOT) and bone marrow transplantation (BMT) (Low & Rotstein,

2011; Sganga, 2011; Silva, 2010). In the US between 1980 and 1997, the death rate due to invasive fungal infections (IFI) increased 320%. The most common fungal pathogens causing serious nosocomial life-threatening infections are *Candida* and *Aspergillus* species (Anaissie, 1992; Lockhart *et al*., 2009; Low & Rotstein, 2011; McNeil *et al*., 2001; Silva, 2010; Walsh & Groll, 1999). Significant increases in IFIhave also been reported with emerging pathogens, including hyaline septated filamentous fungi, such as *Fusarium*, *Acremonium*, *Scedosporium*, *Paecilomyces*, and *Trichoderma*. In addition, yeasts such as *Trichosporon*, *Rhodotorula*, and *Geotrichum,* the mucormycetes *Rhizopus* and *Mucor* species and dematiaceous fungi, including *Pseudallescheria*, *Bipolaris*, *Exophiala*, *Phialophora*and *Cladophialophora*, have all been reported as opportunist pathogens of humans (Pfaller & Diekema, 2004; Ponton *et al*., 2000; Walsh *et al*., 2004; Walsh & Groll, 1999). The mortality rate of hematologic malignancy patients with invasive mould infections is far greater than that associated with yeasts infections with 64% mortality for mucormycosis, 53% for fusariosis, 42% for aspergillosis and 33% for candidiasis (Araujo *et al*., 2010; Pagano *et al*., 2006).

Fusariosis is the second most common invasive fungal infection among high-risk patients after *Aspergillus* (Boutati & Anaissie, 1997; Nucci *et al*., 2004; Stanzani *et al.*, 2007; Walsh & Groll, 1999) and can be cutaneous or disseminated. These infections can occur in both immunocompromised and healthy individuals. In severely immunodeficient patients, invasive infection is more persistent (Jain *et al*., 2011; Nucci & Anaissie, 2007). Because *Fusarium* is unresponsive to standard antifungal therapy, the incidence of the invasive fusariosis significantly increases with intensive use of cytotoxic agents and

immunosuppressive drugs (Nucci *et al*., 2014; Ponton *et al.*, 2000). Some *Fusarium* species comprise complexes of strains that vary in virulence towards humans. *F. solani*, for example, comprises the *F. solani* species complex (FSSC) (Zhang *et al*., 2006). The two most virulent species complexes involved in disseminated fusariosis are FSSC, followed by the *F. oxysporum* species complex (FOSC) (Campbell *et al*., 2013; Mayayo *et al*., 1999; Moore *et al*., 2011; Diepeningen *et al*., 2014). The FSSC is also well known as a complex containing economically important crop pathogens. This species complex is one of the most common causes of fungal keratitis in immunocompetent people. Patients with hematologic malignancies receiving cytotoxic chemotherapy, SOT and stem cell transplant recipients, and patients with extensive burns are at high risk for invasive fusariosis associated with FSSC (Zhang *et al*., 2006).

Pathogenic fungi can make biofilms in environment and even host tissues by proliferating and expanding extensive living forms of hyphae and conidia in an extracellular matrix (ECM) allowing adhesion to surfaces for penetration (Pierce *et al.,* 2008). Accordingly, this ability has identified as a serious clinical issue (Ramage *et al*., 2012). Many of medically important fungi such *Candida* (Deorukhkar *et al*., 2014; Ramage et al., 2012), *Aspergillus* (Kaur & Singh, 2014) and *Fusarium* (Imamura *et al.*, 2008; Mukherjee *et al.,* 2012) that produce biofilms tend to display high resistance to host immunity response and antifungal agents. Recent studies have suggested that biofilm formation is one of the most important pathogenicity virulence factors in invasive mycoses, and associated with high mortality rates (Rajendran *et al*., 2016; Tumbarello *et al*., 2007). Since a biofilm is a surface-associated fungal community, a typical fungal biofilm development involves in four sequential steps including the cell

adhesion to a surface, cells colony formation, maturation into a three-dimensional structure, and dispersion of cells (Ramage *et al.*, 2009), as shown in a model of filamentous (**Figure 1.4**) (Ramage *et al.*, 2011) and in yeasts (Chandra *et al*., 2001) (**Figure 1.5**). Since *Fusarium* keratitis outbreat during 2005/6 in the US, *Fusarium* biofilm formation on soft contact lenses has been studied (Imamura *et al*., 2008; Mukherjee *et al*., 2012). Glycoprotein with mannose residues in the extracellular fibrillar matrix located on the outer cell wall layer of the *Fusarium* spore is thought to be a start point of adhesion and biofilm developing (Kwon & Epstein, 1997; Kwon & Epstein, 1993; Pan *et al.,* 2011; Gauthier & Keller, 2013; Kwon & Epstein, 1997). At present, little is known about *Fusarium* biofilm formation and its role in survival and pathogenicity of the fungus during invasive infections (Peiqian *et al.,* 2014).

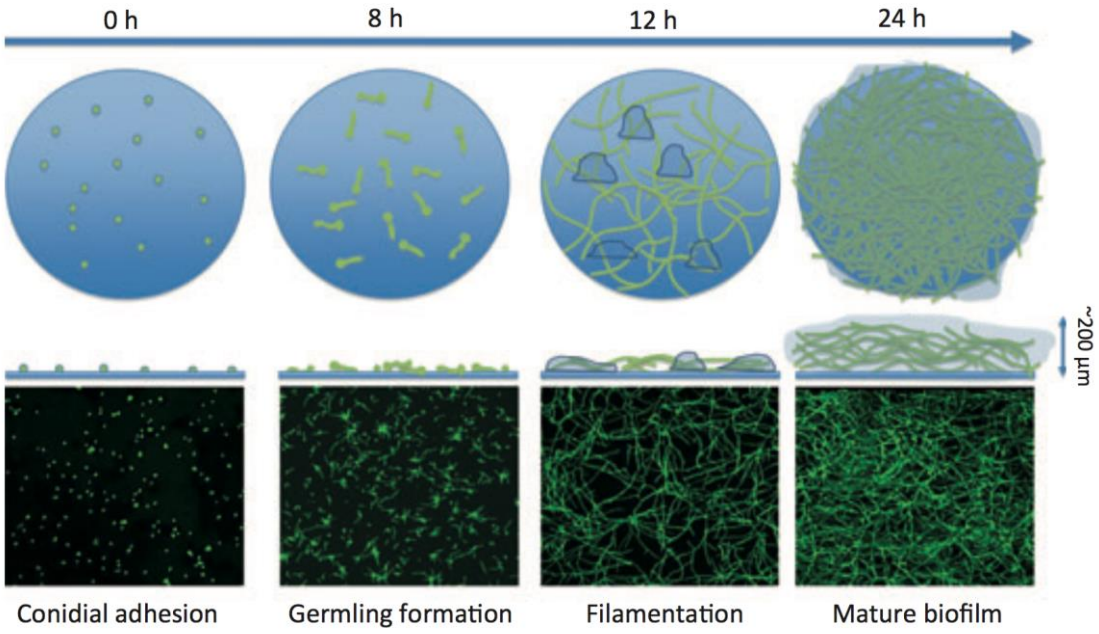


Figure 1.4. *Aspergillus fumigatus* biofilm development. The different phases of biofilm development are represented schematically, from initial adhesion of conidia, germling formation (8 h), a monolayer of intertwined hyphae (12 h) and mature 3D filamentous biomass (c. 200 µm) encased within exopolymeric sub- stance (24 h). (Ramage *et al*., 2011).

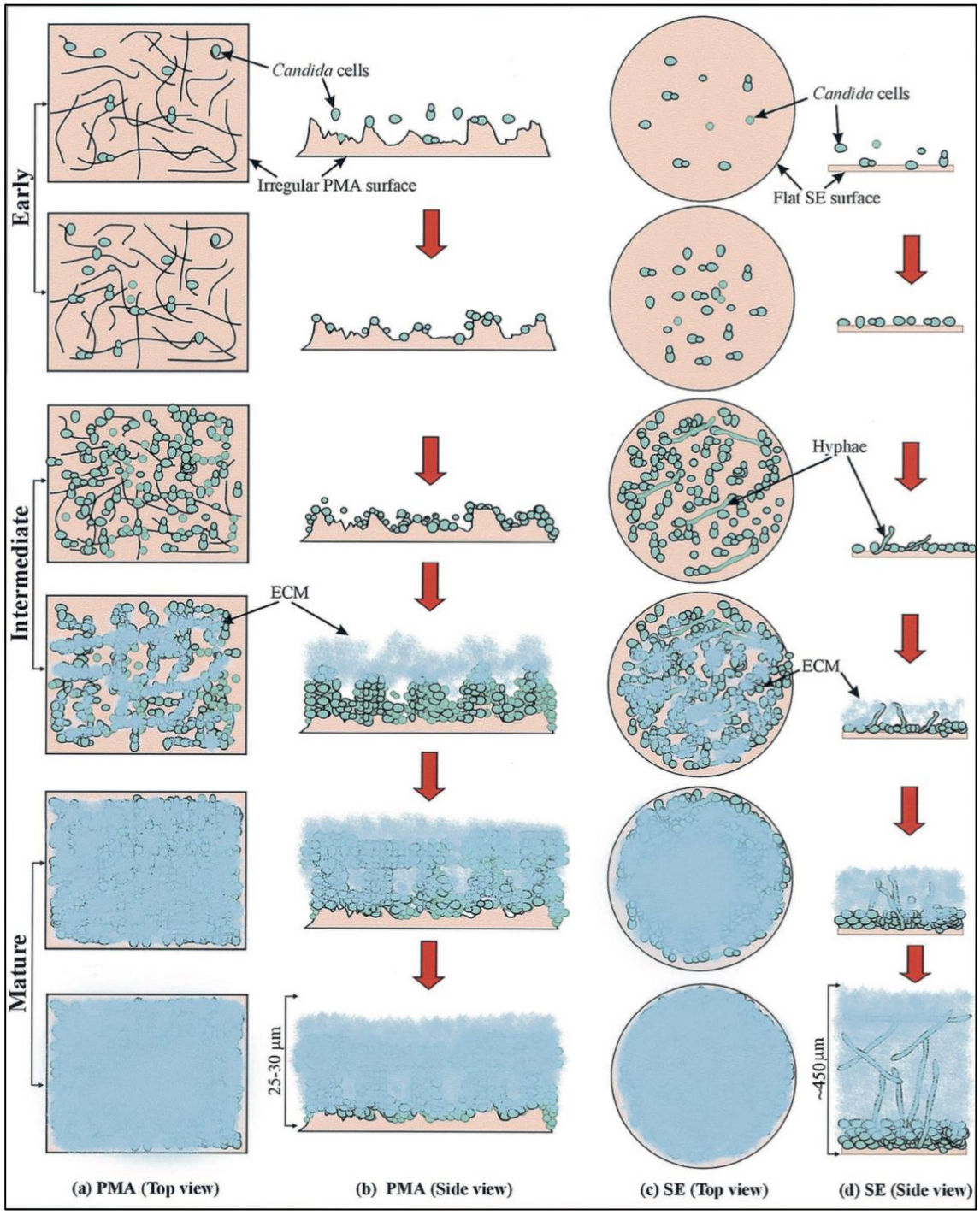


Figure 1.5. Schematic representation of biofilm development in *C. albicans*. (**a** and **b**) Biofilm grown on polymethylmethacrylate (PMA) strips. (**c** and **d**) Biofilm grown on silicone elastomer (SE) disks. Panels (**a**) and (**c**) represent the substrate seen from the top, while panels c and d show the view from the sides of the PMA strip and SE disk, respectively. ECM, extracellular material. (Chandra *et al*., 2001).

1.3 The Human Immune System

The human immune system is comprised of a complex network of immune cells and soluble chemical components that interact effectively to prevent access and defend against fungal invasion. The innate and adaptive immune systems are a vital part of the immune responses towards infectious diseases (Romani, 2004). The innate immune system is the first line of protection (Brown, 2011; Figueiredo *et al*., 2011). It is non-specific, mounting a rapid response to potential pathogens, and it lacks immunological memory. The main components of the innate immunity are a physical barrier, such as the skin, epithelium, and mucus that is produced in the airways and gastrointestinal tract (Romani, 2004; Romani, 2011). In addition, soluble components such as cytokines, complement, antimicrobial compounds and inflammatory mediators interact to deliver immunity (Abbas et al., 2007) alongside innate phagocytic cells such macrophages and neutrophils, natural killer cells, and dendritic cells (Folds, 2008).

The adaptive immune system can be activated once fungal propagules breach the innate immune system. The adaptive immune system provides a specific response and can generate immunological memory for life-long-acquired immunity. Both immune systems cooperate effectively to eliminate infectious pathogens (Williams *et al*., 2012).

1.3.1 The innate immune system and fungal infection

The innate immune system provides an initial line of defence against potential pathogens by recognising PAMPs, foreign antigens, such as carbohydrates and

proteins, unique to pathogens and which evoke a host immune response following recognition by PRRs. Innate immunity to fungi consists of physical barriers, serum factors including complement, cytokines, and natural immunoglobulins, and phagocytic cells (Carvalho *et al*., 2010). These components work efficiently to prevent and control fungal dissemination and halt potential infections. The innate immune response lacks immunological memory and is non-specific, but is rapid. Each part of the innate immune system has an essential role against fungal invasion either directly or indirectly within a complex network of signalling.

The first line of protection is the physical barrier of the skin and the mucosal layer at potential portals of entry for fungi. Therefore, individuals with severe burns are at a high risk of fungal infections (Church *et al.*, 2006). Epithelial tissues participate against microbial invasion via releasing antimicrobial compounds that either inhibit or kill microbial cells whilst also activating cellular innate immunity. Cellular innate immunity comprises phagocytic and antigen-presenting cells including sentinel M∅, circulating polymorphonuclear neutrophils (PMN), natural killer cells (NK), and dendritic cells (DC). Innate immune cells aid in the clearance of infectious agents via phagocytosis and have antigen-presenting functions in the lymphoid organs that stimulate the adaptive immune response. Unlike human cells, fungi have a distinct cell wall that contains chitin, glucans, mannans and glycoproteins (Bowman & Free, 2006). Innate immune cells have specific surface receptors, such as the β-glucan receptor Dectin-1, mannose receptors, and TLRs, for fungal cell wall components and can discriminate between infectious cells and human cells (Folds, 2008; Shoham & Levitz, 2005). Despite this, some pathogenic fungi,

such as *Cryptococcus*, have developed mechanisms to avoid the innate immune system (Voelz & May, 2010). For example; phagocytosed *Cryptococcus* can be multiple inside macrophages and then escape either by a novel non-lytic mechanism (Johnston & May, 2010) or lysis host cells (Alvarez & Casadevall, 2006).

1.3.2 The adaptive immune system and fungal infection

The adaptive immune system differs in some aspects to the innate immune system, not least in that it has immunological memory. Humoral immunity and cell-mediated immunity are the main arms of the adaptive immune system with adaptive cellular immunity consisting of T and B-lymphocytes. Humoral immunity refers to antibodies produced by B-cells. These antibodies can be either non-specific or specific for a certain pathogen, binding to conserved or pathogen-specific epitopes (antigenic determinants) on immunoreactive antigens. Antibodies participate in fungal elimination via a range of mechanisms, including preventing spore adhesion, neutralising extracellular enzymes, inhibiting yeast-hyphal morphological transitions, preventing spore germination, agglutinating and opsonizing fungal spores, and facilitating phagocytosis. There are different classes of antibodies or immunoglobulins (Ig). Ig classes that circulate in the bloodstream include Ig class M (IgM) and IgG. Others, including IgA and IgE, are located within mucous membranes (Romani, 2002). In response to the fungal invasion, T-helper cells (Th) and cytotoxic T-cells (CTLs) differentiate from a common lymphoid progenitor cell. Th cells produce cytokines and interferon-γ (IFN-γ) to activate macrophages to kill phagocytosed pathogens. CTLs play critical roles in the elimination of

pathogens by killing infected host cells (Abbas *et al*., 2007; Blanco & Garcia, 2008).

1.4 Antifungal Treatment

Unlike antibacterial antibiotics, the therapeutic options for managing fungal infections using antifungal compounds are limited, particularly with the dramatic increase in immunocompromised population and diversity of emerging human pathogenic fungi (Kanafani & Perfect, 2008; Richardson & Warnock, 2003). This limitation is associated with a poor prognosis (Ashley, *et. al.*, 2006). The reason behind that is because fungal pathogens were not recognized as serious life threatening agents (Anaissie & Bodey, 1989). Furthermore, fungi and human beings are eukaryotes microorganisms and therefore have closely related to cellular machinery, structure and function. This necessities robust development of antifungal agents with little or minimal to mammalian host cells (Dixon & Walsh, 1996; Masuoka, 2004). Currently, three main classes of antifungal drugs including polyenes, the azoles and the echinocandins have been developed for treatment of fungal infections (Lattif & Swindell, 2010). In addition, there is a miscellaneous group of compounds, such as flucytosine and griseofulvin are also utilised (Richardson & Warnock, 2003). The pharmaceutical form of antifungal are mainly administered orally, intravenous and eye drops (Arthur *et. al*., 2004; Kawamura *et. al*., 2000).

Antifungal agents target only unique fungal cell structure and processes that involve synthesis of cell wall components such as chitin, β-glucans, ergosterol in addition to disrupting the in cell membrane. This selectively kills the fungal

cells without affecting the mammalian cells (**Figure 1.6**) (Free, 2013; Ghannoum & Rice, 1999; Kathiravan *et al*., 2012).

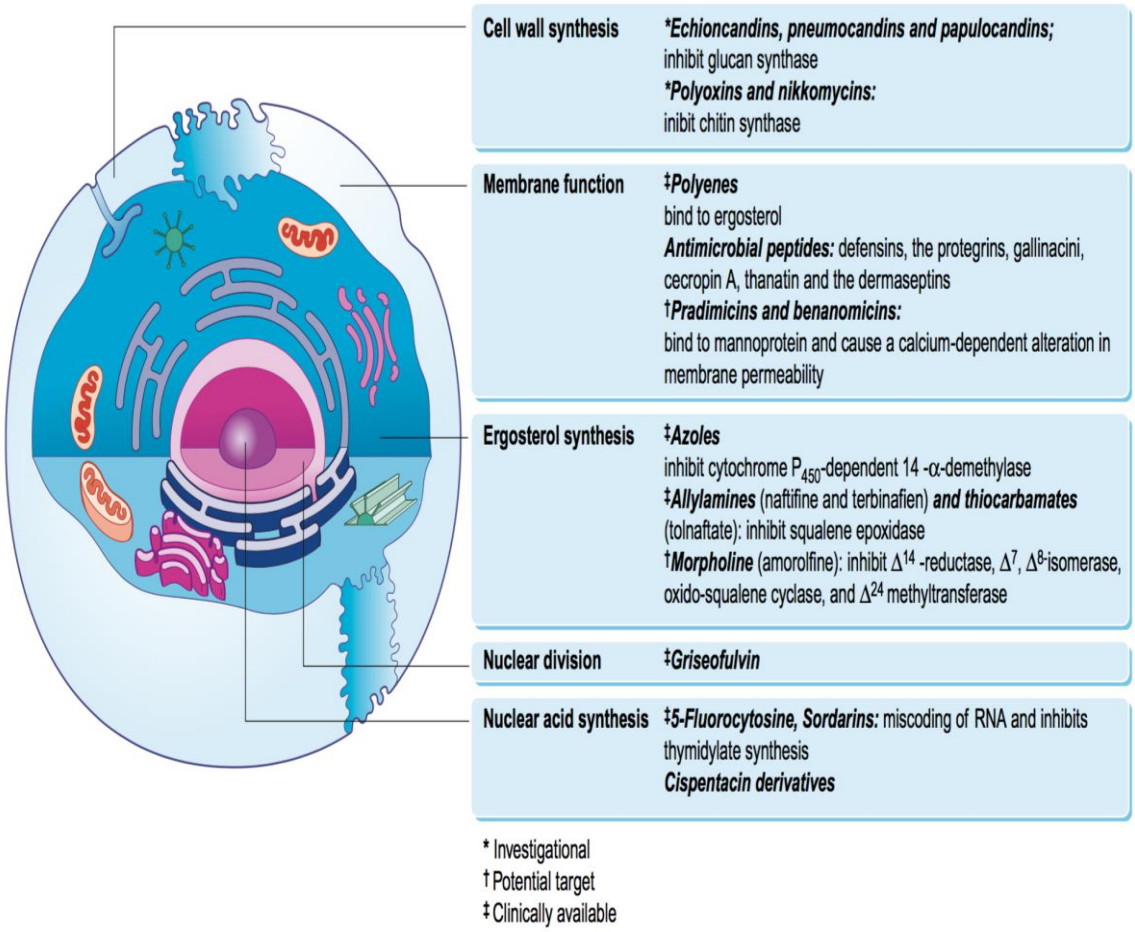


Figure 1. 6. Sites of action of antifungals. (Gubbins & Anaissie, 2009).

Azoles compounds such voriconazole and psoaconazole interfere with the synthesis of ergosterol in the fungal cell membrane through inhibiting the cytochrome P450 enzyme, lanosterol demethylase in fungi (Kanafani & Perfect, 2008). Amphotericin B deoxycholate and its lipid-form belong to polyenes antifungal group and act with ergosterol by making holes in the fungal membrane allowing leakage of essential cytoplasmic materials, such as

potassium, leading to cell death (Ghannoum & Rice, 1999; Kanafani & Perfect, 2008). The echinocandins have a unique mechanism of action, inhibiting β-(1,3)-D-glucan synthase an essential component of the fungal cell wall (Sucher *et. al.*, 2009). In comparison to the azoles and polyenes, the echinocandin group including caspofungin , micafungin and anidulafungin are not widely used for therapy of invasive mycoses due to their large molecular weight and therefore poor absorption through the digestive tract (Lattif & Swindell, 2010).

Pathogenic fungi have developed mechanisms to resist antifungal compounds thereby reducing drug efficiency. The main strategies employed to reduce efficiency of the antifungal compounds by pathogenic fungi include reduction of drug accumulation in the fungal cell, reduction of the target affinity for the drug and changes to the metabolism. (Pemán *et al.*, 2009; Terra *et al*., 2014).

Managing fusariosis is a serious challenge since most of pathogenic *Fusarium* species are resistant to most antifungal agents thereby limiting treatment options for invasive infections (Azor *et. al.*, 2007; Muhammed *et. al.*, 2011; Mukherjee *et. al.*, 2012; Nucci *et. al.*, 2013). There is no clear and optimum treatment strategy for *Fusarium* infections (Blyth *et al*., 2014; Carneiro *et al*., 2011; Liu *et a*l., 2014; Muhammed *et al*., 2011; Nucci & Anaissie, 2007). Therefore, treatment of immunocom-promised patients with disseminated fusariosis is difficult and is compounded by the wide variation of *Fusarium* species complex that leads to variability in response to the antifungal agents (Stanzani *et al*., 2007). That may explain the low survival rate of persistent neutropenic and transplant recipient patients (Nucci *et al*., 2014). However,

some studies showed that Amphotericin B (AMB) was the best active antifungal agent with minimal inhibitory concentration (MIC) no lower than 2 μg/ml (Azor et al., 2007). In contrast, terbinafine (TBF) was the most active drug for treating fusariosis at MIC of 0.60 μg/ml compared to the other rest azole drugs such as ravuconazole (RVC), albaconazole (ABC), itraconazole (ITC), and ketoconazole (KTC), fluconazole (FLC), flucytosine (5-FC) (Azor *et al*., 2009) which had relatively high MIC ranging from 4.24 to 32 μg/ml) . Treatment immunocompromised patients with high intravenous doses of amphotericin B or the lipid formulations is recommended as a first choice therapy for invasive fusariosis (Wheat *et. al.*, 2002) with voriconazole and posaconazole as an alternative therapies (Muhammed *et. al*., 2011). Granulocyte colonies stimulating factor (G-CSF) or granulocyte-macrophage colony stimulating factor (GM-CSF) are also tools for managing fusariosis (Jain *et. al.,* 2011). In conclusion, evidence available indicates there no single effective strategy for treating invasive fusariosis with response to antifungals such azole varying widely (Gubbins & Anaissie, 2009).

1.5 Diagnosis of invasive fungal infections

In comparison with other infectious diseases, the diagnosis of fungal infections (mycosis) is still underscored by the management of the disease. It is a major problematic health issue, especially in the absence of a clear understanding of the epidemiology of fungal infections. The need for accurate and sensitive diagnostic approaches for invasive mycosis is becoming more important, particularly with the dramatic increase in severely immunocompromised patients and the rise in nosocomial infections. Early diagnosis allows timely treatment

with appropriate antifungal drugs and successful management of the fungal disease. Establishing a sensitive and rapid diagnostic test for fungal infections is complicated. Traditional methods of diagnosis that are used include direct microscopy, mycological culture, histopathology, clinical symptoms, radiology and serological tests. The direct microscopic examination is the primary method used to identify fungal propagules in clinical samples visually. Definitive diagnosis of disseminated mycosis requires the recovery and identification of fungal pathogens from samples such as skin, sinuses, lung, and body fluids (in particular blood) using culture and microscopy (Nucci & Anaissie, 2009). Selective stains are used to detect fungal elements. For example, calcofluor white and India ink are used to visualise fungal cells with fluorescence or contrast microscopy (Stevens, 2002).

Identification of fungal pathogens at the species level is often needed for appropriate antifungal treatment. Direct microscopical examination is cheap and widely available but is not able to identify pathogenic fungi at a species level. Furthermore, it requires expertise in medical mycology to discriminate different morphological features of infectious agents (Alexander & Pfaller, 2006; Steinbach *et al*., 2003). A notable example is the invasive hyaline fungal pathogens (causative agents of hyalohyphomycosis). This group of organisms, which includes *Aspergillus*, *Fusarium*, *Scedosporium*, *Penicillium* and *Acremonium* all have hyaline septate hyphae with narrow-angle branching (Arvanitis *et al*., 2014; Tortorano *et al*., 2014; Nguyen *et al*., 2012; Nucci & Anaissie, 2009). Moreover, some pathogenic fungi, such *Acremonium* spp., grow slowly and require around 14 days (Park *et al*., 2011).

Clinical manifestations of deep mycosis are non-specific, and variable, depending on infection site and patient status. For example, severely immunocompromised hosts more frequently undergo fever, chest pain, skin lesions, pulmonary infiltration and other clinical features. A broad range of fungal pathogens, such as *Fusarium*, *Curvularia*, *Bipolaris*, *Exserohilum*, *Alternaria*, *Aspergillus* species and *Pseudallescheria boydii* can all mimic the same clinical signs (Guarner & Brandt, 2011; Park *et al*., 2011; Pauw & Meis, 2005). Radiological findings, including X-ray and computed tomography (CT) scans, are similarly non-specific, but can indicate invasive fungal infection. Therefore, a combination of diagnostic approaches is indispensable to prevent misdiagnosis (Alexander & Pfaller, 2006; Barnes, 2008; Oh *et al*., 2000; Richardson & Warnock, 2003).

The fungal cell wall has a wide variety of antigenic structures that can stimulate the adaptive immune system. In this case, activated B-cells produce antibodies that aid in opsonization and clearance of propagules by phagocytic cells. Fungal-specific serum antibody titers tend to be elevated during fungal infections in patients that can mount a B cell response and so can be used in some instances to detect circulating antigens released during disseminated disease (Blanco & Garcia, 2008; Shoham & Levitz, 2005). However, in patients with impaired immune responses, it is not possible to generate detectable antibody titers against systemic fungal infections (Stevens, 2010).

Conversely, serodiagnostic tests using polyclonal or monoclonal antibodies, generated against fungal antigens in rabbits or mice, have been developed and used to track infections using crude antigen extracts or recombinant proteins

(Brandt & Warnock, 2003). Serodiagnostic tests are rapid and are achieved using immunoassays such as ELISA or immunodiffusion (ID) (Morrison & Warnock, 2007). Because polyclonal antibodies are not specific to a given genus or species (Badiee & Hashemizadeh, 2014), serological assays are less likely to be able to discriminate between the many different fungal pathogens (Ahmad *et al*., 2010; Alexander, 2002; Limper, 2010). For this reason, highly specific monoclonal antibodies generated using hybridoma technology are needed for accurate serological diagnosis. With the broad variety of emerging opportunistic fungal pathogens, conventional diagnostic methods are considered time-consuming, inaccurate, and/or not sensitive enough. This leads to an increased chance of misidentification and mistreatment (Alexander & Pfaller, 2006; Arvanitis *et al*., 2014).

1.5.1 Non-Cultural Based Methods

Non-culture based methods have been developed to improve the accuracy and speed of diagnosis of invasive fungal infections. These methods include molecular (nucleic acid-based) detection methods and fungal antigen detection using mAbs (Alexander & Pfaller, 2006; Kozel & Wickes, 2014).

1.5.1.1 Molecular Diagnosis

The increasing incidence of emerging pathogenic fungi has necessitated the development of more accurate and rapid techniques for fungal diagnosis. DNA sequence-based approaches are considered the pivotal methods in the fungal identification, with a turn-around time of about 24 h (Balajee *et al*.,

2009). Molecular diagnostic techniques are culture-independent methods that do not require morphological features of living cells or colonies for identification. Most of the molecular based tools are based on the use of PCR. This technique amplifies and analyses constant genetic markers of fungal cells that provide rich data for fungal identification (Kozel & Wickes, 2014).

PCR-based techniques are increasingly being used as a rapid tool for detection and amplification of marker genes specific to each fungal species, such as TEF-1α for *Fusarium* species, β-tubulin for *Aspergillus*, endo-polygalacturonase for *Alternaria*, and calmodulin for *P. boydii* (Romanelli *et al*., 2014). Several regions of the fungal genome have been evaluated as potential targets for detection, such as the ITS region of the nuclear ribosomal DNA (rDNA) (Richardson & Warnock, 2003). However, getting sufficient DNA yield from clinical samples is not always feasible (Guarner & Brandt, 2011). Furthermore, it has been shown that PCR-based techniques cannot completely replace culture-based methods due to their variable sensitivities and the possibility of contamination in the diagnostic laboratory (Alexander & Pfaller, 2006).

1.5.1.2 Antigen Detection Using Monoclonal Antibodies

Hybridoma technology enables the produce of highly specific mAbs against a specific antigen *in vitro*, and can make a significant contribution to the improved accuracy and speed of diagnosis for fungal pathogens. This technology was first described by Georges Köhler and Cesar Milstein in 1975 (Köhler & Milstein, 1975) and allows the identification of B-cells secreting antibodies of predefined specificity. Antibody-producing B-cells cannot survive

in vitro unless they are first fused with a myeloma cell, to produce a hybridoma cell. These immortalised B-cells cell lines can then be grown in culture and their antibodies harvested for use in immunodiagnostics (Abbas *et al.,* 2007).

MAbs can identify fungi at the genus (Thornton *et al*., 2002) and species levels (Thornton *et al*., 1993). Highly specific mAbs have been developed to identify pathogenic fungi in soil and plants (Dewey & Thornton, 1995; Dewey *et al*., 1997; Thornton *et al*., 1993; Thornton, 2008a; Thornton *et al*., 2002; Thornton & Talbot, 2006; Thornton *et al*., 2004). For medical purposes, specific mAbs have been developed for the early diagnosis of mycoses, particularly invasive infections (Thornton, 2009a; Thornton, 2008b). Successful diagnosis of human pathogenic fungi using mAbs developed previously against plant pathogens have also been reported (Thornton, 2009b; Thornton, 2008b; Thornton & Wills, 2013).

Many mAbs have been developed for accurate, sensitive and rapid detection of opportunistic fungal pathogens. Specifically, mAbs for *Aspergillus* (Thornton, 2008), *P. boydii* (Thornton, 2009), *Scedosporium prolificans* (Thornton *et al*., 2015), and *Trichosporon asahii* (Davies & Thornton, 2014a) have all been developed. In medical mycology, the availability and accuracy of new diagnostic tests have a significant impact on the management of fungal infections and reduction of mortality levels.

1.6 Focus of This PhD

The ever expanding population of patients with compromised immune systems is at risk of disseminated infections associated with opportunistic fungal pathogens (Crabol *et al.,* 2014; Kriengkauykiat *et al.,* 2011). The most common disseminated fungal infections are caused by species of *Candida* and *Aspergillus*, with considerable rates of morbidity and mortality (Nucci & Marr, 2005). However, some opportunistic fungi such as *Fusarium* and *Scedosporium*, *Rhizopus*, *Mucor*, *Trichosporon*, *Rhodotorula*, *Paecilomyces* and *Exophiala* have emerged as significant causes of life-threatening infections in humans, but relatively little is known about their pathobiologies (Anaissie *et al*., 1989; Boutati & Anaissie, 1997; Kauffman, 2004; Nucci *et a*l., 2002; Pfaller & Diekema, 2004).

Furthermore, these fungi are typically less susceptible to antifungal agents currently used in the clinical setting (Srinivasan *et al.*, 2014). Early diagnosis of these organisms is critical, but rapid and accurate detection remains a serious challenge (Barnes, 2008; Low & Rotstein, 2011).

The aim of this work was to develop and characterise highly *Fusarium*-specific mAb that can be used to detect this important emerging human pathogen and as a tool to study its epidemiology and interaction with cells of the innate immune system.

The specific aims of these investigations were:

1. To characterize a *Fusarium*-specific mAb (mAb ED7) by investigating the chemical nature and cellular localization of its target antigen (**Chapter 2**);

2. To use mAb ED7 to study the interaction of *Fusarium* spores with cells of innate immunity (**Chapter 3**);

3. To use mAb ED7 to identify sources of the pathogen for nosocomial and community-acquired infections (**Chapter 4**).

Chapter 2. Production and Characterization of the *Fusarium*-Specific mAb ED7

2.1 Introduction

2.1.1 *Fusarium* as a Plant Pathogen

Fusarium species are major plant pathogens causing a range of diseases in a large number of economically important crops with yield losses of up to 100% (Burgess, Knight, Tesoriero, & Phan, 2008; Leslie & Summerell, 2006). The genus causes diseases of cereals (Abbas *et al*., 1998; Francis & Burgess, 1977; Moretti, 2009), fruits (Jiménez *et al*., 1993; Zhang *et al*., 1999), and vegetables (Naef & Défago, 2006; Roy, 1997), with species listed in the top 10 fungal plant pathogens according to economic importance including *F. graminearum* (fourth) and *F. oxysporum* (fifth) (Dean *et al.,* 2012). The best known *Fusarium* disease in plants is *Fusarium* Head Blight (FHB), a disease affecting cereal crops, rice and wheat. FHB is caused by *F. oxysporum* (Nganje *et al*., 2004), *F. graminearum* (Goswami & Kistler, 2004), *F. avenaceum* (Yli-Mattila, 2010), *F. culmorum*, *F. pseudograminearum* (Balmas *et al*., 2015) and *F. proliferatum* (O'Donnell *et al*., 2010). Other *Fusarium* diseases include sudden death syndrome (SDS), *Fusarium* wilt, root, stem and seed rots and cankers of many fruits and vegetables including tomato, potato, cotton, cabbage, cucumbers, melons, date palms, peas, and soybeans (Ahmet, 2011; Ajilogba & Babalola, 2013; Gupta *et al.*, 2009; Moretti *et al*., 2002; Vicente *et al.*, 2014; Pilotti *et al.*, 2002; Schroers *et al.*, 2009). A number of *Fusarium* species, for example, *F. solani* and *F. oxysporum*, are capable of infecting both plants and humans (Moore *et al.*, 2011).

2.1.2 *Fusarium* as Agents of Human Infectious Diseases

The most frequently cited fungal pathogens of humans include *Candida*, *Cryptococcus*, *Aspergillus, Fusarium*, *Scedosporium*, *Rhizopus* and *Mucor* (Araujo *et al*., 2010; Esnakula *et al.*, 2013; Pontón *et al.*, 2000). Invasive infections caused by *Fusarium* species (known as fusariosis) mainly occur in immunocompromised humans and, in certain settings, have emerged as the second most common opportunistic pathogen after *Aspergillus* (Boutati & Anaissie, 1997; Nucci *et al.*, 2004; Walsh & Groll, 1999), causing localised and disseminated infections in humans (Nelson *et al.*, 1994; Nucci & Anaissie, 2007).

The first case of disseminated infection due to *F. solani* was reported in an immuno-compromised child in 1973 (Cho *et al.*, 1973). Since then, *F. solani* has been recognised as a serious opportunistic pathogen of humans (Mayayo *et al.*, 1999; Schmidig, 2012; Stanzani *et al.*, 2007). The dramatic increase in fusariosis during the last three decades coincides with the ever expanding populations of severely immune deficient individuals, including those with acquired immune deficiency syndrome (AIDS), BMT and SOT recipients, and those who receiving cytotoxic agents such as immunosuppressive drugs, intensive broad-spectrum antibiotics, radiotherapy and chemotherapy (Dignani & Anaissie, 2004; Muhammed *et al.*, 2011; Schmidig, 2012). Mortality and morbidity rates for disseminated fusariosis are estimated to be between 50-75% (Jensen *et al.*, 2004; Krcmery *et al.*, 1997). However, recent studies have suggested that this could even be as high as 100% (Nucci & Anaissie, 2007). The increasing incidence of invasive fusariosis and high mortality rates are likely due to *Fusarium* virulence factors, the production of mycotoxins and

lytic enzymes, and spore adhesion and biofilm formation. In addition, *Fusarium* is resistant to almost all currently available antifungal drugs (Azor *et al.*, 2007; Nelson *et al.*, 1994; Nucci & Anaissie, 2007; Pujol *et al.*, 1997; Tortorano *et al.*, 2014).

Many other clinically relevant *Fusarium* species include *F. oxysporum, F. verticillioidis, F. moniliforme, F. dimerum, F. proliferatum, F. chlamidosporum, F. sacchari, F. nygamai, F. napiforme, F. antophilum*, and *F. vasinfectum* (Dignani & Anaissie, 2004; Nucci & Anaissie, 2007; Wang *et al.*, 2011). The FSSC group is the most frequent and virulent group involved in invasive infections (Mayayo *et al.*, 1999; O'Donnell *et al.*, 2010; Diepeningen *et al.*, 2014), followed by *F. chlamydosporum* species complex (FCSC), *F. incarnatum/equiseti* species complex (FIESC), FOSC and *F. dimerum* species complex (FDSC) (Balajee *et al.*, 2009; Diepeningen *et al.*, 2014).

Fusarium infections may occur at various sites of the human body, manifesting as superficial, subcutaneous or deep infections. *Fusarium* infections can be localized on the skin (dermatophytosis), cornea (keratitis) (Chang *et al.*, 2006; Cuero, 1980; Gungel *et al.*, 2011; Sekeroglu *et al.*, 2012; Verma & Tuft, 2002), or nails (onychomycosis) (Brasch & Köppl, 2009; Brasch & Shimanovich, 2012; Girmenia *et al.*, 1992). Compared to immunocompetent hosts, infections in immunocompromised patients are typically disseminated, due to the lack of an innate immune response (Bachmeyer, 2007; Bushelman *et al.*, 1995; Cooke *et al.*, 2009; Perez *et al.*, 2007; Yera *et al.*, 2003; Ammari et.al., 1993; Ascioglu *et al.*, 2002; Lucca, 2007; Nucci *et al.*, 2004; Nucci & Anaissie, 2002, 2007; Tortorano *et al.*, 2014; Venditti *et al.*, 1988). Broken skin and the

respiratory tract are the primary portals of entry for *Fusarium* propagules (Legrand *et al.*, 1991; Muhammed *et al.*, 2011; Nucci & Anaissie, 2007). Additionally, *Fusarium* can also cause allergic symptoms during the aerosolization of spores in dusty indoor environments (Noble *et al.*, 1997).

2.1.3 Diagnosis of *Fusarium* infections

2.1.3.1 Morphological Characteristics

As with all other human fungal pathogens, identification of *Fusarium* infection depends on the identification of characteristic morphologic features of cultured samples and microscopic examination of clinical samples from sterile sites or biopsy (Esnakula *et al*., 2013; Marcio Nucci *et al.*, 2004; Marcio Nucci & Anaissie, 2007). However, the morphological appearance of *Fusarium* is highly variable (O'Donnell *et al.*, 1998; Summerell *et al.*, 2003; Diepeningen *et al.*, 2015), and its polyphyletic taxonomy makes it one of the most challenging groups of fungi to categorise (Leslie & Summerell, 2006). Identification of *Fusarium* species using direct microscopical examination can lead to misdiagnosis due to similarities in morphology with other fungal pathogens. Acute-branching septate hyaline hyphae with or without sporulation are characteristics shared between *Fusarium* and other hyalohyphomycetes fungi, including *Aspergillus*, *Scopulariopsis* and *Acremonium* (Jain *et al.*, 2011; Nucci & Anaissie, 2007; Tortorano *et al.*, 2014). Identification based on morphological features requires the preparation of axenic cultures which is time-consuming and delays diagnosis (Dignani & Anaissie, 2004; Muhammed *et al.*, 2011b). Notwithstanding this, mycological culture remains the gold standard test for

fungal detection in clinical laboratories (McClenny, 2005) despite its poor sensitivity compared to other diagnostic procedures.

2.1.3.2 Nucleic Acid-based Techniques

Diagnostic techniques that detect nucleic acids have been used to identify IFIat early stages of clinical manifestations. The need for accurate identification of *Fusarium* at the species level requires construction of an accurate phylogenetic-based classification (O'Donnell, 1996; O'Donnell, 2000). To this end extensive taxonomical studies have been undertaken to identify DNA sequences specific to *Fusarium* species or species complexes (Leslie & Summerell, 2006). Barcoding based on the rDNA ITS region has been used to differentiate closely related species and to identify inter- and intraspecific variation (Bryan *et al*., 1995; O'Donnell, 1996; Seifert, 2009). This technique has enabled accurate discrimination of the most common pathogenic fungi involved in invasive mycoses at the species level (Spiess *et al*., 2007). However, in this setting, the use of ITS sequencing for *Fusarium* species identification is inadequate, as it does not reveal sufficient information for discriminating *Fusarium* species complex, such as FSSC and FOSC (Balajee *et al*., 2009; Geiser *et al*., 2004; O'Donnell *et al*., 2010; O'Donnell & Cigelnik, 1997). For this reason, *Fusarium* protein-encoding genes have been sequenced to identify marker genes for diagnosis, including the TEF-1α gene (Geiser *et al*., 2004; Harrow *et al.*, 2010; Kristensen *et al.*, 2005; Yli-Mattila *et al*., 2002), the β-tubulin gene (Mach *et al*., 2004; Seifert & Lévesque, 2004; Yli-Mattila *et al*., 2004), and two genes encoding the largest and second largest subunits of RNA polymerase (RPB1 and RPB2, respectively) (Boonyuen *et al*., 2011; O'Donnell *et al*., 2010) and

calmodulin (Mulè *et al*., 2004). The TEF-1α gene has been validated as a more informative gene compared to the other encoding genes sequenced. TEF-1α can be used for the identification and phylogenetic analysis of the *Fusarium* genus (O'Donnell *et al*., 1998), but the use of molecular-based techniques for detection of *Fusarium* infections is used alongside other tests for clinical diagnosis (Tortorano *et al*., 2014).

2.1.3.3 Serological Assays

The chemical composition of the fungal cell wall is unique and presents opportunities for the development of diagnostic test based on detection of characteristic signature molecules. While the chemical components of the fungal cell wall are similar at the level of the phylum, the antigenic composition at genus and species levels can vary considerably allowing the identification of genus and species-specific biomarkers for diagnostic test development (Free, 2013). During disseminated infections, antigens are shed into the bloodstream and bronchoalveolar lavage fluid (BAL) (Kozel & Wickes, 2014).

2.1.3.3.1 Detection of 1,3-β-D-Glucan and Galactomannan

Detection of the fungal cell wall components BDG and GM form are the main serological tests commercially available and used widely in clinical settings for invasive fungal pathogens (Kozel & Wickes, 2014). However, the BDG test is not specific and is an insufficient tool for the diagnosis of fusariosis, as *Fusarium* species often cross-react with other species like *Aspergillus*, *Candida, Acremonium* and *Trichosporon*. (Esnakula *et al*., 2013; Ostrosky-Zeichner

et al., 2005; Yoshida *et al*., 1997). In 1992, Stynes described the development of a unique mAb (EB-A2) that strongly reacts the tetra (1→5)- β-D-galactofurano-side, a specific glycoprotein epitope of *Aspergillus* GM (Stynen *et al*., 1992). Ever since the GM test has become a medically important application for the early diagnosis of invasive *Aspergillus* infections, and it is commercially available as an ELISA kit (Kappe & Schulzeberge, 1993; Latge *et al*., 1994; Leeflang *et al*., 2008; Tanriover *et al.*, 2008; Verweij *et a*l., 2006). The GM test cross-reacts with several other invasive pathogenic fungi, including *Penicillium*, *Fusarium*, *Alternaria*, *Histoplasma*, *Trichothecium, Geotrichum, Cladosporium* and *Acremonium* (Aquino *et al*., 2007; Giacchino *et al*., 2006; Huang *et al*., 2007; Kappe & Schulzeberge, 1993; Mikulska *et al.*, 2012; Swanink et al., 1997; Tortorano *et al.*, 2012; Wheat *et al*., 2007). Therefore, serological tests have been of limited benefit for the diagnosis of fusariosis (Muhammed *et al*., 2011).

2.1.3.3.2 Polyclonal and Monoclonal Antibodies

For this reason, diagnosis of invasive mycoses based on cell wall composition using a polyclonal antibody is a routin approach employed in clinical laboratories (Brandt & Warnock, 2003). MAbs are an attractive diagnostic tool for systemic fungal infections and have exclusive specificity for detecting the smallest and unique fungal surface epitopes. This technology saves time and is sensitive for a definitive diagnosis of fungal infections at a genus or species level, and can guide appropriate antifungal therapy usage. Furthermore, antigen tests do not require any immune response. They rely only on the tracking of a fungal cell, or even cell wall debris in the blood stream or other body fluids at any detectable level.

2.1.4 Objective of This Chapter

To date, no studies have been undertaken to develop a diagnostic antibody that can detect human pathogenic strains of *Fusarium*. The purpose of the work presented in this chapter was to generate mAbs specific to a constitutively expressed extracellular antigen from *F. solani* that might be appropriate for tracking human pathogenic strains and to investigate their interaction with cells of the immune system (**Chapter 3**) and during epidemiological studies (**Chapter 4**). This work takes advantage of hybridoma technology, using approaches already successfully exploited in this laboratory for the development of murine mAbs for the detection of invasive fungal infections, including aspergillosis (Thornton, 2010; Thornton, 2008) and scedosporiosis (Thornton, 2009).

2.2 Materials and Methods

2.2.1 Ethics Statement

All animal work described in this study was conducted under a UK Home Office Project License, and was reviewed by the institution's Animal Welfare Ethical Review Board (AWERB) for approval. The work was carried out in accordance with The Animals (Scientific Procedures) Act 1986 Directive 2010/63/EU, and followed all the Codes of Practice which reinforce this law, including all elements of housing, care, and euthanasia of the animals.

2.2.2 Fungal Cultures and Media

Details of the fungi used in this study are in **Table 2.1** All fungi were grown and maintained on PDA or in PDB (PDB; Difco, 254920) 2.4% containing 2% (w/v) agar). For liquid cultures, fungal spores at a concentration of 1×10^3 ml^{-1}, or 3 x 5 mm agar plugs taken from the leading edge of PDA cultures, were inoculated into 250 ml conical flasks containing 100 ml of PDB, or in SDB (SDB; Oxoid; CM0147) (3%). All media was autoclaved at 121°C for 15 min prior to use. Fungi were grown on slopes or on Petri dishes under a 16-h fluorescent light regimen at 27°C.

Table 2.1. Details of fungi used in mAb ED7 specificity tests.

Organism	Isolate no.	Source[a]
Aspergillus cervinus	537.65	CBS
Aspergillus ficuum	555.65	CBS
Aspergillus flavus	91856iii	IMI
Aspergillus fumigatus	AF293	SK
Aspergillus nidulans	A4	FGSC
Aspergillus niger	102.4	CBS
Aspergillus oryzae	672.92	CBS
Aspergillus restrictus	116.5	CBS
Aspergillus terreus var. terreus	601.65	CBS
Botrytis cinerea	R2	CRT
Byssochlamys nivea	153.59	CBS
Candida glabrata	4692	CBS
Candida krusei	5590	CBS
Candida parapsilosis	8836	CBS
Candida tropicalis	1920	CBS
Cryptococcus neoformans (Serotype D)	5728	CBS
Cunninghamella elegans	151.8	CBS
Filobasidiella bacillispora	10865	CBS
Filobasidiella neoformans	10490	CBS
Filobasidiella neoformans	10496	CBS
Fusarium acutatum	402.97	CBS
Fusarium anthophilum	222.76	CBS
Fusarium avenaceum	386.62	CBS
Fusarium cerealis	134.8	CBS
Fusarium chlamydosporium var. chlamydosporium	491.77	CBS
Fusarium culmorum	256.51	CBS
Fusarium dimerum var. dimerum	108944	CBS
Fusarium incarnatum	678.77	CBS
Fusarium nygamai	140.95	CBS
Fusarium oxysporum f.sp. *cucurbitacearum*	254.52	CBS
Fusarium oxysporum f.sp. *lycopersici*	167.3	CBS
Fusarium oxysporum f.sp. *marmaris*	420.8	CBS
Fusarium oxysporum f.sp. *radicis-lycopersici*	872.95	CBS

Fusarium oxysporum f.sp. *vasinfectum*	409.9	CBS
Fusarium proliferatum var. *proliferatum*	181.3	CBS
Fusarium sacchari	183.32	CBS
Fusarium solani	224.34	CBS
Fusarium solani	109696	CBS
Fusarium solani	188.34	CBS
Fusarium solani	115659	CBS
Fusarium solani	117608	CBS
Fusarium solani	119223	CBS
Fusarium solani var. *petroliphilum*	102256	CBS
Fusarium verticillioides	102699	CBS
Geotrichum candidum	115.23	CBS
Haematonectria haematococca	114067	CBS
Haematonectria haematococca	119603	CBS
Haematonectria haematococca	130692	CBS
Lichtheimia corymbifera	T14A (FJ713070)	CBS
Magnusiomyces capitatus	207.83	CBS
Mucor circinellioides f.sp. *circinellioides*	E2A (FJ713065)	CRT
Neosartorya fischeri var. *fischeri*	687.71	CBS
Paecilomyces variotii	339.51	CBS
Penicillium cyclopium	123.14	CBS
Penicillium islandicum	338.48	CBS
Penicillium spinulosum	346.61	CBS
Pichia norvegensis	6564	CBS
Pseudallescheria boydii	835.96	CBS
Pythium insidiosum	673.85	CBS
Rhizomucor miehei	MG4(2) (FJ713069)	CRT
Rhizopus stolonifera	389.95	CBS
Rhodosporidium toruloides	6016	CBS
Rhodotorula mucilaginosa	326	CBS
Scedosporium apiospermum	117407	CBS
Scedosporium aurantiacum	121926	CBS
Scedosporium aurantiacum	118934	CBS
Scedosporium prolificans	102176	CBS
Sporidiobolus salmonicolor	6781	CBS
Trichoderma hamatum	GD12 (AY247559)	CRT

Trichosporon asahii var. *asahii*	8973	CBS
Trichosporon asahii var. *asahii*	5286	CBS
Trichosporon asahii var. *asahii*	7632	CBS
Trichosporon asahii var. *asahii*	5599	CBS
Trichosporon asteroids	6183	CBS
Trichosporon asteroids	7623	CBS
Trichosporon asteroids	2481	CBS
Trichosporon asteroids	7624	CBS
Trichosporon cutaneum	2466	CBS
Trichosporon inkin	7630	CBS
Trichosporon inkin	7655	CBS
Trichosporon loubieri	7065	CBS
Trichosporon ovoides	7556	CBS
Trichosporon mycotoxinovorans	9756	CBS
Wickerhamomyces anomalus	5759	CBS

a CBS = Centraalbureau voor Schimmelcultures, PO Box 85167, 3508 AD Utrecht, The Netherlands; CRT = C.R. Thornton; IMI = International Mycological Institute, Egham, England; SV = S. Krappman, Institute of Microbiology and Genetics, Department of Molecular Microbiology and Genetics, Georg-August-University, Gottingen, Germany.

2.2.3 Preparation of Immunogen and Immunisation Regime

BALB/c mice were immunised with soluble antigens prepared from lyophilized mycelium of a human pathogenic strain of FSSC 1-a (CBS strain 224.34). Conidia were suspended in water after 10-day old PDA slant cultures were flooded with 5 ml MQ-H_2O and gently agitated with an inoculation loop. Conidial suspensions were then filtered through Miracloth to remove mycelium and transferred to 1.5 ml micro-centrifuge tubes. The conidia were washed three times with MQ-H_2O by repeated vortexing and centrifugation at 14,462 *g* for 5 min and finally suspended in MQ-H_2O to give a concentration of 10^6 spores

ml^{-1} solution. Flasks containing 100 ml of sterilised PDB were inoculated with 200 µl of the conidial suspension and incubated with shaking (75 rpm) for 48 h at 26ºC. Hyphal biomass was collected on Miracloth, snap frozen in liquid N_2, and lyophilized. Culture filtrates were retained for gel electrophoresis and Western blotting studies and stored at –20ºC until required. One mg of lyophilized biomass was suspended in 1 ml PBS (0.8% NaCl; 0.02% KCl; 0.115% Na_2HPO_4; 0.02% KH_2PO_4; pH7.2) and the resultant suspension centrifuged for 5 min at 14,462 *g*. The supernatant, containing solubilized antigens, was used as the immunogen and as a source of antigens for hybridoma screening assays. For immunisation, 6-wk-old BALB/c female white mice were given four intraperitoneal injections (300 µl per injection) of antigen extract containing 2.3 mg protein ml^{-1} PBS at 2-wk intervals and a single booster injection five days before fusion.

2.2.4 Production and Screening of Hybridomas and Determination of Antibody Specificity

Hybridoma cells were produced by the method described by Thornton (2001). The myeloma cell line SP2/0-Ag14, in the exponential phase of growth, was prepared at a concentration of 10^7 cells ml^{-1} by enumeration using a haemocytometer. The mouse selected for hybridoma production was euthanised and then doused with 70% (v/v) ethanol. The spleen was removed aseptically and placed in a 30-ml Universal tube containing 10 ml warm 20% TCM. Following this, the spleen was placed in a 9-cm Petri dish containing 15 ml of fresh 20% TCM and the splenocytes released by teasing the tissues with sterile hypodermic needles. The splenocytes were transferred to a sterile

50-ml Falcon tube and, alongside the myeloma cells, were centrifugation at 250 g for 5 min. The supernatants were discarded and the cells were re-suspended in 2.5 ml of RPMI serum-free medium. The two suspensions were combined, mixed, and centrifugation in a sterile round-bottomed 10-ml tube at 250 g for 5 min. Next, the supernatant was aspirated and 0.3 ml of 33% (w/v) PEG solution was added allowing exposure of the cells for 7 min including 1 min centrifugation at 500 g. The PEG was gently removed and then the pellet was slowly re-suspended by adding 5 ml of serum-free medium. The fusion products were diluted into 95 ml of selective medium consisting of 10% TCM with hypoxanthine, aminopterin and thymidine (HAT; Sigma, H0262) supplement and 100 µl volumes of the cell suspension transferred to the wells of 96-well culture plates for incubation at 37^{o}C and 5% CO_2. After 7 days, the cells were fed with 10% TCM supplemented with hypoxanthine and thymidine (HT; Sigma, H0137) and wells containing hybridomas identified subsequently. The cell supernatants were screened by ELISA against of *F. solani* (CBS224.34) antigens immobilized to the wells of Maxisorp microtitre plates (442404; Nunc) (50 µl per well). Selected cell lines were sub-cloned, growing in non-selective 10% TCM, and then preserved by freezing in LN_2.

For antibody specificity tests, fungi were grown on replicate agar slopes and surface washings containing water-soluble antigens prepared as described in Thornton (2001). Protein concentrations, determined spectrophotometrically at 280 nm (Nanodrop, Agilent Technologies Limited, Berkshire, UK), were adjusted to 60 µg ml^{-1} buffer. Fifty µl volumes were then used to coat the wells of microtitre plates. After incubating overnight at 4^{o}C, wells were washed four times with PBST (PBS containing Tween-20, 0.05% (v/v)), once each with PBS

and MQ-H_2O and then air-dried at 23°C in a laminar flow hood. The plates were stored in sealed plastic bags at 4°C in preparation for the screening of hybridoma supernatants by ELISA as described below.

2.2.5 Enzyme-Linked Immunosorbent Assay

Wells containing immobilized antigens were blocked for 10 min with 100 µl of blocking solution 1% (wt/vol) BSA (Sigma A-7030) in PBS and incubated successively with hybridoma tissue culture supernatant (TCS) for 1 h, followed with goat anti-mouse polyvalent (Ig classes IgG, IgA, and IgM) peroxidase conjugate (A0412; Sigma Chemical Company, Poole, United Kingdom) diluted 1 in 1000 in PBST for a further hour. Bound antibody was visualised by incubating wells with TMB (T2885; Sigma) substrate solution (Thornton, 2001) for 30 min. The reactions were stopped by the addition of 3 M H_2SO_4 and absorbance values were determined at 450 nm with an MRX automated microplate reader (Dynex Technologies, Billingshurst, UK). Wells were given four 5-min rinses with PBST between incubations and a final rinse with PBS before addition of the substrate solution. Working volumes were 50 µl per well and control wells were incubated with TCM containing 10% (v/v) FBS. All incubation steps were performed at 23°C in sealed plastic bags. The threshold for detection of the antigen in ELISA was determined from control means (2 x TCM absorbance values) (Sutula *et al*., 1986). These values were consistently in the range (0.05 - 0.10). Consequently, absorbance values > 0.100 were considered as positive for the detection of antigen.

2.2.6 Determination of Ig Subclass and Sub-cloning Procedure

The Ig class of mAbs was determined by using antigen-mediated ELISA. Wells of microtitre plates coated with *F. solani* (CBS224.34) water-soluble antigens from surface washings were incubated successively with ED7 TCS for 1 h, followed with goat anti-mouse IgG_1, IgG_{2a}, IgG_{2b}, IgG_3, IgM, or IgA-specific antiserum (ISO-2; Sigma) diluted 1 in 3000 in PBST for 30 min and rabbit anti-goat peroxidase conjugate diluted 1 in 1000 (A-5420; Sigma) for a further 30 min. Bound antibody was visualised with TMB substrate as described above. Hybridoma cells lines were subcloned three times by limiting dilution according to the method described in Thornton (2001).

2.2.7 Long-term Storage of Hybridoma Cells

Hybridoma cells were frozen for long-term storage in liquid nitrogen (LN_2). Culture plates containing 20 ml 10% TCM were seeded with 5 ml of cell suspension and incubated for 24 h at 37°C and 5% CO_2. Suspended cells were transferred to 15-ml sterile Falcon tubes (Sarstedt, 62.547.254). The contents were pelleted at 2000 rpm for 5 min in a centrifuge (HERMLE 2323K) and the supernatant removed by aspiration. The cell pellet was gently re-suspended in 600 µl of freezing medium (92:8 (vol/vol) FBS: dimethyl sulphoxide (DMSO; Sigma HYBRI-MAX, D-2650). Cell suspensions were transferred at 150 µl volumes into separate cryovials. The vials were frozen overnight in a -80°C freezer, before long-term storage in a Dewar (Tylor-Wharton, XT-20) containing LN_2.

2.2.8 Antigen Characterization

2.2.8.1 Gel Electrophoresis and Western Blotting

For sodium-dodecyl-sulphate-polyacrylamide gel electrophoresis (SDS-PAGE), culture filtrates from 2-d-old PDB shake cultures of *F. solani* (CBS224.34) and *F. oxysporum* f.sp. *lycopersici* (CBS167.30), prepared as described, were diluted in Laemmli buffer (Laemmli, 1970) and were denatured by heating at 95ºC for 10 min. Antigens were separated in 4-20% (w/v) polyacrylamide gradient gels (161-1159; Bio-Rad) for 1.5 h at 23ºC (165V) under denaturing conditions, and pre-stained broad range markers (161-0318; Bio-Rad) were used for molecular weight determinations. For Westerns, separated antigens were transferred electrophoretically to a PVDF membrane (162-0175; Bio-Rad). The membranes were blocked for 16 h at 4ºC with PBSA (PSB + 1% (w/v) BSA) and incubated with ED7 TCS diluted 1 in 2 with PBSA (PBS + 0.5% (w/v) BSA) for 2 h at 23ºC. After washing three times with PBS, membranes were incubated for 1 h with goat anti-mouse IgM (µ - chain specific) alkaline phosphatase conjugate (A-9688; Sigma), diluted 1 in 15,000 in PBSA. After the membranes were washed twice with PBS and once with PBST, the bound antibodies were visualised by incubation in BCIP/NBT substrate solution. Reactions were stopped by immersion in MQ-H_2O and air-dried between sheets of Whatman filter paper.

2.2.8.2 Characterization of Antigen by Enzymatic and Chemical Modifications and by Heating

Water-soluble antigens from surface washings of slopes of *F. solani* (CBS224.34) were prepared as described. Heat stability studies were

conducted by placing tubes of solubilized antigen in a boiling water bath. At 10 min intervals, samples were removed, centrifuged at 14,462 *g* for 5 min, and antigens immobilized to the wells of microtitre plates for assay by ELISA as described. For periodate oxidation, microtitre wells containing immobilized antigens from surface washings of the fungus were incubated with 50 µl of sodium *meta*-periodate solution (20 mM $NaIO_4$ in 50 mM sodium acetate buffer [pH4.5]) or acetate buffer only (control) at 4°C in sealed plastic bags, for time course (0, 5, 10 15, 20 hrs). Plates were given four 3-min PBS washes before processing by ELISA as described. For protease digestions, microtitre wells containing immobilized antigen were incubated with 50 µl of pronase (protease XIV; 0.9 mg ml^{-1} in PBS) or trypsin (1 mg ml^{-1} in MQ-H_2O) solution or MQ-H_2O or PBS only controls respectively for 4 h at 37°C or 4°C. Plates were given four 3-min rinses with PBS and then assayed by ELISA with ED7 TCS as described.

2.2.8.3 Immunofluorescence and Immunogold Electron Microscopy

For immunofluorescence (IF), sterilised slides were coated with a washed spore suspensions of *F. solani* (CBS224.34) containing 1% (w/v) glucose solution and incubated at 27°C for 16 h to allow spore germination and formation of germ tubes. After air-drying, the cells were fixed to the slides as described in (Thornton, 2001) and incubated with ED7 TCS or TCM only (negative control) for 1 h, followed by three 5 min PBS washes. Slides were then incubated with goat anti-mouse polyvalent fluorescein isothiocyanate (FITC) conjugate (diluted 1 in 40 in PBS) (F1010; Sigma) for 30 min. Slides were given three 5 min washes with PBS and mounted in PBS-glycerol mounting medium (F4680; Sigma) beforeoverlaying with coverslips. All incubation steps were performed

at 23°C in a humid environment to prevent evaporation and slides were stored in the dark, at 4°C, prior to examination using an epifluorescence microscope (Olympus IX81) fitted with 495 nm (excitation) and 518 nm (emission) filters for FITC. For immunogold electron microscopy (IEM) the method described in (Thornton & Talbot, 2006) was used. Spores and hyphae of *F. solani* were prepared by incubating washed conidia in 1% (w/v) glucose solution at 27°C for 16 h to allow spore germination and formation of germ tubes. Cells were embedded in LR White resin (Agar Scientific Ltd.) and ultra-thin sections prepared for immunolabeling. Sections immobilized to nickel grids were blocked by immersion in PBST containing 1% (w/v) BSA (PBST-BSA) which had been sterile filtered through a 0.2 µm filter. The grids were washed three times (3 min each) in sterile filtered PBST and then incubated in ED7 TCS or TCM only (negative control) for 1 h. After four washes (3 min each) with sterile filtered PBST, the grids were incubated for a further hour in PBST-BSA containing a 1:20 dilution of goat anti-mouse 20 nm gold conjugate (EM GAF20; BBI Solutions). The grids were washed four times (3 min each) in sterile filtered PBST and then placed on Whatman filter paper to dry. Dried grids were then incubated for 20 min in 2% (w/v) uranyl acetate solution followed by 2% (w/v) lead citrate solution for 4 min. Working volumes were 100 µl and incubation, and washing steps were carried out at 23ºC. Immunostained samples were examined using a Jeol JEM 1400 transmission electron microscope fitted with a Gatan ES 100W CCD camera.

2.2.9 Spore Agglutination Assay

Preliminary investigations showed that mAb ED7 was able to agglutinate spores of *Fusarium*. To study this phenomenon further, a light absorbance assay was developed to enable quantification of the agglutination process. To investigate the wavelength at which spores of *Fusarium* spores absorb light, washed spores of *F. solani* (CBS224.34) were re-suspended at a final concentration of 10^6 ml^{-1} in plastic cuvettes (Greiner Bio-one 613101) using either mAb ED7 TCS or 10% TCM only. Absorbance of spore suspensions was determined across a range of wavelengths from 290 nm to 610 nm, using a Janeway 7300 spectrophotometer (Bibby Scientific Limited, UK). The maximum absorbance of light for spores suspended in TCM (non-agglutinating condition) was found to be 555 nm, and so this wavelength was used for all subsequent *Fusarium* spore agglutination assays. The light absorbance of spore suspensions was recorded every 5 min, following suspension in TCM or in mAb ED7 TCS (agglutinating conditions) up to 110 to 300 min depends on agglutination status compare with the control isolate *F. solani* (CBS224.34).

2.3 Results

2.3.1 Production of Hybridoma Cell Lines, Isotyping of monoclonal Antibody and Specificity

A single fusion was performed, and 389 hybridoma cell lines were screened for specificity against a range of clinically relevant yeasts and moulds (**Table 2.1**). The aim was to identify cell lines secreting mAbs specific to *Fusarium* that could be used to track the fungus in environmental samples containing mixed species of human pathogenic fungi. To this end, a single cell line, ED7, was identified that produced mAb belonging to the immunoglobulin class M (IgM), which was genus-specific, reacting in ELISA tests with antigens from *Fusarium* species and with the *F. solani* teleomorph *Haematonectria haematococca* only (**Figure 2.1 A and B**). It did not cross-react with antigens from a wide range of unrelated mould and yeast species (**Figure 2.1 A**).

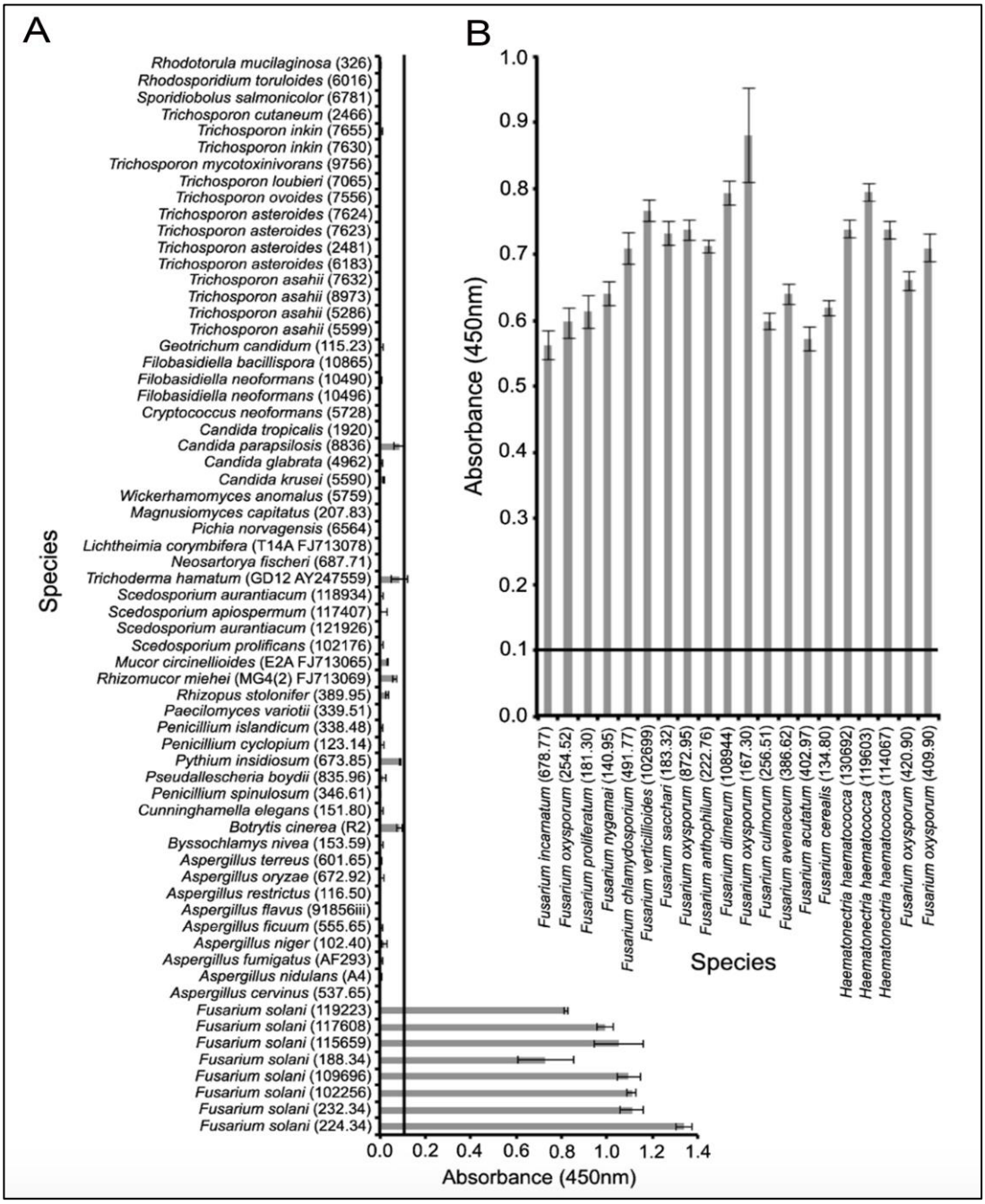


Figure 2.1. Specificity of mAb ED7 determined by ELISA tests of surface washings containing water-soluble antigens from *Fusarium* species and related and unrelated yeasts and moulds. (**A**) ELISA absorbance values at 450 nm for antigens from *F. solani* and unrelated yeasts and moulds and (**B**) for antigens from the *F. solani* teleomorph *Haematonectria haematococca* and related *Fusarium* species. Wells were coated with 60 μg protein ml^{-1} buffer. Bars are the means of three biological replicates ± standard errors and the threshold absorbance value for detection of antigen in ELISA is ≥ 0.100 (indicated by lines on graphs). Numbers in parentheses after species names denote strain numbers with further details of strains provided in Table 2.1.

2.3.2 Western Blotting and Antigen Characterization

Gel electrophoresis and Western blotting studies showed that ED7 binds to a major antigen with a molecular weight of ~200 kDa which is secreted extracellularly by both *F. solani* and *F. oxysporum* (**Figure 2.2**). *F. solani* antigens were subjected to enzymatic (**Figure 2.3 A** and **B**), heat (**Figure 2.3 C**) and chemical (**Figure 2.4**) modifications to characterize the epitope bound by ED7. The lack of reduction in ED7 binding following digestion of immobilized antigen with trypsin (**Figure 2.3 A**) and pronase (**Figure 2.3 B**) shows that it does not bind to a protein epitope. There was no significant decrease in ED7 binding over 70 min of heating, demonstrating that its epitope is heat stable (**Figure 2.3 C**). Reductions in mAb binding following chemical digestion of an antigen with periodate oxidation show that its epitope is carbohydrate and contains vicinal hydroxyl groups (**Figure 2.4**).

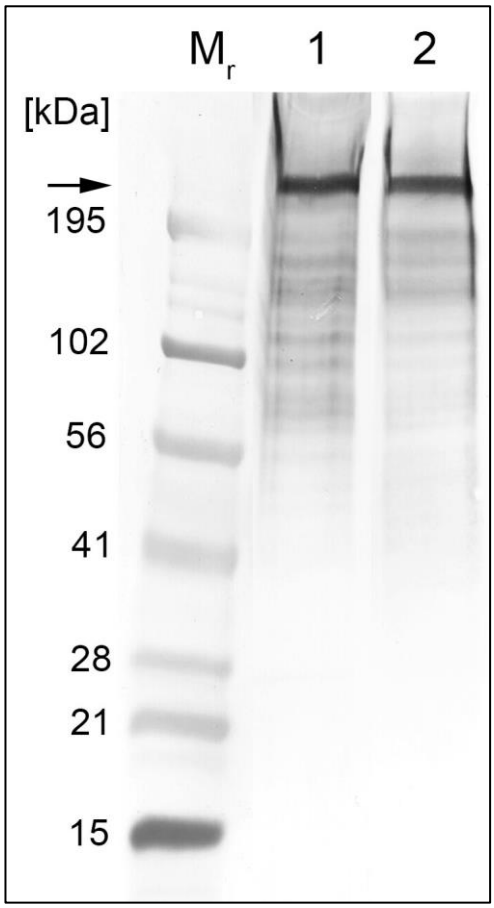


Figure 2. 2. Western immunoblot with ED7 using culture fluid from 2-d-old PDB cultures of *F. solani* (CBS224.34) (**Lane 1**) and *F. oxysporum* f.sp. *lycopersici* (CBS167.30) (**Lane 2**). Wells were loaded with 1.6 µg of protein. M_r denotes molecular weight in kDa. Note the major extracellular antigen with a molecular weight of ~200 kDa.

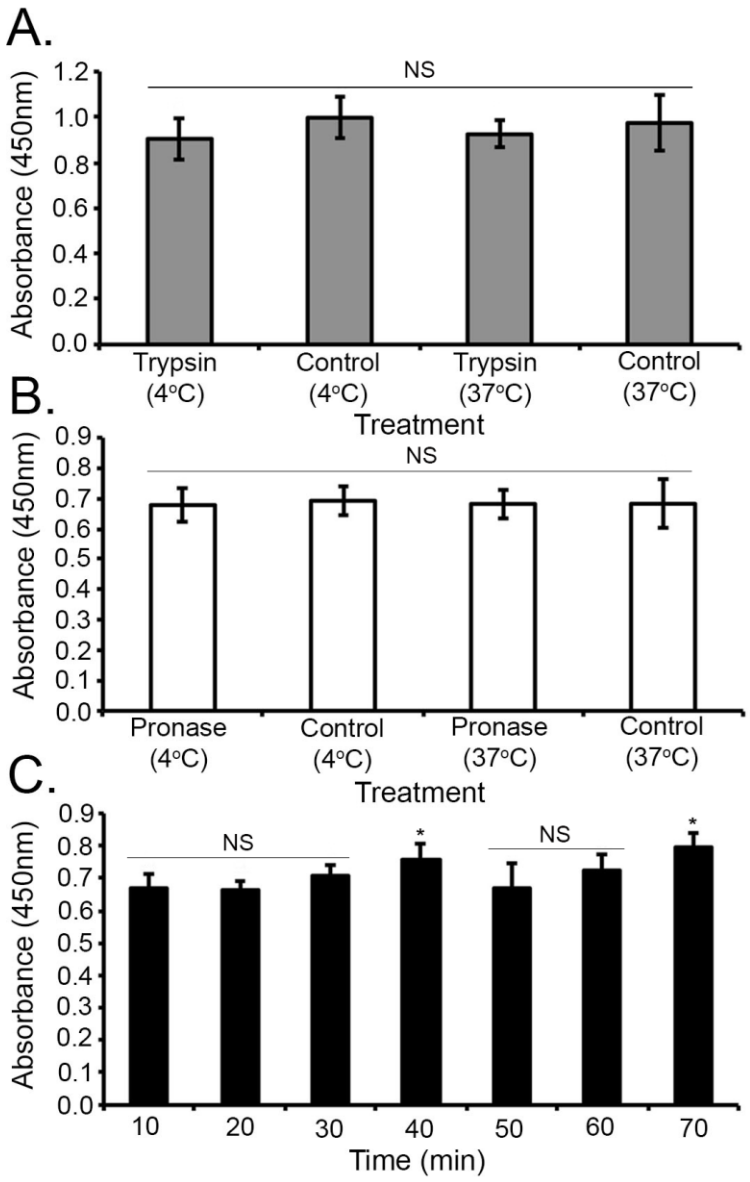


Figure 2. 3. Characterization of antigens bound by mAb ED7. Absorbance values from ELISA tests with ED7 using surface washings containing water-soluble antigens immobilized to the wells of microtitre plates and treated with (**A**): trypsin, (**B**): pronase or PBS only (control) at 4ºC and 37ºC, and (**C**): Stability of the water-soluble ED7 antigen following heating of surface washings at 100 ºC over a 70 min period. Treated antigen was subsequently immobilized to the wells of microtitre plates and assayed by ELISA. Bars are the means of three biological replicates ± SE and bars with the same letter are not significantly different at $p < 0.001$ (ANOVA and Tukey-Kramer test).

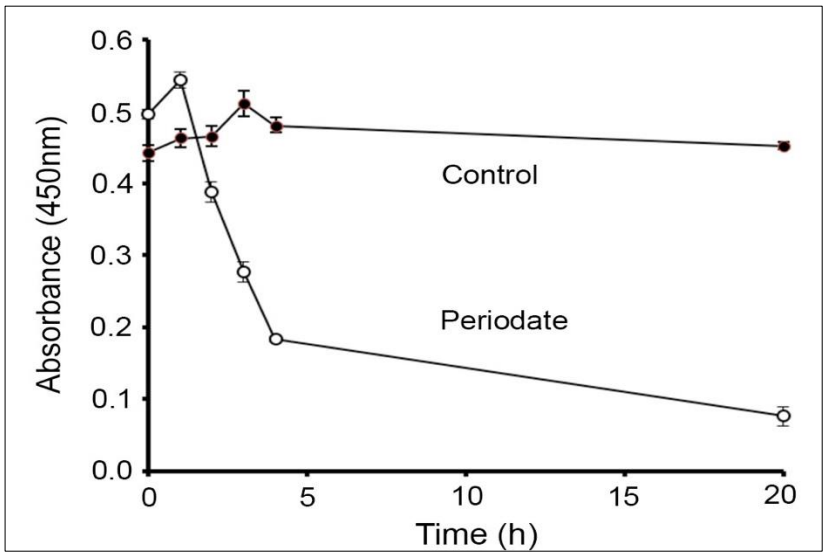


Figure 2. 4. Absorbance values from ELISA tests with ED7 using surface washings containing water-soluble antigens immobilized to the wells of microtitre plates and treated with periodate (open circles) or with acetate only control (closed circles) at 4ºC over a 20 h period. Each point is the mean of three biological replicates ± SE.

2.3.3 Immunofluorescence and Immunogold Electron Microscopy

Immuno-localisation studies using IF showed that the ED7 antigen was present on the surface of spores and hyphae (**Figure 2.5 A to D**), while IEM showed that the antigen was present in the spore and hyphal cell wall in an extracellular matrix surrounding both (**Figure 2.6 A to C**).

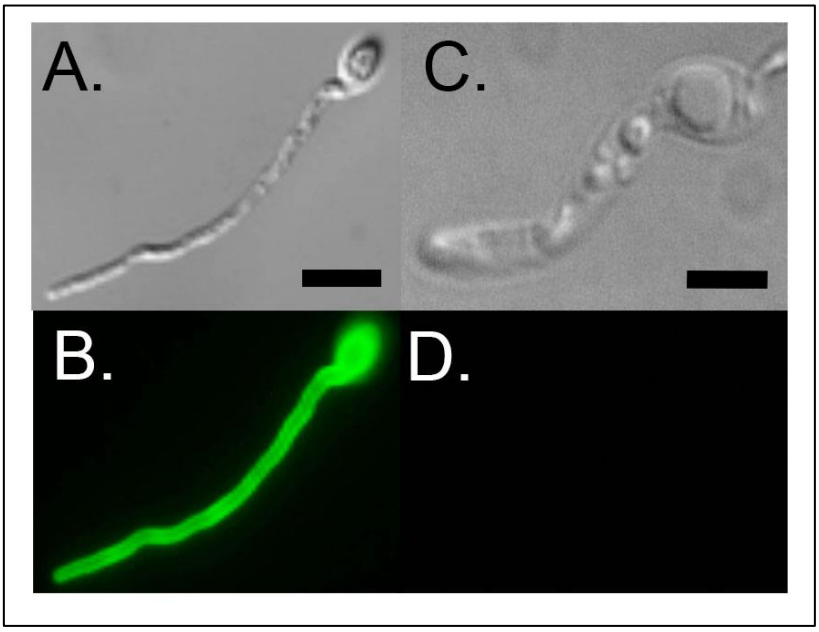


Figure 2. 5. Photomicrographs of *F. solani* (CBS224.34) cells, immunostained with mAb ED (**A, B**) or TCM only (**C**, **D**) and anti-mouse polyvalent Ig fluorescein isothiocyanate. (A) Bright field image of germinated conidium with hypha; (**B**) Same field of view as panel A but examined under epifluorescence. Note the intense staining of the hyphal and conidium cell wall. (**C**) Bright field image of germinated conidium with hypha. (**D**) Same field of view as panel (**C**) but examined under epifluorescence. Scale bar = 8 µm.

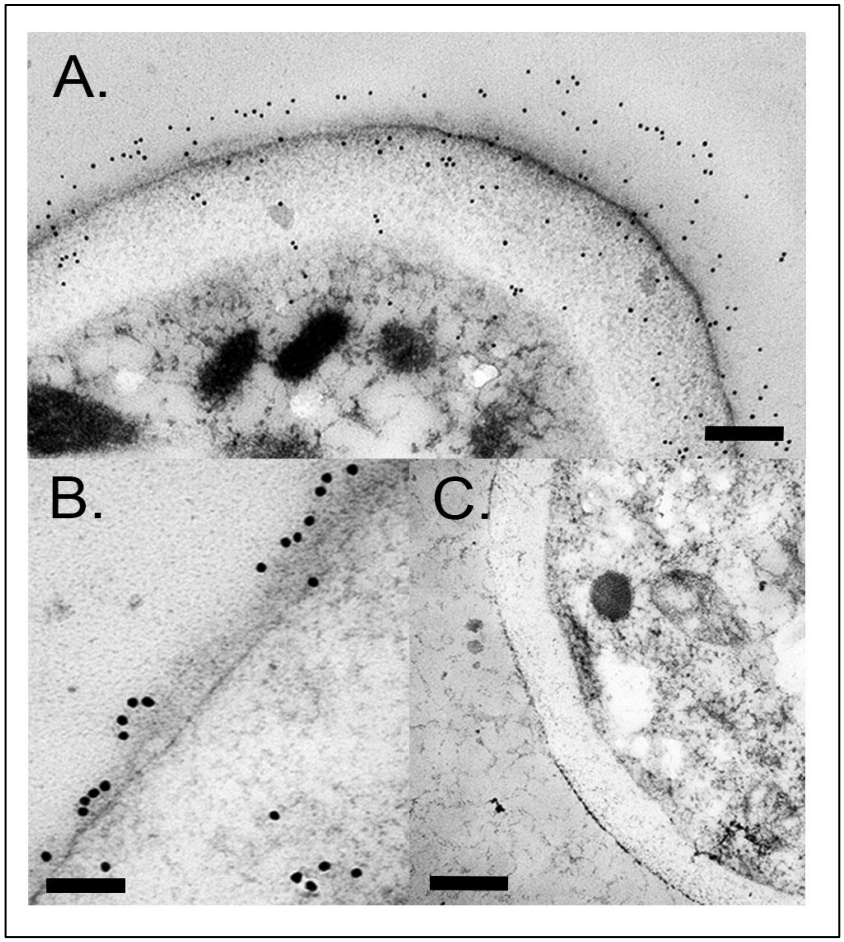


Figure 2. 6. Immunogold labelling of sections of conidia and hyphae of *F. solani* (CBS224.34). (**A**) Transverse section of conidium incubated with ED7 and anti-mouse immunoglobulin 20 nm gold particles, showing antigen in the cell wall in an extracellular fibrial matrix surrounding the spore (Scale bar = 250 nm); (**B**) Longitudinal section of hypha incubated with ED7 and anti-mouse immunoglobulin 20 nm gold particles, showing antigen in the cell wall and in an extracellular matrix surrounding the cell (scale bar = 100 nm); (**C**) Transverse section of a conidium incubated with TCM (negative control) and anti-mouse immunoglobulin 20 nm gold particles, showing a lack of staining by the secondary gold conjugate. Scale bar = 180 nm.

2.3.4 Spore Agglutination Assays

A peak in light absorbance (absorbance value of 0.20) with *F. solani* (CBS224.34) microconidia suspended in 10% TCM was found at 555 nm, while the lowest absorbance (absorbance value of 0.048) was recorded at 340 nm (**Figure 2.7**). Using the wavelength of 555 nm, agglutination of *Fusarium* spores with mAb ED7 was investigated. The result shows that the microconidia of *Fusarium* species suspended in mAb ED7 have two agglutinations and not agglutination trends behaviour as shown in **Table 2.2** and **Appendix 1**. Spores of *F. solani* (CBS224.34) and *F. dimerum* var*. dimerum* (CBS108944) were represented the agglutination trend in mAb ED7 with rapid decreases in absorbance values when compared to the TCM only control (**Figure 2.8** and **Appendix 1**). In contrast, spores of *F. equiseti* (CBS185.34) and *F. solani* (CBS119.223) were represented not agglutinated species by mAb ED7 (**Figure 2.9** and **Appendix 1**).

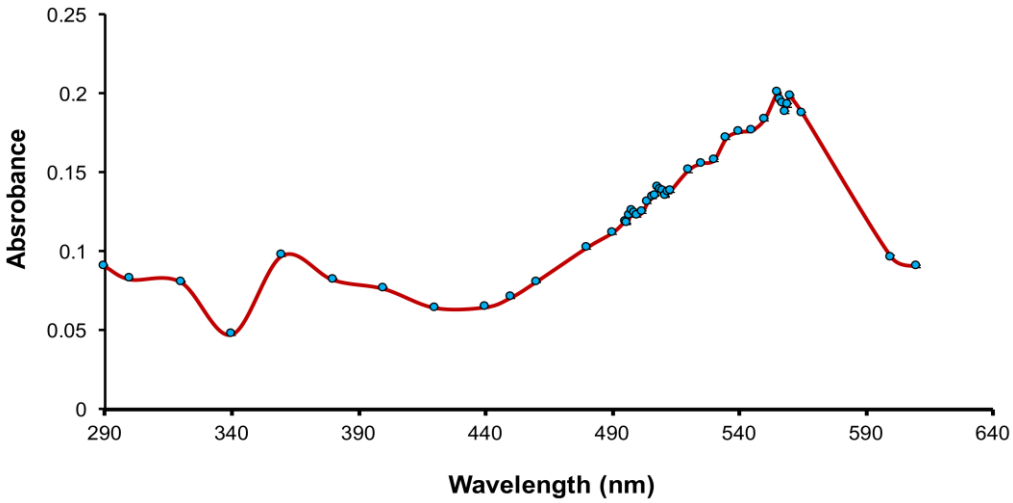


Figure 2. 7. Absorbance of light at different wavelengths by non-agglutinated *F. solani* (CBS224.34) microconidia suspended in 10% TCM at 10^6 spore ml^{-1}. Each point is the mean of 3 replicates ± S.E. Note peak absorbance of spores at 555nm. This wavelength was therefore chosen for subsequent agglutination assays with mAb ED7 and different species, formae speciales and varieties of *Fusarium*.

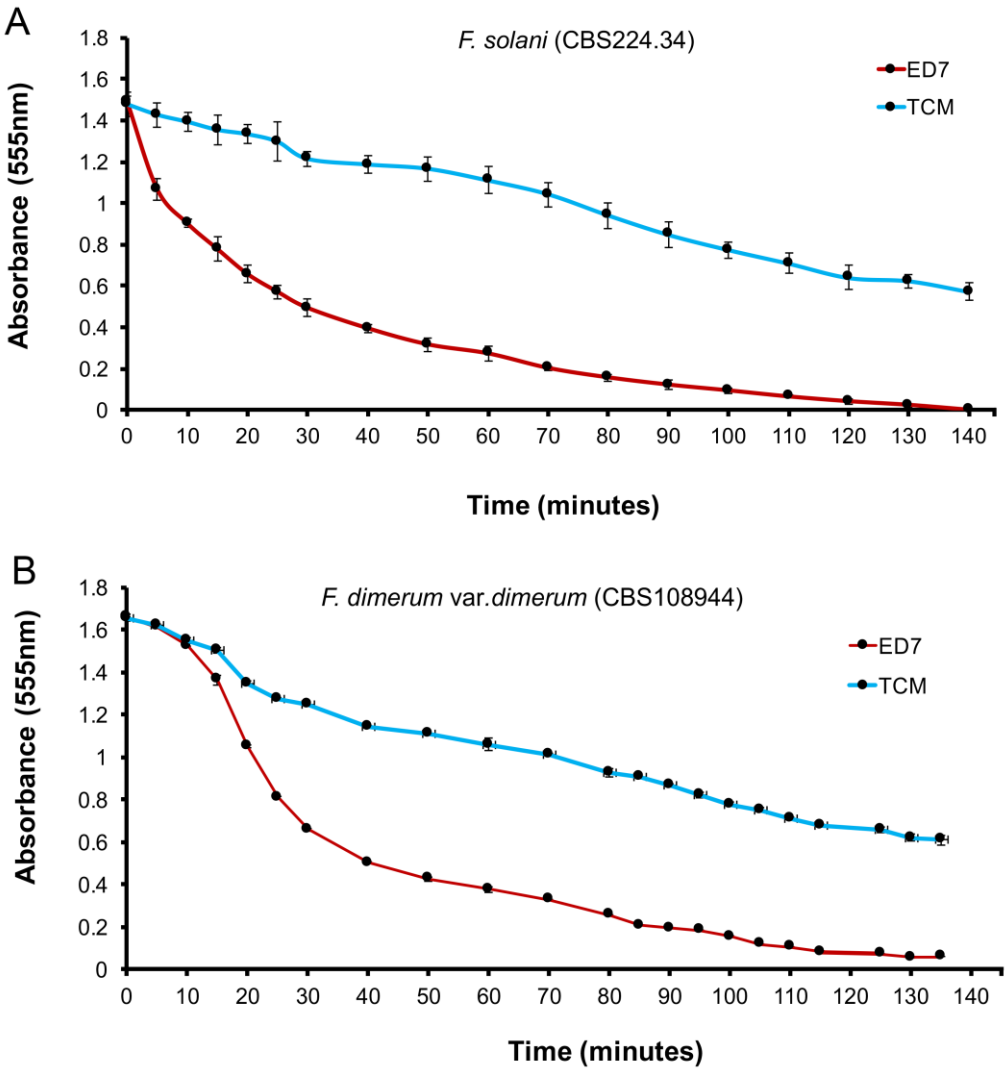


Figure 2. 8. Agglutination of *Fusarium* species microconidia. (**A**) *F. solani* (CBS224.34) and (**B**) *F. dimerum* var. *dimerum* (CBS108944), spores suspended at 10^6 spores/ml in ED7 TCS and 10% TCM. Each point is the mean of 3 replicates ± S.E. Note rapid decrease in absorbance of light at 555 nm due to agglutination of spores by mAb ED7.

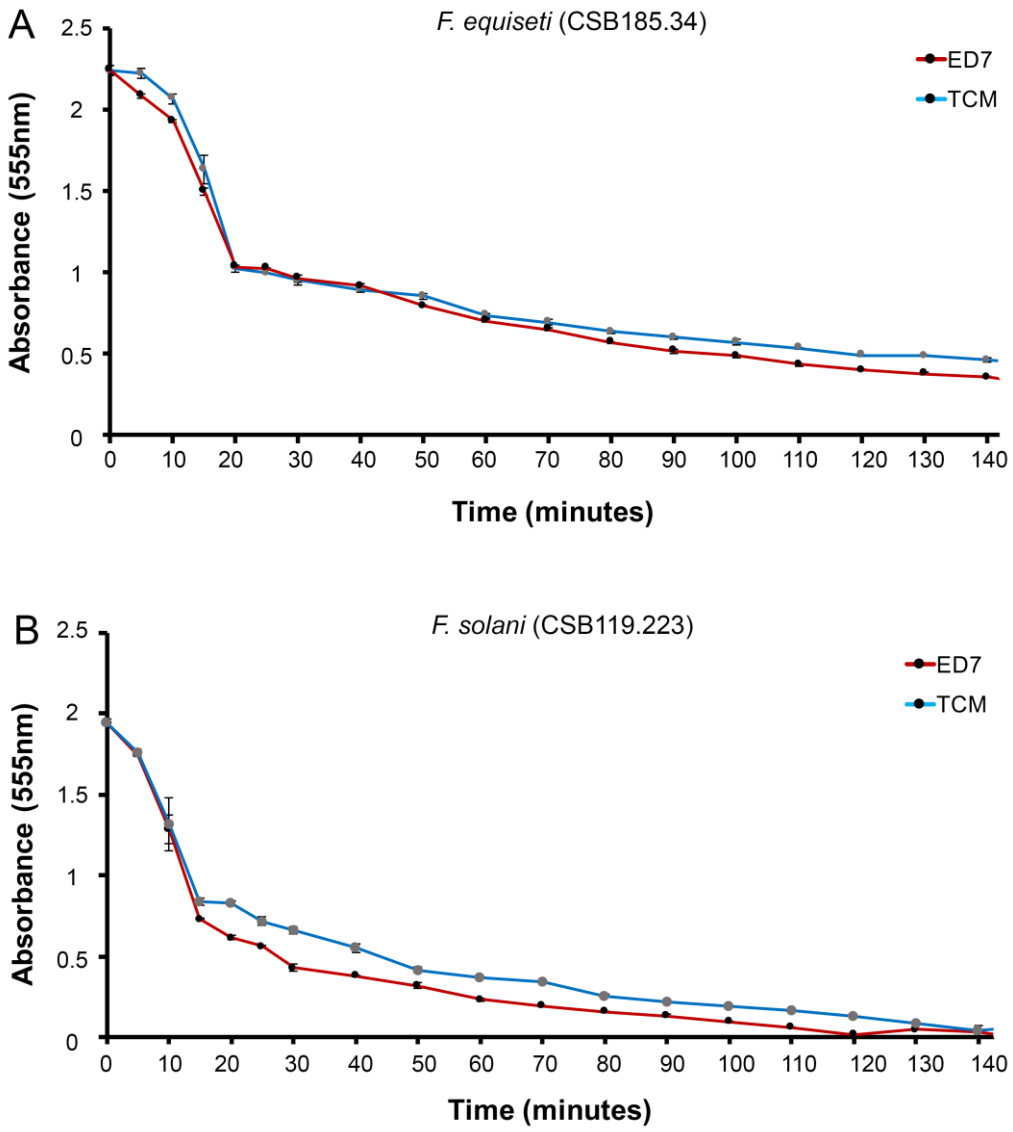


Figure 2. 9. Agglutination of *Fusarium* species microconidia. (**A**) *F. equiseti* (CBS185.34) and (**B**) *F. solani* (CBS119.223) spores in ED7 TCS and 10%TCM. Each point is the mean of 3 replicates ± S.E. Note lack of agglutination of spores of this fungus by mAb ED7.

Table 2.2. Agglutination trend of *Fusarium species* microconidia suspended in mAb ED7 TCS and 10% TCM at 555 nm.

Agglutinated isolate	Weakly agglutinated isolate	Not agglutinated isolate
F. solani (CBS224.34)	*F. proliferatum* var. *proliferatum* (CBS181.30)	*F. chlamydosporium* var. *chlamydosporium* (CBS491.77)
F. dimerum var. *dimerum* (CBS108944)	*F. nygami* (CBS140.93)	*F. equiseti* (CBS185.34)
F. sacchari (CBS183.32)	*F. oxysporum* f.sp. *radicis-lycopersici* (CBS872.95)	*F. solani* (CBS119.223)
F. verticilliodes (CBS102.699)	*F. oxysporum* f.sp. *lycopersici* (CBS167.30)	*F. oxysporum* f.sp. *cucurbitacearum* (CBS254.52)
F. oxysporum var. *vasinfectum* (CBS409.96)	-	*F. incarnatum* (CBS678.77)
F. solani var. *petroliphilum* (CBS102256)	-	*F. solani* (CBS115.659)
F. anthophilum (CBS222.76)	-	-
F. solani (CBS188.34)	-	-
F. solani (CBS117.608)	-	-
F. solani (CBS109.696)	-	-

2.4 Discussion

Monoclonal antibodies are highly specific and can be generated and mass-produced using hybridoma technology. This characteristic feature makes them ideal for several applications, including disease diagnosis, as therapeutic agents and as reagents for a variety of scientific research applications (Li *et al.,* 2010; Modjtahedi et al., 2012; Morgan & Levinsky, 1985).

While mAbs have been developed for detecting and differentiating plant pathogenic *Fusarium* species (Arie *et al.,* 1995, 1991; Banks *et al.,* 1996; Hayashi *et al.,* 1998; Hu *et al.,* 2012, 2013; Wong *et al.,* 1988), few attempts have been made to develop mAbs for the detection of human pathogenic species. Nevertheless, Jensen et al. (2011) recently reported the development of *Fusarium*-specific mAbs for immunohistochemical diagnosis of fusariosis.

The IgM mAbs, which recognise 51 and 63 kDa antigens, reacted strongly with fungal elements in both experimentally infected animals and biopsy samples from patients with fusariosis. The aim of this study was to use hybridoma technology to develop *Fusarium*-specific mAbs for use in tracking human pathogenic species during interactions with cells of the immune system (**Chapter 3**) and during environmental studies (**Chapter 4**). Here, Hybridoma technology was used to generate a mAb against immunogenic antigens from surface washings prepared from *F. solani*, which is one of the most frequent etiologic agents of systemic fusariosis infections in humans.

There were two specific aims required to fulfil this objective. The first was the development of a mAb specific to the genus *Fusarium.* It was important that this mAb lacked cross-reactivity with other related and unrelated pathogenic filamentous fungi and yeasts involved in disseminated mycosis, such as *Aspergillus* (Thornton, 2010a), *Candida* (Marcio Nucci & Marr, 2005), *Scedosporium* (Thornton et al., 2015), *P. boydii* (Thornton, 2009), *Trichosporon* (Davies & Thornton, 2014b), *Rhizopus, Mucor, and Rhizomucor* (Kauffman, 2004).

Because immunogenic antigens such polysaccharides and proteins can be secreted in blood and other body fluids during invasive infections (Thornton, 2008), the second specific aim was to produce a mAb against an extracellular antigen and to investigate its suitability as a diagnostic target for pathogen detection. Using hybridoma technology, murine mAb ED7 was raised against surface antigens from a human pathogenic strain of *F. solani* (strain CBS 224.34).

Using ELISA tests, it was shown that mAb ED7 is genus-specific, reacting with a wide range of species within the genus including the most frequent causes of human fusariosis *F. solani* and *F. oxysporum*. Importantly, it does not cross-react with unrelated opportunistic fungal pathogens, including yeasts and moulds in the genera *Aspergillus* (Thornton & Wills, 2015), *Candida*, *Geotrichum*, *Rhodotorula* and *Trichosporon* (Davies & Thornton, 2014; Miceli *et al.,* 2011), *Cyphellophora* and *Phialophora* (Feng *et al.,* 2014), *Exophiala* (Zeng *et al.,* 2007), *Trichoderma* (Sandoval-Denis *et al.,* 2014), *Engyodontium* (Macêdo *et al.,* 2007; Thamke *et al.,* 2015) and *Mucor* (Petrikkos *et al.,* 2012).

The genus specificity of mAb ED7 and the identification of its target antigen (through heat, chemical and enzymatic modification) as a heat-stable carbohydrate makes it an ideal partnership for tracking pathogenic strains of *Fusarium* in humans and the natural environment. Heat-stable carbohydrate antigens have been used successfully as diagnostic targets for mAb-based detection during human and animal infection (Wiederhold *et al.,* 2013; Rolle *et al.,* 2016) and in environmental monitoring studies (Thornton, 2009; Sharpe *et al.,* 2016).

Immunofluorescence and immunogold electron microscopy showed that the antigen bound by the mAb ED7 is located in the outer cell wall of hyphae and spores in a microfibrillar matrix that surrounds microconidia. The extracellular microfibrillar matrix of *F. solani* spores was first reported in 1975 (Tewari & Skoropad, 1975). It was the first immunological study conducted to investigate a matrix-associated antigen in *Fusarium* species. A putative 90 kDa glycoprotein is thought to be associated with the adhesive properties of macroconidia of the plant pathogen *Haematonectria haematococca* (the teleomorph stage of *F. solani*) (Kwon & Epstein, 1993), but adhesion negative strains (Att) of the fungus also produce the 90 kDa antigen. Furthermore, Att strains produce a conidial tip mucilage, suggesting that the 90 kDa glycoprotein is not the only molecule involved in adhesion (Schuerger & Mitchell, 1993). In a subsequent study, Hayashi and co-workers (Hayashi *et al.,* 1998) showed that a mAb (API9-2), raised against a *F. oxysporum* hyphal antigen, can recognise the antigenic carbohydrate, mannan, with a molecular weight of ~55 kDa. From the characterization work conducted here, I have shown that the antigen bound by mAb ED7 is an immunodominant, heat-stable, carbohydrate. Western blotting

studies demonstrated that the antigen is extracellular and has a molecular weight of ~200 kDa, indicating that the antigen recognised by mAb ED7 may be a novel high molecular weight antigenic structure. Agglutination by the mAb shows that it binds to a previously uncharacterized component on the spore surface and may be associated with the spore agglutination process described previously.

Previous studies have investigated the process of spore adhesion and agglutination prior to germination. The process of spore adhesion occurs naturally prior to spore germination in *F. solani* and is thought to be essential to successful plant infection by the fungus. It has been shown that ungerminated conidia agglutinate during attachment to host roots, before spore mucilage is produced and prior to spore germination. Consequently, adhesion and agglutination allow the macroconidia to come into contact with the host root after which germination and infection occur (Schuerger & Mitchell, 1993). A similar process is believed to occur in *H. haematococca*. Host nutrients stimulate spores to become adhesion competent, allowing macroconidia to adhere and agglutinate at their apices during host tissue attachment (Epstein & Nicholson, 2006; Jones & Epstein, 1990).

The process of mAb ED7 agglutination was investigated with a strain of *F. solani* whose spore production is dominated by microconidia. The agglutinating activity of mAb ED7 was not universal, with certain *Fusarium* species, and even strains of *F. solani*, failing to agglutinate with the antibody. This is consistent with previous studies that have shown that strains of *F. solani*

differ in spore adhesion and agglutination properties (Epstein & Nicholson, 2006).

Similar to other fungal pathogens (Tucker & Talbot, 2001), the initial stages of *Fusarium* infection include spore adhesion, germination, and hyphal network establishment, followed by penetration of the host cells (Stephens *et al*., 2008). The lack of spore adhesion by mAb ED7 in certain isolates of the fungus warrants further investigations of the spatio-temporal expression of the antigen in agglutinating and non-agglutinating strains of the pathogen.

Summary of Key Findings:

1. A single B-cell line, ED7, produces mAb belonging to the IgM class, which is genus-specific, *Fusarium*-specific, reacting with antigens from *Fusarium* species and it does not cross-react with antigens from a wide range of unrelated mould and yeast species.
2. The mAb ED7 binds to an antigen with a molecular weight of ~200 kDa. The antigen epitope is a carbohydrate contains vicinal hydroxyl groups, not protein and a heat.
3. The ED7 antigen is located in an extracellular microfibrillar matrix in the outer cell wall surrounding both the of hyphae and spores.
4. The *Fusarium* species microconidia suspended in mAb ED7 have two agglutinations and non-agglutination trends at wavelength of 555 nm.

Chapter 3. Alveolar Macrophage Phagocytosis and Killing of *F. solani* Expressing Green Fluorescent Protein (GFP)

3.1 Introduction

Fusarium species are emerging as lethal human pathogens and constitute an important cause of morbidity and mortality, particularly in severely immunocompromised patients. The number of systemic *Fusarium* infections in humans has increased since the mid-1970s (Cho *et al*., 1973). This upward trend has coincided with an expanding population of immune impaired patients, including neutropenic patients with haematological malignancies, recipients of haematopoietic stem cell transplants (HSCTs) or SOTs, patients with diabetes, AIDS patients, and those having toxic chemotherapy or radiotherapy treatments (Dignani & Anaissie, 2004; Guarro *et al*., 2000; O'Donnell *et al*., 2007; Walsh *et al*., 2004; Zhang *et al*., 2006). *F. solani* is the most frequently reported species, involved in approximately 50% of fusariosis cases, and followed by *F. oxysporum* and *F. verticillioides* (Jain et al., 2011; Marcio Nucci & Anaissie, 2007).

Fusarium infections can be superficial, local or disseminated (Marcio Nucci & Anaissie, 2002). In addition to producing allergens and many types of mycotoxins (that cause respiratory infections and organ damage) (Li *et al*., 2011), deaths due to invasive fusariosis are being increasingly reported due, in part, to inherent resistance to many of the antifungal drugs that are currently available for clinical treatment (Azor *et al*., 2007; Tortorano *et al*., 2014).

Diagnosis of disseminated fusariosis is also a big challenge. Morphological identification using traditional microscopic and cultural base methods can lead to misdiagnosis, especially in the absence of macroconidia that are characteristic of some *Fusarium* species. These methods are time-consuming and required extensive knowledge of mycology (Alexander & Pfaller, 2006). Additionally, recent studies have shown serodiagnostic tests, such as GM and BDG tests, are not especially effective for diagnosing *Fusarium* infections (Esnakula *et al*., 2013).

3.1.1 Immunity to Fungal Infections

The skin is the first physical barrier of the innate immune system and is critical in isolating the sterile internal tissues from the external environment. This physical barrier prevents fungal invasion and provides an initial immunity to fungi such as *Fusarium*. Fungal propagules have to adhere and penetrate epithelial tissue under the skin or mucous membranes, to develop into deep-seated infections and that can lead to tissue damage (Williams *et al*., 2012). As a result of tissue damage, broad-spectrum antimicrobial peptides (AMPs), such as defensins, cathelicidins and collectins, are secreted by epithelial cells to eliminate fungal infections. AMPs play an integral role in host defence by destroying fungal cells directly through binding to the cell membrane or cell wall carbohydrates, or indirectly via activation and recruitment of innate immune phagocytic cells (Kolar *et al*., 2013; McDermott, 2009). Cellular innate immunity includes residential macrophages and circulated neutrophils. These cells are involved in the innate immune host defence and are considered a core immune response to fungal invasions (Bain *et al*., 2014; Grazziutti *et al*., 2005). PRRs on

phagocytic cells are able to recognise effectively a variety of polysaccharide antigens found on the fungal cell wall. These antigens are known as PAMPs. Common examples include β-glucan and mannan (Figueiredo *et al*., 2011; McCann *et al.*, 2005). After antigen recognition, AMØ are associated with the clearance of *Fusarium* propagules via phagocytosis (Brakhage *et al.*, 2010; Roilides *et al.*, 2006; Walsh & Groll, 1999). Macrophages and neutrophils can damage *Fusarium* hyphae by producing Interferon gamma (IFNγ), granulocyte-colony stimulating factor (G-CSF) and granulocyte-macrophage-colony-stimulating factor (GM-CSF) (Gaviria *et al*., 1999).

TLRs expressed on the surface of phagocytic cells bind to β-glucan and mannose, and play a crucial role in the recognition of *Fusarium* cell wall components. These receptors mediate the phagocytosis process and stimulate the adaptive immune response (Gordon & Read, 2002; Guo *et al*., 2008; Jin *et al*., 2007; Shoham & Levitz, 2005; Tarabishy *et al*., 2008). Furthermore, these receptors cooperate in a complex network to activate the production of cytokines and chemokines, including tumour necrosis factor (TNF), interleukins (IL-1α, IL-1β, IL-6) and macrophage inflammatory proteins (MIP-2 and 1) to attract other innate phagocytic cells to the site of invasion (Abbas *et al*., 2007). Recent studies have shown a novel way of neutrophil-mediated killing of pathogenic bacteria, via the release of extracellular networks (NETs) (Brinkmann *et al*., 2004). Since then, subsequent studies have revealed the effector role of NETs in trapping pathogenic fungi and eliminating infections, such as *Aspergillus fumigatus* (Bruns *et al*., 2010), and *Candida albicans* (Branzk *et al*., 2014) infections. This unique phenomenon has also been reported in macrophages as an initial process for host defence, resulting in

microorganism clearance and destruction via the release of macrophage extracellular traps (METs). MET formation is induced by microbial cell invasion, such as by *Mannheimia haemolytica* (Aulik *et al.*, 2012; Hellenbrand *et al*., 2013) and *Candida albicans* (Liu *et al*., 2014). The MET is a structure of nuclear DNA fibres supported with active proteins. This trap has fungistatic and fungicidal affectivity to prevents fungal dissemination, and may recruit other phagocytic cells to the infection site (Aulik *et al*., 2012, Guimarães-Costa *et al*., 2012, Brakhage *et al*., 2010).

In healthy individuals, innate immune responses can detect and clear most fungal invaders within a short time (Blanco & Garcia, 2008). However, disseminated *Fusarium* infections often occur in immunocompromised patients and these patients have defective or completely lack an innate immune response, often due to having immunosuppressive therapy (Abbas *et al.,* 2007; Jain *et al.,* 2011; Nucci & Anaissie, 2007; Roilides *et al.,* 2006).

3.1.2 GFP Transformation

GFP isolated from the luminescent jellyfish, *Aequorea victoria*, absorbs blue light, and then re-emits it as green fluorescence. This characteristic feature has been used to monitor microbial cells via gene expression, without affecting natural microbial behaviour (Chalfie *et al.,* 1994). This technique has also been used as a reporter for macromolecular localization in bacterial cells (Margolin, 2000). Furthermore, a synthetic GFP method has been successfully used as a reporter gene expression tool, for transformant cell isolation and cellular gene expression quantification. This has allowed the study of host-fungal pathogen

interactions and the assessment fungal or yeast biomass (Cormack *et al.,* 1997; Lorang *et al.,* 2001). In addition, GFP techniques have been used for yeast cell quantitative analysis (Barelle *et al.,* 2004) and have been applied to many filamentous fungi for monitoring growth, behaviour in different environments, and to study fungal pathogenicity and assess antifungal activity (Bae & Knudsen, 2000; Knudsen & Dandurand, 2014; Spellig *et al.,* 1996).

Studies have shown the application of GFP techniques to quantitatively investigate fungi and follow the plant infection processes of a number of *Fusarium* plant pathogens, including *F. oxysporum* (Inoue *et al.,* 2002; Lagopodi *et al.,* 2002; Zhang *et al.,* 2013), *F. equiseti* (Maciá-Vicente *et al.,* 2009), *F. graminearum* (Skadsen & Hohn, 2004) and *F. verticillioides* (Oren *et al.,* 2003). Additionally, transformation with GFP is considered to be an appropriate tool for studying the phagocytic cell response of the cellular innate immune system against fungal pathogen invasion *in vitro*. Expression of GFP facilitates fungal propagule visualisation, allowing the assessment of the efficiency of pathogenic fungal cell internalisation and clearance. This method has been used for both *Aspergillus* (Wasylnka & Moore, 2002) and *Candida* (Katragkou *et al.,* 2010) species. To the best of my knowledge, no studies have been undertaken to transform *F. solani* with GFP or to use it to track *F. solani* during phagocytosis by AMØ.

3.1.3 Aims and objectives

Presently, very little is known about the cellular innate immune response to human pathogenic *Fusarium* species, particularly *F. solani*. AMØs and the

phagocytic processes of engulfment and destruction are important topics that are under-studied (Gaviria *et al.,* 1999a; Schäfer *et al.,* 2014). Recognition of PAMPs present in the pathogen cell wall, via PRRs on the phagocytic cell membrane, is the initial step for engulfment and pathogen clearance (Brown, 2011; Luigina Romani, 2011). Some studies have been conducted to identify immunodominant epitopes on surface antigens of pathogenic fungi and to follow antigen production during the activation of the phagocytic cells of the innate immune system (Figueiredo *et al.,* 2011; Karthikeyan *et al.,* 2011; Tarabishy *et al.,* 2008). Identification of the molecular form of the antigenic structures of the fungal spore cell wall is incompletely defined. Knowledge of these immunodominant molecules is required to understand their role in inducing pro-inflammatory cytokine and chemokine responses through host cell receptor binding. Identification and characterization of fungal cell wall antigenic epitopes that evoke immune response might lead to their use as vaccines to immunise human patients against fungal infections (Levitz *et al.,* 2014). They may also drive novel therapeutic approaches to prevent or control opportunistic fungal infections, such as *F. solani*.

The overall aim of the work in this chapter was to investigate the innate immune response to *F. solani* spores by studying phagocytosis of conidia by mouse AMØs. No studies examining the phagocytic clearance of *F. solani* by AMØs have been conducted and there is an apparent need to evaluate innate immune cell responses to this emerging opportunistic pathogen since it is the most frequent *Fusarium* species causing life-threating disease among immunocompromised patients. To achieve this aim, the following objectives were identified:

1.Generation of a *F. solani* strain expressing GFP to enable the interaction of spores with AMØ to be visualised; To develop a live-cell imaging system, to allow the phagocytosis of *F. solani* spores by AMØ to be investigated in real-time; To determine the effect of spore agglutination by mAb ED7 in the phagocytic process.Current techniques to quantify fungal biomass, based on measurements of metabolic activity (Wallander *et al.,* 2013), do not allow for discrimination between the metabolic activities of the fungus and of the engulfing phagocytes. Consequently, fungus-specific techniques need to be developed to monitor the effects of phagocytosis on fungal proliferation. Here, a murine AMØ method was developed that allows the monitoring and quantification of *F*. *solani* germination and hyphal development during phagocytosis in the presence and absence or the spore-agglutinating mAb ED7. This novel approach allowed the quantitative determination of phagocytosis of the fungus under these conditions.

3.2 Materials and Methods

3.2.1 Fungal Transformation with GFP

3.2.1.1 Protoplast preparation

F. solani (CBS224.34) conidia were harvested from 7-day old PDA plates after flooding with 5 ml SMQ-H_2O, conidia suspension was filtered through Miracloth then washed three times with SMQ-H_2O. Washed conidia were suspended in 20 ml of PDB media and incubated in Petri dishes at a concentration of 10^6 spore ml^{-1}. Plates were incubated at for 48 h at 27°C, the mycelium collected by filtering through sterile Miracloth (Calbiochem), rinsed with SMQ-H_2O and dried with paper towel.

Fungal protoplasts were prepared by re-suspending 0.3 g of fresh and washed mycelium in filter-sterilized enzyme solution (1.2 mg chitinase (Sigma), 7 mg lyticase (Sigma), 44 mg cellulase (Sigma), in 2.4 ml of mannitol osmoticum (5 ml 1M $CaCl_2$, 9.11 g mannitol, 1.06 g MES [2-(N- Morpholino) ethanesulfonic acid hydrate], 95 ml dH_2O, pH adjusted to 5.5 with KOH) in a 15-ml Falcon tube (Becton Dickinson). The suspensions were mixed by shaking at 225 rpm for 25 min on a flat-bed shaker (New Brunswick Scientific, USA) at RT. Protoplast suspensions were filtered through sterile Miracloth, washed with 5 ml of sterile osmotic buffer, and then centrifuged at 3,044 *g* for 5 min at 4°C. The protoplast pellets were washed twice by centrifugation and re-suspension and were finally suspended in 300 µl of sterile osmoticum. The protoplast numbers were determined by using a haemocytometer.

3.2.1.2 Fungal Transformation

For *F. solani* (CBS224.34) protoplast transformation, 300 µl of washed protoplasts, at a concentration 10^7 ml^{-1}, were mixed with 6 µg of the DNA plasmid that containing a modified hygromycin resistance gene under the control of the *Aspergillus nidulans TRPC* promoter together with the *SGFP* gene under the control of the *Pyrenophora tritici-repentis TOXA* gene promoter was isolated from pCT74 (was provided kindly by N. Talbot group – Bioscience, the University of Exeter, UK) previously suspended in 34 µl of SMQ (**Figure 3.1**). As a negative control, the same concentration of protoplasts was re-suspended in osmotic buffer only. Both suspensions were incubated on ice for 20 min, and then each tube was treated with 130 µl of polyethylene glycol solution (40% PEG 8000 in osmoticum). Next, the tubes were mixed gently by inversion, followed by incubation for 30 min at RT. The treated protoplasts were mixed gently with cell wall regeneration medium containing molten PDA (42°C) and 2.2 µg/ml of hygromycin B (Caliobiochem) as a selective agent, and 0.8% (w/v) sucrose), poured into five Petri dishes, and then incubated for 24 h at 26°C in the dark. Putative *Fusarium* transformants were selected by adding a thin overlay of molten PDA, containing hygromycin B at the same concentration 2.2 µg/ml, on the top of the plates. Single spore isolates of putative transformants were produced on PDA-hygromycin plates and transferred to PDA slopes for long-term storage. Expression of the GFP protein in putative transformants (hereafter referred to as FS-GFP transformants) was determined by examining tissue sections under epifluorescence by using an Olympus microscope (IX81, Visitron System, GmbH), fitted with a 365 nm UV excitation filter and a GFP absorption filter. Bright-field and fluorescence images

were captured using a Photometrics Cool SNAP HQ2 camera (Roper Scientific, Germany).

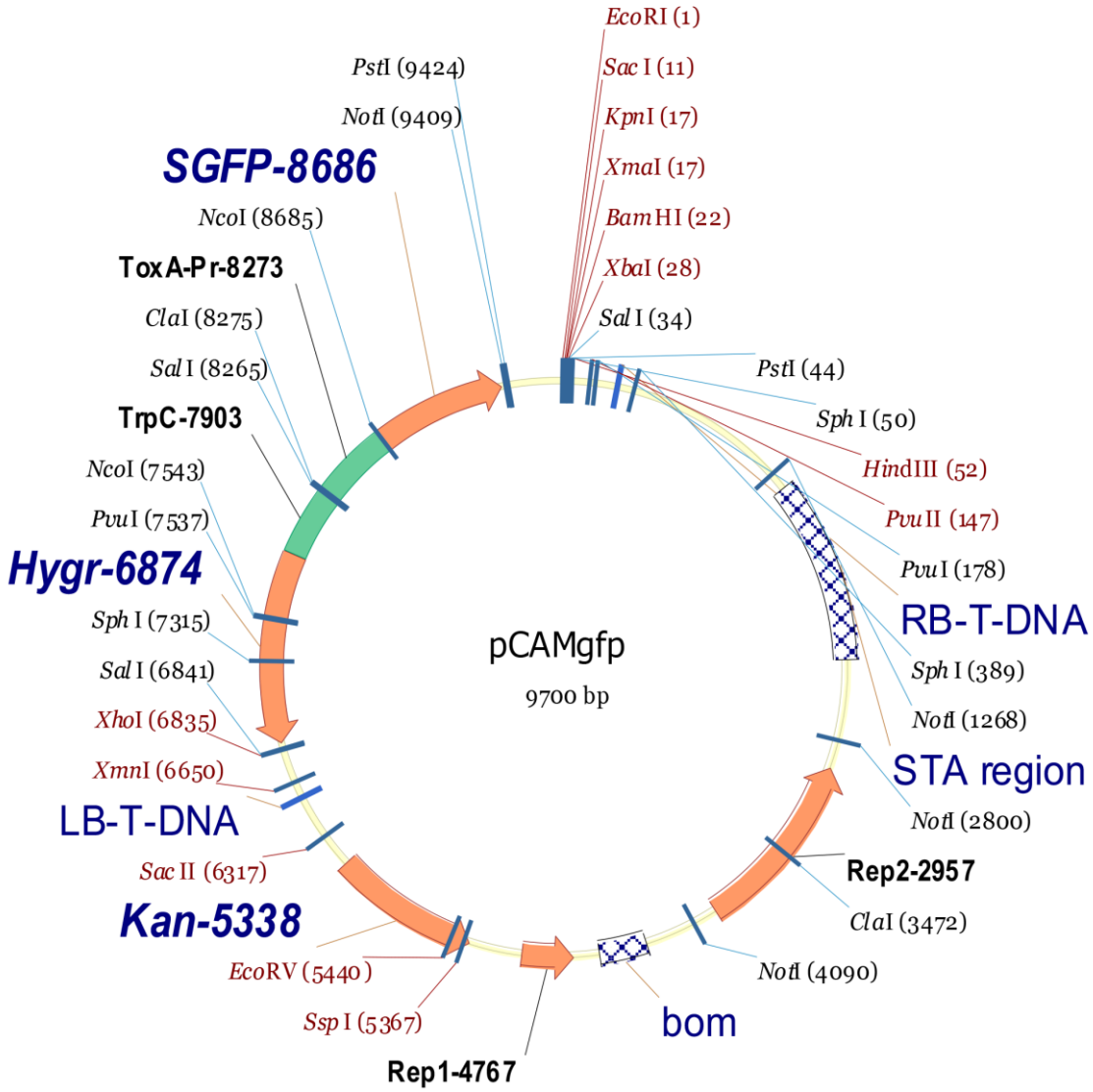


Figure 3.1. Schema of the vector hygro-GFP construction. (Sesma & Osbourn, 2004)

3.2.1.3 Determination of GFP protein expression by Enzyme-Linked Immunosorbent Assay

Expression of the GFP protein in fluorescent-positive FS-GFP transformants was further established by confirmatory ELISA, using the ELISA protocol essentially as described previously (**Section 2.2.5**). Wells were blocked for 10 min with 100 µl of blocking solution (1% (w/v) Bovine Serum Albumin (BSA; Sigma, A-7030) in PBS, and then incubated for 1 h with 50 µl of the anti-GFP mAb class IgG (Ref-11814460001; Roche, Germany) diluted 1:1 at a final concentration 0.02 mg/ml in PBS with Tween 20 (PBST; PBS containing 0.05% (v/v) Tween-20 (polyoxyethylene-sorbitan monolaurate) Sigma; P7949)). The goat anti-mouse polyvalent (Ig classes IgG, IgA, and IgM) peroxidase conjugate (A-0412; Sigma Chemical Company, Poole, United Kingdom) was used as secondary antibody. Control wells were incubated with PBST only.

3.2.2 Morphological studies

3.2.2.1 Hyphal Growth and Spore Production

The hyphal growth of wild-type *F. solani* (FS) and FS-GFP transformants was determined by inoculating replicate PDA plates centrally with 5-mm agar plugs of inoculum taken from the leading edge of 5-day old fungal cultures. Plates were incubated at 27°C, and colony diameters were measured every 4 days for 12 days. For spore counts, wild-type and GFP transformants plate cultures were flooded with 5 ml SMQ H_2O and spore concentration determined by using haemocytometer at dilution factor 1:500 after 1, 4, 8 and 12 days.

3.2.2.2 Quantification of Hyphal Biomass, Antigen Production and Spore Agglutination with mAb ED7

Hyphal biomass and antigen production were quantified on the abundance of fungal propagules for the wild type, *F. solani* (CBS224.34), and FS-GFP transformants isolate. Longer-term (21 days) growth experiments were constituted by inoculating three replicates of 250 ml flasks containing 100 ml PDB with 1 X 10^3 spore ml^{-1} of the isolate. Flasks were incubated with shaking (New Brunswick Scientific, USA) at 125 rpm for 21 days at 27°C. Culture filtrates and hyphal biomass were collected every 3 days by straining the contents of the flasks through a sterile Miracloth and transferred into fresh Falcon tubes. The dry weights of hyphal biomass were determined in milligramme (mg) using scale (Mettler Toledo AB54-5/Facts, Switzerland) after drying at 70°C for 16 h. Culture filtrates containing soluble antigens were stored at -20°C until used to perform soluble antigens production using ELISA as described in (**Section 2.2.5**). For testing spore agglutination, the protocol followed was previously described in (**Section 2.2.9**).

3.2.3 Live Cell Imaging of Spore Agglutination

F. solani spores were harvested and washed as described previously. One hundred-µl of either mAb ED7 (spore agglutinating) TCS, were mixed with 6 µl of washed spore suspension at a concentration of 10^6 spore ml^{-1}. Control samples received 10% TCM only. The agglutination process was captured with an Olympus microscope (IX81, Visitron System, GmbH) and images were recorded every 10 sec for 35 min using a Photometrics CoolSNAP HQ2 camera (Roper Scientific, Germany). Image sequences were combined using the

Metamorph software package (MDS Analytical Technologies, Winnersh, UK). Both agglutinated and unagglutinated spores were counted manually in 5 independent fields of view with three replicates for each mAb ED7 and TCM the control, and the percentage of non-agglutinated spores (S%) were plotted as bar charts after 30 mins using this formula:

S% = (Number of Single Spores / Total Number of Spore) X 100

3.2.4 Murine Alveolar Macrophage cell line MH-S

3.2.4.1 Details and Culture of Mouse Alveolar Macrophages

The mouse AMØ cell line MH-S (ATCC CRL-2019) used in this study was developed from mouse lung tissues (Mbawuike & Herscowitz, 1989). The AMØ cell line was derived by SV40 transformation of an adherent cell-enriched population of mouse AMØ and retains properties of AMØ including phagocytosis and typical macrophage morphology. The cell line is esterase positive and peroxidase negative, Fc receptor positive, expresses the complement receptor CD11b (Mac-1), Class II antigens (I-A) and T antigen, and produces the pro-inflammatory cytokine interleukin-1.

The AMØ cell line was cultured for 48 h in 25 cm^2 tissue culture flasks, containing 10 ml of MH-S tissue culture medium (MH-S TCM; RPMI-1640 medium, 10%(v/v) heat-inactivated fetal bovine serum (ΔFBS; Biosera), 2 mM L-glutamine, 0.05 mM 2-mercaptoethanol, and the antibiotics penicillium and streptomycin, which was prepared by dissolving 0.3 g penicillin-G (Duchefa Biochemie, P0142) and 0.5 g streptomycin sulphate (Melford, S0148) in 5 ml

MQ-H_2O, followed by filter sterilization through a 0.2 µm filter disc. The cells were incubated in a CO_2 incubator (San

3.2.4.2 Quantification of Phagocytic Percentage and Phagocytic Index of Engulfed Spores

Spores of FS-GFP transformants were harvested from 5-day old PDA Petri-dish cultures by flooding the plates with 5 ml of sterile SMQ-H_2O and stroking the surface with a sterile L-shaped spreader. Next, suspensions were filtered through sterile Miracloth (Calbiochem) to remove fragments of mycelium, and the spores were pelleted by centrifugation at 14,489 *g* for 5 min. Spores were washed twice with sterile SMQ-H_2O and re-suspended in 1 ml of MHS TCM.

AMØ cell concentration was determined with a hemocytometer. Cells were diluted to 5 x 10^5 ml^{-1} in MH-S TCM-ΔFBS and inoculated into 8-well culture µ-Slides (ibidi, 80826, Germany) for use in microscopy. Slide cultures were incubated for 24 h at 37°C and 5% CO_2. Cells received fresh 10% MH-S TCM-ΔFBS containing washed FS-GFP transformant spores at a final concentration of 10^6 spore ml^{-1}. Inoculated slides were incubated for 2, 12 or 24 h, after which wells were washed 3 times with sterile, warmed PBS, to remove any un-engulfed spores. Phagocytosis of FS-GFP transformant spores was quantified by counting 100 AMØ cells in each of five random fields of view (x100 magnification), using an inverted an Olympus epifluorescence microscope (IX81, Visitron System, GmbH), fitted with a 365 nm UV excitation filter and a 420 nm GFP absorption filter.

The phagocytosis percentage (P%) and phagocytosis index (Pi) were then determined for each treatment, according to the following formula, where:

P% is the percentage of cells containing spores, calculated according to the formula:

P% = [(B+C+D+E+F) / (A+B+C+D+E+F)] x 100, where:

A: is the number of cells with no spores.
B: is the number of cells with 1 spore.
C: is the number of cells with 2 spores.
D: is the number of cells with 3 spores.
E: is the number of cells with 4 spores.
F: is the number of cells with >5 spores.

Pi is the average number of phagocytosed spores per macrophage, calculated according to the formula:

Pi = (B+2C+3D+4E+5F) / (B+C+D+E+F), where:

A: is the number of cells with no spores.
B: is the number of cells with 1 spore.
C: is the number of cells with 2 spores.
D: is the number of cells with 3 spores.
E: is the number of cells with 4 spores.
F: is the number of cells with >5 spores.

Spores that remained attached to AMØ cells after 3x PBS washes, but had been 50% ingested, were considered to have been phagocytosed (Gill-Lamaignere *et al.,* 2002).

3.2.5 Live Cell Imaging of Spore Phagocytosis

A model for live cell imaging of FS-GFP spore phagocytosis by AMØ and evaluation of P% and Pi following agglutination with mAb ED7 was established using 8-well culture µ-Slides (ibidi, 80826, Germany) as shown in **Figure 3.2**. The slides were modified by creating cross channels between well numbers 1 and 2 and 3 and 4 using a heated 1.25 mm syringe needle. This allowed media connectivity between the wells. Wells number 1 and 3 were inoculated with fresh cultures of AMØ cells in MH-S TCM-ΔFBS at a concentration of 10^4 cells ml^{-1}. Washed spores of the FS-GFP transformant were re-suspended in MH-S TCM-ΔFBS, and the concentration was adjusted to 10^4 spore ml^{-1} prior to phagocyte challenge. A confocal microscope (Zeiss LSM 510 META), fitted with an environmental chamber heated to 37°C and 5% CO_2 and 488 nm excitation/ emission filters, was used for live cell imaging of spore phagocytosis. MH-S TCM-ΔFBS bathing the AMØ cells in wells 1 and 3 was aspirated and replaced with the MH-S TCM spore suspension. Well numbers 2 and 4 were then filled with mAb ED7 TCS or TCM-ΔFBS only, respectively. Images of spore agglutination and phagocytosis by the AMØ cells were then captured every 60 sec, for a total of 8 h, with DIC optics (Plan-Apochromat 63x/1.40 Oil DIC M27), and 488 nm laser excitation.

3.2.6 Statistical analysis

The data was analysed using the statistical programme Minitab (Minitab Express, Minitab®, Coventry, UK). Statistical differences between treatments and controls were established by t-test or means of one-way analysis of variance (ANOVA). Post hoc Tukey-Kramer analysis was then performed to

distinguish which groups were significantly different from one another. Results of the test had levels of significance indicate: NS = non-significant, and significant when *p*-values were * < 0.05, ** < 0.01, *** < 0.001.

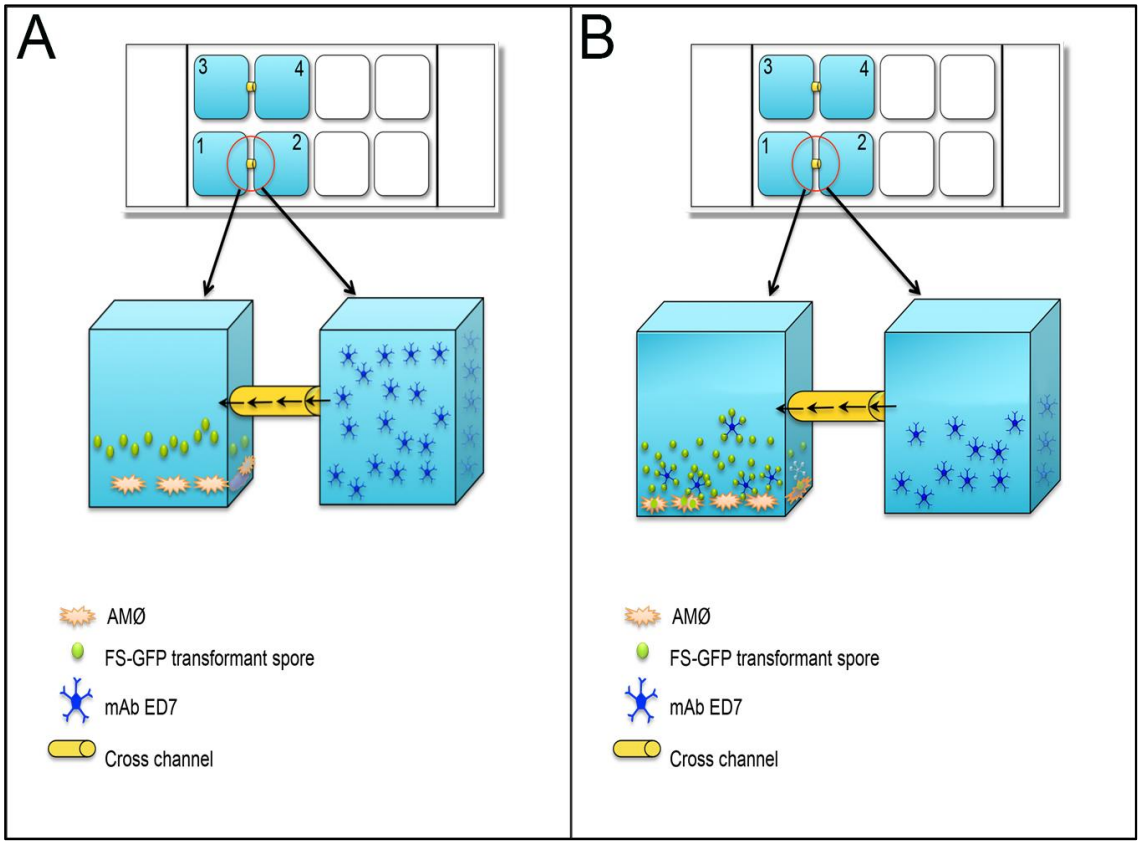


Figure 3.2. Schematic diagram showing the experimental system developed for real-time imaging of phagocytosis of FS-GFP spores during agglutination with the Fusarium-specific mAb ED7. Adherent AMØ cells in wells 1 and 3 were challenged with FS-GFP spores, while ED TCS or TCM-ΔFBS only was placed in wells 2 and 4 respectively. Antibody permeating through the cross channel into well 1 allowed the effect of spore agglutination on AMØ phagocytosis to be quantified by calculating P% and Pi of fluorescent spores.

3.3 Results

3.3.1 Transformation of *F. solani* (CBS224.34) with GFP

F. solani (CBS224.34) produced a typical spherical protoplast as shown in **Figure 3.3**. Putative FS-GFP transformants appeared as white colonies under hygromycin selection, compared to no growth of wild-type control protoplasts. Under epifluorescence, putative FS-GFP transformants had green fluorescent spores and hyphae (**Figure 3.4 A to D**), when compared to the untransformed wild-type strain, which showed no fluorescence (**Figure 3.4 E and F**). Stable FS-GFP transformants were obtained following single spore isolation and repeated sub-culturing onto PDA without hygromycin.

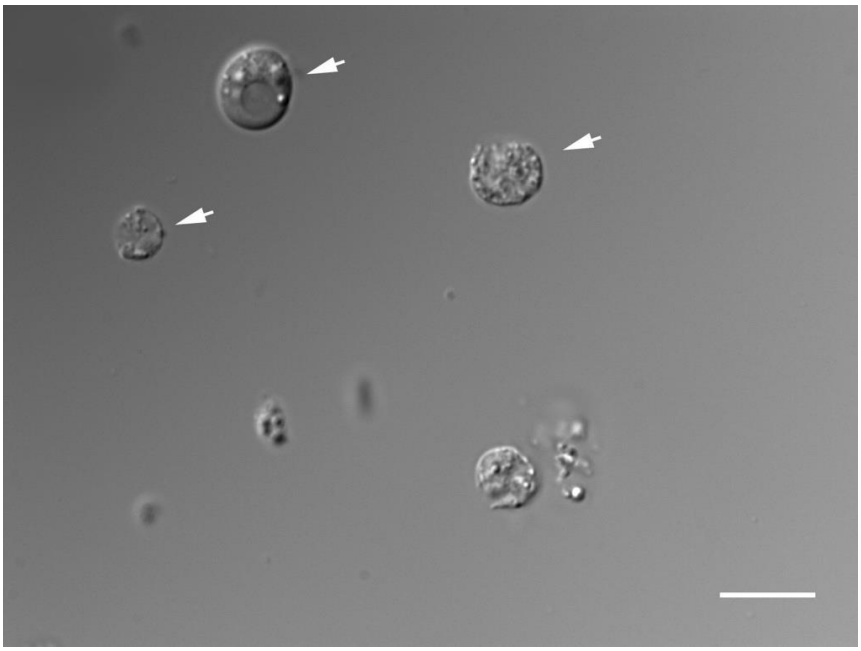


Figure 3.3. Selection of hygromycin-resistant FS-GFP strains following the transformation of *F. solani* protoplasts. Typical *F. solani* protoplast. Scale bars = 10 µm.

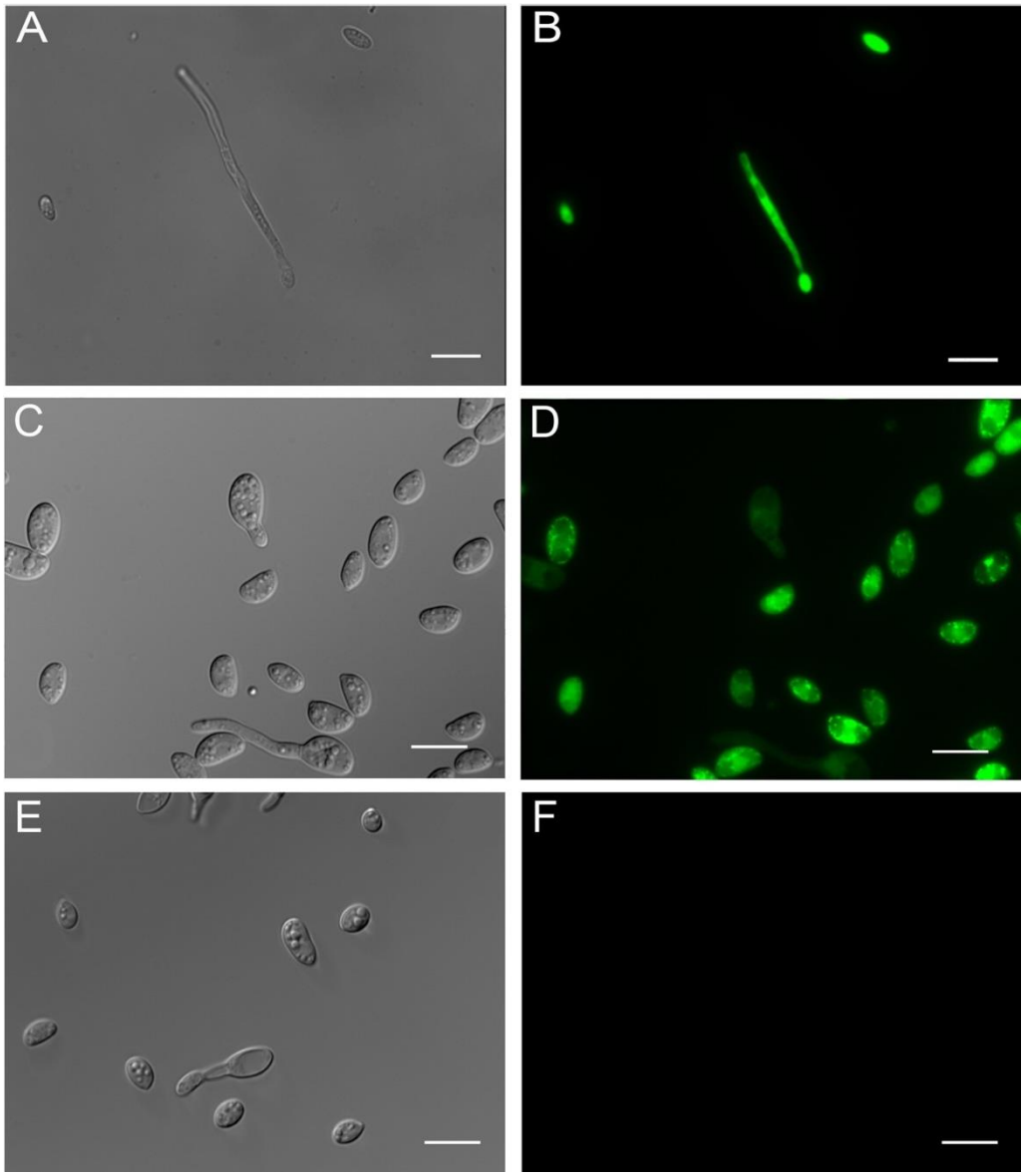


Figure 3.4. Green fluorescent protein expression in a stable FS-GFP transformant, following single spore isolation and several rounds of sub-culturing onto PDA without hygromycin selection. (**A**) Brightfield image of ungerminated and germinated spore with germ tube. (**B**) The same image as (**A**), but examined under epifluorescence. (**C**) Brightfield image of spores. (**D**) The same image as (**C**), but examined under epifluorescence. (**E**) Brightfield image of control wild-type *F. solani* (CBS224.34) spores and germlings. (**F**) The same image as (**E**), but examined under epifluorescence. Note the lack of fluorescence of wild-type propagules. The magnification for panel **A** and **B** is x60 and x100 for the rest panels. Scale bars = 10 μm.

3.3.2 Detection of GFP Expression in FS-GFP Transformants by Using ELISA

Expression of GFP in fluorescent FS-GFP transformants was confirmed by ELISA tests using anti-GFP antiserum. The ELISA absorbance values at 450 nm of antigens extracted from cultures of the FS-GFP strain compare with the wild-type are shown in (**Figure 3.5**)**.** The absorbance value for the transformants (A_{450} 1.87) is significantly greater than the control strain (A_{450} 0.183) when analysed using the Student's t-test (p <0.01). The ELISA therefore confirms GFP expression in this fluorescent FS-GFP transformants.

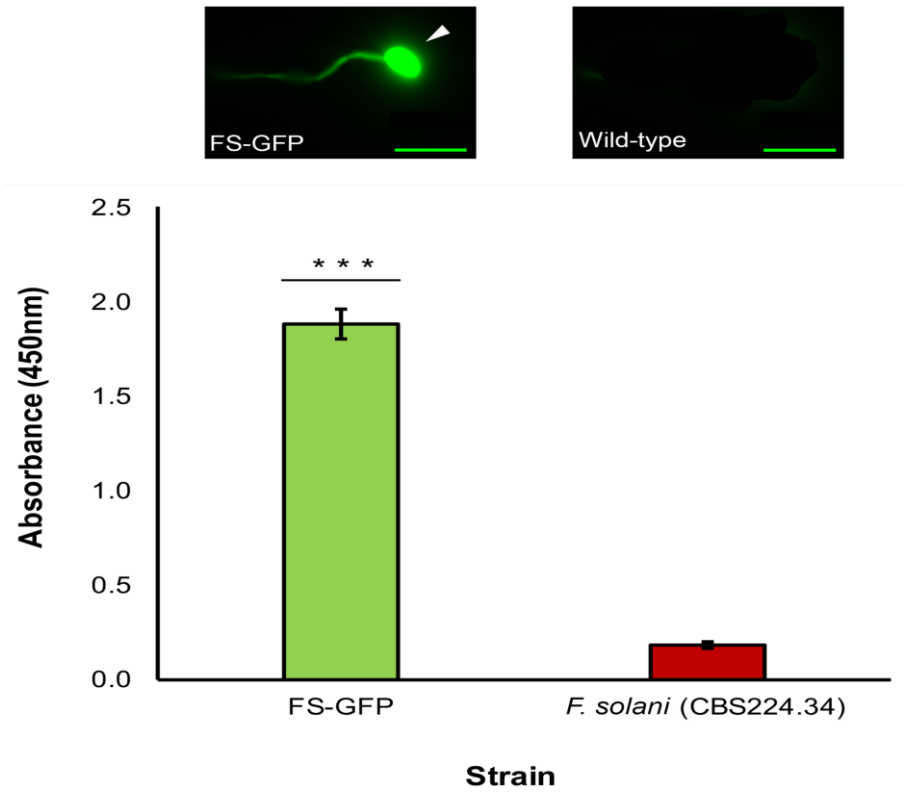


Figure 3.5. Absorbance values from ELISA tests of a stable fluorescent FS-GFP transformant and the wild-type strain (CBS224.34). Each bar is the mean of 3 replicate samples ± S.E. The level of significance indicates: *** < 0.001. Scale bars = 20 μm.

3.3.3 Phenotypic Analysis of FS-GFP Transformant and Wild-type Strain

Linear growth of the FS-GFP transformant and *F. solani* (CBS224.34) was determined by measuring colony diameters over a 12-day period (**Figure 3.6**). Three replicate culture plates for both strains were scanned daily with an Epson Perfection V750 Pro scanner. The results showed that colony diameters were similar for both strains with no significant differences over the experimental period (p >0.05) (**Figure 3.7 A**). The FS-GFP transformant had a significantly reduced spore concentration compared to the wild-type strain when grown under the same conditions (**Figure 3.7 B**).

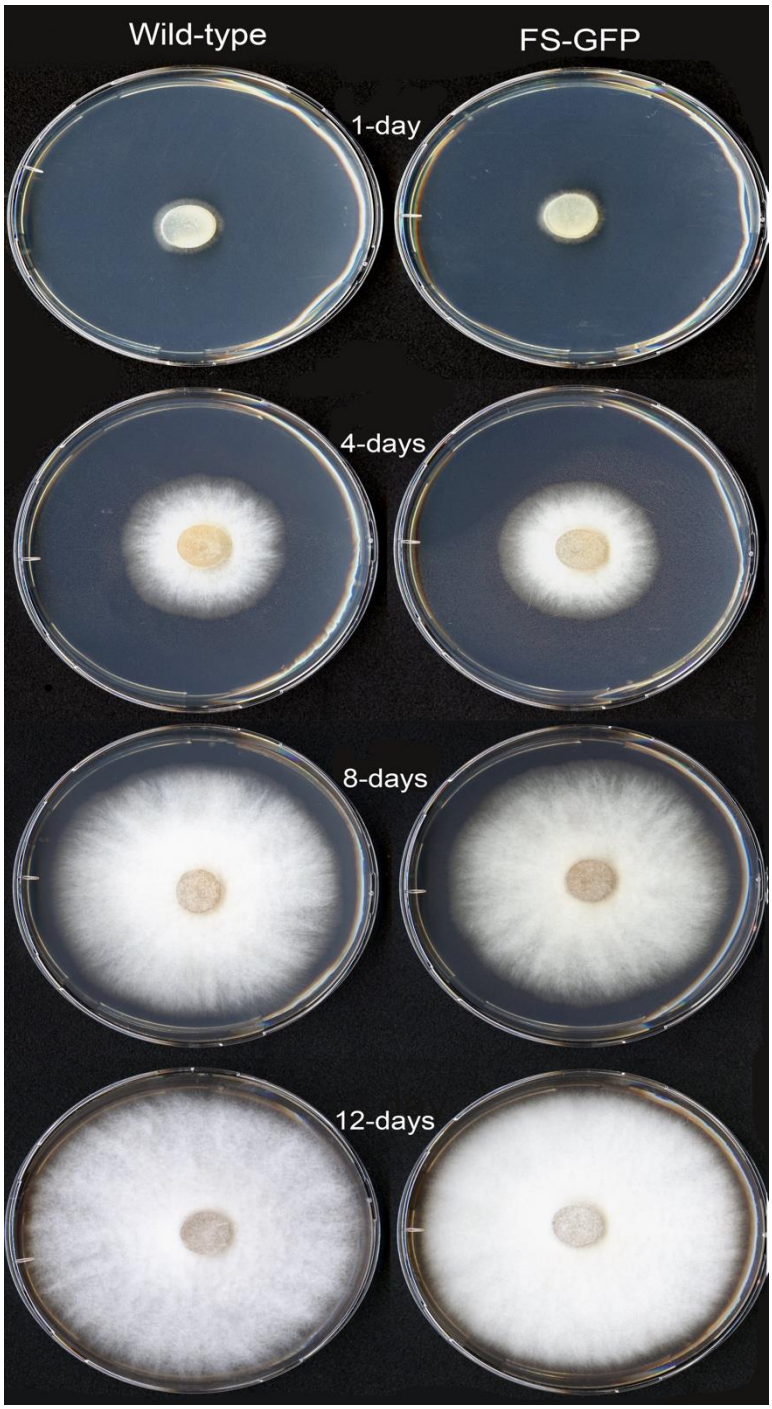


Figure 3.6. Colony morphologies of wild-type *F. solani* (CBS224.34) and the stable FS-GFP transformant when grown on PDA over a 12-day period. The two strains have similar morphologies, showing that transformation with GFP has had no discernible effect on gross morphology.

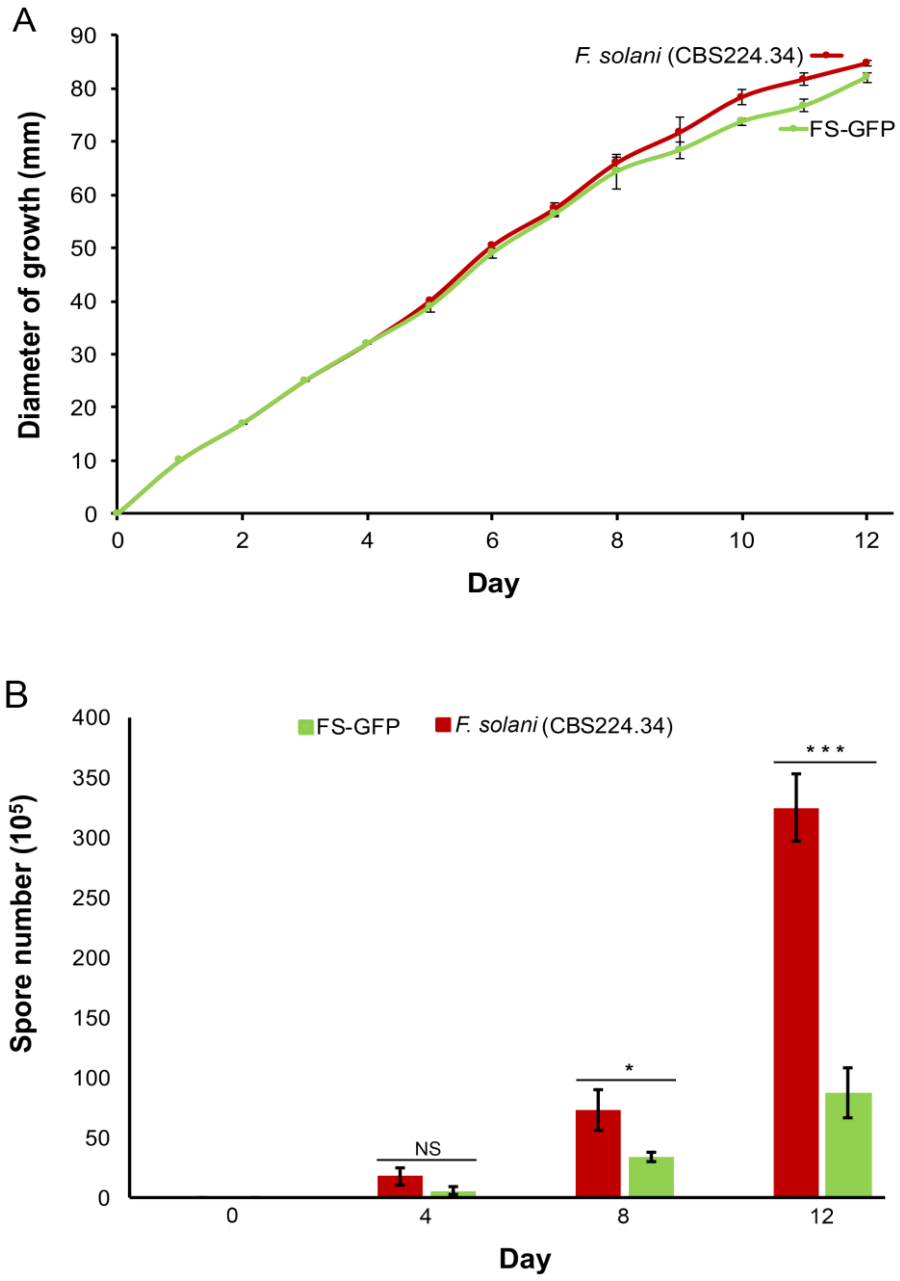


Figure 3.7. Time course of wild-type *F. solani* (CSB224.34) and putative GFP strain over 12 days growth on PDA. (**A**) colony diameters (mm), the chart shows the similarity in linear growth of both the wild-type and FS-GFP strain. (**B**) spore production, note a decrease in spore production by the FS-GFP strain. Each point is the mean of 3 replicates ± S.E. The levels of significance indicate: NS= non-significant, * < 0.05, *** < 0.001.

3.3.4 Antigen Production, Biomass Accumulation and Spore Agglutination

Antigen production (determine by ELISA) by the FS-GFP transformant did not differ significantly to the wild-type strain during the 21 days sampling period (p >0.05) (**Figure 3.8 A**). However, biomass accumulation of the FS-GFP transformant was significantly reduced in PDB cultures compared to the wild-type strain *F. solani* (CBS224.34) at (p <0.05) (**Figure 3.8 B**). MAb ED7 strongly agglutinated spores from both the wild-type *F. solani* strain (CBS224.34) (**Figure 3.9 A**) and the FS-GFP transformant (**Figure 3.9 B**), compared to the TCM control. This shows that transformation of the fungus with GFP had not altered the agglutination properties of its spores.

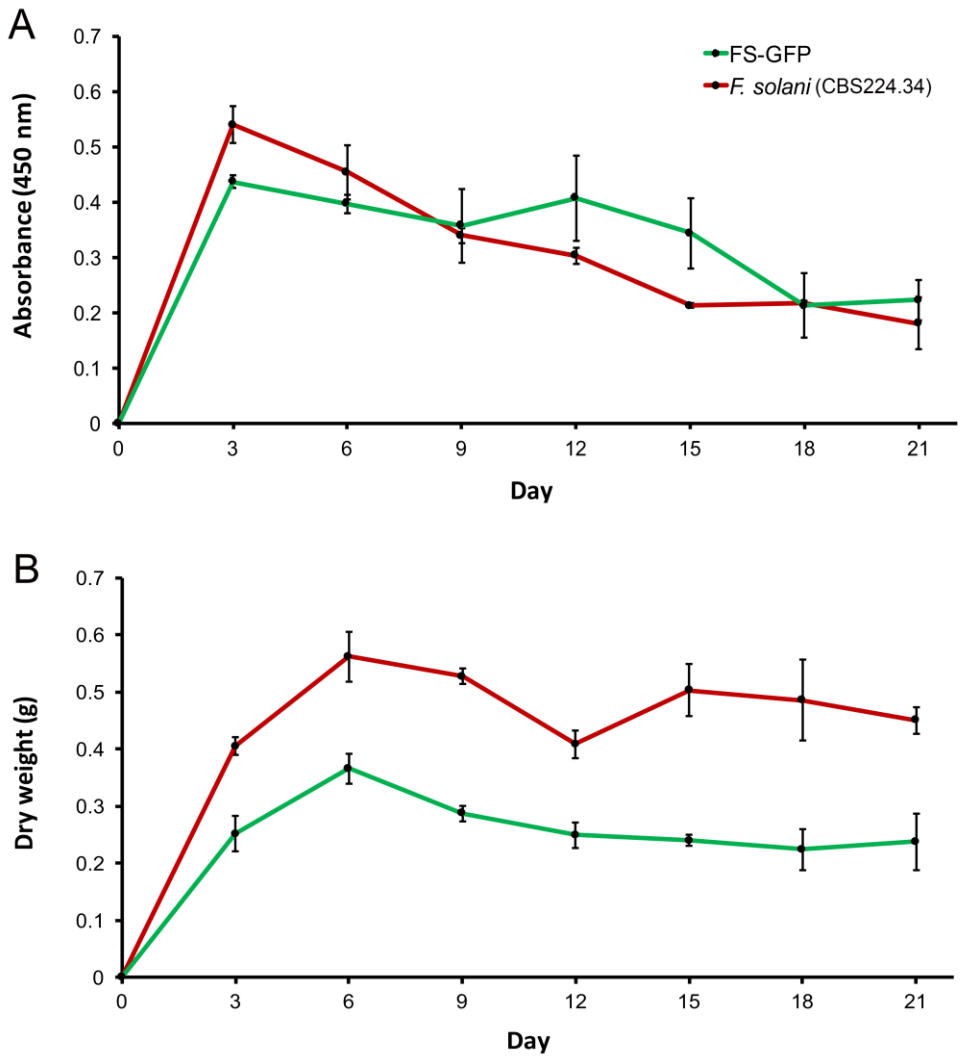


Figure 3.8. (**A**) Antigen production by the wild-type strain and FS-GFP transformant determined by ELISA over the same time period. (**B**) Biomass accumulation of *F. solani* (CBS224.34) and the FS-GFP transformant during 21 days growth in PDB. Each point is the mean of 3 replicates ± S.E.

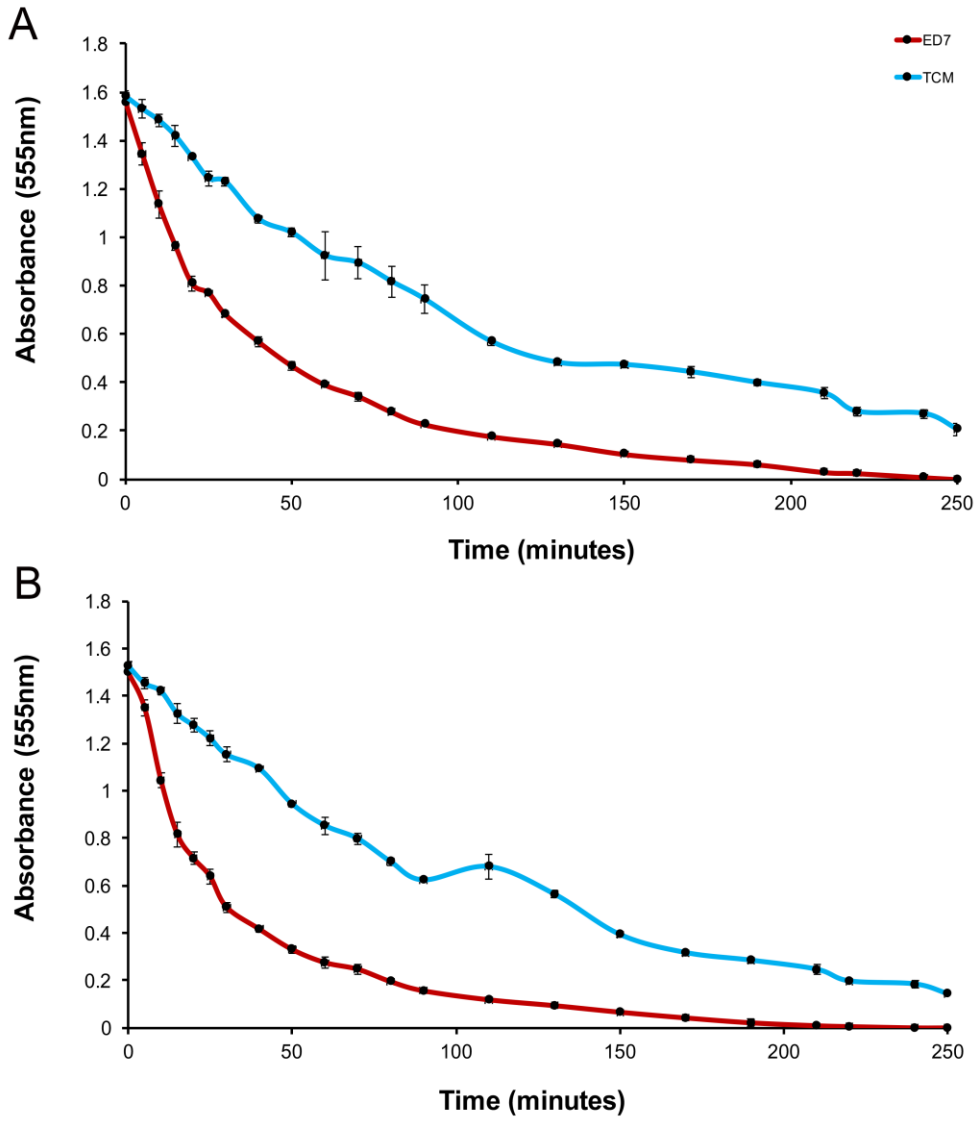


Figure 3.9. Agglutination of wild-type *F. solani* (CBS 224.34) spores (**A**) and spores of the FS-GFP transformant (**B**) by mAb ED7. Note the rapid decline in absorbance for both strains in the presence of the antibody, but the lack of agglutination by in TCM only. Each point is the mean of 3 replicates ± S.E.

3.3.5 Live Cell Imaging of Spore Agglutination

Live cell imaging showed that *F. solani* (CBS224.34) spores agglutinated within 5 min of re-suspension in mAb ED7 (**Figure 3.10 A**). Agglutination was not exhibited in TCM (**Figure 3.10 B**). Movies demonstrating the agglutination phenomenon over 30 min are archived at 35 mins. Single spores were quantified manually using Patina software version 1.0.1 (Atek, Inc., Patina App.com) (**Figure 3.10 C**).

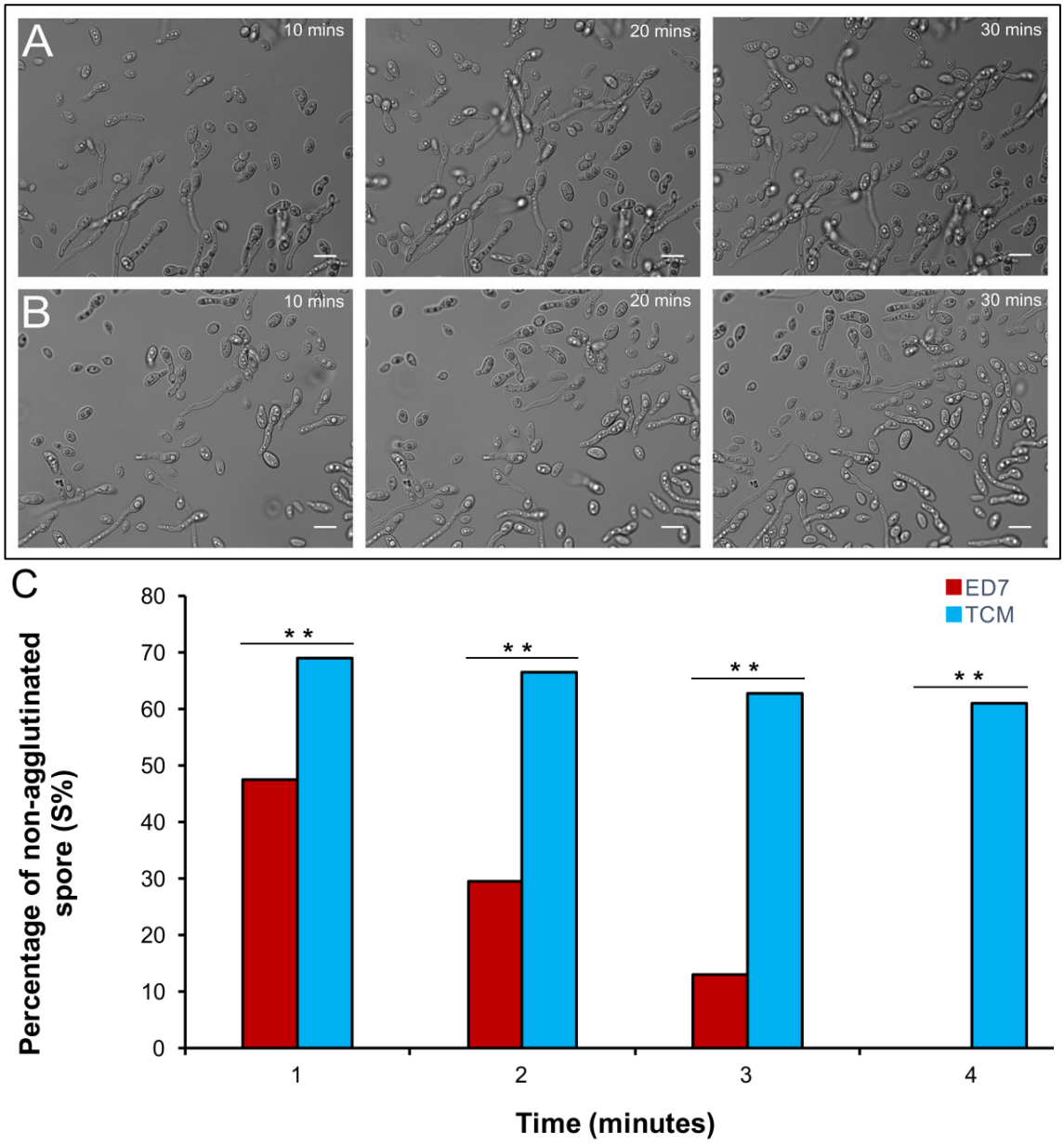


Figure 3.10. Agglutination of *F. solani* (CBS224.34) spores in TCS of mAb ED7 (**A**) and TCM only (**B**) over a 30 min sampling period. Note agglutination with mAb ED7, but the lack of agglutination with TCM. Scale bars = 10 µm. (**C**) Quantification of the agglutination process. The percentage of non-agglutinated spores were calculated from 7 fields of view at each time point, three biological replicates for each treatment. The number of single spores was significantly decreased in the ED7 mAb treatment. The level of significance indicate: ** < 0.01.

3.3.6 Live Cell Imaging of Spore Phagocytosis

Phagocytosis of FS-GFP spores by AMØs was quantified by determining the P% and Pi. The experiments were conducted with the fluorescently-labelled strain to facilitate spore counting and to allow live cell imaging of the phagocytic process. There was an increase over time in the mean number of engulfed spores per AMØ, both with mAb ED7-treated (agglutinated) and with control TCM-treated (non-agglutinated) spores (**Figure 3.11 A**). There was a significant reduction in the mean number of spores per AMØ at 2 h under agglutinating conditions compared to TCM only and no significant differences in the treatments thereafter. This shows that spore agglutination delayed the phagocytic process. This finding was corroborated by the significantly greater number of AMØs at 2 h, in the ED7 treatment compared to the control, which contained no spores (**Figure 3.11 B**). Thereafter, there were no significant differences in treatments.

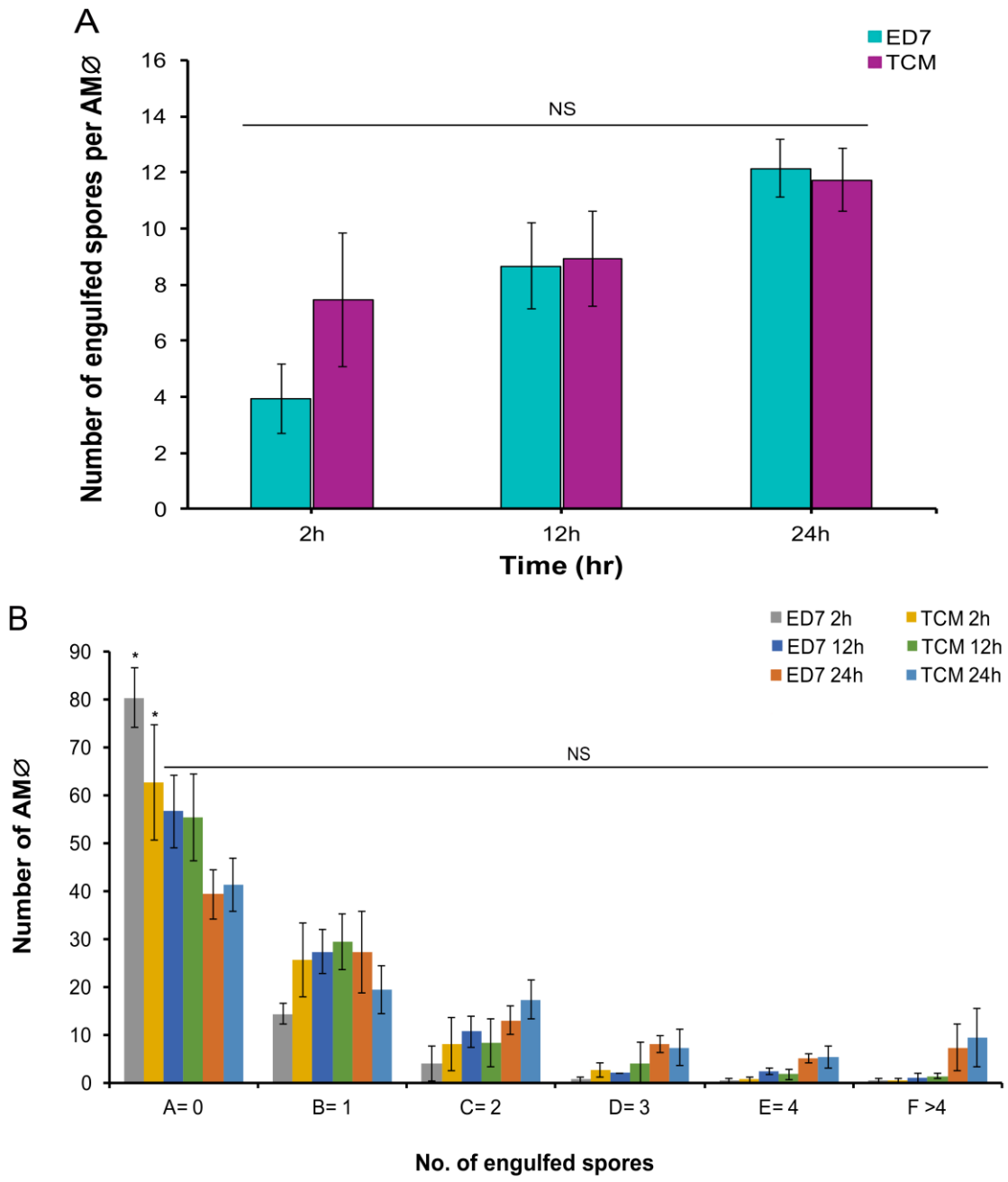


Figure 3.11. Phagocytosis of FS-GFP microconidia by AMØ at 2h, 12h, and 24 h under agglutinating (mAb ED7) or non-agglutinating (10%TCM-ΔFCS only) conditions. (**A**) Mean number of engulfed FS-GFP spores per AMØ, and (**B**) Numbers of AMØs containing (0, 1, 2, 3, 4 or >4) spores at each time point. Each bar is the mean of three replicates ± S.E. The levels of significance indicate: NS = non-significant, * < 0.05.

3.3.7 Phagocytic Percentage and Phagocytic Index

The P% and the Pi values for were not significantly different (p >0.05) between the FS-GFP spores treated with mAb ED7 or with TCM only at 2, 12 and 24 h as shown in **Figure 3.12 A** and **Figure 3.12 B**. Phagocytic percentages increased over time under both conditions and while there was no significant increase in P% over time under non-agglutinating conditions (TCM only), there was a significant increase in P% between 2 h and 12 h under agglutinating conditions. This further illustrates the delay in phagocytosis due to spore agglutination by mAb ED7.

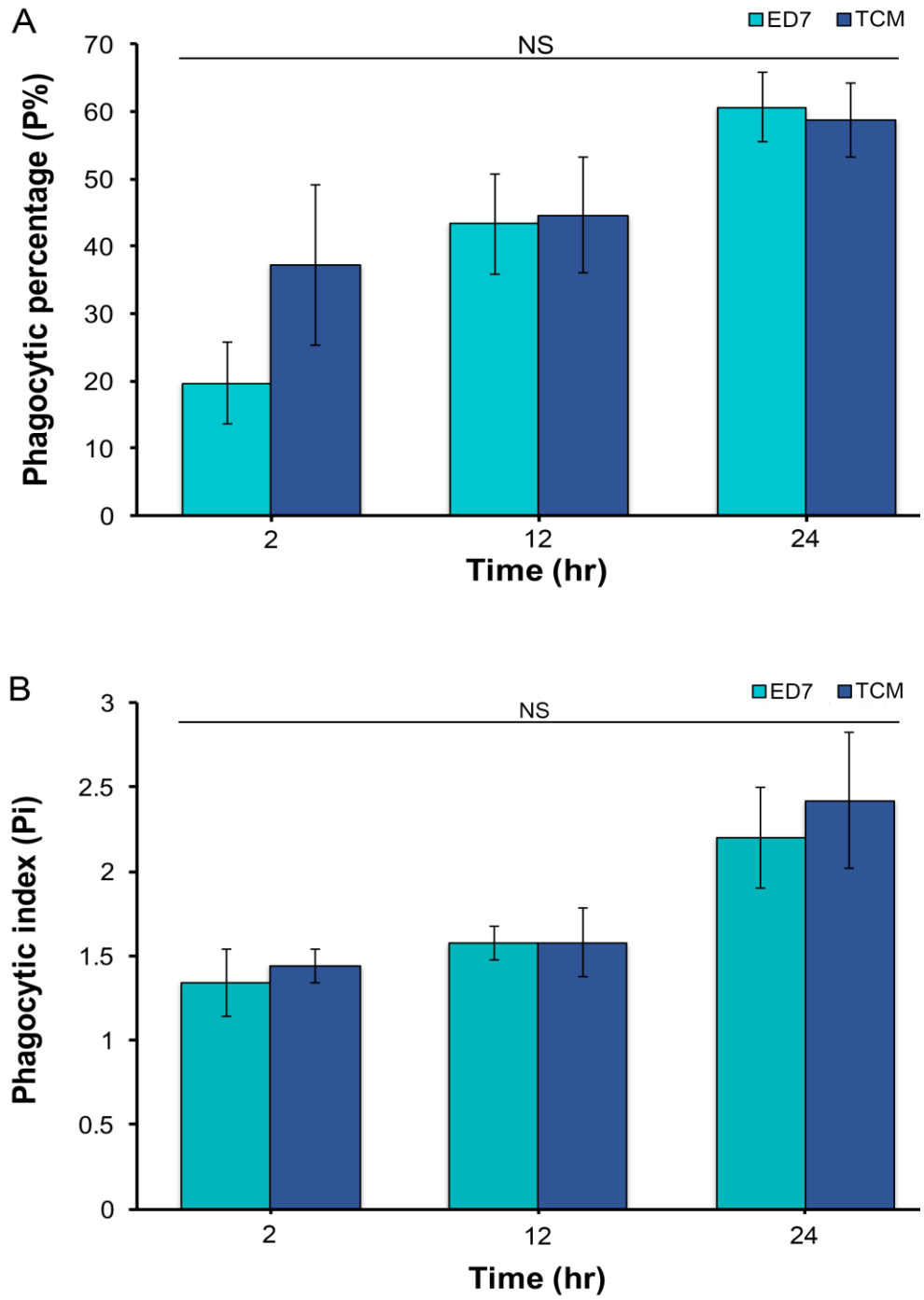


Figure 3.12. Phagocytic activity of FS-GFP microconidia by AM∅ at 2h, 12h, and 24 h under agglutinating (mAb ED7) or non-agglutinating (10% TCM-ΔFCS only) conditions. (**A**) Phagocytic percentage (P%), and (**B**) Phagocytic index (Pi). Each bar is the mean of three biological replicates ± S.E. The levels of significance indicate: NS = non-significant ($p > 0.05$).

3.4 Discussion

The fungal genus *Fusarium* contains opportunistic fungal pathogens that have emerged over recent decades as causative agents of life-threatening disease in humans (van Diepeningen *et al.,* 2015). *F. solani* is the most common and virulent species and is associated with high rates of mortality and morbidity in individuals with impaired immunity. Effector cells of innate immunity (macrophages and neutrophils) are a critical first-line of defence against infection by the fungus. In the absence of effective phagocytosis and clearance of infectious propagules, the fungus can disseminate, causing the frequently fatal invasive disease fusariosis. *Fusarium* infectious propagules (spores) enter the human body mainly through the respiratory tract, wounds, catheters, trauma sites and contaminated contact lenses (Mukherjee *et al*., 2012; Marcio Nucci & Anaissie, 2007; Sanz *et al*., 1993).

Phagocytic cells in the lungs AMØ typically phagocytose and destroy spores and, along with neutrophils, are highly efficient barrier preventing spore germination and dissemination from the lung tissues (Bain *et al.,* 2014). The mechanisms of AMØ recognition and destruction of mould spores have been investigated in detail in *Aspergillus fumigatus*, the cause of invasive pulmonary aspergillosis. Fungal recognition by AMØ is the first step of the innate immune response that leads to phagocytosis and intracellular intra-cellular degradation of inhaled conidia of *A. fumigatus* (Nicola *et al.,* 2008). The antigenic fungal cell wall components including β-glucans, chitin and mannose or galactomannan are the first point of contact and can be recognised as PAMPs by PRRs that present on the phagocytic cell surface (Chotirmall *et al.,* 2013). PRRs that include different recognition receptors such as TLR2 and TLR4, dectin-1 and 2,

and mannose receptor (MR) are able to detect invading *Aspergillus* species (Gersuk *et al.,* 2006; Luther & Ebel, 2006; Park & Mehrad, 2009). The PRRs and PAMPs interaction induces generation of AMPs such as defensins and production of inflammatory cytokines such as interleukins group, TNF-α and IFNγ that can play a key role in recognising fungal elements, recruiting other phagocytes and activating adaptive immune response resulting in increased fungal clearance via phagocytosis (Blanco & Garcia, 2008; Brieland *et al.,* 2001; Hegedüs & Marx, 2013; Luther & Ebel, 2006; Park & Mehrad, 2009; Phadke & Mehrad, 2005).

Following this interaction, AMØs engulf conidia in an actin-dependent manner within phagosome that fuse with lysosomes leading to the killing of conidia by oxidative such as and non-oxidative ways depends on phagolysosome acidification (Agapov *et al.,* 2009; Brakhage *et al.,* 2010; Ibrahim-Granet *et al.,* 2003; Nicola *et al.,* 2008). However, *Aspergillus* species have developed inhibitory mechanism against phagolysosome acidification effect resulting in conidia germinating, macrophage pierces and escape (Dagenais & Keller, 2009; Slesiona *et al.,* 2012).

Macrophages like other leukocytes produce METs like a network to further control *A. fumigatus* proliferation that forming of the nuclear DNA with antimicrobial molecules to surround and trap fungi (Aulik *et al.,* 2012; Bruns *et al.,* 2010). Despite an increasing knowledge of the mechanisms of innate immunity to other mould pathogens, our understanding of the interactions between phagocytic immune cells and spores of pathogenic *Fusarium* species remains limited.

To study the interactions between *F. solani* propagules and AMØ, the major phagocytes present in lung alveoli, a GFP-expressing isolate of *F. solani* was constructed. This allowed the process of spore phagocytosis to be more easily observed in live cell imaging investigations.

The use of the GFP-expression technique to study the interaction and phagocytosis process between macrophages and fungi has been reported in *Aspergillus fumigatus* (Wasylnka & Moore, 2002) and *C. albicans* (Katragkou *et al.,* 2010). Also, this tool was used to investigate plant infections by pathogenic *Fusarium* species, including *F. oxysporum* (Lagopodi *et al.,* 2002), *F. equiseti* (Maciá-Vicente *et al.,* 2009), *F. graminearum* (Skadsen & Hohn, 2004), and *F. verticillioides* (Oren *et al.,* 2003).

To the best of my knowledge, this work has two novel aspects not previously undertaken by others: (**1**) *F. solani* was transformed with GFP to allow the phagocytosis process by AMØ to be studied in real-time. This work demonstrates that GFP is an excellent reporter system in *F. solani.* (**2**) An *in-vitro* system was developed to mimic *Fusarium*-innate immune cell interactions. These two developments enabled me to track internalization of FS-GFP spores by mouse AMØ using live-cell imaging for up to 8 hr, and (to assess the effect of spore agglutination by the *Fusarium*-specific mAb ED7 on the phagocytosis process.

In this study, a total of four hygromycin-B resistant colonies were recovered on selection plates for *F. solani* GFP transformation. In contrast to the wild-type strain, the putative transformants displayed GFP expression under epifluorescence. One of the four transformants (FS-GFP) was selected for the

phagocytosis studies. The GFP expression and hygromycin-B resistance of the selected transgenic isolate were stable after five generations on non-selective media, with the fluorescent protein expressed in both fungal hyphae and conidia. Previous studies have reported similar observations in *F. oxysporum* f. sp. *radicis-lycopersici* GFP transformants (Lagopodi *et al.,* 2002) and *F. equesiti* (Maciá-Vicente *et al.,* 2009). Apart from a decrease in spore and biomass production, compared to the wild-type strain, the FS-GFP transformant had a similar morphological appearance in axenic culture and had similar antigenic properties, both regarding extracellular antigen production and spore agglutination by mAb ED7. This gave confidence that the FS-GFP strain could be used to investigate phagocytosis of antigenic *F. solani* spores and also to determine how spore agglutination might affect the phagocytic process.

Opsonization and agglutination of micro-organisms by antibodies can have a profound effect on phagocytosis by complexing infectious propagules and increasing their accessibility to macrophages and neutrophils. Besides antibodies have a significant role in microbial toxin neutralization, they are able to coat and opsonize microbial cell surface leading to enabling efficient recognition by phagocytic cells, then promote and accelerate pathogen uptaking by efficiently (Casadevall & Pirofski, 2012; Lindow *et al.,* 2011; McCullough *et al.,* 1988; van Kessel *et al.,* 2014). Furthermore, antibodies can prevent fungal biofilm and cell adherence resulting in biomass agglutination and facilitating phagocytosis (Casadevall & Pirofski, 2007; Martinez & Casadevall, 2005; Mishra *et al.,* 2014).

Opsonized fungal cell with human serum antibody or a specifically targeted antibody can be recognised by mediated phagocytosis receptor such as Fc receptors (Fc-Rs) located on the phagocytic cell surface (Levitz, 2010; van Kessel *et al.,* 2014; Wellington *et al.,* 2003; Wellington *et al.,* 2007). Recent studies have shown that the shape and size of agglutinated microbial cells by antibodies have a critical impact on phagocytosis rate of macrophages cells (Litvack *et al.,* 2011). A large agglutinated biomass exceeds macrophage cells can inhibit and block engulfment (Champion & Mitragotri, 2006; Litvack *et al.,* 2011).

In this study, I investigated the effect of spore agglutination by using two measures of AMØ phagocytic efficiency, P% and Pi, under agglutinating and non-agglutinating conditions. P% is the percentage of cells containing spores, while Pi is the average number of phagocytosed spores per macrophage.

Using this technique, no significant overall variation was found in P% and Pi for agglutinated and non-agglutinated spores. However, the percentage of AMØ that did not engulf spores was higher after the spores were treated with mAb ED7 during the first two hours of challenge. This suggests that spore agglutination by mAb ED7 delays the process of phagocytosis. There are two possible explanations for this delay. The first is that the antibody blocks AMØ recognition of surface antigen that binds via cell surface receptors. The cell line used in this study, MH-S, employs the complement receptor CR3 (CD11b/CD18) also to recognize fungal β-D-glucan and mannan (Xia *et al.,* 1999). While the extracellular antigen bound by mAb ED7 has been shown to be an immunodominant high molecular weight glycoprotein (**see Section 2.3.2,**

Chapter 2), its full molecular identity and involvement in phagocyte recognition have yet to be determined. Notwithstanding this, it is feasible that ED7 blocks receptor binding to an immunodominant antigen on the spore surface is thereby delaying recognition and engulfment by the phagocyte. An alternative explanation for the delay in phagocytosis is physical impairment. The increase in fungal biomass due to spore agglutination may simply prevent efficient uptake by the AMØ. A combination of reduced recognition by cell surface receptors and physical impairment may also account for the delay. Currently, little is known about the cell wall components of *F. solani* involved in recognition and uptake by macrophages, which warrants further investigation.

The work conducted here shows that murine AMØs efficiently internalize FS-GFP conidia and germinated spores, even despite delays in phagocytosis due to spore agglutination Indeed, single AMØ were able to phagocytose up to 12 spores. However, it was observed that engulfed FS-GFP conidia were able to germinate and continued to grow within the AMØ, ultimately leading to their escape from the macrophages and host cell lysis. Attempts were made by neighbouring macrophages to engulf developing hyphae, but no apparent damage appeared to occur to hyphae, which continued to proliferate into a hyphal network. Similar observations of cell lysis and death of murine macrophages by hyphae have been reported in the related fungus *F.oxysporum* (Schäfer *et al.,* 2014) and the dimorphic yeast *Candida albicans* (Bain *et al.,* 2012). Further analysis of the time-lapse movies generated here revealed a very rapid expulsion of FS-GFP conidia from intact macrophages. Importantly, this phenomenon during *F. solani* phagocytosis was only observable because of the real-time imaging system employed here. While the process of

vomocytosis has been reported in the yeasts *Cryptococcus neoformans* (Alvarez & Casadevall, 2006; Ma *et al*., 2006) and *C. albicans* (Bain *et al*., 2012), this is the first time this phenomenon has been reported for a mould pathogen and might explain, in part, how *F. solani* is able to escape innate immune cell killing.

Summary of Key Findings:

1. Transformation of *F. solani* (CBS224.34) with GFP marker is a convenient, fast, and effective approach for studying the cellular innate immunity–fungus interaction without using chemical stains. To the best of my knowledge, this work is novel.
2. The level of gfp expression in single-spore isolated transformant is high and stable for long term in the vegetative and reproductive forms of the fungus, including the mycelium and microconidia.
3. The putative FS-GFP transformants colony has similar morphology, growth and antigenic characteristics to the wild-type isolate. However, there is a significantly reduction in spore production compare to the wild-type strain.
4. The developed *in-vitro* system to mimic *Fusarium*-innate immune cell interactions is simple, effective and can be used for visualization and live cell imaging of the phagocytosis process in real-time. To the best of my knowledge, this system is novel.
5. The FS-GFP spore agglutination by *Fusarium*-specific mAb ED7 delays the phagocytic process by AMØs compare with the control 10% TCM.

Chapter 4. Tracking Human Pathogenic *Fusarium* species in Sink Drain Biofilms by Using mAb ED7

4.1 Introduction

Species in the fungal genus *Fusarium* are ubiquitous environmental moulds, and pathogens of both plants and animals (Thornton & Wills, 2015; Zhang *et al.,* 2006). In immunocompromised humans, such as patients with haematological malignancies and HSCT and SOT recipients, *Fusarium* species are significant emerging pathogens, causing a frequently fatal disseminated disease known as fusariosis with an associated mortality rate of 50 - 100% (Boutati & Anaissie, 1997; Dignani & Anaissie, 2004; Girmenia *et al.,* 2000; Jensen *et al*. 2004; Musa *et al.,* 2000; Nucci & Anaissie, 2007; O'Donnell *et al.,* 2008), and have appeared in some tertiary cancer centres as the second most common mould pathogen after *Aspergillus* (Boutati & Anaissie, 1997; Walsh & Groll, 1999). Regardless of immunocompetency, *Fusarium* species can cause localised nail infections (onychomycosis) (Arrese *et al.,* 1996), bone and joint infections (Koehler *et al.,* 2014), infections of burn wounds (Latenser, 2003), skin infections (Bodey *et al.,* 2002; Gupta *et al.,* 2000; Gurusidappa & Mamatha, 2011; Nucci & Anaissie, 2002), and are the most frequent cause of fungal keratitis (Jurkunas *et al.,* 2009), leading to progressive corneal destruction and endophthalmitis, with loss of vision or even loss of the affected eye (Dursun *et al.,* 2003; Edelstein *et al.,* 2012).

A recent outbreak of fungal keratitis in the USA, Singapore and Hong Kong was associated with contact lens solution contaminated with multiple strains of *Fusarium* and which led to a visual loss in many patients and the need

for corneal transplantation (Chang *et al.,* 2006). While such outbreaks are rare, disseminated *Fusarium* infections and keratomycosis have increased in frequency over the past decade (Koehler *et al.,* 2014) and an increasing body of evidence suggests that the main environmental sources of human pathogenic *Fusarium* species are contaminated water systems (Adams *et al.,* 2013; Anaissie *et al.,* 2001; Anaissie *et al.,* 2002; Doggett, 2000; Mehl & Epstein, 2008).

4.1.2 Environmental Sources of Human Pathogenic *Fusarium* Species

Increasing incidences of nosocomial fungal infections caused by *Candida, Aspergillus, Mucor*, *Rhizopus*, *Fusarium*, *Acremonium*, *Alternaria*, *Bipolaris* and *Curvularia* species have been reported (Fridkin & Jarvis, 1996; Scheel *et al.,* 2013), necessitating improved identification and monitoring of hospitals for potentially infectious propagules.

As with many fungal pathogens, *Fusarium* species have adapted to survive in a wide range of ecosystems, with the dispersal of spores into terrestrial and aquatic habitats (Short *et al.,* 2011) through air, soil, wind, rain, river water and sewage (Defra, 2011). Additionally, a number of studies have recovered pathogenic *Fusarium* species from plumbing fixtures and it is hypothesised that microbial biofilms on fixtures may serve as important reservoirs of infectious *Fusarium* propagules in hospitals and domestic environments (Anaissie *et al.,* 2001; Mehl & Epstein, 2008; Scheel *et al.,* 2013; Short *et al.,* 2011; Zhang *et al.,* 2006). The idea of water-borne infections has been suggested for other

opportunistic fungal pathogens such as *Aspergillus* (Anaissie *et al.,* 2002; Gangneux *et al.,* 2002), *Cladosporium* (Mesquita-Rocha *et al.,* 2013), *Exophiala* (Marcio Nucci *et al*., 2002), *Candida* and Penicillium (Arvanitidou *et al*., 1999).

Tracking environmental sources of pathogenic fungi is critical to the prevention and control of outbreaks of hospital infections (Anaissie *et al.,* 2001). A number of pathogenic *Fusarium* species have been recovered from naturally-infested water samples, including *F. anthophilum, F. acuminatum, F. chlamydosporum, F. culmorum, F. equiseti, F. verticillioides, F. oxysporum, F. dimerum, F. proliferatum, F. solani, F. sambucinum* and *F. sporotrichioides* (Anaissie *et al.,* 2001; Palmero *et al.,* 2009; Sautour *et al.,* 2012). However, the most frequent human pathogenic species recovered from water tanks, sink drains, shower heads and plumbing systems in hospitals and domestic residences belong to the FSSC, FOSC and FDSC (Anaissie *et al.,* 2001; Arvanitidou *et al.,* 1999; Hageskal *et al.,* 2006; Mehl & Epstein, 2007; O'Donnell *et al.,* 2007, 2009, 2010; Sautour *et al.,* 2012; Schroers *et al.,* 2009; Short *et al.,* 2011; Steinberg *et al.,* 2015; Zhang *et al.,* 2006). Immuno-compromised individuals exposed to these water-borne species complexes are at increased risk of disseminated fusariosis (Anaissie *et al.,* 2003; Carneiro *et al.,* 2011; Garnica & Nucci, 2013; Nucci & Anaissie, 2007; O'Donnell *et al.,* 2004).

4.1.3 *Fusarium* Biofilms

Fusarium species complexes can form thick biofilms in plumbing systems in which the fungal spores and hyphae are proliferate and enmeshed in an extracellular matrix allowing adhesion to surfaces (Pierce *et al.,* 2008). The

formation of biofilms is thought to contribute to *Fusarium* antifungal resistance and pathogenicity (Mukherjee *et al.,* 2012). Fungal spore adhesion to living cells or surfaces is a crucial step to the establishment of biofilms and the development infections (Ramage *et al.,* 2009). *Fusarium* spores adhere to surfaces through interaction with an extracellular fibrillar matrix located on the outer layer of the spore cell wall. The process of adhesion starts within few minutes and construction a biofilm is completed within a few hours (Kwon & Epstein, 1997; Kwon & Epstein, 1993; Pan *et al.,* 2011). The extracellular matrix typically consists of glycoprotein with mannose residues that act as an adhesive for spore attachment (Gauthier & Keller, 2013; Kwon & Epstein, 1997). Involvement of extracellular matrices in adhesion of other pathogenic fungi has also been reported for *Aspergillus* (Wasylnka & Moore, 2000; Yang *et al.,* 2000), *Penicillium* (Srinoulprasert *et al.,* 2009), *Candida* (Tournu & Van Dijck, 2011) and *Rhizopus* (Bouchara et al., 1996). At present, little is known about *Fusarium* biofilm formation and its role in survival and pathogenicity of the fungus (Peiqian *et al.,* 2014).

4.1.4 Objective of Chapter 4

In Chapter 2 of this thesis, I described the development and characterization of the *Fusarium*-specific mAb, ED7, that binds to an abundant extracellular glycoprotein antigen present in a fibrillar matrix surrounding the spores and hyphae of *F. solani*. Furthermore, I showed that the antibody was able to agglutinate spores of *F. solani* and other pathogenic species. In this Chapter, I set out to determine whether mAb ED7 could be used to track the fungus in environmental samples by detecting the water-soluble diagnostic antigen in

swabs of communal and hospital sink drain biofilms. By using the mAb in an ELISA, I show that it can differentiate *Fusarium* species from other human pathogenic yeasts and moulds present in mixed fungal communities in sink drain biofilms. In doing so, I show that the ELISA represents a simple method for specific detection of *Fusarium* species in environmental reservoirs and for identifying water systems contaminated with the fungus.

The work described in this chapter and accompanying work in Chapter 2 forms the basis of a paper published in 2016 in the journal *Environmental Microbiology* (Al-Maqtoofi, M. and Thornton, C.R. [2016] Detection of human pathogenic *Fusarium* species in hospital and communal sink biofilms by using a highly specific mAb. *Environmental Microbiology*, in press. DOI:10.1111/1462-2920.13233) and which is presented at the rear of this thesis.

4.2 Materials and Methods

4.2.1 Study Location

A total of 65 drain and water samples were collected between March and December 2014 from communal sinks located on the University of Exeter Streatham Campus (Exeter, Devon, UK) and from sinks located at the Royal Devon & Exeter tertiary care hospital (Exeter, Devon, UK). At the start of the study, 33 sink drain samples from six locations on the University campus were collected. An additional 32 samples were then collected from five areas of the hospital, namely the Intensive care unit (ICU), Intensive Therapy Unit (ITU), Haematology Unit, Ophthalmology Unit and Oncology unit. Permission for sink sampling at the Royal Devon and Exeter Hospital was granted by the Director of Infection Prevention and Control.

4.2.2 Collection of Biofilm Swabs and Water Samples

Sink drain biofilms were collected using sterile 15-cm cotton-tipped applicators (Technical Service Consultants Ltd., UK) as illustrated in **Figure 4.1** and **Figure 4.2**. The cotton tips were wetted with sterile PBS buffer, inserted into the drain pipes through the sink strainers and used to scour the interior surface of drain pipes for approximately 10 sec. Applicators with visible detritus were immersed into 1.5 ml microcentrifuge tubes containing 1 ml of sterile PBS buffer to dislodge biofilm debris (**Figure 4.1 A to C**). For sink water samples, 1 ml of tap water was collected in sterile microcentrifuge tubes. Sealed tubes were immediately transferred to the laboratory for processing by mycological culture and ELISA. Samples were vortexed vigorously for 1 min (**Figure 4.1 D to E**), centrifuged at 14,462 *g* for 10 min and supernatants (**Figure 4.1 F to G**)

transferred to clean tubes prior and stored at – 20 °C before processing by ELISA. For mycological culture pelleted biofilm debris (**Figure 4.1 H**) were re-suspended in 250 µl sterile PBS buffer, homogenized by vortex, and 200 µl of suspension spread on the surface of PDA containing 0.1 mg/ml of the broad-spectrum antibiotic Rifampicin (Cat. No. 195490, MB Biomedicals LLC, Germany) using a sterile L-shape spreader. The culture plates were incubated at 26 °C, fungal colonies were separated on the basis of gross morphological characteristics (**Figure 4.1 I**) and were sub-cultured on PDA until axenic cultures were generated (**Figure 4.1 J**). A reference code was allocated to each of the 225 strains recovered, and isolates were maintained as PDA slopes at room temperature before identification by ITS sequencing and TEF-1α polymerase chain reaction.

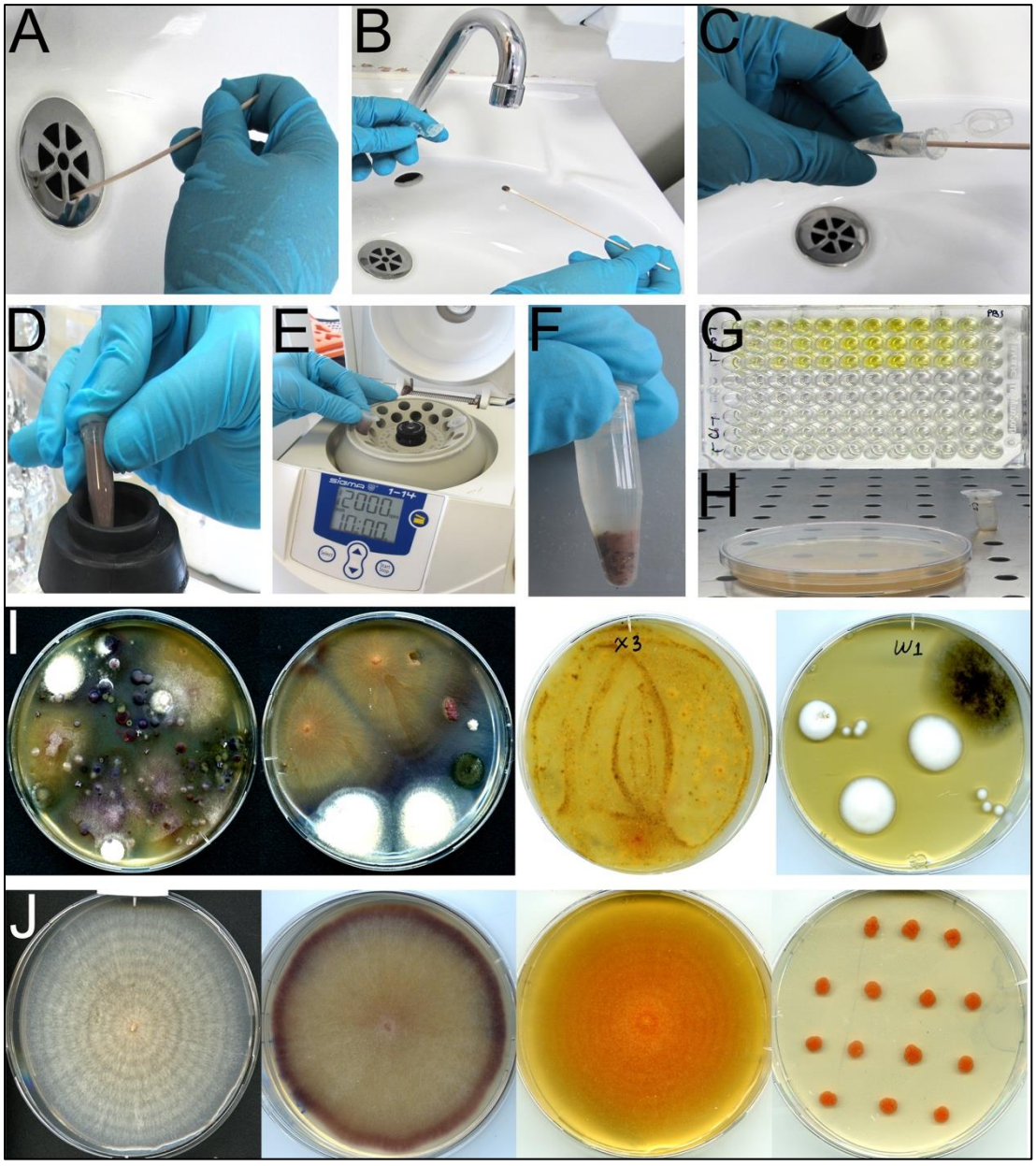


Figure 4.1. Biofilm sampling protocol and preparation of axenic cultures of recovered fungi. (**A - C**) Recovery of biofilms from sink drain using a cotton-tipped swab and immersion of biofilm-coated tip in PBS. (**D**) Vigorous vortexing of biofilm debris, (**E**) pelleted debris and (**F**) supernatant containing soluble antigens following centrifugation to test by (**G**) ELISA using mAb ED7 and **(H**) debris culturing on PDA supplemented with Rifampicin. (**I**) Typical mycological culture is showing different morphologies of culturable fungi on mixed culture plates. (**J**) Fungi were separated on the basis of gross morphologies and were sub-cultured until axenic cultures were obtained.

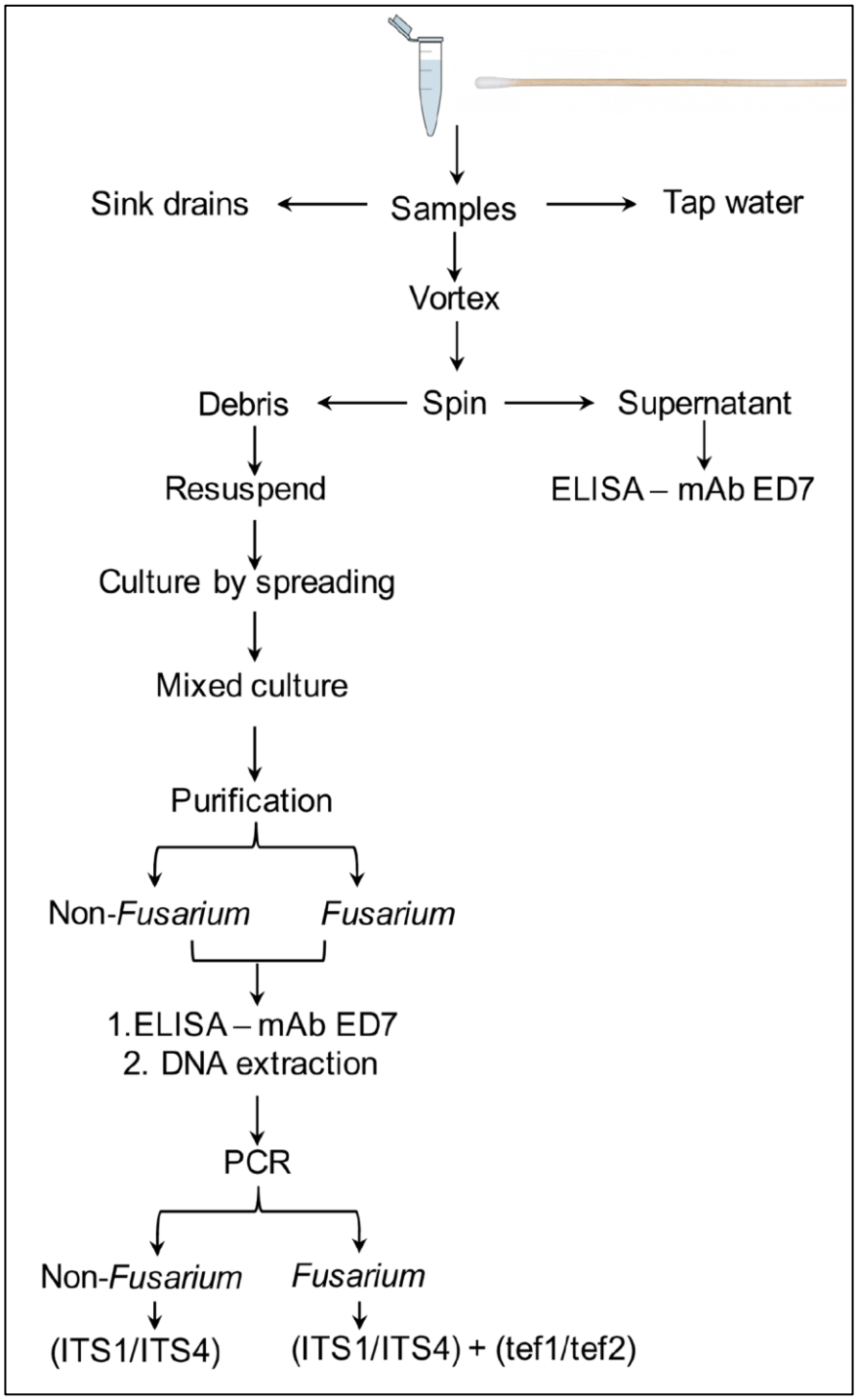


Figure 4.2. Schematic diagram showing diagnostic work-flow for identification of *Fusarium* species and unrelated yeasts and moulds recovered from sink drain biofilms.

4.2.3 Immunodetection of *Fusarium* Species in Sink Swabs and Mixed and Axenic Cultures by ELISA

The ELISA described in Chapter 2 was used for detection of the ED7 diagnostic antigen in swab samples (**Table 4.1** and **Table 4.2**; Swab-ELISA) and mixed cultures and axenic cultures (**Table 4.1** and **Table 4.2**; Mixed culture-ELISA and Axenic culture-ELISA, respectively). Swab samples stored at -20^{o}C were thawed and 50 µl samples transferred to the wells of microtitre plates for overnight antigen immobilization and assay by ED7 ELISA as described. Crude antigen extracts were prepared as surface washings from mixed cultures and axenic cultures and, following overnight antigen immobilization of 50 µl samples to the wells of microtitre plates, similarly assayed by ED7 ELISA.

4.2.4 Fungal DNA Extraction

A rapid method for fungal DNA extraction was developed from the protocol used by Wand and co-workers (Wang *et al.*, 1993). For a collection of fungal biomass, axenic PDA slope cultures were flooded with 3 ml of sterile MQ-H_2O and the surfaces stroked using sterile pipette tips. The washings containing spores and hyphal fragments were transferred to 1.5 ml micro-centrifuge tubes and tissues were pelleted by centrifugation at 14,462 *g* for 5 min. The supernatants were discarded, and 50 – 100 mg of biomass were transferred to clean microcentrifuge tubes to which were added 500 µl of 350 mM NaOH. The mixtures were vortexed for 30 sec, and 10 µl of suspension were diluted in 490 µl of 100 mM Tris-HCl, pH 8.0. All steps were performed at room temperature and the mixtures were stored at – 20°C before use.

4.2.4.1 Identification of Fungi by Analysis of the ITS Regions of the rRNA-encoding Gene Unit

Fungi were identified by sequencing of the ITS1-5.8S-ITS2 region of the rRNA-encoding gene unit (White *et al.,* 1990) according to procedures described elsewhere (Thornton *et al.,* 2002), using the primers ITS1ext (5'-GTAACAAGGT TTCCGTAGGTG-3') and ITS4 ext (5'TTCTTTTCCTCCGCTTATTGATATGC-3'). PCR mixtures contained the following components: 1 µl DNA template, 12.5 µl of GoTaq® G2 Green Master Mix DNA polymerase (Promega, MF7112), 9.5 µl of nuclease free water (Promega) and 1 µl of each at 20 picomoles (pmol). The PCR cycling parameters (**Figure 4.3**) were: an initial denaturation step for 5 min at 95°C; 35 cycles of 30 sec at 94°C (denaturation), 30 sec at the 55°C (annealing), 1.5 min at 72°C (extension), followed by a final 10 min extension step at 72°C (Thornton *et al.,* 2002). Species identity was predicted based on >95% sequence identity (E-value = 0.0) (Altschul *et al.,* 1997) of the ITS1-5.8S-ITS2 region of recovered species to species recorded in GenBank.

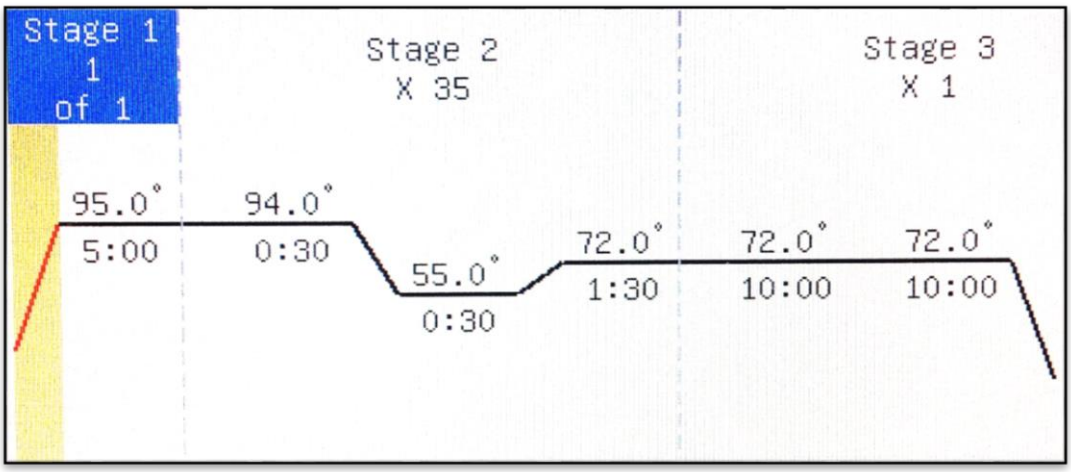


Figure 4.3. PCR cycling parameters for ITS region amplification.

4.2.4.2 Nucleotide Sequence Accession Numbers

Newly determined ITS sequences (**Appendix 3**) were submitted to GenBank and the ITS accession numbers KT876496 to KT876723 were obtained. Species designations of recovered fungi are shown in **Table 4.1**.

4.2.4.3 Identification of *Fusarium* Species Complexes by Using Translation Elongation Factor-1α PCR

Fusarium species were further identified to species complex level by using the forward primer tef-1 (5'-ATGGGTAAGGA(A/G)GACAAGAC-3') and reverse primer tef-2 (5'-GGA(G/A)GTACCAGT(G/C)ATCATGTT-3'), which amplify an ~700 bp region of TEF-1α, the single-locus identification tool in *Fusarium* (Geiser *et al.,* 2004). PCR reactions were carried out in a total volume of 25 µl consisting of 1 µl DNA at a concentration of 30 - 75 ng μl^{-1}, 12.5 µl of GoTaq® Green Master Mix DNA polymerase (Promega, MF7112), 9.5 µl of nuclease free water (Promega) and 1 µl of each primer at 20 pmol. The following cycling parameters (**Figure 4.4**) were used: an initial denaturation step at 95°C for 8 min; 35 cycles of 15 sec at 95°C (denaturation); 20 sec at 54°C (annealing), 1 min at 72°C (extension) followed by a final 5 min extension step at 72°C. Phylogenetic sub-groups of *Fusarium* species (**Table 4.3**) were determined by interrogation of the FUSARIUM-ID v. 1.0 database (isolate.fusariumdb.org) (O'Donnell *et al.,* 2010), with the newly acquired TEF-1α sequences (**Appendix 4**).

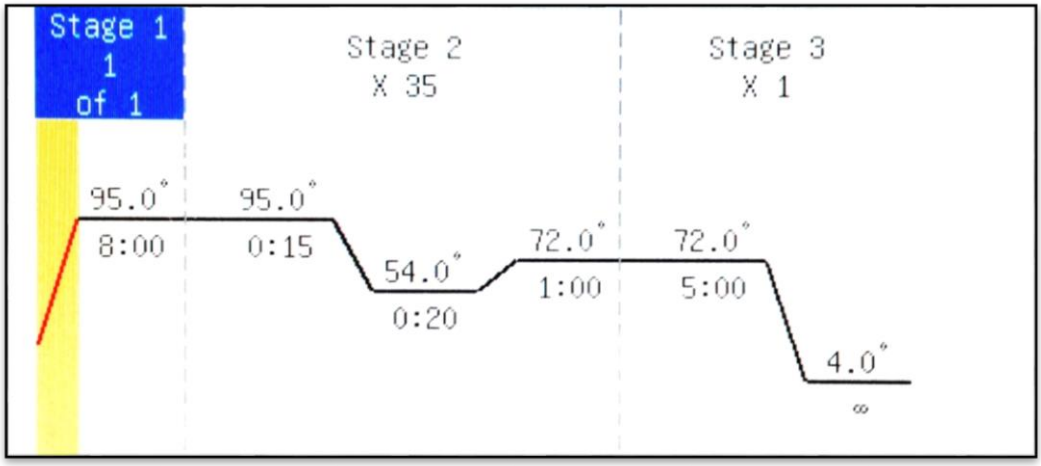


Figure 4.4. PCR cycling parameters for TEF gene amplification.

4.2.5 Phylogenetic Analysis

A total of 225 ITS sequences were compared with those deposited in the National Center for Biotechnology Information (NCBI) databases by using NCBI BLAST® (https://blast.ncbi.nlm.nih.gov). A combination of software were utilized for basic editing and analysis to establish the phylogeny tree (**Appendix 2**) including SeaView Version 4.5.4 (http://doua.prabi.fr/software/seaview) (Manolo Gouy, Laboratoire de Biométrie et Biologie Evolutive CNRS/ Université de Lyon "Licensed under the GNU General Public Licence."), FigTree Version 1.4.2 (http://tree.bio.ed.ac.uk/software/figtree/) (Andrew Rambaut Institute of Evolutionary Biology, University of Edinburgh), REFGENE bioinformatics tool (http://richardslab.exeter.ac.uk/refgen.html) and TREENAMER bioinformatics tool(http://richardslab.exeter.ac.uk/treenamer.html) as published by (Leonard *et al.*, 2009).

ITS sequences were blasted in NCBI, and at least 8 of the closest species based on high identity (Ident) percentage were selected and downloaded in FASTA format and saved as (Plain.txt) file by Microsoft word. The accession

number for NCBI isolates was extracted by loading the last file on REFGENE website, and data were saved as a (*.csv) file and text file. A master file was established by adding ITSs and NCBI (accession numbers) DNA sequences data within one file by word office and finally saved as the (plain.txt) file format. For DNA sequences alignment, Master file was uploaded on MAFFT website (http://mafft.cbrc.jp/alignment/server/) (Katoh & Standley, 2013) and aligned DNA sequences were saved as FASTA format file on word Microsoft Office and then as a (plain.txt) format. Next, for extra basic edition and construction of phylogenetic trees the Master (plain.txt) files were exhibited by SeaView software, followed by serial of process to select one of the closest alignment to each ITS sequence and built pre-final phylogeny tree (PhyML) files and then were saved with (*.mase) extension. Last files were displayed by FigTree programme and saved.

Following, NCBI isolate names were re-extracted from accession numbers to identify ITS data by TREENAMER website through uploading two files were generated previously (*.csv) file and (PhyML) file as a result, Newick file saved on word Microsoft office and converted to (plain.txt) to be displayed on FigTree. For cosmetic editing of phylogeny tree, Inkscape v 0.91 software (https://inkscape.org/en/download/) (Free Software Foundation, Inc., 59 Temple Place, Suite 330, Boston, MA 02111-1307 USA) was used. ITS sequence did not match any of NCBI sequence was considered novel species.

4.3 Results

4.3.1 Immunodetection of *Fusarium* Species In Sink Swabs

Monoclonal antibody ED7 was highly specific for the three human pathogenic species of *Fusarium*, *F. solani*, *F. oxysporum* and *F. dimerum*, which were culturable from 75% of the sink swabs (**Table 4.1 and Table 4.2**). ELISA tests of the saline sink swabs showed that 52% contained detectable levels of *Fusarium* antigen (**Table 4.1** and **Table 4.2**), with ELISA absorbance values in the range ≥0.100 (the threshold value for antigen detection) and up to 1.500. In four hospital samples (samples S47, S48 and S49 from ophthalmology and sample S64 from oncology) *Fusarium* strains could not be recovered for identification by ITS sequencing despite detection of the diagnostic antigen in swab samples with absorbance values of 0.264, 0.530, 0.187 and 0.193 respectively (**Table 4.1**). This was likely due to the *Fusarium* isolates being outgrown in the mixed culture plates by faster growing or more abundant unrelated fungi. Importantly, ED7 was shown not to cross-react with unrelated fungi (axenic culture absorbance values of ≤ 0.100 in all cases) including the human pathogenic yeast or yeast-like fungi *Candida*, *Exophiala*, *Meyerozyma*, *Rhodotorula*, *Trichosporon*, the human pathogenic hyaline or dematiaceous moulds *Aspergillus*, *Phialophora*, *Phoma*, *Trichoderma*, and the human pathogenic mucormycete *Mucor* (**Table 4.1**). The remaining 93% of samples positive for *Fusarium* antigen, either at the swab stage or following periods of biological amplification in mixed or axenic cultures (**Table 4.2**), yielded strains of the three *Fusarium* species. In addition to drain swabs, water samples were collected from the taps of sinks in the hospital haematology and oncology units and the main water tanks feeding the ophthalmology unit. The ED7 diagnostic

antigen could not be detected in any of the water samples directly. However, the oncology samples W57 and W60 contained *Fusarium* strains that belonged to FDSC ET-gr. and were detectable by ELISA at the mixed culture stage (**Table 4.3**). The sink biofilms corresponding to these water samples were also positive at the swab ELISA stage (**Table 4.1**).

4.3.2 Identification of Fungi by Analysis of the ITS Regions of the rRNA-Encoding Gene Unit and by Translation Elongation Factor-1α PCR

There was 100% agreement between *Fusarium* genus identification by ELISA and species identification by ITS sequencing (**Table 4.1**). The species of *F. solani* and *F. oxysporum* recovered from sink swabs were subsequently shown by TEF-1α PCR (**Table 4.3**) to belong to FSSC 1-a, 1-c, 2-a, 2-v, 5-d, 5-k, 9-a, 15-a, 20-d and FOSC 16, 33, 99, 111, 126, 134, 183 (**Table 4.3**). All of the recovered *F. dimerum* isolates belonged to the FDSC ET-gr (**Table 4.3**). ITS analysis of axenic cultures (**Table 4.1**) showed that a number of sink samples (e.g. S2, S6, S17, S19, S21, S24, S30, S38) contained mixtures of *Fusarium* species, while ITS and TEF-PCR analysis (**Table 4.1** and **Table 4.3**) showed that others contained mixtures of species complexes of the same species (e.g. S8, S9, S25, S28). Only two of the tap samples (oncology W57 and W60) contained *Fusarium* strains which were shown to belong to FDSC ET-gr.

4.3.3 Frequencies and Distributions of *Fusarium* Species Complexes

By combining the ITS and TEF-PCR data, the dominant species complex recovered from the University sink drain biofilms was shown to be FOSC (47.6%), followed by FSSC (11.2%) and then FDSC (7.4%) (**Figure 4.5 A**). At the RD&E hospital, FDSC (27.1%) was found the most frequent grouping, followed by FSSC (9.3%) and then by FOSC (3.3%) (**Figure 4.5 A**). Different species complexes dominated in the different hospital units tested (**Figure 4.5 B**).

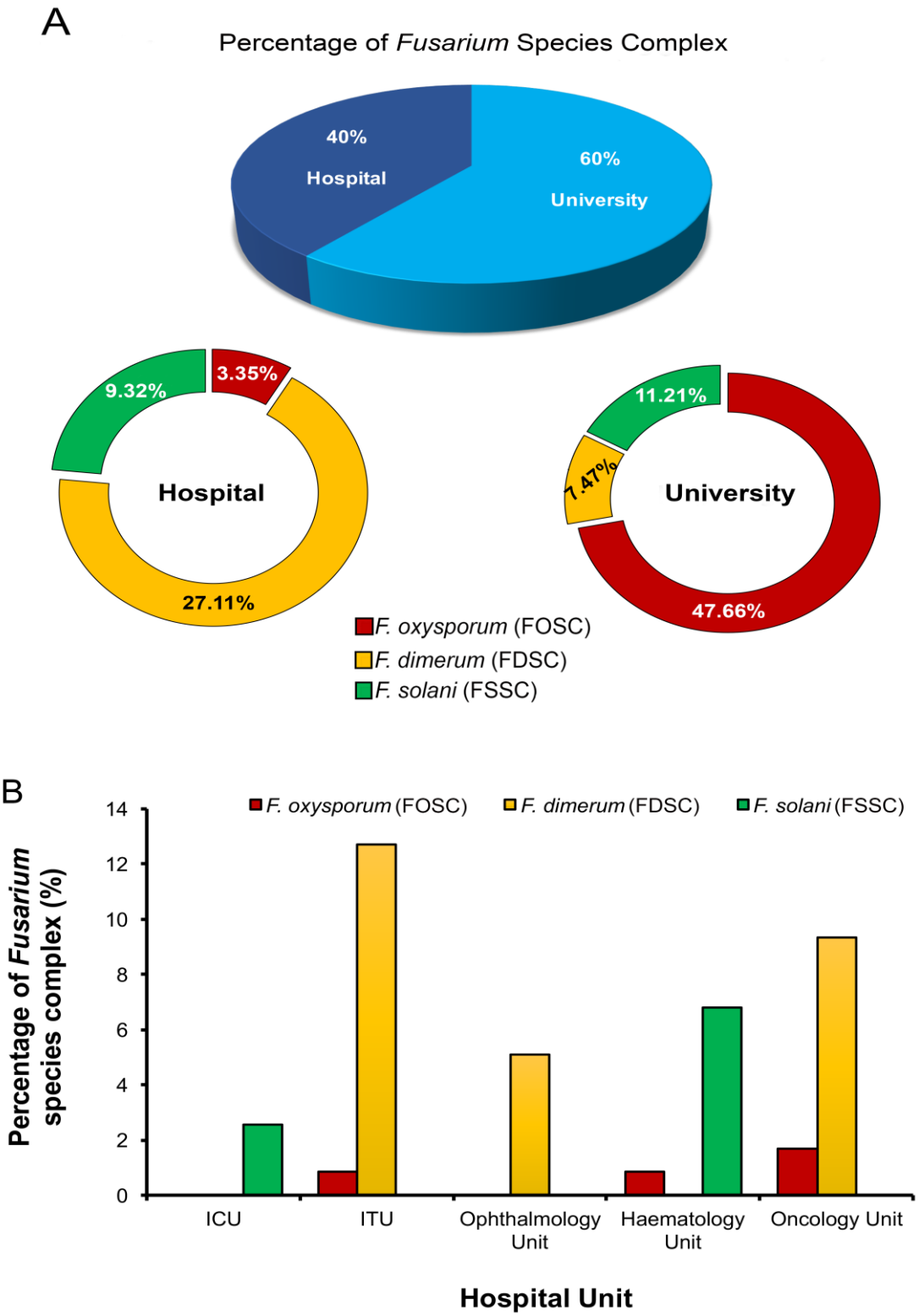


Figure 4.5. Frequency of *F.* species complex recovered from sink drain biofilms based on ITS and TEF-PCR identification. (**A**) depends on collection area and (**B**) depends on RD&E hospital units. The total number of samples is 225. FOSC, *F. oxysporum* species complex; FDSC, *F. dimerum* species complex; FSSC, *F. solani* species complex.

Table 4. 1. Locations and identities of sink swabs and water samples and results of ELISA tests and fungal identification based on ITS sequencing. The ITS sequences corresponding to each isolate are shown in **Appendix 3**.

Sink ID	Location (Source)[a]	Swab ELISA abs (450nm)[b]	Mixed culture ELISA abs (450nm)[b]	Isolate No.	Axenic culture ELISA abs (450nm)[b]	Identification based on ITS sequencing[c]	GenBank Accession No.
A1	University (S1)	0.0763	0.0575	A1-1	1.3736	*Fusarium oxysporum*	KT876668
	University (S1)			A1-2	0.7646	*Fusarium oxysporum*	KT876662
	University (S1)			A1-3	0.0196	*Penicillium crustosum*	KT876719
	University (S1)			A1-4	0.0139	*Penicillium expansum*	KT876718
A2	University (S2)	0.5472	0.0045	A2-1	1.5723	*Fusarium solani*	KT876635
	University (S2)			A2-3	0.6554	*Fusarium oxysporum*	KT876690
	University (S2)			A2-5	1.1312	*Fusarium solani*	KT876631
A3	University (S3)	0.0053	0.0077	A3-1	0.0082	*Trichosporon domesticum*	KT876717
	University (S3)			A3-2	0.0162	*Cadophora fastigiata*	KT876615
	University (S3)			A3-3	0.0014	*Cyphellophora oxyspora*	KT876613
	University (S3)			A3-4	0.0093	*Penicillium crustosum*	KT876714
A4	University (S4)	0.0297	0.0049	A4-1	0.0105	*Phoma herbarum*	KT876697
	University (S4)			A4-2	0.0079	*Penicillium echinulatum*	KT876710
	University (S4)			A4-3	0.0029	*Cytobasidium slooffiae*	KT876704
	University (S4)			A4-6	0.0060	*Trichoderma asperellum*	KT876620
A5	University (S5)	0.0206	0.0039	A5-1	1.1815	*Fusarium oxysporum*	KT876692
	University (S5)			A5-2	0.0034	*Penicillium crustosum*	KT876715
	University (S5)			A5-5	0.0088	*Aspergillus niger*	KT876702
	University (S5)			A5-6	1.0630	*Fusarium oxysporum*	KT876667
	University (S5)			A5-7	0.0037	*Rhodotorula mucilaginosa*	KT876700
	University (S5)			A5-8	0.0088	*Cyphellophora oxyspora*	KT876614
A6	University (S6)	0.0251	0.0412	A6-1	1.1130	*Fusarium oxysporum*	KT876648
	University (S6)			A6-2	0.9410	*Fusarium oxysporum*	KT876678
	University (S6)			A6-3	0.6377	*Fusarium oxysporum*	KT876688
	University (S6)			A6-4	1.0020	*Fusarium solani*	KT876640
A7	University (S7)	0.1716	0.0082	A7-1	0.0098	*Exophiala pisciphila*	KT876618
	University (S7)			A7-2	0.0096	*Penicillium brevicompactum*	KT876695
	University (S7)			A7-3	1.0556	*Fusarium oxysporum*	KT876684
	University (S7)			A7-4	1.0077	*Fusarium oxysporum*	KT876671
A8	University (S8)	0.0431	0.0008	A8-1	1.1045	*Fusarium oxysporum*	KT876672
	University (S8)			A8-2	0.9707	*Fusarium oxysporum*	KT876654

	University (S8)			A8-3	0.0091	*Trichoderma atroviride*	KT876622
A9	University (S9)	0.3806	0.0052	A9-1	0.9087	*Fusarium solani*	KT876639
	University (S9)			A9-2	0.8849	*Fusarium solani*	KT876638
	University (S9)			A9-3	0.9004	*Fusarium solani*	KT876632
	University (S9)			A9-4	0.8093	*Fusarium solani*	KT876636
A10	University (S10)	0.0034	0.0028	A10-1	0.0188	*Rhodotorula slooffiae*	KT876705
	University (S10)			A10-2	0.0020	*Exophiala pisciphila*	KT876616
B1	University (S11)	0.0231	0.0005	B1-1	0.6064	*Fusarium dimerum*	KT876625
	University (S11)			B1-6	0.8138	*Fusarium dimerum*	KT876628
B2	University (S12)	0.0200	0.0020	B2-1	0.5678	*Fusarium dimerum*	KT876626
	University (S12)			B2-5	0.4827	*Fusarium dimerum*	KT876624
B3	University (S13)	0.0091	0.0023	B3-4	0.0026	*Phoma herbarum*	KT876696
B4	University (S14)	0.0163	0.0008	B4-1	0.6992	*Fusarium dimerum*	KT876627
B5	University (S15)	0.0132	0.0003	B5-1	0.8008	*Fusarium oxysporum*	KT876674
	University (S15)			B5-2	0.0048	*Mucor circinelloides*	KT876701
	University (S15)			B5-3	0.8851	*Fusarium oxysporum*	KT876677
B6	University (S16)	0.0229	0.0002	B6-1	0.8193	*Fusarium oxysporum*	KT876676
	University (S16)			B6-2	0.7582	*Fusarium oxysporum*	KT876661
B7	University (S17)	0.0395	0.0014	B7-1	0.8201	*Fusarium oxysporum*	KT876675
	University (S17)			B7-6	0.7758	*Fusarium dimerum*	KT876623
B8	University (S18)	0.0133	0.0011	B8-1	0.7347	*Fusarium solani*	KT876637
	University (S18)			B8-7	0.0095	*Rhodosporidium babjevae*	KT876706
B9	University (S19)	0.0212	0.0048	B9-1	0.0051	*Meyerozyma guilliermondii*	KT876707
	University (S19)			B9-2	0.8104	*Fusarium oxysporum*	KT876657
	University (S19)			B9-3	0.6302	*Fusarium solani*	KT876634
	University (S19)			B9-5	0.0017	*Penicillium crustosum*	KT876720
B10	University (S20)	0.5467	0.0394	B10-1	0.7467	*Fusarium oxysporum*	KT876680
	University (S20)			B10-2	0.7667	*Fusarium oxysporum*	KT876659
	University (S20)			B10-6	0.0071	*Candida parapsilosis*	KT876703
	University (S20)			B10-7	0.0251	*Meyerozyma guilliermondii*	KT876711
	University (S20)			B10-9	0.0031	*Cystobasidium slooffiae*	KT876712
	University (S20)			B10-12	0.0251	*Trichosporon asteroides*	KT876713
C1	University (S21)	0.0083	0.0269	C1-1	0.0044	*Clavispora lusitaniae*	KT876708
	University (S21)			C1-3	0.8875	*Fusarium oxysporum*	KT876682
	University (S21)			C1-4	0.8884	*Fusarium dimerum*	KT876629
	University (S21)			C1-7	0.0003	*Exophiala pisciphila*	KT876617
C2	University (S22)	0.2977	0.0047	C2-1	0.9443	*Fusarium oxysporum*	KT876658
	University (S22)			C2-4	0.9825	*Fusarium oxysporum*	KT876694
	University (S22)			C2-7	0.8677	*Fusarium oxysporum*	KT876687
C3	University (S23)	0.0808	0.0116	C3-1	0.9853	*Fusarium oxysporum*	KT876683
	University (S23)			C3-2	1.0160	*Fusarium oxysporum*	KT876693
	University (S23)			C3-4	0.7485	*Fusarium oxysporum*	KT876644
C4	University (S24)	0.1113	0.0202	C4-1	0.8930	*Fusarium oxysporum*	KT876670

	University (S24)			C4-2	0.0028	*Candida intermedia*	KT876709
	University (S24)			C4-5	0.9008	*Fusarium dimerum*	KT876630
C5	University (S25)	0.5741	0.0097	C5-1	0.9874	*Fusarium oxysporum*	KT876652
	University (S25)			C5-2	1.0996	*Fusarium oxysporum*	KT876656
	University (S25)			C5-3	1.1236	*Fusarium oxysporum*	KT876649
	University (S25)			C5-4	1.0355	*Fusarium oxysporum*	KT876669
	University (S25)			C5-5	1.0672	*Fusarium oxysporum*	KT876660
C6	University (S26)	0.0603	0.0348	C6-1	1.0135	*Fusarium oxysporum*	KT876665
	University (S26)			C6-2	0.9362	*Fusarium oxysporum*	KT876681
	University (S26)			C6-3	0.8898	*Fusarium oxysporum*	KT876673
	University (S26)			C6-4	1.0577	*Fusarium oxysporum*	KT876686
	University (S26)			C6-5	0.9445	*Fusarium oxysporum*	KT876646
C7	University (S27)	0.1279	0.0113	C7-1	0.9724	*Fusarium oxysporum*	KT876666
C8	University (S28)	0.0607	0.0035	C8-1	0.8694	*Fusarium oxysporum*	KT876685
	University (S28)			C8-2	0.9599	*Fusarium oxysporum*	KT876655
	University (S28)			C8-3	0.0031	*Penicillium expansum*	KT876716
	University (S28)			C8-4	0.0049	*Phoma herbarum*	KT876698
	University (S28)			C8-6	0.9003	*Fusarium oxysporum*	KT876679
C9	University (S29)	0.0310	0.0001	C9-1	1.0488	*Fusarium oxysporum*	KT876651
	University (S29)			C9-2	0.9254	*Fusarium oxysporum*	KT876645
	University (S29)			C9-3	0.9711	*Fusarium oxysporum*	KT876664
	University (S29)			C9-4	1.035	*Fusarium oxysporum*	KT876647
	University (S29)			C9-5	0.9604	*Fusarium oxysporum*	KT876663
C10	University (S30)	0.1002	0.0333	C10-1	0.9254	*Fusarium oxysporum*	KT876689
	University (S30)			C10-2	1.0198	*Fusarium solani*	KT876642
	University (S30)			C10-4	0.9007	*Fusarium solani*	KT876641
	University (S30)			C10-7	1.0697	*Fusarium solani*	KT876643
CRT-1	University (S31)	0.6175	0.1129	CRT1-1	0.4639	*Fusarium oxysporum*	KT876691
	University (S31)			CRT1-2	0.9086	*Fusarium oxysporum*	KT876650
	University (S31)			CRT1-3	0.7503	*Fusarium oxysporum*	KT876652
CRT-2	University (S32)	0.0303	0.0021	CRT2-1	0.0040	*Trichoderma asperellum*	KT876619
	University (S32)			CRT2-2	0.0417	*Trichoderma asperellum*	KT876621
CRT-3	University (S33)	0.0663	0.0028	CRT3-1	0.0256	*Phoma herbarum*	KT876699
R1	ICU (S34)	0.0285	0.3620	R1-1	1.5057	*Fusarium solani*	KT876550
	ICU (S34)			R1-2	1.5154	*Fusarium solani*	KT876551
	ICU (S34)			R1-3	1.5555	*Fusarium solani*	KT876549
R2	ICU (S35)	0.0071	0.0147	R2-4	0.0096	*Clonostachys rosea*	KT876552
	ICU (S35)			R2-5	0.0018	*Clonostachys rosea*	KT876553
	ICU (S35)			R2-6	0.0035	*Clonostachys rosea*	KT876554
R3	ICU (S36)	0.0391	0.045	R3-1	0.0006	*Trichoderma asperellum*	KT876548
R5	ITU (S37)	0.7291	0.7724	R5-1	1.5606	*Fusarium dimerum*	KT876561
	ITU (S37)			R5-2	1.3481	*Fusarium dimerum*	KT876565
	ITU (S37)			R5-3	1.4990	*Fusarium dimerum*	KT876567
R6	ITU (S38)	1.2086	0.3691	R6-1	1.3388	*Fusarium dimerum*	KT876572
	ITU (S38)			R6-2	1.4734	*Fusarium dimerum*	KT876563
	ITU (S38)			R6-3	1.4312	*Fusarium dimerum*	KT876562
	ITU (S38)			R6-9	1.2648	*Fusarium oxysporum*	KT876557

R7	ITU (S39)	0.1121	0.8186	R7-1	1.3266	*Fusarium dimerum*	KT876570
	ITU (S39)			R7-2	1.2352	*Fusarium dimerum*	KT876564
R8	ITU (S40)	1.0157	0.3612	R8-1	1.2399	*Fusarium dimerum*	KT876568
	ITU (S40)			R8-2	1.1552	*Fusarium dimerum*	KT876566
	ITU (S40)			R8-3	1.1856	*Fusarium dimerum*	KT876558
R9	ITU (S41)	0.0391	0.0477	R9-3	0.0113	*Exophiala phaeomuriformis*	KT876555
R10	ITU (S42)	0.0549	0.0097	R10-2	0.0072	*Cadophora fastigiata*	KT876556
	ITU (S42)			R10-6	0.0124	*Coniochaeta fasciculata*	KT876721
X2	ITU (S43)	0.0630	1.5751	X2-2	1.1492	*Fusarium dimerum*	KT876571
	ITU (S43)			X2-4	1.2505	*Fusarium dimerum*	KT876560
	ITU (S43)			X2-5	1.1397	*Fusarium dimerum*	KT876569
	ITU (S43)			X2-6	1.1716	*Fusarium dimerum*	KT876559
X3	Ophthalmology Unit (S44)	1.0313	1.5035	X3-1	1.1353	*Fusarium dimerum*	KT876509
	Ophthalmology Unit (S44)			X3-2	0.0224	*Candida parapsilosis*	KT876498
	Ophthalmology Unit (S44)			X3-3	1.1856	*Fusarium dimerum*	KT876512
X4	Ophthalmology Unit (S45)	1.4582	1.4049	X4-1	1.1322	*Fusarium dimerum*	KT876511
	Ophthalmology Unit (S45)			X4-2	1.1523	*Fusarium dimerum*	KT876500
	Ophthalmology Unit (S45)			X4-3	0.0622	*Candida parapsilosis*	KT876508
	Ophthalmology Unit (S45)			X4-4	0.1245	*Gloeotinia temulenta*	KT876515
X5	Ophthalmology Unit (S46)	0.3325	1.4466	X5-2	1.0654	*Fusarium dime rum*	KT876510
	Ophthalmology Unit (S46)			X5-3	0.0599	*Rhodotorula mucilaginosa*	KT876501
	Ophthalmology Unit (S46)			X5-4	1.0862	*Fusarium dimerum*	KT876513
X6	Ophthalmology Unit (S47)	0.2640	1.3729	X6-1	0.0616	*Candida paraphilia's*	KT876499
	Ophthalmology Unit (S47)			X6-2	0.063	*Engyodontium album*	KT876522
	Ophthalmology Unit (S47)			X6-3	0.0603	*Engyodontium album*	KT876521
	Ophthalmology Unit (S47)			X6-4	0.0657	*Exophiala phaeomuriformis*	KT876504
X8	Ophthalmology Unit (S48)	0.5299	0.0223	X8-2	0.0118	*Cladosporium macrocarpum*	KT876506
	Ophthalmology Unit (S48)			X8-3	0.0985	*Gloeotinia temulenta*	KT876514
	Ophthalmology Unit (S48)			X8-4	0.0662	*Engyodontium album*	KT876523
X9	Ophthalmology Unit (S49)	0.1872	0.0061	X9-1	0.0686	*Engyodontium album*	KT876520
	Ophthalmology Unit (S49)			X9-2	0.0611	*Candida parapsilosis*	KT876496
	Ophthalmology Unit (S49)			X9-3	0.0641	*Candida parapsilosis*	KT876497
	Ophthalmology Unit (S49)			X9-4	0.0677	*Exophiala dermatitidis*	KT876503
	Ophthalmology Unit (S49)			X9-5	0.0705	*Engyodontium album*	KT876519

	Ophthalmology Unit (S49)			X9-7	0.0660	*Exophiala pisciphila*	KT876502
X10	Ophthalmology Unit (T1)	0.0132	0.0159	X10-1	0.0868	*Cladosporium sphaerospermum*	KT876507
	Ophthalmology Unit (T1)			X10-3	0.0130	*Engyodontium album*	KT876518
E1	Ophthalmology Unit (T2)	0.0117	0.0883	W1-1	0.0114	*Paraconiothyrium fuckelii*	KT876505
	Ophthalmology Unit (T2)			W1-2	0.0139	*Engyodontium album*	KT876517
	Ophthalmology Unit (T2)			W1-3	0.0108	*Engyodontium album*	KT876516
K1	Haematology Unit (S50)	0.2694	0.0093	K1-1	0.0172	*Trichoderma asperellum*	KT876534
K2	Haematology Unit (S51)	0.7466	1.1746	K2-1	0.0144	*Trichoderma viride*	KT876533
	Haematology Unit (S51)			K2-3	1.1385	*Fusarium solani*	KT876543
	Haematology Unit (S51)			K2-4	1.2487	*Fusarium solani*	KT876542
K6	Haematology Unit (S52)	1.0621	1.3090	K6-1	1.1544	*Fusarium oxysporum*	KT876541
	Haematology Unit (S52)			K6-4	0.0071	*Candida parapsilosis*	KT876524
K8	Haematology Unit (S53)	1.0120	1.2628	K8-1	1.2376	*Fusarium solani*	KT876722
	Haematology Unit (S53)			K8-2	1.2983	*Fusarium solani*	KT876723
	Haematology Unit (S53)			K8-6	0.0084	*Cadophora fastigiata*	KT876531
	Haematology Unit (S53)			K8-7	0.0051	*Trichoderma asperellum*	KT876535
K9	Haematology Unit (S54)	0.9843	1.2903	K9-1	0.0046	*Candida parapsilosis*	KT876525
	Haematology Unit (S54)			K9-2	0.0090	*Rhodotorula dairenensis*	KT876526
	Haematology Unit (S54)			K9-4	0.0073	*Cadophora fastigiata*	KT876532
	Haematology Unit (S54)			K9-5	1.1652	*Fusarium solani*	KT876547
	Haematology Unit (S54)			K9-6	1.0316	*Fusarium solani*	KT876545
K10	Haematology Unit (S55)	0.8638	1.0753	K10-1	1.0684	*Fusarium solani*	KT876546
	Haematology Unit (S55)			K10-2	1.0386	*Fusarium solani*	KT876544
	Haematology Unit (W50)	0.0082	0.0451	G1-2	0.0097	*Exophiala pisciphila*	KT876529
G2	Haematology Unit (W51)	0.0581	0.0553	G2-1	0.0104	*Engyodontium album*	KT876540
	Haematology Unit (W51)			G2-3	0.0062	*Exophiala pisciphila*	KT876530
G7	Haematology Unit (W52)	0.0125	0.0118	G6-2	0.0161	*Exophiala castellanii*	KT876528
G8	Haematology Unit (W53)	0.0075	0.0219	G8-1	0.0065	*Engyodontium album*	KT876538
G9	Haematology Unit (W54)	0.0002	0.0164	G9-2	0.0005	*Gloeotinia temulenta*	KT876537
G10	Haematology Unit (W55)	0.0021	0.0182	G10-1	0.0051	*Engyodontium album*	KT876539

H1	Oncology Unit (S56)	0.4822	1.2287	H1-1	0.9022	*Fusarium dimerum*	KT876595
	Oncology Unit (S56)			H1-3	0.0151	*Pichia kudriavzevii*	KT876578
H2	Oncology Unit (S57)	1.0579	1.1650	H2-1	0.9312	*Fusarium dimerum*	KT876590
	Oncology Unit (S57)			H2-5	0.0061	*Magnusiomyces capitatus*	KT876611
H3	Oncology Unit (S58)	1.0938	1.2091	H3-1	0.8909	*Fusarium oxysporum*	KT876584
	Oncology Unit (S58)			H3-4	0.0075	*Candida palmioleophila*	KT876573
	Oncology Unit (S58)			H3-5	0.0140	*Rhodotorula glutinis*	KT876598
H4	Oncology Unit (S59)	0.0253	1.0856	H4-1	0.8884	*Fusarium dimerum*	KT876596
	Oncology Unit (S59)			H4-3	0.0004	*Magnusiomyces capitatus*	KT876612
H5	Oncology Unit (S60)	1.0856	1.0193	H5-1	1.1148	*Fusarium dimerum*	KT876593
	Oncology Unit (S60)			H5-3	0.005	*Candida tropicalis*	KT876574
H6	Oncology Unit (S61)	0.5256	1.1898	H6-1	1.1238	*Fusarium dimerum*	KT876594
H7	Oncology Unit (S62)	1.0789	1.1588	H7-1	1.1513	*Fusarium oxysporum*	KT876583
	Oncology Unit (S62)			H7-3	0.0045	*Candida tropicalis*	KT876575
H8	Oncology Unit (S63)	0.0953	0.0152	H8-1	0.0063	*Phoma herbarum*	KT876580
	Oncology Unit (S63)			H8-3	0.0126	*Candida albicans*	KT876577
	Oncology Unit (S63)			H8-4	0.0088	*Rhodotorula mucilaginosa*	KT876599
H9	Oncology Unit (S64)	0.1926	0.0162	H9-1	0.0152	*Exophiala dermatitidis*	KT876581
	Oncology Unit (S64)			H9-2	0.0049	*Candida orthopsilosis*	KT876576
	Oncology Unit (S64)			H9-3	0.0109	*Pichia kudriavzevii*	KT876579
H10	Oncology Unit (S65)	0.4961	1.1005	H10-1	0.9606	*Fusarium dimerum*	KT876589
	Oncology Unit (S65)			H10-4	0.0163	*Trametes ochracea*	KT876608
P1	Oncology Unit (W56)	0.0030	0.0247	P1-2	0.0082	*Exophiala dermatitidis*	KT876582
	Oncology Unit (W56)			P1-3	0.0024	*Trametes versicolor*	KT876603
P2	Oncology Unit (W57)	0.0061	1.0891	P2-1	0.8451	*Fusarium dimerum*	KT876587
	Oncology Unit (W57)			P2-2	0.9429	*Fusarium dimerum*	KT876597
	Oncology Unit (W57)			P2-3	0.8627	*Fusarium dimerum*	KT876588
P3	Oncology Unit (W58)	0.0016	0.0202	P3-1	0.0177	*Trametes ochracea*	KT876607
P4	Oncology Unit (W59)	0.0035	0.0125	P4-1	0.0164	*Beauveria bassiana*	KT876586
	Oncology Unit (W59)			P4-2	0.0084	*Gliomastix polychroma*	KT876584

P5	Oncology Unit (W60)	0.0025	1.1746	P5-1	0.8563	*Fusarium dimerum*	KT876591
	Oncology Unit (W60)			P5-2	0.8066	*Fusarium dimerum*	KT876592
	Oncology Unit (W60)			P5-3	0.0024	*Piptoporus betulinus*	KT876609
P7	Oncology Unit (W62)	0.0011	0.0417	P7-1	0.0097	*Trametes versicolor*	KT876605
P8	Oncology Unit (W63)	0.0044	0.0060	P8-1	0.0144	*Trametes versicolor*	KT876606
	Oncology Unit (W63)			P8-2	0.0106	*Stereum gausapatum*	KT876601
P9	Oncology Unit (W64)	0.0022	0.0163	P9-1	0.0101	*Stereum gausapatum*	KT876600
	Oncology Unit (W64)			P9-2	0.0068	*Trametes versicolor*	KT876604
	Oncology Unit (W64)			P9-3	0.0140	*Stereum gausapatum*	KT876602
P10	Oncology Unit (W65)	0.0051	0.0280	P10-1	0.0032	*Phaeophlebiopsis peniophoroides*	KT876610

a. S, sink; W, tap water from corresponding sink number; T, water sample from main tank.

b. Threshold absorbance value for detection in ELISA ≥0.100. Shading indicates earliest point in sampling process at which antigen was detectable in ELISA tests with ED7.

c. *Fusarium* strains in bold further characterized by TEF-1α PCR analysis (**Table 4.3** and **Appendix 4**).

Table 4. 2. Summary of ELISA tests and mycological culture of sink swabs.

Total no. sinks swabbed	No. swab samples yielding fungi after mycological culture (%)[a]	No. samples positive for *Fusarium* antigen at Swab-ELISA stage (%)	No. samples positive for *Fusarium* antigen by Mixed culture-ELISA stage (%)	No. samples positive for *Fusarium* antigen by Axenic culture-ELISA stage (%)	No. antigen-positive samples yielding *Fusarium* spp. after mycological culture (%)	No. antigen-positive samples not yielding *Fusarium* spp. after mycological culture (%)
65	65 (100%)	34 (52%)	37 (57%)	54 (83%)	50 (93%)	4 (7%)

a. *Fusarium* spp. and/or unrelated fungi.

Table 4. 3. Summary of species complex identities following TEF-1α PCR analysis of *Fusarium* isolates recovered from sinks. The TEF-1α gene sequences corresponding to each isolate are shown in **Appendix 4**.

Isolate No.	Location	TEF-1α ID
A1-1	University	*F. oxysporum* species complex 33
A1-2	University	*F. oxysporum* species complex 33
A2-1	University	*F. solani* species complex 1-a
A2-3	University	*F. oxysporum* species complex 33
A2-5	University	*F. solani* species complex 1-a
A5-1	University	*F. oxysporum* species complex 33
A5-6	University	*F. oxysporum* species complex 33
A6-1	University	*F. oxysporum* species complex 33
A6-2	University	*F. oxysporum* species complex 33
A6-3	University	*F. oxysporum* species complex 33
A6-4	University	*F. solani* species complex 9-a
A7-3	University	*F. oxysporum* species complex 183
A7-4	University	*F. oxysporum* species complex 126
A8-1	University	*F. oxysporum* species complex 33
A8-2	University	*F. oxysporum* species complex 33
A9-1	University	*F. solani* species complex 5-d
A9-2	University	*F. solani* species complex 5-d
A9-3	University	*F. solani* species complex 1-c
A9-4	University	*F. solani* species complex 1-a
B1-1	University	*F. dimerum* species complex ET-gr.
B1-6	University	*F. dimerum* species complex ET-gr.
B2-1	University	*F. dimerum* species complex ET-gr.
B2-5	University	*F. dimerum* species complex ET-gr.
B4-1	University	*F. dimerum* species complex ET-gr.
B5-1	University	*F. oxysporum* species complex 16
B5-3	University	*F. oxysporum* species complex 16
B6-1	University	*F. oxysporum* species complex 16
B6-2	University	*F. oxysporum* species complex 16
B7-1	University	*F. oxysporum* species complex 16
B7-6	University	*F. dimerum* species complex ET-gr.

B8-1	University	*F. solani* species complex 15-a
B9-2	University	*F. oxysporum* species complex 99
B9-3	University	*F. solani* species complex 1-a
B10-1	University	*F. oxysporum* species complex 33
B10-2	University	*F. oxysporum* species complex 33
C1-3	University	*F. oxysporum* species complex 33
C1-4	University	*F. dimerum* species complex ET-gr.
C2-1	University	*F. oxysporum* species complex 33
C2-4	University	*F. oxysporum* species complex 33
C2-7	University	*F. oxysporum* species complex 33
C3-1	University	*F. oxysporum* species complex 134
C3-2	University	*F. oxysporum* species complex 134
C3-4	University	*F. oxysporum* species complex 134
C4-1	University	*F. oxysporum* species complex 33
C4-5	University	*F. dimerum* species complex ET-gr.
C5-1	University	*F. oxysporum* species complex 33
C5-2	University	*F. oxysporum* species complex 126
C5-3	University	*F. oxysporum* species complex 33
C5-4	University	*F. oxysporum* species complex 33
C5-5	University	*F. oxysporum* species complex 33
C6-1	University	*F. oxysporum* species complex 33
C6-2	University	*F. oxysporum* species complex 33
C6-3	University	*F. oxysporum* species complex 33
C6-4	University	*F. oxysporum* species complex 33
C6-5	University	*F. oxysporum* species complex 33
C7-1	University	*F. oxysporum* species complex 134
C8-1	University	*F. oxysporum* species complex 134
C8-2	University	*F. oxysporum* species complex 134
C8-6	University	*F. oxysporum* species complex 33
C9-1	University	*F. oxysporum* species complex 134
C9-2	University	*F. oxysporum* species complex 134
C9-3	University	*F. oxysporum* species complex 134
C9-4	University	*F. oxysporum* species complex 134
C9-5	University	*F. oxysporum* species complex 134
C10-1	University	*F. oxysporum* species complex 111
C10-2	University	*F. solani* species complex 2-v
C10-4	University	*F. solani* species complex 2-v

C10-7	University	*F. solani* species complex 2-v
CRT1-1	University	*F. oxysporum* species complex 33
CRT1-2	University	*F. oxysporum* species complex 33
CRT1-3	University	*F. oxysporum* species complex 33
R1-1	ICU	*F. solani* species complex 1-a
R1-2	ICU	*F. solani* species complex 1-a
R1-3	ICU	*F. solani* species complex 1-a
R5-1	ITU	*F. dimerum* species complex ET-gr.
R5-2	ITU	*F. dimerum* species complex ET-gr.
R5-3	ITU	*F. dimerum* species complex ET-gr.
R6-1	ITU	*F. dimerum* species complex ET-gr.
R6-2	ITU	*F. dimerum* species complex ET-gr.
R6-3	ITU	*F. dimerum* species complex ET-gr.
R6-9	ITU	*F. solani* species complex 20-d
R7-1	ITU	*F. dimerum* species complex ET-gr.
R7-2	ITU	*F. dimerum* species complex ET-gr.
R8-1	ITU	*F. dimerum* species complex ET-gr.
R8-2	ITU	*F. dimerum* species complex ET-gr.
R8-3	ITU	*F. dimerum* species complex ET-gr.
X2-2	ITU	*F. dimerum* species complex ET-gr.
X2-4	ITU	*F. dimerum* species complex ET-gr.
X2-5	ITU	*F. dimerum* species complex ET-gr.
X2-6	ITU	*F. dimerum* species complex ET-gr.
X3-1	Ophthalmology Unit	*F. dimerum* species complex ET-gr.
X3-3	Ophthalmology Unit	*F. dimerum* species complex ET-gr.
X4-1	Ophthalmology Unit	*F. dimerum* species complex ET-gr.
X4-2	Ophthalmology Unit	*F. dimerum* species complex ET-gr.
X5-2	Ophthalmology Unit	*F. dimerum* species complex ET-gr.
X5-4	Ophthalmology Unit	*F. dimerum* species complex ET-gr.
K2-3	Haematology Unit	*F. solani* species complex 5-k
K2-4	Haematology Unit	*F. solani* species complex 5-k
K6-1	Haematology Unit	*F. oxysporum* species complex 33
K8-1	Haematology Unit	*F. solani* species complex 5-k
K8-2	Haematology Unit	*F. solani* species complex 5-k
K9-5	Haematology Unit	*F. solani* species complex 2-a
K9-6	Haematology Unit	*F. solani* species complex 2-a
K10-1	Haematology Unit	*F. solani* species complex 2-a

K10-2	Haematology Unit	*F. solani* species complex 2-a
H1-1	Oncology Unit	*F. dimerum* species complex ET-gr.
H2-1	Oncology Unit	*F. dimerum* species complex ET-gr.
H3-1	Oncology Unit	*F. oxysporum* species complex 33
H4-1	Oncology Unit	*F. dimerum* species complex ET-gr.
H5-1	Oncology Unit	*F. dimerum* species complex ET-gr.
H6-1	Oncology Unit	*F. dimerum* species complex ET-gr.
H7-1	Oncology Unit	*F. oxysporum* species complex 33
H10-1	Oncology Unit	*F. dimerum* species complex ET-gr.
P2-1	Oncology Unit	*F. dimerum* species complex ET-gr.
P2-2	Oncology Unit	*F. dimerum* species complex ET-gr.
P2-3	Oncology Unit	*F. dimerum* species complex ET-gr.
P5-1	Oncology Unit	*F. dimerum* species complex ET-gr.
P5-2	Oncology Unit	*F. dimerum* species complex ET-gr.

4.4. Discussion

The genus *Fusarium* comprises ubiquitous environmental moulds capable of infecting plants and humans (Zhang *et al*., 2006). Unlike agriculture, where the most economically damaging pathogens are considered to be *F. graminearum* and *F. oxysporum* (Dean *et al*., 2012), the species most commonly cited as human pathogens belong to the FSSC, responsible for 50% of reported infections in humans, followed by strains in the FOSC (Torres and Kontoyiannis, 2011). The FDSC is less frequently reported as causing human disease, but it is similarly capable of causing disseminated infections in immunocompromised patients (Bigley *et al*., 2004; Schroers *et al*., 2009).

While the natural habitats of plant pathogenic *Fusarium* strains are well characterized as soil and decaying plant material, habitats providing direct human exposure to infectious propagules are largely unexplored. The increasing frequency of opportunistic fungal infections in humans means that improved surveillance methods are needed to identify environmental reservoirs of pathogens to limit the exposure of vulnerable individuals to potentially infective propagules. For *Fusarium*, there is a growing body of evidence to suggest that domestic and municipal water systems are potential reservoirs of human pathogenic strains in the FSSC, FOSC and FDSC groups (Short *et al*., 2011).

Accurate techniques that can be used to identify the fungus in environmental samples containing mixed populations of fungi are currently lacking and, while nucleic acid-based technologies have been developed for the differentiation of *Fusarium* from other human pathogenic species and to identify *Fusarium*

species complexes, such techniques have typically been used in retrospective analysis of axenic cultures collected during human and environmental population studies (Bouchara *et al*., 2009; Steinmann *et al*., 2011; Lackner *et al*., 2012). Furthermore, these studies have often employed *Fusarium*-selective medium that eliminate other fungi present in polymicrobial communities (Short *et al*., 2011). While mAbs and antibody fragments have been developed for detecting and differentiating *Fusarium* species *in vitro* or *in planta* (Wong *et al*., 1988; Arie *et al*., 1991, 1995; Danks *et al*., 1996; Hayashi *et al*., 1998; Hu *et al*., 2012, 2013), no attempts have been made to use mAbs to track human pathogenic strains in environmental samples. Jensen *et al*. (2011) recently reported the development of *Fusarium*-specific mAbs for immunohistochemical diagnosis of fusariosis. The IgM mAbs, which recognise 51 and 63 kDa antigens, reacted strongly with fungal elements in both experimentally infected animals and biopsy samples from patients with fusariosis sepsis and dissemination to the skin.

In this prospective study, we set out to determine whether human pathogenic species of *Fusarium* could be identified in sink drains directly by using crude antigen extracts of biofilms and detection using a genus-specific IgM mAb, ED7, that binds to an extracellular ~ 200kDa carbohydrate antigen present on the surface of spores and hyphae (**Chapter 2**). While the function of the antigen is currently unknown we were able, in ELISA tests, to detect its presence in 52% of swab samples and, following biological amplification of biofilms on a non-selective mycological medium, were able to identify additional biofilm samples containing pathogenic strains of *Fusarium*. This is the first time, to our knowledge, that the mAb-based detection method has been used to track

Fusarium in environmental samples. The mAb was able to differentiate *Fusarium* from a broad spectrum of unrelated fungi, including the human pathogens *Aspergillus* (Thornton and Wills, 2015), *Candida*, *Geotrichum*, *Rhodotorula* and *Trichosporon* (Davies and Thornton, 2014; Miceli *et al*., 2011), *Cyphellophora* and *Phialophora* (Feng *et al*., 2014), *Exophiala* (Zeng *et al*., 2007), *Trichoderma* (Sandoval-Denis *et al*., 2014), *Engyodontium* (Macêdo *et al*., 2007; Thamke *et al*., 2015) and *Mucor* (Petrikkos *et al*., 2012), several of which have been reported previously to inhabit biofilms in water distribution systems (Dogget, 2000). The 100% accuracy of the ED7 ELISA, confirmed by using ITS sequencing and TEF PCR analysis of recovered isolates, demonstrates its robustness in detecting potentially infectious *Fusarium* species in polymicrobial communities. Importantly, ED7 reacted with all of the species complex strains isolated including the most common clinical pathotypes of *Fusarium,* FSSC 1-a, FOSC 33 and FDSC ET-gr (Schroers *et al*., 2009; Short *et al*., 2011).

While the ED7 ELISA was able to identify *Fusarium* to the level of genus only, the simplicity of the mAb-based approach to detection, even when combined with a standard mycological isolation procedure, means that a recognised environmental niche of this group of pathogenic fungi can be monitored readily. The widespread occurrence of human pathogenic *Fusarium* species in sinks of a tertiary care hospital and sinks of a heavily populated university campus, show that indoor plumbing-associated biofilms and water sources are an unseen source of *Fusarium* infectious propagules for nosocomial and community-acquired infections of vulnerable individuals, an observation consistent with previous studies (Annaisie *et al*., 2011; Short *et al*., 2011). While

no cases of fusariosis were reported during the course of this study, the close proximity of the patients to hospital sinks colonised with both pathogenic fusaria and with other opportunistic fungal pathogens is a serious concern given the known vulnerability of immunocompromised individuals to invasive fungal infections.

Summary of Key Findings:

1. The mAb ED7 is 100% accurate in detecting *Fusarium* species antigens by ELISA in mixed community of fungal biofilms with no cross-react with unrelated human pathogenic fungi or yeasts.

2. Three major human pathogenic *Fusarium* species belong to *F. solani*, *F. oxysporum* and *F. dimerum* were isolated at 52% out of a total number 225 of fungi recovered from sink swabs and water system samples that collected across a University campus and tertiary care hospital units.

3. The ITS sequencing 100% validated identification of *Fusarium* genus by ELISA.

4. The TEF-1α analysis data for identification of *Fusarium* at species complex level showed that FSSC 1-a, FOSC 33 and FDSC ET-gr are the most clinical frequent pathotypes recovered from swabs and water samples.

5. The dominant species complex recovered from the University sink drain biofilms related to FOSC (47.6%), followed by FSSC (11.2%) and then FDSC (7.4%). At the RD&E hospital, FDSC (27.1%) was found the most frequent grouping, followed by FSSC (9.3%) and then by FOSC (3.3%).

Chapter 5. General Discussion

5.1 Epidemiology of fungal infections

Over the past decade, the incidence of disseminated infections caused by opportunistic fungal pathogens has increased in line with the ever expanding populations of at-risk patients including BMT and SOT recipients, AIDS patients and those receiving broad-spectrum antibiotics, high-dose corticosteroids and aggressive anti-cancer treatments (Badiee *et al*., 2009; Crabol *et al*., 2014; Slavin & Grist, 2013).

Aspergillus and *Candida* species are the most frequently cited opportunistic pathogens of immune impaired humans (Menzin *et al*., 2009; Nucci & Marr, 2005; Pagano *et al*., 2006; Pfaller *et al*., 2006) which, along with other moulds and yeasts, are responsible for high rates of mortality and morbidity in susceptible patient populations (Dictar *et al.*, 2000; Menzin *et al*., 2009; Montagna *et al.*, 2013). Indeed, *Fusarium* species are the second most common opportunistic fungi after *Aspergillus*, involved in superficial infections of the skin and eyes but also as invasive pathogens of the severely immunocompromised (Boutati & Anaissie, 1997; Nucci *et al*., 2004; van Diepeningen *et al*., 2015). The principle portal of entry of *Fusarium* infectious propagules is the respiratory tract through the inhalation of indoor or outdoor air laden with air-borne conidia, the skin, and the digestive system through ingestion of contaminated water or food (Araujo *et al*., 2010; Legrand *et al*., 1991; Lockhart *et al*., 2009; Nucci & Anaissie, 2007). *Fusarium* species cause a range of infections including dermatomycosis, keratitis, onychomycosis for both immunocompetent and impaired immune system individuals such neutropenic patients (Brasch & Köppl, 2009; Brasch & Shimanovich, 2012;

Chang *et al*., 2006; Cuero, 1980; Girmenia *et al*., 1992; Godoy *et al*., 2004; Gungel *et al*., 2011; Sekeroglu *et al*., 2012; Verma & Tuft, 2002; Zhang *et al*., 2006). Invasive fusariosis among immunocompromised patients with haematological disorders commonly associated with relatively high mortality rate 50% to ~90% (Dignani & Anaissie, 2004; Horn *et al*., 2014; Hsiue *et al*., 2010; Stempel *et al*., 2015).

The epidemiology of fusariosis infections varies and corresponding to many factors such the host health situation and geographic region (Garnica & Nucci, 2013). Given the ubiquity of the *Fusarium* species in the environment, climate factors such wind and rain are more likely play an effective way in the *Fusarium* spores dispersion by which fusariosis may potentially be acquired in the clinic or non-clinic areas (Dignani & Anaissie, 2004; Garnica & Nucci, 2013; Palmero *et al.*, 2010). In prospective studies, pathogenic *Fusarium* species in the USA were recovered from a hospital water system including sink biofilms, tap water and water storage tanks, shower and sink drains, shower heads and sink faucet aerators from hospital air and other environments (Anaissie *et al*., 2001; Short *et al*., 2011). Recently, similar this observation has been reported at a tertiary care hospital in the UK (Al-Maqtoofi & Thornton, 2016). More than 70% of clinical *Fusarium* species that isolated from water system in the hospitals were genetically similar to those isolated from the environment and that suggested the geographical widespread (O'Donnell *et al.*, 2004).

5.2 Diagnosis and Treatment of *Fusarium* Infections

Diagnosis and treatment of fusariosis in immunocompromised patients is a real challenge due to the lack of accurate diagnostic test and resistance to practically all available antifungal drugs (Muhammed *et al*., 2011; Thornton, 2011; van Diepeningen *et al*., 2015). Diagnosis of the *Fusarium* infections is critical for timely and appropriate treatment. At present, the gold standard for detection of fusariosis and other IFI is mycological culture of the fungus from blood samples or from biopsy samples. As with any IFI, diagnosis must confirm to standards set out in the European Organization for Research and Treatment of Cancer and the the National Institute of Allergy and Infectious Diseases Mycoses Study Group (EORTC/MSG diagnostic criteria. In 2002, the EORTC/MSG classified potential cases of IFI into possible, probable and proven case to o facilitate drawing a policy for epidemiological studies, clinical practice guide, treatment and diagnosis tests validation (Ascioglu *et al*., 2002). Based on that, fungus propagules has to be detected from a specimen of sterile site by histological analysis or culture based method to be considered as proven infection (Ascioglu *et al*., 2002; De Pauw *et al*., 2008; Tsitsikas *et al.*, 2012). Possible and probable delimitations depends on a host factor for immune deficiency patients, clinical symptoms and microbiological observations including culture based method with microscopic analysis and non–culture-based diagnostic tests by high resolution scan, molecular detection, and through fungal antigen detection using ELISA test for GM and BDG (De Pauw *et al*., 2008). Therefore, isolation of *Fusarium* from blood or other sterile site biopsy with clinical symptoms is recognized as proven invasive infection, while recovering of *Fusarium* from respiratory tract secretion with demonstrating

clinical signs can be defined as probable (Garnica *et al*., 2015; Nucci *et al*., 2004).

Clinically, skin lesions and positive blood cultures for the mould consider characteristic feature of invasive fusariosis (Jain *et al.*, 2011; Nucci & Anaissie, 2007). This procedure is time-consuming, require skilled mycologists and have poor diagnostic sensitivities since *Fusarium* species exhibit similar morphological appearances with other hyalohyphomycete fungi such as *Aspergillus* leading to misdiagnosis (Tortorano *et al.*, 2014; van Diepeningen *et al.*, 2015;). An added complication is that skin lesions, which are the most common clinical presentation associated with disseminated fusariosis, appear at the later stage of infection (Boutati & Anaissie, 1997; Dignani & Anaissie, 2004).

Commercially, GM and BDG assay cross react with other emerging pathogens resulting in losing specificity and sensitivity to use for diagnosis of *Fusarium* at species or even genus level (Mikulska *et al.*, 2012; Nucci *et al*., 2014; Tortorano *et al*., 2012; Yoshida, 2006;). In addition, Recently, an immunochromatographic lateral-flow device (LFD) has been developed using a highly specific mouse mAb, JF5, specific to a protein epitope expressed on an extracellular glycoprotein antigen of *Aspergillus* in human serum with no cross reactivity with other clinically important fungal pathogens, including *Candida*, *Cryptococcus neoformans*, *F. solani*, *Penicillium marneffei*, *P. boydii*, and *Rhizopus oryzae* (Thornton, 2008). The mAb JF5 has been engineered to draw a new view for monitoring *Aspergillus* infection and treatment progress

through positron emission tomography and magnetic resonance (immune PET/MR) imaging (Rolle *et al*., 2016).

Molecular amplification technique using PCR might provide a promising tool for the IFI diagnosis (Lass-Flörl *et al*., 2013; Lau *et al*., 2007), with consideration of avoiding misdiagnosis due DNA contamination (Barnes, 2008; Donnelly, 2006; Dornbusch *et al*., 2010). Using imaging methods such chest radiography and computed tomography (CT) for diagnosis of invasive fusariosis among immunocompromised patients has limited help for early diagnosis and track the treatment progress (Barnes, 2008; van Diepeningen *et al*., 2015; Marom *et al*., 2008).

The lack of diagnostic test for the *Fusarium* can be due to missing the general infrequency of the infection cases over world wild (Guarro, 2013). In addition, developing species-specific or genus-specific monoclonal antibodies is problematic because *Fusarium* shares the antigenic cell wall determinants, such as GM and BDG with other related and unrelated pathogenic fungi (Aquino *et al*., 2007; Kedzierska, 2007; Khan *et al.,* 2008; Mikulska *et al*., 2012; Odabasi *et al.*, 2004; Ostrosky-Zeichner *et al*., 2005; Tortorano *et al*., 2012). There are few studies managed to generate mAb specific for *Fusarium* to quantify fungal biomass in plant hosts (Arie *et al.*, 1995; Gan *et al*., 1997; Hayashi *et a*l., 1998) or *Fusarium* mycotoxins that affected economical crops (Hill *et al*., 2006; Maragos *et al.*, 2013). However, these works have never been validated for medical usage against a broad range of clinically important opportunistic pathogenic filamentous fungi or yeasts. Therefore, this study provides powerful findings in developing a rapid and accurate immunodiagnostic test can

specifically recognize epitope with a single glycoprotein epitope expressed on the extracellular carbohydrate antigen and located on the *Fusarium* propagules with a molecular mass of ~200 kDa with no cross reactivity with other pathogenic fungi. This fitful the urgent needs for early, rapid and accurate diagnostic test specific for *Fusarium*, in particular *F. solani*, that can lead to manage fusariosis infections (Araujo *et al*., 2010; Hue *et al*., 1999; Nucci *et al*., 2003; Richardson & Warnock, 2003; Roilides *et al.*, 2007; Stevens, 2002; van Diepeningen *et al*., 2015). There is only one study mentioned using developed a mAb, IgM, can bind to two antigens of *Fusarium* with 51 and 63 kDa in infected animal model with no cross reactivity with other fungal pathogens including *Aspergillus*, *Candida* and *Scedesporum* (Jensen *et al*., 2011).

Macrophages are professional phagocytes that play a key role as the first line of the cellular innate immunity that response to *Fusarium* invaders. AMØs are able to recognize and internalize fungal propagules through phagocytosis leading to eliminate fungal infections and dissemination (Brakhage *et al.,* 2010; Figueiredo *et al.,* 2011; Roilides *et al.,* 2006). Thus**,** patients with an impaired immune system due to having cytotoxic therapy will be at the highest risk for developing disseminated invasive fusariosis (Cooke *et al.,* 2009; Lucca, 2007; Nucci *et al.,* 2004; Nucci & Anaissie, 2002; Perez-Perez *et al.,* 2007; Tortorano *et al.,* 2014; Yera *et al.,* 2003). Currently, little is known about the interaction between *Fusarium* species and macrophages (Nucci & Anaissie, 2007; Schäfer *et al.,* 2014). Here, in this phagocytosis of *F. solani* by the MH-S murine macrophage cell line was investigated and the findings demonstrate the ability of AMØs for uptaking spores and germinated spores. Moreover, this data highlighted two novel observations. First, the ability of phagocytosed spores to germinate inside

AMØ phagosome resulting in macrophage lysis and escape. Second, scape of internalized spore without macrophage lysis. These observations might indicate to a way of *Fusarium* resist the fungicidal mechanisms of phagosome including reactive oxygen species (ROS) and AMPs for developing disseminated disease. To our knowledge, this is the first analysis of the phagocytosis process for the important emerging opportunist human pathogen *F. solani.* These results suggested the need of more studies on the interaction between *F. solani* and the AMØs to better understand the recogniation and clearnce mechanisms by the cellular innate immunity for managing *Fusarium* infections.

The *Fusarium* species most commonly cited as human pathogens belong to species complex including FSSC, causes 50% of human infections, followed by FOSC (Torres & Kontoyiannis, 2011). The FDSC is less frequently reported as causing human disease, but it is similarly capable of causing disseminated infections in severely immunosuppressive patients (Bigley *et al.,* 2004; Vismer *et al.,* 2002). Identification the environmental reservoirs of pathogenic *Fusarium* species complex is essential for understanding the epidemiology of the *Fusarium* prevalence (Zhang *et al.*, 2006). There is increasing trend of opportunistic fusariosis infections in humans associates with surveillance methods within environmental reservoirs of fungal propagules (Raad *et al.*, 2002). Recent studies showed that domestic and municipal water systems are potential reservoirs of human pathogenic strains in the FSSC, FOSC and FDSC groups (Short *et al.,* 2011). Here, we set out to determine whether human pathogenic species of *Fusarium* could be identified in sink drains directly by using crude antigen extracts of biofilms and detection using the mAb, ED7 by ELISA. The results showed that the mAb ED7 able to react with *Fusarium*

antigens in swab samples but no other unrelated fungal antigens existed within biofilm structures in water distribution systems such as *Aspergillus* and *Candida*. Interestingly, the *Fusarium* species were predominately recovered from more than 50% of swab and sink samples. Similar observation was reported during screening of *Fusarium* pathogens in hospital water system (Anaissie *et al*., 2001)**.**

The abundance of the *Fusarium* species in water system indicates to two main noteworthy features. First, pathogenic *Fusarium* species are able to adapt to urban aquatic environment of hospital and the university campus. Second, the *Fusarium* species performed strong resistance to antifungal water supply treatment. These features might result in nosocomial outbreak infections particularly in a hospital area associated with water distribution system. It has been suggested that emerging pathogenic *Fusarium* species are able to adapt in hospital and non-hospital water system (Sautour *et al.*, 2012; Steinberg *et al.*, 2015).

Validation analysis using ITS sequencing and amplification of TEF gene by PCR analysis of recovered isolates confirmed the 100% accuracy and sensitivity of ED7 for detection a unique epitope of the *Fusarium* genus including medically important species and species complex such as FSSC 1-a, FSSC 2, FOSC 33 and FDSC ET-gr in a mixture of fungal biofilms communities in skins of a tertiary care hospital and sinks of a heavily populated university campus. It is worth to mention that all *Fusarium* species were recovered from haematology unit sink drains related to FSSC. Similar observation had been reported (Horn *et al.*, 2014; Scheel *et al.*, 2013). This result revealed an unseen

source and the widespread occurrence of human pathogenic *Fusarium* infectious propagules in sinks and water system of a clinic and non-clinic environments that could cause outbreak nosocomial infections. This observation was reported in previous studies (Anaissie *et al*., 2001; Short *et al*., 2011). To this end, detection of opportunistic fungal pathogens including *Fusarium* and other filamentous and yeasts increases awareness of invasive mycosis among immunocompromised individuals.

Future Work

This thesis describes the development of a novel immunodiagnostic assay for tracking the emerging human pathogen *Fusarium,* by using a newly developed genus-specific mAb, ED7. Future studies should attempt to develop a rapid diagnostic test for the pathogen, employing lateral-flow technology already successfully used for other opportunistic pathogens of humans (Thornton, 2008). This technology could have a significant impact in *Fusarium* diagnostics, delivering accurate and fast detection of the pathogen.

To better understand cellular immune responses to *Fusarium solani*, the live cell imaging studies reported here should be extended. Furthermore, the chemical nature of the epitope bound by the spore agglutinating mAb ED7 should be investigated as this may provide insights into the recognition of surface antigens of infectious propagules by cells of the innate immune system.

Finally, by combining mAb ED7 with the molecular methods of species and sub-species identification described here, a better understanding of the epidemiology of *Fusarium* and routes of exposure of susceptible individuals to

human pathogenic strains, may help to control nosocomial and community acquired infections.

BIBLIOGRAPHY

Abbas, A., Lichtman, A., & Pillai, S. (2007). Cellular and molecular immunology, (International Edition, 6th). West B. Saunders Company.

Abbas, H. K., Cartwright, R. D., Shier, W. T., Abouzied, M. M., Bird, C. B., Rice, L. G., Meredith, F. I. (1998). Natural Occurrence of Fumonisins in Rice with *Fusarium* Sheath Rot Disease. *Plant Disease*, *82*(1): 22–25.

Adams, D. J. (2004). Fungal cell wall chitinases and glucanases. *Microbiology*, *150*(Pt 7): 2029–35.

Adams, R. I., Miletto, M., Taylor, J. W., & Bruns, T. D. (2013). The Diversity and Distribution of Fungi on Residential Surfaces. *PLoS ONE*, *8*(11) : e78866.

Agapov, E., Battaile, J. T., Tidwell, R., Hachem, R., Patterson, G. A., Pierce, R. A., Holtzman, M. J. (2009). Macrophage Chitinase 1 Stratifies Chronic Obstructive Lung Disease. *American Journal of Respiratory Cell and Molecular Biology*, *41*(4): 379–384.

Aguirre, J., Hansberg, W., & Navarro, R. (2006). Fungal responses to reactive oxygen species. *Medical Mycology*, *44*(Suppl. 1): S101–S107.

Ahmet, A. (2011). Checklist of Fusarium Species Reported from Turkey. *Mycotaxon*, *116*(1): 479.

Aimanianda, V., Clavaud, C., Simenel, C., Fontaine, T., Delepierre, M., & Latgé, J. P. (2009). Cell Wall β-(1,6)-Glucan of *Saccharomyces cerevisiae*. *The Journal of Biological Chemistry*, *284*(20): 13401–13412.

Ajilogba, C. F., & Babalola, O. O. (2013). Integrated Management Strategies for Tomato Fusarium Wilt. *Biocontrol Science*, *18*(3): 117–127.

Al-Maqtoofi, M., & Thornton, C. R. (2016). Detection of human pathogenic

Fusarium species in hospital and communal sink biofilms by using a highly specific monoclonal antibody. *Environmental Microbiology*. doi: 10.1111/1462-2920.13233.

Alexander, B. D., & Pfaller, M. A. (2006). Contemporary Tools for the Diagnosis and Management of Invasive Mycoses. *Clinical Infectious Diseases*, *43*(Suppl. 1): S15–S27.

Altschul, S. F., Madden, T. L., Schäffer, A. A., Zhang, J., Zhang, Z., Miller, W., & Lipman, D. J. (1997). Gapped BLAST and PSI-BLAST: a new generation of protein database search programs. *Nucleic Acids Research*, *25*(17): 3389–3402.

Alvarez, M., & Casadevall, A. (2006). Phagosome Extrusion and Host-Cell Survival after *Cryptococcus neoformans* Phagocytosis by Macrophages. *Current Biology*, *16*(21): 2161–2165.

Anaissie, E. J., Bodey, G. P., & Rinaldi, M. G. (1989). Emerging fungal pathogens. *European Journal of Clinical Microbiology and Infectious Diseases*, *8*(4): 323–330.

Anaissie, E. J., Kuchar, R. T., Rex, J. H., Francesconi, A., Kasai, M., Müller, F. M., Walsh, T. J. (2001). Fusariosis associated with pathogenic *Fusarium* species colonization of a hospital water system: a new paradigm for the epidemiology of opportunistic mold infections. *Clinical Infectious Diseases*, *33*(11): 1871–8.

Anaissie, E. J., Penzak SR, & Dignani M. (2002). The hospital water supply as a source of nosocomial infections: A plea for action. *Archives of Internal Medicine*, *162*(13): 1483–1492.

Anaissie, E. J., Stratton, S. L., Dignani, M. C., Lee, C., Summerbell, R. C., Rex, J. H., Walsh, T. J. (2003). Pathogenic molds (including *Aspergillus*

species) in hospital water distribution systems: a 3-year prospective study and clinical implications for patients with hematologic malignancies. *Blood*, *101*(7): 2542–2546.

Anaissie, E. J., Stratton, S. L., Dignani, M. C., Summerbell, R. C., Rex, J. H., Monson, T. P., Walsh, T. J. (2002). Pathogenic *Aspergillus* Species Recovered from a Hospital Water System: A 3-Year Prospective Study. *Clinical Infectious Diseases*, *34*(6): 780–789.

Anaissie, E., & Bodey. (1989). Nosocomial fungal infections. Old problems and new challenges. *Infectious Disease Clinics of North America*, *3*(4) : 867–882.

Aquino, V. R., Goldani, L. Z., & Pasqualotto, A. C. (2007). Update on the contribution of galactomannan for the diagnosis of invasive aspergillosis. *Mycopathologia*, *163*(4): 191–202.

Araujo, R., Pina-Vaz, C., & Rodrigues, A. G. (2010). Mould Infections: A Global Threat to Immunocompromised Patients. In *Combating Fungal Infections: Problems and Remedy* (pp. 1–19). Springer-Verlag, Berlin, Heidelberg: Springer.

Arie, T., Hayashi, Y., Nagatani, A., Furuya, M., & Yamaguchi, I. (1991). Production and partial characterization of monoclonal antibodies against *Fusarium oxysporum* 860926a. *Annals of the Phytopathological Society of Japan*, *57*.

Arie, T., Hayashi, Y., Yoneyama, K., Nagatani, A., Furuya, M., & Yamaguchi, I. (1995). Detection of *Fusarium* spp. in plants with monoclonal antibody. *Annals of the Phytopathological Society of Japan*, *61*.

Arrese, J. E., Piérard-Franchimont, C., & Piérard, G. E. (1996). Fatal hyalohyphomycosis following *Fusarium* onychomycosis in an

immunocompromised patient. *The American Journal of Dermatopathology*, *18*(2): 196–198.

Arthur, R. R., Drew, R. H., & Perfect, J. R. (2004). Novel modes of antifungal drug administration. *Expert Opinion on Investigational Drugs*, *13*(8) : 903–932.

Arvanitidou, M., Kanellou, K., Constantinides, T. C., & Katsouyannopoulos, V. (1999). The occurrence of fungi in hospital and community potable waters. *Letters in Applied Microbiology*, *29*(2): 81–84.

Ascioglu, S., Rex, J. H., de Pauw, B., Bennett, J. E., Bille, J., Crokaert, F., Mycoses Study Group of the National Institute of Allergy and Infectious Diseases. (2002). Defining opportunistic invasive fungal infections in immunocompromised patients with cancer and hematopoietic stem cell transplants: an international consensus. *Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America, 34(1)* : 7–14.

Ascioglu, S., Rex, J. H., de Pauw, B., Bennett, J. E., Bille, J., Crokaert, F., Mycoses Study Group of the National Institute of Allergy and Infectious Diseases. (2002). Defining opportunistic invasive fungal infections in immunocompromised patients with cancer and hematopoietic stem cell transplants: an international consensus. *Clinical Infectious Diseases:*

Ashley, E. S. D., Lewis, R., Lewis, J. S., Martin, C., & Andes, D. (2006). Pharmacology of Systemic Antifungal Agents. *Clinical Infectious Diseases*, *43*(Suppl. 1): S28–S39.

Ashman, R. B., Farah, C. S., Wanasaengsakul, S., Hu, Y., Pang, G., & Clancy, R. L. (2004). Innate versus adaptive immunity in *Candida albicans* infection. *Immunology and Cell Biology*, *82*(2): 196–204.

Aulik, N. A., Hellenbrand, K. M., & Czuprynski, C. J. (2012). Mannheimia haemolytica and its leukotoxin cause macrophage extracellular trap formation by bovine macrophages. *Infection and Immunity*, *80*(5) : 1923–1933.

Azor, M., Gené, J., Cano, J., & Guarro, J. (2007). Universal In Vitro Antifungal Resistance of Genetic Clades of the *Fusarium solani* Species Complex. *Antimicrobial Agents and Chemotherapy*, *51*(4): 1500–1503.

Azor, M., Gené, J., Cano, J., Manikandan, P., Venkatapathy, N., & Guarro, J. (2009). Less-Frequent *Fusarium* Species of Clinical Interest: Correlation between Morphological and Molecular Identification and Antifungal Susceptibility. *Journal of Clinical Microbiology*, *47*(5): 1463–1468.

Bachmeyer, C. (2007). Deep cutaneous infection by *Fusarium solani* in a healthy child. *Journal of the American Academy of Dermatology.*, *57*(6): 1095–1096.

Badiee, P., Kordbacheh, P., Alborzi, A., Malekhoseini, S., Ramzi, M., Mirhendi, H., Shakiba, E. (2009). Study on invasive fungal infections in immunocompromised patients to present a suitable early diagnostic procedure. *International Journal of Infectious Diseases*, *13*(1): 97–102.

Bae, Y. S., & Knudsen, G. R. (2000). Cotransformation of *Trichoderma harzianum* with β-Glucuronidase and Green Fluorescent Protein Genes Provides a Useful Tool for Monitoring Fungal Growth and Activity in Natural Soils. *Applied and Environmental Microbiology*, *66*(2): 810–815.

Bain, J. M., Lewis, L. E., Okai, B., Quinn, J., Gow, N. A. R., & Erwig, L. P. (2012). Non-lytic expulsion/exocytosis of *Candida albicans* from macrophages. *Fungal Genetics and Biology*, *49*(9): 677–678.

Bain, J., Gow, N. A. R., & Erwig, L. P. (2014). Novel insights into host-fungal

pathogen interactions derived from live-cell imaging. *Seminars in Immunopathology*, *37*(2): 131–139.

Balajee, S. A., Borman, A. M., Brandt, M. E., Cano, J., Cuenca-Estrella, M., Dannaoui, E., Wickes, B. L. (2009). Sequence-Based Identification of *Aspergillus*, *Fusarium*, and *Mucorales* Species in the Clinical Mycology Laboratory: Where Are We and Where Should We Go from Here? *Journal of Clinical Microbiology*, *47*(4): 877–884.

Balmas, V., Scherm, B., Marcello, A., Beyer, M., Hoffmann, L., Migheli, Q., & Pasquali, M. (2015). *Fusarium* species and chemotypes associated with Fusarium head blight and Fusarium root rot on wheat in Sardinia. *Plant Pathology*, 64(4): 972–979.

Banks, J. N., Rizvi, R. H., Barker, I., Turner, J. A., Rahman, S., & Northway, B. J. (1996). Specific monoclonal antibodies to *Fusarium* species and microdochium nivale. *Food and Agricultural Immunology*, *8*(4) : 249–268.

Barelle, C. J., Manson, C. L., MacCallum, D. M., Odds, F. C., Gow, N. A. R., & Brown, A. J. P. (2004). GFP as a quantitative reporter of gene regulation in *Candida albicans*. *Yeast*, 21(4): 333-40.

Barnes, R. A. (2008). Early diagnosis of fungal infection in immunocompromised patients. *Journal of Antimicrobial Chemotherapy*, *61*(suppl 1): i3–i6.

Barreto-Bergter, E., & Figueiredo, R. T. (2014). Fungal glycans and the innate immune recognition. *Frontiers in Cellular and Infection Microbiology*, *4* : 145.

Bérdy, J. (2005). Bioactive Microbial Metabolites. *The Journal of Antibiotics*, *58*(1): 1–26.

Bigley, V. H., Duarte, R. F., Gosling, R. D., Kibbler, C. C., Seaton, S., & Potter, M. (2004). *Fusarium dimerum* infection in a stem cell transplant recipient treated successfully with voriconazole. *Bone Marrow Transplantation*, *34*(9): 815–817.

Blanco, J. L. & Garcia, M. E (2008). Immune response to fungal infections. *Veterinary Immunology and Immunopathology, 125*: 47–70.

Blanco, J. L., & Garcia, M. E. (2008). Immune response to fungal infections.

Blyth, C. C., Gilroy, N. M., Guy, S. D., Chambers, S. T., Cheong, E. Y., Gottlieb, T., Thursky, K. A. (2014). Consensus guidelines for the treatment of invasive mould infections in haematological malignancy and haemopoietic stem cell transplantation, 2014. *Internal Medicine Journal*, *44*(12b): 1333–1349.

Bodey, G. P., Boktour, M., Mays, S., Duvic, M., Kontoyiannis, D., Hachem, R., & Raad, I. (2002). Skin lesions associated with *Fusarium* infection. *Journal of the American Academy of Dermatology*, *47*(5): 659–666.

Boonyuen, N., Chuaseeharonnachai, C., Suetrong, S., Sri-indrasutdhi, V., Sivichai, S., Jones, E. B. G., & Pang, K. L. (2011). Savoryellales (Hypocreomycetidae, Sordariomycetes): a novel lineage of aquatic ascomycetes inferred from multiple-gene phylogenies of the genera *Ascotaiwania, Ascothailandia*, and *Savoryella. Mycologia,* 103(6) : 1351–1371.

Booth, C. (1971). *The genus Fusarium*. Commonwealth Agricultural Bureaux.

Bottalico, A. (1998). *Fusarium* diseases of cereals: species complex and related mycotoxin profiles, in Europe. *Journal of Plant Pathology*, *80*(2):85–103.

Bouchara, J. P., Oumeziane, N. A., Lissitzky, J. C., Larcher, G., Tronchin, G., & Chabasse, D. (1996). Attachment of spores of the human pathogenic

fungus Rhizopus oryzae to extracellular matrix components. *European Journal of Cell Biology*, *70*(1): 76–83.

Bouchara, J.-P., Hsieh, H. Y., Croquefer, S., Barton, R., Marchais, V., Pihet, M., & Chang, T. C. (2009). Development of an Oligonucleotide Array for Direct Detection of Fungi in Sputum Samples from Patients with Cystic Fibrosis. *Journal of Clinical Microbiology*, *47*(1): 142–152.

Boutati, E. I., & Anaissie, E. J. (1997). *Fusarium*, a significant emerging pathogen in patients with hematologic malignancy: ten years' experience at a cancer center and implications for management. *Blood*, *90*(3): 999–1008.

Bowman, S. M., & Free, S. J. (2006). The structure and synthesis of the fungal cell wall. *Bioessays*, *28*(8): 799–808.

Brakhage, A. A., Bruns, S., Thywissen, A., Zipfel, P. F., & Behnsen, J. (2010). Interaction of phagocytes with filamentous fungi. *Current Opinion in Microbiology*, *13*(4): 409–415.

Brandt, M. E., & Warnock, D. W. (2003). Laboratory aspects of medical mycology. In W. E. Dismukes, P. G. Pappas, & J. D. Sobel (Eds.): *Clinical Mycology* (pp. 3–22). Oxford University Press.

Branzk, N., Lubojemska, A., Hardison, S. E., Wang, Q., Gutierrez, M. G., Brown, G. D., & Papayannopoulos, V. (2014). Neutrophils sense microbe size and selectively release neutrophil extracellular traps in response to large pathogens. *Nature Immunology*, *15*(11): 1017–1025.

Brasch, J., & Köppl, G. (2009). Persisting onychomycosis caused by *Fusarium solani* in an immunocompetent patient. *Mycoses*, *52*(3): 285–6.

Brasch, J., & Shimanovich, I. (2012). Persistent fingernail onychomycosis caused by *Fusarium proliferatum* in a healthy woman. *Mycoses*, *55*(1)

: 86–89.

Brase, S., Encinas, A., Keck, J., & Nising, C. F. (2009). Chemistry and biology of mycotoxins and related fungal metabolites. *Chem Rev*, *109*(9) : 3903–90.

Brieland, J. K., Jackson, C., Menzel, F., Loebenberg, D., Cacciapuoti, A., Halpern, J., O'Garra, A. (2001). Cytokine Networking in Lungs of Immunocompetent Mice in Response to Inhaled *Aspergillus fumigatus*. *Infection and Immunity*, *69*(3): 1554–1560.

Brinkmann, V., Reichard, U., Goosmann, C., Fauler, B., Uhlemann, Y., Weiss, D. S., Zychlinsky, A. (2004). Neutrophil Extracellular Traps Kill Bacteria. *Science*, *303*(5663): 1532–1535.

Brown, A. J. P., Brown, G. D., Netea, M. G., & Gow, N. A. R. (2014). Metabolism impacts upon Candida immunogenicity and pathogenicity at multiple levels. *Trends in Microbiology*, *22*(11): 614–622.

Brown, G. D. (2011). Innate Antifungal Immunity: The Key Role of Phagocytes. *Annual Review of Immunology*, *29*, 1–21.

Brown, G. D., Denning, D. W., Gow, N. A. R., Levitz, S. M., Netea, M. G., & White, T. C. (2012). Hidden killers: human fungal infections. *Science Translational Medicine*, *4*(165): 165rv13.

Bruns, S., Kniemeyer, O., Hasenberg, M., Aimanianda, V., Nietzsche, S., Thywißen, A., Gunzer, M. (2010). Production of Extracellular Traps against *Aspergillus fumigatus* In Vitro and in Infected Lung Tissue Is Dependent on Invading Neutrophils and Influenced by Hydrophobin RodA. *PLoS Pathogens*, *6*(4): e1000873.

Bruns, S., Kniemeyer, O., Hasenberg, M., Aimanianda, V., Nietzsche, S., Thywißen, A., Gunzer, M. (2010). Production of Extracellular Traps

against *Aspergillus fumigatus* In Vitro and in Infected Lung Tissue Is Dependent on Invading Neutrophils and Influenced by Hydrophobin RodA. *PLoS Pathogens*, *6*(4): e1000873.

Bryan, G. T., Daniels, M. J., & Osbourn, A. E. (1995). Comparison of fungi within the Gaeumannomyces-Phialophora complex by analysis of ribosomal DNA sequences. *Applied and Environmental Microbiology*, *61*(2): 681–689.

Burgess, L. W., Knight, T. E., Tesoriero, L., & Phan, H. T. (2008). *Diagnostic manual for plant diseases in Vietnam* (Australian Centre for International Agricultural Research, Vols. 1–ACIAR Monograph No. 129,). Canberra 2008.

Bushelman, S. J., Callen, J. P., Roth, D. N., & Cohen, L. M. (1995). Disseminated *Fusarium solani* infection. *Journal of the American Academy of Dermatology*, *32*(2, Part 2): 346–351.

Campbell, C. K., Johnson, E. M., & Warnock, D. W. (2013). *Identification of Pathogenic Fungi* (2nd Edition). A John Wiley & Sons, Ltd., Publication.

Campbell, S., & Landry, M. L. (2006). Rapid Antigen Tests. In *Advanced Techniques in Diagnostic Microbiology* (pp. 23–41). Springer US.

Carneiro, H. A., Coleman, J. J., Restrepo, A., & Mylonakis, E. (2011). *Fusarium* Infection in Lung Transplant Patients. *Medicine*, *90*(1): 69–80.

Carvalho, A., Cunha, C., Pasqualotto, A. C., Pitzurra, L., Denning, D. W., & Romani, L. (2010). Genetic variability of innate immunity impacts human susceptibility to fungal diseases. *International Journal of Infectious Diseases*, *14*(6): e460–e468.

Casadevall, A., & Pirofski, L. (2007). Antibody-Mediated Protection through Cross-Reactivity Introduces a Fungal Heresy into Immunological

Dogma. *Infection and Immunity*, *75*(11): 5074–5078.

Casadevall, A., & Pirofski, L. (2012). A new synthesis for antibody-mediated immunity. *Nature Immunology*, *13*(1): 21–28.

Chalfie, M., Tu, Y., Euskirchen, G., Ward, W. W., & Prasher, D. C. (1994). Green fluorescent protein as a marker for gene expression. *Science*, *263*(5148): 802–805.

Champion, J. A., & Mitragotri, S. (2006). Role of target geometry in phagocytosis. *Proceedings of the National Academy of Sciences of the United States of America*, *103*(13): 4930–4934.

Chandra, J., Kuhn, D. M., Mukherjee, P. K., Hoyer, L. L., McCormick, T., & Ghannoum, M. A. (2001). Biofilm Formation by the Fungal Pathogen *Candida albicans*: Development, Architecture, and Drug Resistance. *Journal of Bacteriology*, *183*(18): 5385–5394.

Chang, D. C., Grant, G. B., O'Donnell, K., Wannemuehler, K. A., Noble-Wang, J., Rao, C. Y., Lewis, F. M. (2006). Multistate outbreak of *Fusarium* keratitis associated with use of a contact lens solution. *JAMA: The Journal of the American Medical Association*, *296*(8): 953–963.

Chen, K., & Kolls, J. K. (2013). T Cell–Mediated Host Immune Defenses in the Lung. *Annual Review of Immunology*, *31*, 605–633.

Cho, C. T., Vats, T. S., Lowman, J. T., Brandsberg, J. W., & Tosh, F. E. (1973). *Fusarium solani* infection during treatment for acute leukemia. *Journal of Pediatrics*, *83*(6): 1028–1031.

Chotirmall, S. H., Al-Alawi, M., Mirkovic, B., Lavelle, G., Logan, P. M., Greene, C. M., McElvaney, N. G. (2013). *Aspergillus*-Associated Airway Disease, Inflammation, and the Innate Immune Response, *Aspergillus*-Associated Airway Disease, Inflammation, and the Innate Immune

Response. *BioMed Research International, BioMed Research International*, *2013,* e723129.

Chow, T. Y. K., & Käfer, E. (1993). A rapid method for isolation of total nucleic acids from *Aspergillus nidulans*. *Fungal Genet. Newsl.*, *40*, 25–27.

Church, D., Elsayed, S., Reid, O., Winston, B., & Lindsay, R. (2006). Burn Wound Infections. *Clinical Microbiology Reviews*, *19*(2): 403–434.

Cooke, N. S., Feighery, C., Armstrong, D. K., Walsh, M., & Dempsey, S. (2009). Cutaneous *Fusarium solani* infection in childhood acute lymphoblastic leukaemia. *Clin Exp Dermatol*, *34*(5): e117–9.

Cormack, B. P., Bertram, G., Egerton, M., Gow, N. A. R., Falkow, S., & Brown, A. J. P. (1997). Yeast-enhanced green fluorescent protein (yEGFP): a reporter of gene expression in Candida albicans. *Microbiology*, *143*(2): 303–311.

Crabol, Y., Lortholary, O., Crabol, Y., & Lortholary, O. (2014). Invasive Mold Infections in Solid Organ Transplant Recipients, Invasive Mold Infections in Solid Organ Transplant Recipients. *Scientifica, Scientifica*, *2014,* e821969.

Cryptococcus neoformans Escapes Macrophages by a Phagosome Emptying Mechanism That Is Inhibited by Arp2/3 Complex-Mediated Actin Polymerisation. *PLoS Pathog*, *6*(8): e1001041.

Cuero, R. G. (1980). Ecological distribution of *Fusarium solani* and its opportunistic action related to mycotic keratitis in Cali, Colombia. *Journal of Clinical Microbiology*, *12*(3): 455–61.

Dagenais, T. R. T., & Keller, N. P. (2009). Pathogenesis of *Aspergillus fumigatus* in Invasive Aspergillosis. *Clinical Microbiology Reviews*, *22*(3): 447–465.

Davies, G. E., & Thornton, C. R. (2014). Differentiation of the Emerging Human Pathogens *Trichosporon asahii* and *Trichosporon asteroides* from Other Pathogenic Yeasts and Moulds by Using Species-Specific Monoclonal Antibodies. *PLoS ONE*, *9*(1): e84789

De Pauw, B., Walsh, T. J., Donnelly, J. P., Stevens, D. A., Edwards, J. E., Calandra, T., Bennett, J. E. (2008). Revised Definitions of Invasive Fungal Disease from the European Organization for Research and Treatment of Cancer/Invasive Fungal Infections Cooperative Group and the National Institute of Allergy and Infectious Diseases Mycoses Study Group (EORTC/MSG) Consensus Group. *Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America*, *46*(12): 1813–1821.

Dean, R., Van Kan, J. A., Pretorius, Z. A., Hammond-Kosack, K. E., Di Pietro, A., Spanu, P. D., Foster, G. D. (2012). The Top 10 fungal pathogens in molecular plant pathology. *Mol Plant Pathol*, *13*(4): 414–30.

Defra, (Department for Environment Food & Rural Affairs). (2011). *A Review of Fungi in Drinking Water and the Implications for Human Health* (Final report No. Reference: WD 0906) (pp. 1–107). Bio Intelligence Service, Paris, France.

Deorukhkar, S. C., Saini, S., Mathew, S., Deorukhkar, S. C., Saini, S., & Mathew, S. (2014). Virulence Factors Contributing to Pathogenicity of *Candida tropicalis* and Its Antifungal Susceptibility Profile, Virulence Factors Contributing to Pathogenicity of *Candida tropicalis* and Its Antifungal Susceptibility Profile. *International Journal of Microbiology, International Journal of Microbiology*, *2014, 2014*, e456878.

Desjardins, A. E., & Proctor, R. H. (2007). Molecular biology of *Fusarium*

mycotoxins. *Int J Food Microbiol*, *119*(1-2): 47–50.

Dictar, M. O., Maiolo, E., Alexander, B., Jacob, N., & Verón, M. T. (2000). Mycoses in the transplanted patient. *Medical Mycology*, *38 Suppl 1*, 251–258.

Dignani, M., & Anaissie, E. (2004). Human Fusariosis. *Clinical Microbiology and Infection*, *10*(s1): 67–75.

Dixon, D. M., & Walsh, T. J. (1996). Antifungal Agents. In S. Baron (Ed.), *Medical Microbiology* (4th ed.). Galveston (TX): University of Texas Medical Branch at Galveston.

Dóczi, I., Gyetvai, T., Kredics, L., & Nagy, E. (2004). Involvement of *Fusarium* spp. in fungal keratitis. *Clinical Microbiology and Infection*, *10*(9), 773–776.

Doggett, M. S. (2000). Characterization of Fungal Biofilms within a Municipal Water Distribution System. *Applied and Environmental Microbiology*, *66*(3): 1249–1251.

Donnelly, J. P. (2006). Polymerase Chain Reaction for Diagnosing Invasive Aspergillosis: Getting Closer but Still a Ways to Go. *Clinical Infectious Diseases*, *42*(4): 487–489.

Donnio, A., Van Nuoi, D. N. 'Guyen, Catanese, M., Desbois, N., Ayeboua, L., & Merle, H. (2007). Outbreak of keratomycosis attributable to *Fusarium solani* in the French West Indies. *American Journal of Ophthalmology*, *143*(2): 356–358.

Dornbusch, H. J., Groll, A., & Walsh, T. J. (2010). Diagnosis of invasive fungal infections in immunocompromised children. *Clinical Microbiology and Infection*, *16*(9): 1328–1334.

Dursun, D., Fernandez, V., Miller, D., & Alfonso, E. C. (2003). Advanced

Fusarium keratitis progressing to endophthalmitis. *Cornea*, *22*(4) : 300–303.

Edelstein, S. L., Akduman, L., Durham, B. H., Fothergill, A. W., & Hsu, H. Y. (2012). Resistant *Fusarium* keratitis progressing to endophthalmitis. *Eye & Contact Lens*, *38*(5): 331–335.

Epstein, A. B. (2007). In the aftermath of the *Fusarium* keratitis outbreak: What have we learned? *Clinical Ophthalmology (Auckland, N.Z.)*, *1*(4) : 355–366.

Epstein, L., & Nicholson, R. L. (2006). Adhesion and Adhesives of Fungi and Oomycetes. In D. A. M. Smith & D. J. A. Callow (Eds.), *Biological Adhesives* (pp. 41–62). Springer Berlin Heidelberg.

Erwig, L. P., & Gow, N. A. R. (2016). Interactions of fungal pathogens with phagocytes. *Nature Reviews Microbiology*, *14*(3): 163–176.

Esnakula, A. K., Summers, I., & Naab, T. J. (2013). Fatal Disseminated Infection in a Human Immunodeficiency Virus Positive Patient. *Case Reports in Infectious Diseases*, *2013*, e379320.

Feng, P., Lu, Q., Najafzadeh, M. J., Ende, A. H. G. G. van den, Sun, J., Li, R., Hoog, G. S. de. (2014). *Cyphellophora* and its relatives in *Phialophora*: biodiversity and possible role in human infection. *Fungal Diversity*, *65*(1): 17–45.

Figueiredo, R. T., Carneiro, L. A. M., & Bozza, M. T. (2011). Fungal Surface and Innate Immune Recognition of Filamentous Fungi. *Frontiers in Microbiology*, *2*(248): 1–14.

Folds, J. D. (2008). Overview of Immunity. In M. R. G. O'Gorman & A. D. Donnenberg (Eds.), *Handbook of Human Immunology* (2nd edition, pp. 1–28). CRC Press.

Fontaine, T., Simenel, C., Dubreucq, G., Adam, O., Delepierre, M., Lemoine, J., Latge, J. P. (2000). Molecular organization of the alkali-insoluble fraction of *Aspergillus fumigatus* cell wall. *Journal of Biological Chemistry*. 8;275(36): 27594-607.

Fontana, C., Gaziano, R., Favaro, M., Casalinuovo, I., Pistoia, E., & Di Francesco, P. (2012). (1-3)-β-D-Glucan vs Galactomannan Antigen in Diagnosing Invasive Fungal Infections (IFIs). *The Open Microbiology Journal*, *6*, 70–73.

Francis, R. G., & Burgess, L. W. (1977). Characteristics of two populations of *Fusarium roseum* "Graminearum" in Eastern Australia. *Transactions of the British Mycological Society*, *68*(3): 421–427.

Free, S. J. (2013). Fungal cell wall organization and biosynthesis. *Advances in Genetics*, *81*, 33–82.

Fridkin, S. K., & Jarvis, W. R. (1996). Epidemiology of nosocomial fungal infections. *Clinical Microbiology Reviews*, *9*(4): 499–511.

Gan, Z., Marquardt, R. R., Abramson, D., & Clear, R. M. (1997). The characterization of chicken antibodies raised against *Fusarium* spp. by enzyme-linked immunosorbent assay and immunoblotting. *International Journal of Food Microbiology*, *38*(2-3): 191–200.

Gangneux, J. P., Bretagne, S., Cordonnier, C., Datry, A., Derouin, F., Grillot, R., Poirot, J. L. (2002). Prevention of Nosocomial Fungal Infection: The French Approach. *Clinical Infectious Diseases*, *35*(3): 343–346.

Garnica, M., & Nucci, M. (2013). Epidemiology of Fusariosis. *Current Fungal Infection Reports*, *7*(4): 301–305.

Garnica, M., Cunha, M. O. da, Portugal, R., Maiolino, A., Colombo, A. L., & Nucci, M. (2015). Risk Factors for Invasive Fusariosis in Patients With

Acute Myeloid Leukemia and in Hematopoietic Cell Transplant Recipients. *Clinical Infectious Diseases*, *60*(6): 875–880.

Gauthier, G. M., & Keller, N. P. (2013). Crossover fungal pathogens: the biology and pathogenesis of fungi capable of crossing kingdoms to infect plants and humans. *Fungal Genetics and Biology: FG & B*, *61*, 146–157.

Gaviria, J. M., van Burik, J. A., Dale, D. C., Root, R. K., & Liles, W. C. (1999a). Comparison of interferon-gamma, granulocyte colony-stimulating factor, and granulocyte-macrophage colony-stimulating factor for priming leukocyte-mediated hyphal damage of opportunistic fungal pathogens. *J Infect Dis*, *179*(4): 1038–41.

Gaviria, J. M., van Burik, J. A., Dale, D. C., Root, R. K., & Liles, W. C. (1999b). Modulation of neutrophil-mediated activity against the pseudohyphal form of Candida albicans by granulocyte colony-stimulating factor (G-CSF) administered in vivo. *J Infect Dis*, *179*(5): 1301–4.

Geiser, D. M., del Mar Jiménez-Gasco, M., Kang, S., Makalowska, I., Veeraraghavan, N., Ward, T. J., O'donnell, K. (2004). FUSARIUM-ID v. 1.0: A DNA Sequence Database for Identifying *Fusarium*. *European Journal of Plant Pathology*, *110*(5/6): 473–479.

Gersuk, G. M., Underhill, D. M., Zhu, L., & Marr, K. A. (2006). Dectin-1 and TLRs Permit Macrophages to Distinguish between Different *Aspergillus fumigatus* Cellular States. *The Journal of Immunology*, *176*(6): 3717–3724.

Ghannoum, M. A., & Rice, L. B. (1999). Antifungal Agents: Mode of Action, Mechanisms of Resistance, and Correlation of These Mechanisms with Bacterial Resistance. *Clinical Microbiology Reviews*, *12*(4): 501–517.

Giacchino, M., Chiapello, N., Bezzio, S., Fagioli, F., Saracco, P., Alfarano, A., Girmenia, C. (2006). *Aspergillus* Galactomannan Enzyme-Linked Immunosorbent Assay Cross-Reactivity Caused by Invasive Geotrichum capitatum. *Journal of Clinical Microbiology*, *44*(9): 3432–3434.

Gil-Lamaignere, C., Roilides, E., Mosquera, J., Maloukou, A., & Walsh, T. J. (2002). Antifungal Triazoles and Polymorphonuclear Leukocytes Synergize To Cause Increased Hyphal Damage to *Scedosporium prolificans* and *Scedosporium apiospermum*. *Antimicrobial Agents and Chemotherapy*, *46*(7): 2234–2237.

Gilbert, A. S., Wheeler, R. T., & May, R. C. (2015). Fungal Pathogens: Survival and Replication within Macrophages. *Cold Spring Harbor Perspectives in Medicine*, *5*(7): a019661.

Girmenia, C., Arcese, W., Micozzi, A., Martino, P., Bianco, P., & Morace, G. (1992). Onychomycosis as a possible origin of disseminated *Fusarium solani* infection in a patient with severe aplastic anemia. *Clinical Infectious Diseases*, *14*(5): 1167–1167.

Girmenia, C., Pagano, L., Corvatta, L., Mele, L., Del Favero, A., & Martino for the Gimema Infection Programme, P. (2000). The epidemiology of fusariosis in patients with haematological diseases. *British Journal of Haematology*, *111*(1): 272–276.

Godoy, P., Nunes, E., Silva, V., Tomimori-Yamashita, J., Zaror, L., & Fischman, O. (2004). Onychomycosis caused by *Fusarium solani* and *Fusarium oxysporum* in São Paulo, Brazil. *Mycopathologia*, *157*(3): 287–90.

Gordon, S., & Read, R. (2002). Macrophage defences against respiratory tract infections The immunology of childhood respiratory infections. *British*

Medical Bulletin, *61*(1): 45–61.

Gorscak, J. J., Ayres, B. D., Bhagat, N., Hammersmith, K. M., Rapuano, C. J., Cohen, E. J., Chu, D. S. (2007). An Outbreak of *Fusarium* Keratitis Associated With Contact Lens Use in the Northeastern United States: *Cornea*, *26*(10): 1187–1194.

Goswami, R. S., & Kistler, H. C. (2004). Heading for disaster: *Fusarium graminearum* on cereal crops. *Mol Plant Pathol*, *5*(6): 515–25.

Goswami, R. S., & Kistler, H. C. (2004). Heading for disaster: *Fusarium graminearum* on cereal crops. *Molecular Plant Pathology, 5*(6): 515–25.

Grazziutti, M., Anaissie, E. J., & Wingard, J. R. (2005). Overview of Host Defenses: Innate and Acquired Immunity. In J. R. Wingard & E. J. Anaissie (Eds.), *Fungal Infections in the Immunocompromised Patient* (pp. 1–44). Boca Raton London New York Singapore: Taylor & Francis Group.

Green, B. J., Beezhold, D. H., Green, B. J., & Beezhold, D. H. (2011). Industrial Fungal Enzymes: An Occupational Allergen Perspective, Industrial Fungal Enzymes: An Occupational Allergen Perspective. *Journal of Allergy, Journal of Allergy*, *2011,* e682574.

Guarro, J. (2013). Fusariosis, a complex infection caused by a high diversity of fungal species refractory to treatment. *European Journal of Clinical Microbiology & Infectious Diseases: Official Publication of the European Society of Clinical Microbiology*, *32*(12): 1491–1500.

Guarro, J., Nucci, M., Akiti, T., & Gené, J. (2000). Mixed Infection Caused by Two Species of *Fusarium* in a Human Immunodeficiency Virus-Positive Patient. *J. Clin. Microbiol.*, *38*(9): 3460–3462.

Gubbins, P. O., & Anaissie, E. J. (2009). CHAPTER 7 - Antifungal therapy. In

Clinical Mycology (Second Edition) (pp. 161–195). Edinburgh: Churchill Livingstone.

Gungel, H., Eren, M. H., Pinarci, E. Y., Altan, C., Baylancicek, D. O., Kara, N., Susever, S. (2011). An outbreak of *Fusarium solani* endophthalmitis after cataract surgery in an eye training and research hospital in Istanbul. *Mycoses*, *54*(6): e767–74.

Guo, H., Wu, X., Fu-shin, X. Y., & Zhao, J. (2008). Toll-like receptor 2 mediates the induction of IL-10 in corneal fibroblasts in response to *Fusarium solu*. *Immunology and Cell Biology*, *86*(3): 271–276.

Gupta, A. K., Baran, R., & Summerbell, R. C. (2000). *Fusarium* infections of the skin. *Curr Opin Infect Dis*, *13*(2): 121–128.

Gupta, V. K., Misra, A. K., Gaur, R., Pandey, R., & Chauhan, U. (2009). Studies of genetic polymorphism in the isolates of *Fusarium solani*. *Australian Journal Crop Science*, *3*, 101–106.

Gurusidappa, S., & Mamatha, H. (2011). Fusarial skin lesion in immunocompromised. *Indian Journal of Cancer*, *48*(1): 116.

Hageskal, G., Knutsen, A. K., Gaustad, P., Hoog, G. S. de, & Skaar, I. (2006). Diversity and Significance of Mold Species in Norwegian Drinking Water. *Applied and Environmental Microbiology*, *72*(12): 7586–7593.

Hall, R. A., Bates, S., Lenardon, M. D., MacCallum, D. M., Wagener, J., Lowman, D. W., Gow, N. A. R. (2013). The Mnn2 Mannosyltransferase Family Modulates Mannoprotein Fibril Length, Immune Recognition and Virulence of Candida albicans. *PLOS Pathog*, *9*(4): e1003276.

Harrow, S. A., Farrokhi-Nejad, R., Pitman, A. R., Scott, I. A. W., Bentley, A., Hide, C., & Cromey, M. G. (2010). Characterisation of New Zealand *Fusarium* populations using a polyphasic approach differentiates the *F.*

avenaceum/F. acuminatum/F. tricinctum species complex in cereal and grassland systems. *Fungal Biology*, *114*(4): 293–311.

Hayashi, Y., Arie, T., Yoneyama, K., & Yamaguchi, I. (1998). Characterization of the antigenic determinant on *Fusarium oxysporum* recognized by a genus-specific monoclonal antibody. *The Journal of General and Applied Microbiology*, *44*(1): 43–47.

Hegedüs, N., & Marx, F. (2013). Antifungal proteins: More than antimicrobials? *Fungal Biology Reviews*, *26*(4): 132–145.

Heinekamp, T., Thywißen, A., Macheleidt, J., Keller, S., Valiante, V., & Brakhage, A. A. (2013). *Aspergillus fumigatus* melanins: interference with the host endocytosis pathway and impact on virulence. *Frontiers in Microbiology*,*3*, 440.

Held, J., Schmidt, T., Thornton, C. R., Kotter, E., & Bertz, H. (2013). Comparison of a novel Aspergillus lateral-flow device and the Platelia® galactomannan assay for the diagnosis of invasive aspergillosis following haematopoietic stem cell transplantation. *Infection*, *41*(6): 1163–1169.

Hellenbrand, K. M., Forsythe, K. M., Rivera-Rivas, J. J., Czuprynski, C. J., & Aulik, N. A. (2013). *Histophilus somni* causes extracellular trap formation by bovine neutrophils and macrophages. *Microbial Pathogenesis*, *54*, 67–75.

Hill, N. S., Schwarz, P., Dahleen, L. S., Neate, S. M., Horsley, R., Glenn, A. E., & O'Donnell, K. (2006). ELISA Analysis for in Barley. *Crop Science*, *46*(6): 2636.

Hohl, T. M., & Feldmesser, M. (2007). *Aspergillus fumigatus*: Principles of Pathogenesis and Host Defense. *Eukaryotic Cell*, *6*(11): 1953–1963.

Horn, D. L., Freifeld, A. G., Schuster, M. G., Azie, N. E., Franks, B., &

Kauffman, C. A. (2014). Treatment and outcomes of invasive fusariosis: review of 65 cases from the PATH Alliance® registry. *Mycoses*, *57*(11): 652–658.

Houang, E., Lam, D., Fan, D., & Seal, D. (2001). Microbial keratitis in Hong Kong: relationship to climate, environment and contact-lens disinfection. *Transactions of The Royal Society of Tropical Medicine and Hygiene*, *95*(4): 361–367.

Hsiue, H. C., Ruan, S. Y., Kuo, Y. L., Huang, Y. T., & Hsueh, P. R. (2010). Invasive infections caused by non-*Aspergillus* moulds identified by sequencing analysis at a tertiary care hospital in Taiwan, 2000–2008. *Clinical Microbiology and Infection*, *16*(8): 1204–1206.

Hu, Y., Farah, C. S., & Ashman, R. B. (2006). Effector function of leucocytes from susceptible and resistant mice against distinct isolates of Candida albicans. *Immunology and Cell Biology*, *84*(5): 455–460.

Hu, Z. Q., Li, H. P., Zhang, J. B., Huang, T., Liu, J. L., Xue, S., Liao, Y. C. (2013). A phage-displayed chicken single-chain antibody fused to alkaline phosphatase detects *Fusarium* pathogens and their presence in cereal grains. *Analytica Chimica Acta*, *764*, 84–92.

Hu, Z. Q., Liu, J. L., Li, H. P., Xing, S., Xue, S., Zhang, J. B., Liao, Y. C. (2012). Generation of a highly reactive chicken-derived single-chain variable fragment against *Fusarium verticillioides* by phage display. *International Journal of Molecular Sciences*, *13*(6): 7038–7056.

Huang, Y. T., Hung, C. C., & Hsueh, P.-R. (2007). *Aspergillus* galactomannan antigenemia in penicilliosis marneffei: *AIDS*, *21*(14): 1990–1991.

Hue, F.-X., Huerre, M., Rouffault, M. A., & De Bievre, C. (1999). Specific detection of *Fusarium* species in blood and tissues by a PCR technique.

J. Clin. Microbiol., *37*(8): 2434–2438.

Ibrahim-Granet, O., Philippe, B., Boleti, H., Boisvieux-Ulrich, E., Grenet, D., Stern, M., & Latgé, J. P. (2003). Phagocytosis and Intracellular Fate of *Aspergillus fumigatus* Conidia in Alveolar Macrophages. *Infection and Immunity*, *71*(2): 891–903.

Ibrahim, M. M., Vanini, R., Ibrahim, F. M., Martins, W. de P., Carvalho, R. T. de C., Castro, R. S. de, & Rocha, E. M. (2011). Epidemiology and medical prediction of microbial keratitis in southeast Brazil. *Arquivos Brasileiros de Oftalmologia*, *74*(1): 7–12.

Imamura, Y., Chandra, J., Mukherjee, P. K., Lattif, A. A., Szczotka-Flynn, L. B., Pearlman, E., Ghannoum, M. A. (2008). *Fusarium* and *Candida albicans* Biofilms on Soft Contact Lenses: Model Development, Influence of Lens Type, and Susceptibility to Lens Care Solutions. *Antimicrobial Agents and Chemotherapy*, *52*(1): 171–182.

Inoue, I., Namiki, F., & Tsuge, T. (2002). Plant Colonization by the Vascular Wilt Fungus *Fusarium oxysporum* Requires FOW1, a Gene Encoding a Mitochondrial Protein. *The Plant Cell*, *14*(8): 1869–1883.

Jain, P. K., Gupta, V. K., Misra, A. K., Gaur, R., Bajpai, V., & Issar, S. (2011). Current Status of *Fusarium* Infection in Human and Animal. *Asian Journal of Animal and Veterinary Advances*, *6*(3): 201–227.

Jensen, H. M. E., Aalbæk, B., Jungersen, G., Hartvig, T., Moser, C., Rozell, B., & Blennow, O. (2011). Immunohistochemical diagnosis of fusariosis with monoclonal antibodies. *Mycoses-Free Communications*, *54* (Poster in 5th Congress on Trends in Medical Mycology, Valencia, Spain) : 51–60.

Jensen, T. G., Gahrn-Hansen, B., Arendrup, M., & Bruun, B. (2004). *Fusarium*

fungaemia in immunocompromised patients. *Clinical Microbiology and Infection*, *10*(6): 499–501.

Jiménez, M., Logrieco, A., & Bottalico, A. (1993). Occurrence and Pathogenicity of *Fusarium* Species in Banana Fruits. *Journal of Phytopathology*, *137*(3): 214–220.

Jin, X., Qin, Q., Tu, L., Zhou, X., Lin, Y., & Qu, J. (2007). Toll-like receptors (TLRs) expression and function in response to inactivate hyphae of *Fusarium solani* in immortalized human corneal epithelial cells. *Molecular Vision*, *13*, 1953.

Johnston, S. A., & May, R. C. (2010). The Human Fungal Pathogen *Cryptococcus neoformans* Escapes Macrophages by a Phagosome Emptying Mechanism That Is Inhibited by Arp2/3 Complex-Mediated Actin Polymerisation. *PLoS Pathog*, *6*(8): e1001041.

Jones, M. J., & Epstein, L. (1990). Adhesion of Macroconidia to the Plant Surface and Virulence of Nectria haematococca. *Applied and Environmental Microbiology*, *56*(12): 3772–3778.

Jung, B., Lee, S., Ha, J., Park, J.-C., Han, S.-S., Hwang, I., Lee, J. (2013). Development of a Selective Medium for the Fungal Pathogen *Fusarium graminearum* Using Toxoflavin Produced by the Bacterial Pathogen Burkholderia glumae. *The Plant Pathology Journal*, *29*(4): 446–450.

Jurkunas, U., Behlau, I., & Colby, K. (2009). Fungal keratitis: changing pathogens and risk factors. *Cornea*, *28*(6): 638–643.

Kanafani, Z. A., & Perfect, J. R. (2008). Resistance to Antifungal Agents: Mechanisms and Clinical Impact. *Clinical Infectious Diseases*, *46*(1): 120–128.

Kappe, R., & Schulzeberge, A. (1993). New cause for false-positive results with the pastorex *Aspergillus* antigen latex agglutination-test. *Journal of Clinical Microbiology*, *31*(9): 2489–2490.

Karthikeyan, R. S., Leal, S. M., Prajna, N. V., Dharmalingam, K., Geiser, D. M., Pearlman, E., & Lalitha, P. (2011). Expression of innate and adaptive immune mediators in human corneal tissue infected with *Aspergillus* or *Fusarium*. *The Journal of Infectious Diseases*, *204*(6): 942–50.

Kathiravan, M. K., Salake, A. B., Chothe, A. S., Dudhe, P. B., Watode, R. P., Mukta, M. S., & Gadhwe, S. (2012). The biology and chemistry of antifungal agents: A review. *Bioorganic & Medicinal Chemistry*, *20*(19): 5678–5698.

Katoh, K., & Standley, D. M. (2013). MAFFT multiple sequence alignment software version 7: improvements in performance and usability. *Molecular Biology and Evolution*, *30*(4): 772–80.

Katragkou, A., Kruhlak, M. J., Simitsopoulou, M., Chatzimoschou, A., Taparkou, A., Cotten, C. J., Roilides, E. (2010). Interactions between Human Phagocytes and Candida albicans Biofilms Alone and in Combination with Antifungal Agents. *Journal of Infectious Diseases*, *201*(12) : 1941–1949.

Kauffman, C. A. (2004). Zygomycosis: Reemergence of an Old Pathogen. *Clinical Infectious Diseases*, *39*(4): 588–590.

Kaur, S., & Singh, S. (2014). Biofilm formation by *Aspergillus fumigatus*. *Medical Mycology*, *52*(1): 2–9.

Kawamura, S., Maesaki, S., Tomono, K., Tashiro, T., & Kohno, S. (2000). Clinical Evaluation of 61 Patients with Pulmonary Aspergilloma. *Internal Medicine*, *39*(3): 209–212.

Kedzierska, A. (2007). (1 --> 3)-beta-D-glucan--a new marker for the early serodiagnosis of deep-seated fungal infections in humans. *Polish Journal of Microbiology / Polskie Towarzystwo Mikrobiologów = The Polish Society of Microbiologists*, *56*(1): 3–9.

Khan, Z. U., Ahmad, S., & Theyyathel, A. M. (2008). Diagnostic value of DNA and (1 -> 3)-beta-D-glucan detection in serum and bronchoalveolar lavage of mice experimentally infected with *Fusarium oxysporum*. *J. Med. Microbiol.*, *57*(1): 36–42.

Khor, W. B., Aung, T., Saw, S. M., Wong, T. Y., Tambyah, P. A., Tan, A. L., Tan, D. T. H. (2006). AN outbreak of *Fusarium* keratitis associated with contact lens wear in singapore. *JAMA*, *295*(24): 2867–2873.

Klis, F. M., de Groot, P., & Hellingwerf, K. (2001). Molecular organization of the cell wall of Candida albicans. *Medical Mycology*, *39 Suppl 1*: 1–8.

Knudsen, G. R., & Dandurand, L. M. C. (2014). Ecological Complexity and the Success of Fungal Biological Control Agents, Ecological Complexity and the Success of Fungal Biological Control Agents. *Advances in Agriculture, Advances in Agriculture*, *2014,* e542703.

Koehler, P., Tacke, D., & Cornely, O. A. (2014). Bone and joint infections by *Mucorales*, *Scedosporium*, *Fusarium* and even rarer fungi. *Critical Reviews in Microbiology*, *42(1)*: 158-171.

Köhler, G., & Milstein, C. (1975). Continuous cultures of fused cells secreting antibody of predefined specificity. *Nature*, *256*: 495–497.

Kolar, S. S. N., Baidouri, H., Hanlon, S., & McDermott, A. M. (2013). Protective Role of Murine β-Defensins 3 and 4 and Cathelin-Related Antimicrobial Peptide in *Fusarium solani* Keratitis. *Infection and Immunity*, *81*(8):

2669–2677.

Kontoyiannis, D. P., Marr, K. A., Park, B. J., Alexander, B. D., Anaissie, E. J., Walsh, T. J., Pappas, P. G. (2010). Prospective Surveillance for Invasive Fungal Infections in Hematopoietic Stem Cell Transplant Recipients, 2001–2006: Overview of the Transplant-Associated Infection Surveillance Network (TRANSNET) Database. *Clinical Infectious Diseases*, *50*(8): 1091–1100.

Kozel, T. R., & Wickes, B. (2014). Fungal Diagnostics. *Cold Spring Harbor Perspectives in Medicine*, *4*(4): a019299.

Krcmery, V., Jesenska, Z., Spanik, S., Gyarfas, J., Nogova, J., Botek, R., Trupl, J. (1997). Fungaemia due to *Fusarium* spp. in cancer patients. *J Hosp Infect*, *36*(3): 223–8.

Kriengkauykiat, J., Ito, J. I., & Dadwal, S. S. (2011). Epidemiology and treatment approaches in management of invasive fungal infections. *Clinical Epidemiology*, *3*, 175–191.

Kristensen, R., Torp, M., Kosiak, B., & Holst-Jensen, A. (2005). Phylogeny and toxigenic potential is correlated in *Fusarium* species as revealed by partial translation elongation factor 1 alpha gene sequences. *Mycological Research*, *109*(2): 173–186.

Kwon, Y. H., & Epstein, L. (1993). A 90-kDa glycoprotein associated with adhesion of *Nectria haematococca* macroconidia to substrata. *Molecular Plant Microbe Interactions*, *6*, 481–481.

Kwon, Y. H., & Epstein, L. (1997). Involvement of the 90 kDa glycoprotein in adhesion of *Nectria haematococca* macroconidia. *Physiological and Molecular Plant Pathology*, *51*(5): 287–303.

Lackner, M., Najafzadeh, M. J., Sun, J., Lu, Q., & de Hoog, G. S. (2012). Rapid

Identification of *Pseudallescheria* and *Scedosporium* Strains by Using Rolling Circle Amplification. *Applied and Environmental Microbiology*, *78*(1): 126–133.

Laemmli, U. K. (1970). Cleavage of structural proteins during the assembly of the head of bacteriophage T4. *Nature*, *227*(5259): 680–685.

Lagopodi, A. L., Ram, A. F. J., Lamers, G. E. M., Punt, P. J., Van den Hondel, C. A. M. J. J., Lugtenberg, B. J. J., & Bloemberg, G. V. (2002). Novel Aspects of Tomato Root Colonization and Infection by *Fusarium oxysporum* f. sp. *radicis-lycopersici* Revealed by Confocal Laser Scanning Microscopic Analysis Using the Green Fluorescent Protein as a Marker. *Molecular Plant-Microbe Interactions*, *15*(2): 172–179.

Lass-Flörl, C., Mutschlechner, W., Aigner, M., Grif, K., Marth, C., Girschikofsky, M., Nachbaur, D. (2013). Utility of PCR in Diagnosis of Invasive Fungal Infections: Real-Life Data from a Multicenter Study. *Journal of Clinical Microbiology*, *51*(3): 863–868.

Latenser, B. A. (2003). *Fusarium* infections in burn patients: a case report and review of the literature. *The Journal of Burn Care & Rehabilitation*, *24*(5): 285–288.

Latgé, J. P. (2001). The pathobiology of *Aspergillus fumigatus*. *Trends in Microbiology*, *9*(8): 382–389.

Latgé, J. P. (2010). Tasting the fungal cell wall. *Cellular Microbiology*, *12*(7): 863–872.

Latge, J. P., Kobayashi, H., Debeaupuis, J. P., Diaquin, M., Sarfati, J., Wieruszeski, J. M., Fournet, B. (1994). Chemical and immunological characterization of the extracellular galactomannan of *Aspergillus fumigatus*. *Infect Immun*, *62*(12): 5424–5433.

Lattif, A., & Swindell, K. (2010). History of Antifungals. In *Antifungal therapy* (pp. 1–10). New York [u.a.: Informa Healthcare.

Lau, A., Chen, S., Sorrell, T., Carter, D., Malik, R., Martin, P., & Halliday, C. (2007). Development and Clinical Application of a Panfungal PCR Assay To Detect and Identify Fungal DNA in Tissue Specimens. *Journal of Clinical Microbiology*, *45*(2): 380–385.

Lavigne, L. M., Albina, J. E., & Reichner, J. S. (2006). Beta-glucan is a fungal determinant for adhesion-dependent human neutrophil functions. *Journal of Immunology (Baltimore, Md.: 1950)*, *177*(12): 8667–8675.

Leck, A. K., Thomas, P. A., Hagan, M., Kaliamurthy, J., Ackuaku, E., John, M., Johnson, G. J. (2002). Aetiology of suppurative corneal ulcers in Ghana and south India, and epidemiology of fungal keratitis. *The British Journal of Ophthalmology*, *86*(11): 1211–1215.

Leeflang, M. M., Debets-Ossenkopp, Y. J., Visser, C. E., Scholten, R. J. P. M., Hooft, L., Bijlmer, H. A., Vandenbroucke-Grauls, C. M. (2008). Galactomannan detection for invasive aspergillosis in immunocompromized patients. *The Cochrane Database of Systematic Reviews*, (4): CD007394.

Legrand, C., Anaissie, E., Hashem, R., Nelson, P., Bodey, G. P., & Ro, J. (1991). Experimental fusarial hyalohyphomycosis in a murine model. *Journal of Infectious Diseases*, *164*(5): 944–948.

Leonard, G., Stevens, J. R., & Richards, T. A. (2009). REFGEN and TREENAMER: Automated Sequence Data Handling for Phylogenetic Analysis in the Genomic Era. *Evolutionary Bioinformatics*, *2009*(5): 1–5.

Leslie, J. F., & Summerell, B. A. (2006). *The Fusarium laboratory manual*.

Blackwell Publishing.

Levitz, S. M. (1992). Overview of host defenses in fungal infections. *Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America*, *14 Suppl 1*, S37–42.

Levitz, S. M. (2010). Innate recognition of fungal cell walls. *PLoS Pathogens*, *6*(4): e1000758.

Levitz, S. M., Huang, H., Ostroff, G. R., & Specht, C. A. (2014). Exploiting fungal cell wall components in vaccines. *Seminars in Immunopathology*, *37*(2): 199–207.

Li, X. S., Sato, T., Ooiwa, Y., Kusumi, A., Gu, J. D., & Katayama, Y. (2010). Oxidation of elemental sulfur by *Fusarium solani* strain THIF01 harboring endobacterium *Bradyrhizobium* sp. *Microb Ecol*, *60*(1): 96–104.

Li, Y., Wang, Z., Beier, R. C., Shen, J., De Smet, D., De Saeger, S., & Zhang, S. (2011). T-2 toxin, a trichothecene mycotoxin: review of toxicity, metabolism, and analytical methods. *Journal of Agricultural and Food Chemistry*, *59*(8): 3441–3453.

Lin, H. C., Chu, P. H., Kuo, Y. H., & Shen, S. C. (2005). Clinical experience in managing *Fusarium solani* keratitis. *Int J Clin Pract*, *59*(5): 549–54.

Lindow, J. C., Fimlaid, K. A., Bunn, J. Y., & Kirkpatrick, B. D. (2011). Antibodies in Action: Role of Human Opsonins in Killing *Salmonella enterica* Serovar Typhi. *Infection and Immunity*, *79*(8): 3188–3194.

Litvack, M. L., Post, M., & Palaniyar, N. (2011). IgM Promotes the Clearance of Small Particles and Apoptotic Microparticles by Macrophages. *PLoS ONE*, *6*(3): e17223.

Litvinov, N., Silva, M. T. N. da, Heijden, I. M. van der, Graça, M. G., Oliveira, L.

M. de, Fu, L., Levin, A. S. (2015). An outbreak of invasive fusariosis in a children's cancer hospital. *Clinical Microbiology and Infection*, *21*(3): 268.e1–268.e7.

Liu, P., Wu, X., Liao, C., Liu, X., Du, J., Shi, H., Liu, M. (2014). Escherichia coli and Candida albicans Induced Macrophage Extracellular Trap-Like Structures with Limited Microbicidal Activity. *PLoS ONE*, *9*(2): e90042.

Liu, X. C., Lu, B. H., Wang, Xue, & Gao, J. (2015). First Report of *Fusarium solani* Causing Stem and Root Rot on *Foeniculum vulgare* in Jilin Province of China. *Plant Disease*, 100(1): 210.

Liu, Y. S., Wang, N. C., Ye, R. H., & Kao, W. Y. (2014). Disseminated *Fusarium* infection in a patient with acute lymphoblastic leukemia: A case report and review of the literature. *Oncology Letters*, *7*(2): 334–336.

Lockhart, S. R., Diekema, D. J., & Pfaller, M. A. (2009). CHAPTER 1 - The epidemiology of fungal infections. In E. J. A. R. M. A. Pfaller (Ed.), *Clinical Mycology (second edition)* (pp. 1–14). Edinburgh: Churchill Livingstone.

Lorang, J., Tuori, R., Martinez, J., Sawyer, T., Redman, R., Rollins, J., Dickman, M. (2001). Green fluorescent protein is lighting up fungal biology. *Applied and Environmental Microbiology*, *67*(5): 1987–1994.

Low, C. Y., & Rotstein, C. (2011). Emerging fungal infections in immunocompromised patients. *F1000 Medicine Reports*, *3*.

Lowe, R. G. T., & Howlett, B. J. (2012). Indifferent, Affectionate, or Deceitful: Lifestyles and Secretomes of Fungi. *PLoS Pathogens*, *8*(3).

Lu, X., Gao, Y., Zhang, L., Du, M., Li, S., Wang, T., & Gao, H. (2013). Aetiology analyses of 334 cases fungal keratitis. *[Zhonghua Yan Ke Za Zhi]*

Chinese Journal of Ophthalmology, *49*(1): 12–15.

Lucca, A. J. D. (2007). Harmful fungi in both Agriculture and Medicine. *Revista Iberoamericana de Micología*, *24*, 3–13.

Luther, K., & Ebel, F. (2006). Toll-like receptors: Recent advances, open questions and implications for aspergillosis control. *Medical Mycology*, *44*(Suppl. 1): S219–S227.

Ma, H., Croudace, J. E., Lammas, D. A., & May, R. C. (2006). Expulsion of Live Pathogenic Yeast by Macrophages. *Current Biology*, *16*(21): 2156–2160.

Macêdo, D. P. C., Neves, R. P., Souza-Motta, C. M. de, & Magalhães, O. M. C. (2007). Engyodontium album fungaemia: the first reported case. *Brazilian Journal of Microbiology*, *38*(1): 110–112.

Mach, R. L., Kullnig-Gradinger, C. M., Farnleitner, A. H., Reischer, G., Adler, A., & Kubicek, C. P. (2004). Specific detection of *Fusarium langsethiae* and related species by DGGE and ARMS-PCR of a β-tubulin (tub1) gene fragment. *International Journal of Food Microbiology*, *95*(3): 333–339.

Maciá-Vicente, J. G., Jansson, H.-B., Talbot, N. J., & Lopez-Llorca, L. V. (2009). Real-time PCR quantification and live-cell imaging of endophytic colonization of barley (Hordeum vulgare) roots by *Fusarium equiseti* and *Pochonia chlamydosporia*. *The New Phytologist*, *182*(1): 213–228.

Maragos, C. M., Kurtzman, C., Busman, M., Price, N., & McCormick, S. (2013). Development and Evaluation of Monoclonal Antibodies for the Glucoside of T-2 Toxin (T2-Glc). *Toxins*, *5*(7): 1299–1313.

Margolin, W. (2000). Green fluorescent protein as a reporter for macromolecular localization in bacterial cells. *Methods (San Diego, Calif.)*, *20*(1): 62–72.

Marom, E. M., Holmes, A. M., Bruzzi, J. F., Truong, M. T., O'Sullivan, P. J., & Kontoyiannis, D. P. (2008). Imaging of Pulmonary Fusariosis in Patients with Hematologic Malignancies. *American Journal of Roentgenology*, *190*(6): 1605–1609.

Martinez, L. R., & Casadevall, A. (2005). Specific Antibody Can Prevent Fungal Biofilm Formation and This Effect Correlates with Protective Efficacy. *Infection and Immunity*, *73*(10): 6350–6362.

Masuoka, J. (2004). Surface Glycans of Candida albicans and Other Pathogenic Fungi: Physiological Roles, Clinical Uses, and Experimental Challenges. *Clinical Microbiology Reviews*, *17*(2): 281–310.

Mayayo, E., Pujol, I., & Guarro, J. (1999). Experimental pathogenicity of four opportunist *Fusarium* species in a murine model. *J Med Microbiol*, *48*(4): 363–6.

Mbawuike, I. N., & Herscowitz, H. B. (1989). MH-S, a murine alveolar macrophage cell line: morphological, cytochemical, and functional characteristics. *Journal of Leukocyte Biology*, *46*(2): 119–127.

McCann, F., Carmona, E., Puri, V., Pagano, R. E., & Limper, A. H. (2005). Macrophage internalization of fungal beta-glucans is not necessary for initiation of related inflammatory responses. *Infection and Immunity*, *73*(10): 6340–6349.

McClenny, N. (2005). Laboratory detection and identification of *Aspergillus* species by microscopic observation and culture: the traditional approach. *Medical Mycology*, *43*(S1): 125–128.

McCullough, K. C., Parkinson, D., & Crowther, J. R. (1988). Opsonization-enhanced phagocytosis of foot-and-mouth disease virus. *Immunology*, *65*(2): 187–191.

McDermott, A. M. (2009). The Role of Antimicrobial Peptides at the Ocular Surface. *Ophthalmic Research*, *41*(2): 60–75.

Mehl, H. L., & Epstein, L. (2007). *Fusarium solani* species complex isolates conspecific with *Fusarium solani* f. sp. *cucurbitae* race 2 from naturally infected human and plant tissue and environmental sources are equally virulent on plants, grow at 37 degrees C and are interfertile. *Environ Microbiol*, *9*(9): 2189–99.

Mehl, H. L., & Epstein, L. (2008). Sewage and community shower drains are environmental reservoirs of *Fusarium solani* species complex group 1, a human and plant pathogen. *Environ Microbiol*, *10*(1): 219–27.

Menzin, J., Meyers, J. L., Friedman, M., Perfect, J. R., Langston, A. A., Danna, R. P., & Papadopoulos, G. (2009). Mortality, length of hospitalization, and costs associated with invasive fungal infections in high-risk patients. *American Journal of Health-System Pharmacy: AJHP: Official Journal of the American Society of Health-System Pharmacists*, *66*(19): 1711–1717.

Mesquita-Rocha, S., Godoy-Martinez, P. C., Gonçalves, S. S., Urrutia, M. D., Carlesse, F., Seber, A., Colombo, A. L. (2013). The water supply system as a potential source of fungal infection in paediatric haematopoietic stem cell units. *BMC Infectious Diseases*, *13*, 289.

Miceli, M. H., Díaz, J. A., & Lee, S. A. (2011). Emerging opportunistic yeast infections. *The Lancet. Infectious Diseases*, *11*(2): 142–151.

Mikulska, M., Furfaro, E., Del Bono, V., Gualandi, F., Raiola, A. M., Molinari, M. P., Viscoli, C. (2012). Galactomannan testing might be useful for early diagnosis of fusariosis. *Diagn Microbiol Infect Dis*, *72*(4): 367–9.

Mishra, N. N., Ali, S., & Shukla, P. K. (2014). A monoclonal antibody against 47.2 kDa cell surface antigen prevents adherence and affects biofilm formation of *Candida albicans*. *World Journal of Microbiology and Biotechnology*, *31*(1): 11–21.

Modjtahedi, H., Ali, S., & Essapen, S. (2012). Therapeutic application of monoclonal antibodies in cancer: advances and challenges. *British Medical Bulletin*, *104*(1): 41–59.

Montagna, M. T., Caggiano, G., Lovero, G., De Giglio, O., Coretti, C., Cuna, T., Puntillo, F. (2013). Epidemiology of invasive fungal infections in the intensive care unit: results of a multicenter Italian survey (AURORA Project). *Infection*, *41*(3): 645–653.

Moore, D., Robson, G. D., & Trinci, A. P. (2011). *21st Century Guidebook to Fungi with CD*. Cambridge University Press.

Moretti, A. (2009). Taxonomy of *Fusarium* genus: A continuous fight between lumpers and splitters. *Zbornik Matice Srpske Za Prirodne Nauke*, (117): 7–13.

Moretti, A., Belisario, A., Tafuri, A., Ritieni, A., Corazza, L., & Logrieco, A. (2002). Production of Beauvericin by Different Races of *Fusarium Oxysporum* f. sp. *Melonis*, The *Fusarium* Wilt Agent of Muskmelon. *European Journal of Plant Pathology*, *108*(7): 661–666.

Morgan, G., & Levinsky, R. J. (1985). Monoclonal antibodies in diagnosis and treatment. *Archives of Disease in Childhood*, *60*(2): 96–98.

Morrison, C. J., & Warnock, D. W. (2007). Serodiagnosis: antibody and antigen detection. In J. A. Maertens & K. A. Marr (Eds.) (Vol. 47, pp. 65–119). informa healthcare.

Mossavi-Ayatolahi, S., Farajzadeh, S., Fekri, A., Vares, B., & Fekri, S. (2009).

Cutaneous infection by *Fusarium solani* in an addict man with active hepatitis C. *Iranian Journal of Dermatology*, *12*(Suppl. 3): S12–S15.

Muhammed, M., Anagnostou, T., Desalermos, A., Kourkoumpetis, T. K., Carneiro, H. A., Glavis-Bloom, J., Mylonakis, E. (2013). *Fusarium* Infection. *Medicine*, *92*(6): 305–316.

Muhammed, M., Carneiro, H., Coleman, J., & Mylonakis, E. (2011). The challenge of managing fusariosis. *Virulence*, *2*(2): 91–96.

Mukherjee, P. K., Chandra, J., Yu, C., Sun, Y., Pearlman, E., & Ghannoum, M. A. (2012). Characterization of *Fusarium* keratitis outbreak isolates: contribution of biofilms to antimicrobial resistance and pathogenesis. *Invest Ophthalmol Vis Sci*, *53*(8): 4450–7.

Mulè, G., Susca, A., Stea, G., & Moretti, A. (2004). A Species-Specific PCR Assay Based on the Calmodulin Partial Gene for Identification of *Fusarium Verticillioides*, *F. Proliferatum* and *F. Subglutinans*. *European Journal of Plant Pathology*, *110*(5-6): 495–502.

Musa, Al Eisa, Halim, Sahovic, Gyger, Chaudhri, Aljurf. (2000). The spectrum of *Fusarium* infection in immunocompromised patients with haematological malignancies and in non-immunocompromised patients: a single institution experience over 10 years. *British Journal of Haematology*, *108*(3): 544–548.

Naef, A., & Défago, G. (2006). Population structure of plant-pathogenic *Fusarium* species in overwintered stalk residues from Bt-transformed and non-transformed maize crops. *European Journal of Plant Pathology*, *116*(2): 129–143.

Naznin, H. A., Kimura, M., Miyazawa, M., & Hyakumachi, M. (2013). Analysis of Volatile Organic Compounds Emitted by Plant Growth-Promoting

Fungus *Phoma* sp. GS8-3 for Growth Promotion Effects on Tobacco. *Microbes and Environments*, *28*(1): 42–49.

Nelson, P. E., Desjardins, A. E., & Plattner, R. D. (1993). Fumonisins, Mycotoxins Produced by *Fusarium* Species: Biology, Chemistry, and Significance. *Annual Review of Phytopathology*, *31*(1): 233–252.

Nelson, P. E., Dignani, M. C., & Anaissie, E. J. (1994). Taxonomy, biology, and clinical aspects of *Fusarium* species. *Clin. Microbiol. Rev.*, *7*(4): 479–503.

Netea, M. G., Joosten, L. A. B., van der Meer, J. W. M., Kullberg, B. J., & van de Veerdonk, F. L. (2015). Immune defence against Candida fungal infections. *Nature Reviews Immunology*, *15*(10): 630–642.

Nganje, W. E., Bangsund, D. A., Leistritz, F. L., Wilson, W. W., & Tiapo, N. M. (2004). Regional Economic Impacts of *Fusarium* Head Blight in Wheat and Barley. *Review of Agricultural Economics*, *26*(3): 332–347.

Nguyen, M. H., Wissel, M. C., Shields, R. K., Salomoni, M. A., Hao, B., Press, E. G., Clancy, C. J. (2012). Performance of Candida Real-time Polymerase Chain Reaction, β-D-Glucan Assay, and Blood Cultures in the Diagnosis of Invasive Candidiasis. *Clinical Infectious Diseases*, *54*(9): 1240–1248.

Nicola, A. M., Casadevall, A., & Goldman, D. L. (2008). Fungal killing by mammalian phagocytic cells. *Current Opinion in Microbiology*, *11*(4): 313–317.

Noble, J. A., Crow, S. A., Ahearn, D. G., & Kuhn, F. A. (1997). Allergic fungal sinusitis in the southeastern USA: involvement of a new agent *Epicoccum nigrum* Ehrenb. ex Schlecht. 1824. *J Med Vet Mycol*, *35*(6): 405–9.

Nucci, M., & Anaissie, E. (2002). Cutaneous infection by *Fusarium* species in healthy and immunocompromised hosts: implications for diagnosis and management. *Clinical Infectious Diseases*, *35*(8): 909–920.

Nucci, M., & Anaissie, E. (2007). *Fusarium* infections in immunocompromised patients. *Clinical Microbiology Reviews*, *20*(4): 695–704.

Nucci, M., & Anaissie, E. (2009). Hyalohyphomycosis. In E. J. Anaissie, M. R. McGinnis, & M. A. Pfaller (Eds.), *Clinical mycology* (pp. 313–332). Elsevier Health Sciences.

Nucci, M., & Marr, K. A. (2005). Emerging Fungal Diseases. *Clinical Infectious Diseases*, *41*(4): 521–526.

Nucci, M., Anaissie, E. J., Queiroz-Telles, F., Martins, C. A., Trabasso, P., Solza, C., Souza, C. A. (2003). Outcome predictors of 84 patients with hematologic malignancies and *Fusarium* infection. *Cancer*, *98*(2): 315–319.

Nucci, M., Carlesse, F., Cappellano, P., Varon, A. G., Seber, A., Garnica, M., Colombo, A. L. (2014). Earlier Diagnosis of Invasive Fusariosis with *Aspergillus* Serum Galactomannan Testing. *PLoS ONE*, *9*(1): e87784.

Nucci, M., Marr, K. A., Queiroz-Telles, F., Martins, C. A., Trabasso, P., Costa, S., Pasquini, R. (2004). *Fusarium* infection in hematopoietic stem cell transplant recipients. *Clinical Infectious Diseases*, *38*(9): 1237–1242.

Nucci, M., Marr, K. A., Vehreschild, M. J. G. T., de Souza, C. A., Velasco, E., Cappellano, P., Anaissie, E. (2013). Improvement in the outcome of invasive fusariosis in the last decade. *Clinical Microbiology and Infection*, *20*(6): 580–585.

Nucci, M., Tiyomi, A., Gloria, B., Fernanda, S., Revankar, S. G., Brian, L. W., Patterson, T. F. (2002). Nosocomial Outbreak of *Exophiala jeanselmei*

Fungemia Associated with Contamination of Hospital Water. *Clinical Infectious Diseases*, *34*(11): 1475–1480.

O'Donnell, K. (1996). Progress towards a phylogenetic classification of *Fusarium*. *Sydowia*, *48*(1): 57–70.

O'Donnell, K. (2000). Molecular phylogeny of the Nectria haematococca-Fusarium solani species complex. *Mycologia*, *92*(5): 919–938.

O'Donnell, K., & Cigelnik, E. (1997). Two Divergent Intragenomic rDNA ITS2 Types within a Monophyletic Lineage of the Fungus *Fusarium* Are Nonorthologous. *Molecular Phylogenetics and Evolution*, *7*(1): 103–116.

O'Donnell, K., Cigelnik, E., & Casper, H. H. (1998). Molecular Phylogenetic, Morphological, and Mycotoxin Data Support Reidentification of the Quorn Mycoprotein Fungus as *Fusarium venenatum*. *Fungal Genetics and Biology*, *23*(1): 57–67.

O'Donnell, K., Cigelnik, E., & Nirenberg, H. I. (1998). Molecular Systematics and Phylogeography of the *Gibberella fujikuroi* Species Complex. *Mycologia*, *90*(3): 465–493.

O'Donnell, K., Gueidan, C., Sink, S., Johnston, P. R., Crous, P. W., Glenn, A., Sarver, B. A. J. (2009). A two-locus DNA sequence database for typing plant and human pathogens within the *Fusarium oxysporum* species complex. *Fungal Genetics and Biology: FG & B*, *46*(12): 936–948.

O'Donnell, K., Kistler, H. C., Cigelnik, E., & Ploetz, R. C. (1998). Multiple evolutionary origins of the fungus causing Panama disease of banana: concordant evidence from nuclear and mitochondrial gene genealogies. *Proceedings of the National Academy of Sciences of the United States of America*, *95*(5): 2044–9.

O'Donnell, K., Sarver, B. A. J., Brandt, M., Chang, D. C., Noble-Wang, J., Park, B. J., Ward, T. J. (2007). Phylogenetic Diversity and Microsphere Array-Based Genotyping of Human Pathogenic Fusaria, Including Isolates from the Multistate Contact Lens-Associated U.S. Keratitis Outbreaks of 2005 and 2006. *Journal of Clinical Microbiology*, *45*(7): 2235–2248.

O'Donnell, K., Sutton, D. A., Fothergill, A., McCarthy, D., Rinaldi, M. G., Brandt, M. E., Geiser, D. M. (2008). Molecular phylogenetic diversity, multilocus haplotype nomenclature, and in vitro antifungal resistance within the *Fusarium solani* species complex. *J Clin Microbiol*, *46*(8): 2477–90.

O'Donnell, K., Sutton, D. A., Rinaldi, M. G., Magnon, K. C., Cox, P. A., Revankar, S. G., Robinson, J. S. (2004). Genetic Diversity of Human Pathogenic Members of the *Fusarium oxysporum* Complex Inferred from Multilocus DNA Sequence Data and Amplified Fragment Length Polymorphism Analyses: Evidence for the Recent Dispersion of a Geographically Widespread Clonal Lineage and Nosocomial Origin. *Journal of Clinical Microbiology*, *42*(11): 5109–5120.

O'Donnell, K., Sutton, D. A., Rinaldi, M. G., Sarver, B. A. J., Balajee, S. A., Schroers, H. J., Geiser, D. M. (2010). Internet-Accessible DNA Sequence Database for Identifying Fusaria from Human and Animal Infections. *Journal of Clinical Microbiology*, *48*(10): 3708–3718.

Odabasi, Z., Mattiuzzi, G., Estey, E., Kantarjian, H., Saeki, F., Ridge, R. J., Ostrosky-Zeichner, L. (2004). beta-D-glucan as a diagnostic adjunct for invasive fungal infections: Validation, cutoff development, and performance in patients with acute myelogenous leukemia and myelodysplastic syndrome. *Clinical Infectious Diseases*, *39*(2) : 199–205.

Oh, Y. W., Effmann, E. L., & Godwin, J. D. (2000). Pulmonary Infections in Immunocompromised Hosts: The Importance of Correlating the Conventional Radiologic Appearance with the Clinical Setting. *Radiology*, *217*(3): 647–656.

Ojha, S., Tandon, A., Shukla, D., Saraswat, N., & Joshi, S. (2015). Etiology of suppurative corneal ulcers in rural population of Northern India. *Indian Journal of Clinical and Experimental Ophthalmology*, *1*(2): 62–67.

Oren, L., Ezrati, S., Cohen, D., & Sharon, A. (2003). Early Events in the *Fusarium verticillioides*-Maize Interaction Characterized by Using a Green Fluorescent Protein-Expressing Transgenic Isolate. *Applied and Environmental Microbiology*, *69*(3): 1695–1701.

Ostrosky-Zeichner, L., Alexander, B. D., Kett, D. H., Vazquez, J., Pappas, P. G., Saeki, F., Rex, J. H. (2005). Multicenter clinical evaluation of the (1 -> 3) beta-D-glucan assay as an aid to diagnosis of fungal infections in humans. *Clinical Infectious Diseases*, *41*(5): 654–659.

Pagano, L., Caira, M., Candoni, A., Offidani, M., Fianchi, L., Martino, B., Nosari, A. (2006). The epidemiology of fungal infections in patients with hematologic malignancies: the SEIFEM-2004 study. *Haematologica*, *91*(8): 1068–1075.

Palmero, D., Iglesias, C., de Cara, M., Lomas, T., Santos, M., & Tello, J. C. (2009). Species of *Fusarium* Isolated from River and Sea Water of Southeastern Spain and Pathogenicity on Four Plant Species. *Plant Disease*, *93*(4): 377–385.

Palmero, D., Rodríguez, J. M., Cara, M. de, Camacho, F., Iglesias, C., & Tello, J. C. (2010). Fungal microbiota from rain water and pathogenicity of *Fusarium* species isolated from atmospheric dust and rainfall dust.

Journal of Industrial Microbiology & Biotechnology, *38*(1): 13–20.

Pan, X., Wang, Y., Zhou, Q., Chen, P., Xu, Y., Chen, H., & Xie, L. (2011). Activation of focal adhesion kinase enhances the adhesion of *Fusarium solani* to human corneal epithelial cells via the tyrosine-specific protein kinase signaling pathway. *Mol Vis*, *17*, 638–46.

Park, S. J., & Mehrad, B. (2009). Innate Immunity to *Aspergillus* Species. *Clinical Microbiology Reviews*, *22*(4): 535–551.

Parks, L. W., & Casey, W. M. (1995). Physiological Implications of Sterol Biosynthesis in Yeast. *Annual Review of Microbiology*, *49*(1): 95–116.

Pasanen, A.-L., Yli-Pietilä, K., Pasanen, P., Kalliokoski, P., & Tarhanen, J. (1999). Ergosterol Content in Various Fungal Species and Biocontaminated Building Materials. *Applied and Environmental Microbiology*, *65*(1): 138–142.

Peiqian, L., Xiaoming, P., Huifang, S., Jingxin, Z., Ning, H., & Birun, L. (2014). Biofilm formation by *Fusarium oxysporum* f. sp. *cucumerinum* and susceptibility to environmental stress. *FEMS Microbiology Letters*, *350*(2), 138–145.

Pemán, J., Cantón, E., & Espinel-Ingroff, A. (2009). Antifungal drug resistance mechanisms. *Expert Review of Anti-Infective Therapy*, *7*(4), 453–460.

Pérez Vicente, L., Dita, M., & Martinez De La Parte, E. (2014). *Technical Manual: Prevention and diagnostic of Fusarium Wilt (Panama disease) of banana caused by Fusarium oxysporum f. sp. cubense Tropical Race 4 (TR4)*. FAORegional Workshop on the Diagnosis of *Fusarium* Wilt (Panama disease) caused by *Fusarium oxysporum* f. sp. *cubense* Tropical Race 4: Mitigating the Threat and Preventing its Spread in the

Caribbean, St. Augustine, Trinidad and Tobago, 05-09/05/2014: FAO.

Perez-Perez, L., Pereiro, M., Jr., Sanchez-Aguilar, D., & Toribio, J. (2007). Ulcerous lesions disclosing cutaneous infection with *Fusarium solani*. *Acta Derm Venereol*, *87*(5): 422–4.

Perfect, J. R. (2012). The impact of the host on fungal infections. *American Journal of Medicine*, *125*(1 Suppl.): S39–51.

Petrikkos, G., Skiada, A., Lortholary, O., Roilides, E., Walsh, T. J., & Kontoyiannis, D. P. (2012). Epidemiology and Clinical Manifestations of Mucormycosis. *Clinical Infectious Diseases*, *54*(Suppl. 1): S23–S34.

Pfaller, M. A., & Diekema, D. J. (2004). Rare and Emerging Opportunistic Fungal Pathogens: Concern for Resistance beyond Candida albicans and *Aspergillus fumigatus*. *Journal of Clinical Microbiology*, *42*(10): 4419–4431.

Pfaller, M. A., Pappas, P. G., & Wingard, J. R. (2006). Invasive Fungal Pathogens: Current Epidemiological Trends. *Clinical Infectious Diseases*, *43*(Suppl. 1): S3–S14.

Phadke, A. P., & Mehrad, B. (2005). Cytokines in host defense against *Aspergillus*: recent advances. *Medical Mycology*, *43*(Suppl. 1) : S173–S176.

Pierce, C. G., Uppuluri, P., Tristan, A. R., Wormley, F. L., Mowat, E., Ramage, G., & Lopez-Ribot, J. L. (2008). A simple and reproducible 96-well plate-based method for the formation of fungal biofilms and its application to antifungal susceptibility testing. *Nature Protocols*, *3*(9): 1494–1500.

Pilotti, M., Ponzio, V., & Motta, E. (2002). Disorders of Platanus acerifolia in Italy associated with *Fusarium solani*. *Forest Pathology*, *32*, 249–264.

Ponton, J., Rüchel, R., Clemons, K. V., Coleman, D. C., Grillot, R., Guarro, J., Sullivan, D. J. (2000). Emerging pathogens. *Medical Mycology*, *38*(Suppl. 1): 225–236.

Poole, G. J., Smiley, R. W., Walker, C., Huggins, D., Rupp, R., Abatzoglou, J., Paulitz, T. C. (2013). Effect of Climate on the Distribution of *Fusarium* spp. Causing Crown Rot of Wheat in the Pacific Northwest of the United States. *Phytopathology*, *103*(11): 1130–1140.

Pujol, I., Guarro, J., Gené, J., & Sala, J. (1997). In-vitro antifungal susceptibility of clinical and environmental *Fusarium* spp. strains. *J Antimicrob Chemother*, *39*(2): 163–7.

Raad, I., Tarrand, J., Hanna, H., Albitar, M., Janssen, E., Boktour, M., Rolston, K. (2002). Epidemiology, Molecular Mycology, and Environmental Sources of Fusarium Infection in Patients with Cancer. *Infection Control & Hospital Epidemiology*, *23*(09): 532–537.

Rajendran, R., Sherry, L., Nile, C. J., Sherriff, A., Johnson, E. M., Hanson, M. F., Ramage, G. (2016). Biofilm formation is a risk factor for mortality in patients with *Candida albicans* bloodstream infection—Scotland, 2012–2013. *Clinical Microbiology and Infection*, *22*(1): 87–93.

Ramage, G., Mowat, E., Jones, B., Williams, C., & Lopez-Ribot, J. (2009). Our Current Understanding of Fungal Biofilms. *Critical Reviews in Microbiology*, *35*(4): 340–355.

Ramage, G., Rajendran, R., Gutierrez-Correa, M., Jones, B., & Williams, C. (2011). *Aspergillus* biofilms: clinical and industrial significance. *FEMS Microbiology Letters*, *324*(2): 89–97.

Ramage, G., Rajendran, R., Sherry, L., Williams, C., Ramage, G., Rajendran, R., Williams, C. (2012). Fungal Biofilm Resistance, Fungal Biofilm

Resistance. *International Journal of Microbiology, International Journal of Microbiology*, e528521.

Richardson, D. M., & Warnock, D. W. (2003). *Fungal Infection: Diagnosis and Management*.

Roilides, E., Dotis, J., & Katragkou, A. (2007). *Fusarium* and *Scedosporium*: emerging fungal pathogens. In K. Kavanagh (Ed.), *New Insights in Medical Mycology* (pp. 267–285). Springer.

Roilides, E., Lyman, C. A., Armstrong, D., Stergiopoulou, T., Petraitiene, R., & Walsh, T. J. (2006). Deoxycholate amphotericin B and amphotericin B lipid complex exert additive antifungal activity in combination with pulmonary alveolar macrophages against *Fusarium solani*. *Mycoses*, *49*(2): 109–13.

Roilides, E., Lyman, C. A., Armstrong, D., Stergiopoulou, T., Petraitiene, R., & Walsh, T. J. (2006). Deoxycholate amphotericin B and amphotericin B lipid complex exert additive antifungal activity in combination with pulmonary alveolar macrophages against Fusarium solani. *Mycoses*, *49*(2): 109–113.

Rolle, A.-M., Hasenberg, M., Thornton, C. R., Solouk-Saran, D., Männ, L., Weski, J., Wiehr, S. (2016). ImmunoPET/MR imaging allows specific detection of *Aspergillus fumigatus* lung infection in vivo. *Proceedings of the National Academy of Sciences*, *113*(8): E1026–E1033.

Romani, L. (2004). Immunity to fungal infections. *Nat Rev Immunol*, *4*(1): 1–23.

Romani, L. (2011). Immunity to fungal infections. *Nature Reviews Immunology*, *11*(4): 275–288.

Rougeron, A., Schuliar, G., Leto, J., Sitterlé, E., Landry, D., Bougnoux, M. E., Giraud, S. (2015). Human-impacted areas of France are environmental

reservoirs of the *Pseudallescheria boydii*/*Scedosporium apiospermum* species complex. *Environmental Microbiology*, *17*(4): 1039–1048.

Roy, K. (1997). *Fusarium solani* on soybean roots: nomenclature of the causal agent of sudden death syndrome and identity and relevance of *F. solani* form B. *Plant Disease*, *81*(3): 259–266.

Sandoval-Denis, M., Sutton, D. A., Cano-Lira, J. F., Gené, J., Fothergill, A. W., Wiederhold, N. P., & Guarro, J. (2014). Phylogeny of the Clinically Relevant Species of the Emerging Fungus *Trichoderma* and Their Antifungal Susceptibilities. *Journal of Clinical Microbiology*, *52*(6): 2112–2125.

Sanz, F., Soledad Cuétara, M., del Palacio, A., Campbell, C. K., Johnson, L., Urruzunu, P., & Pía Roiz, M. (1993). [Problems found in the isolation of mycelial fungi (*Fusarium solani*) from blood culture (Bacter NR-860)]. *Enferm Infecc Microbiol Clin*, *11*(9): 490–3.

Sautour, M., Edel-Hermann, V., Steinberg, C., Sixt, N., Laurent, J., Dalle, F., Bonnin, A. (2012). *Fusarium* species recovered from the water distribution system of a French university hospital. *International Journal of Hygiene and Environmental Health*, *215*(3): 286–292.

Schäfer, K., Bain, J. M., Di Pietro, A., Gow, N. A. R., & Erwig, L. P. (2014). Hyphal growth of phagocytosed *Fusarium oxysporum* causes cell lysis and death of murine macrophages. *PLoS ONE*, *9*(7).

Scheel, C. M., Hurst, S. F., Barreiros, G., Akiti, T., Nucci, M., & Balajee, S. A. (2013). Molecular analyses of *Fusarium* isolates recovered from a cluster of invasive mold infections in a Brazilian hospital. *BMC Infectious Diseases*, *13*(1): 49.

Schmidig, D. (2012). *Epidemiology and Outcome of Human Fusariosis as*

Reflected in Published Cases of the Disease. University of Zurich.

Schroers, H. J., O'Donnell, K., Lamprecht, S. C., Kammeyer, P. L., Johnson, S., Sutton, D. A., Summerbell, R. C. (2009). Taxonomy and phylogeny of the *Fusarium dimerum* species group. *Mycologia*, *101*(1): 44–70.

Schuerger, A., & Mitchell, D. (1993). Influence of Mucilage Secreted by Macroconidia of *Fusarium solani* f. sp. *phaseoli* on Spore Attachment to Roots of *Vigna radiata* in Hydroponic Nutrient Solution. *Phytopathology*, *83*(11): 1162–1170.

Segal, A. W. (2005). How Neutrophils Kill Microbes. *Annual Review of Immunology*, *23*, 197–223.

Seifert, K. A. (2009). Progress towards DNA barcoding of fungi. *Molecular Ecology Resources*, *9*: 83–89.

Seifert, K. A., & Lévesque, C. A. (2004). Phylogeny and Molecular Diagnosis of Mycotoxigenic Fungi. *European Journal of Plant Pathology*, *110*(5-6): 449–471.

Sesma, A., & Osbourn, A. E. (2004). The rice leaf blast pathogen undergoes developmental processes typical of root-infecting fungi. *Nature*, *431*(7008): 582–586.

Sganga, G. (2011). Fungal infections in immunocompromised patients. *Mycoses*, *54*, 1–3.

Shoham, S., & Levitz, S. M. (2005). The immune response to fungal infections. *Br J Haematol*, *129*(5): 569–82.

Short, D. P., O'Donnell, K., Zhang, N., Juba, J. H., & Geiser, D. M. (2011). Widespread occurrence of diverse human pathogenic types of the fungus *Fusarium* detected in plumbing drains. *J Clin Microbiol*, *49*(12): 4264–72.

Silva, R. F. e. (2010). Chapter 8 - Fungal infections in immunocompromised patients. *Jornal Brasileiro de Pneumologia*, *36*(1): 142–147.

Skadsen, R. W., & Hohn, T. M. (2004). Use of *Fusarium graminearum* transformed with gfp to follow infection patterns in barley and Arabidopsis. *Physiological and Molecular Plant Pathology*, *64*(1): 45–53.

Skiada, A., Pagano, L., Groll, A., Zimmerli, S., Dupont, B., Lagrou, K., Petrikkos, G. (2011). Zygomycosis in Europe: analysis of 230 cases accrued by the registry of the European Confederation of Medical Mycology (ECMM) Working Group on Zygomycosis between 2005 and 2007. *Clinical Microbiology and Infection*, *17*(12): 1859–1867.

Slavin, M. A., & Grist, L. M. (2013). Invasive Fungal Infections: A Continuing Challenge Report from the 17th International Symposium on Infections in the Immunocompromised Host, Genoa, Italy, 24–27 July 2012. *Current Fungal Infection Reports*, *7*(1): 83–88.

Slesiona, S., Gressler, M., Mihlan, M., Zaehle, C., Schaller, M., Barz, D., Brock, M. (2012). Persistence versus Escape*: Aspergillus terreus* and *Aspergillus fumigatus* Employ Different Strategies during Interactions with Macrophages. *PLoS ONE*, *7*(2).

Spellig, T., Bottin, A., & Kahmann, R. (1996). Green fluorescent protein (GFP) as a new vital marker in the phytopathogenic fungus *Ustilago maydis*. Molecular and General Genetics MGG, 252(5): 503–509.

Spellig, T., Bottin, A., & Kahmann, R. (1996). Green fluorescent protein (GFP) as a new vital marker in the phytopathogenic fungusUstilago maydis. *Molecular and General Genetics MGG*, *252*(5): 503–509.

Spiess, B., Seifarth, W., Hummel, M., Frank, O., Fabarius, A., Zheng, C., Buchheidt, D. (2007). DNA Microarray-Based Detection and Identification of Fungal Pathogens in Clinical Samples from Neutropenic Patients. *Journal of Clinical Microbiology*, *45*(11): 3743–3753.

Srinivasan, A., Lopez-Ribot, J. L., & Ramasubramanian, A. K. (2014). Overcoming antifungal resistance. *Drug Discovery Today. Technologies*, *11*, 1–116.

Srinoulprasert, Y., Pongtanalert, P., Chawengkirttikul, R., & Chaiyaroj, S. C. (2009). Engagement of *Penicillium marneffei* conidia with multiple pattern recognition receptors on human monocytes. *Microbiology and Immunology*, *53*(3): 162–172.

Stanzani, M., Tumietto, F., Vianelli, N., & Baccarani, M. (2007). Update on the treatment of disseminated fusariosis: Focus on voriconazole. *Therapeutics and Clinical Risk Management*, *3*(6): 1165.

Steinberg, C., Laurent, J., Edel-Hermann, V., Barbezant, M., Sixt, N., Dalle, F., Sautour, M. (2015). Adaptation of *Fusarium oxysporum* and *Fusarium dimerum* to the specific aquatic environment provided by the water systems of hospitals. *Water Research*, *76*, 53–65.

Steinmann, J., Schmidt, D., Buer, J., & Rath, P. M. (2011). Discrimination of *Scedosporium prolificans* against *Pseudallescheria boydii* and *Scedosporium apiospermum* by semiautomated repetitive sequence-based PCR. *Medical Mycology*, *49*(5): 475–483.

Stempel, J. M., Hammond, S. P., Sutton, D. A., Weiser, L. M., & Marty, F. M. (2015). Invasive Fusariosis in the Voriconazole Era: Single-Center 13-Year Experience. *Open Forum Infectious Diseases*, *2*(3): ofv099.

Stephens, A. E., Gardiner, D. M., White, R. G., Munn, A. L., & Manners, J. M. (2008). Phases of Infection and Gene Expression of *Fusarium graminearum* During Crown Rot Disease of Wheat. *Molecular Plant-Microbe Interactions*, *21*(12): 1571–1581.

Stevens, D. A. (2002). Diagnosis of fungal infections: current status. *The Journal of Antimicrobial Chemotherapy*, *49 Suppl 1*, 11–19.

Stynen, D., Goris, A., Sarfati, J., & Latgé, J. P. (1995). A new sensitive sandwich enzyme-linked immunosorbent assay to detect galactofuran in patients with invasive aspergillosis. *Journal of Clinical Microbiology*, *33*(2): 497–500.

Stynen, D., Sarfati, J., Goris, A., Prévost, M. C., Lesourd, M., Kamphuis, H., Latgé, J. P. (1992). Rat monoclonal antibodies against *Aspergillus* galactomannan. *Infection and Immunity*, *60*(6): 2237–2245.

Sucher, A. J., Chahine, E. B., & Balcer, H. E. (2009). Echinocandins: the newest class of antifungals. *The Annals of Pharmacotherapy*, *43*(10): 1647–1657.

Summerell, B. A., Salleh, B., & Leslie, J. F. (2003). A Utilitarian Approach to *Fusarium* Identification. *Plant Disease*, *87*(2): 117–128.

Sutula, C. L., Gillett, J. M., Morrisey, S. M., & Ramsdell, D. C. (1986). Interpreting ELISA data and establishing the positive-negative threshold. *Plant Disease*, *70*, 722–726.

Swanink, C. M., Meis, J. F., Rijs, A. J., Donnelly, J. P., & Verweij, P. E. (1997). Specificity of a sandwich enzyme-linked immunosorbent assay for detecting *Aspergillus* galactomannan. *Journal of Clinical Microbiology*, *35*(1): 257–260.

Tanriover, M. D., Ascioglu, S., Altun, B., & Uzun, O. (2008). Galactomannan on

the stage: prospective evaluation of the applicability in routine practice and surveillance. *Mycoses*, *53*(1): 16–25.

Tarabishy, A. B., Aldabagh, B., Sun, Y., Imamura, Y., Mukherjee, P. K., Lass, J. H., Pearlman, E. (2008). MyD88 regulation of *Fusarium* keratitis is dependent on TLR4 and IL-1R1 but not TLR2. *The Journal of Immunology*, *181*(1): 593–600.

Taylan Sekeroglu, H., Erdem, E., Yagmur, M., Gumral, R., Ersoz, R., Ilkit, M., & Harbiyeli, I. (2012). Successful medical management of recalcitrant *Fusarium solani* keratitis: molecular identification and susceptibility patterns. *Mycopathologia*, *174*(3): 233–7.

Terra, L., Abreu, P. A., Teixeira, V. L., Paixão, I. C. P., Pereira, R., Leal, B., Castro, H. C. (2014). Mycoses and Antifungals: reviewing the basis of a current problem that still is a biotechnological target for marine products. *Marine Biotechnology*, *1*, 12.

Tewari, J., & Skoropad, W. (1975). Fine structure of the macroconidia of *Fusarium solani*. *Canadian Journal of Botany*, *53*(19): 2134–2146.

Thamke, D., Mendiratta, D., Dhabarde, A., & Shukla, A. (2015). Mycotic keratitis due to Engyodontium album: First case report from India. *Indian Journal of Medical Microbiology*, *33*(2): 303.

Theel, E. S., & Doern, C. D. (2013). β-d-Glucan Testing Is Important for Diagnosis of Invasive Fungal Infections. *Journal of Clinical Microbiology*, *51*(11): 3478–3483.

Thornton, C. R. (2001). Immunological methods for fungi. In N. J. Talbot (Ed.), *Molecular and cellular biology of filamentous fungi: a practical approach* (Vol. 249, pp. 227–257). Oxford: Oxford University Press.

Thornton, C. R. (2008). Development of an immunochromatographic lateral-flow

device for rapid serodiagnosis of invasive aspergillosis. *Clin Vaccine Immunol*, *15*(7): 1095–105.

Thornton, C. R. (2008). Development of an Immunochromatographic Lateral-Flow Device for Rapid Serodiagnosis of Invasive Aspergillosis. *Clinical and Vaccine Immunology*, *15*(7): 1095–1105.

Thornton, C. R. (2009). Tracking the emerging human pathogen *Pseudallescheria boydii* by using highly specific monoclonal antibodies. *Clinical and Vaccine Immunology*, *16*(5): 756–764.

Thornton, C. R. (2010a). Detection of invasive aspergillosis. *Advances in Applied Microbiology*, *70*, 187–216.

Thornton, C. R. (2010b). Environmental fungi as a cause of human mycoses. *Cultue*, *31*(2): 1–8.

Thornton, C. R. (2011). Environmental fungi as a cause of human mycoses. *Biomedical Scientist*, *55*(10): 716–21.

Thornton, C. R. (2012). Serological techniques for diagnosis. In *Fungal Plant Pathogens (Principles and Protocols Series)* (Vols. 1, The Food and Environment Research Agency (Fera), P. Beales, The Food and Environment Research Agency (Fera), K. Hughes, The Food and Environment Research Agency (Fera), UK, pp. 159–177). Wallingford, UK: CABI.

Thornton, C. R., & Talbot, N. J. (2006). Immunofluorescence microscopy and immunogold EM for investigating fungal infections of plants. *Nat. Protoc.*, *5*, 2506–2511.

Thornton, C. R., & Wills, O. E. (2015). Immunodetection of Fungal and Oomycete Pathogens: Established and Emerging Threats to Human Health, Animal Welfare and Global Food Security. *Critical Reviews in*

Microbiology, *41*(1): 27-51.

Thornton, C. R., Pitt, D., Wakley, G. E., & Talbot, N. J. (2002). Production of a monoclonal antibody specific to the genus *Trichoderma* and closely related fungi, and its use to detect *Trichoderma* spp. in naturally infested composts. *Microbiology*, *148*(5): 1263–1279.

Thornton, C. R., Ryder, L. S., Le Cocq, K., & Soanes, D. M. (2015). Identifying the emerging human pathogen *Scedosporium prolificans* by using a species-specific monoclonal antibody that binds to the melanin biosynthetic enzyme tetrahydroxynaphthalene reductase. *Environmental Microbiology*, *17*(4): 1023–1038.

Timling, I., Walker, D. A., Nusbaum, C., Lennon, N. J., & Taylor, D. L. (2014). Rich and cold: diversity, distribution and drivers of fungal communities in patterned-ground ecosystems of the North American Arctic. *Molecular Ecology*, *23*(13): 3258–3272.

Torres, H. A., & Kontoyiannis, D. P. (2011). Hyalohyphomycoses (Hyaline Moulds). In C. A. Kauffman, P. G. Pappas, J. D. Sobel, & W. E. Dismukes (Eds.), *Essentials of Clinical Mycology* (pp. 281–304). Springer New York.

Tortorano, A. M., Esposto, M. C., Prigitano, A., Grancini, A., Ossi, C., Cavanna, C., & Cascio, G. L. (2012). Cross-Reactivity of *Fusarium* spp. in the *Aspergillus* Galactomannan Enzyme-Linked Immunosorbent Assay. *Journal of Clinical Microbiology*, *50*(3): 1051–1053.

Tortorano, A. M., Prigitano, A., Esposto, M. C., Arsic Arsenijevic, V., Kolarovic, J., Ivanovic, D., ECMM Working Group. (2014). European Confederation of Medical Mycology (ECMM) epidemiological survey on invasive infections due to *Fusarium* species in Europe. *European*

Journal of Clinical Microbiology & Infectious Diseases: Official Publication of the European Society of Clinical Microbiology, *33*(9): 1623–1630.

Tortorano, A. M., Richardson, M., Roilides, E., van Diepeningen, A., Caira, M., Munoz, P., Lass-Flörl, C. (2014). ESCMID and ECMM joint guidelines on diagnosis and management of hyalohyphomycosis: *Fusarium* spp., *Scedosporium* spp. and others. *Clinical Microbiology and Infection*, *20*, 27–46.

Tournu, H., & Van Dijck, P. (2011). Candida Biofilms and the Host: Models and New Concepts for Eradication. *International Journal of Microbiology*, *2012*, e845352.

Tsitsikas, D. A., Morin, A., Araf, S., Murtagh, B., Johnson, G., Vinnicombe, S., Agrawal, S. G. (2012). Impact of the revised (2008) EORTC/MSG definitions for invasive fungal disease on the rates of diagnosis of invasive aspergillosis. *Medical Mycology*, *50*(5): 538–542.

Tucker, S. L., & Talbot, N. J. (2001). Surface Attachment and Pre-Penetration Stage Development by Plant Pathogenic Fungi. *Annual Review of Phytopathology*, *39*(1): 385–417.

Tumbarello, M., Posteraro, B., Trecarichi, E. M., Fiori, B., Rossi, M., Porta, R., Sanguinetti, M. (2007). Biofilm Production by *Candida* Species and Inadequate Antifungal Therapy as Predictors of Mortality for Patients with Candidemia. *Journal of Clinical Microbiology*, *45*(6): 1843–1850.

van Diepeningen, A. D. van, Al-Hatmi, A. M. S., Brankovics, B., & Hoog, G. S. de. (2014). Taxonomy and Clinical Spectra of *Fusarium* Species: Where Do We Stand in 2014? *Current Clinical Microbiology Reports*, *1*(1-2)

: 10–18.

van Diepeningen, A. D. van, Brankovics, B., Iltes, J., Lee, T. A. J. van der, & Waalwijk, C. (2015). Diagnosis of Infections: Approaches to Identification by the Clinical Mycology Laboratory. *Current Fungal Infection Reports*, *9*(3): 135–143.

van Diepeningen, A. D., Feng, P., Ahmed, S., Sudhadham, M., Bunyaratavej, S., & de Hoog, G. S. (2015). Spectrum of *Fusarium* infections in tropical dermatology evidenced by multilocus sequencing typing diagnostics. *Mycoses*, *58*(1): 48–57.

van Kessel, K. P. M., Bestebroer, J., & van Strijp, J. A. G. (2014). Neutrophil-mediated phagocytosis of Staphylococcus aureus. *Microbial Immunology*, *5*, 467.

Verma, S., & Tuft, S. J. (2002). *Fusarium solani* keratitis following LASIK for myopia. *Br J Ophthalmol*, *86*(10): 1190–1.

Verweij, P. E., & Mennink-Kersten, M. A. (2006). Issues with galactomannan testing. *Medical Mycology*, *44*(s1): 179–183.

Vismer, H. F., Marasas, W. F. O., Rheeder, J. P., & Joubert, J. J. (2002). *Fusarium dimerum* as a cause of human eye infections. *Medical Mycology*, *40*(4): 399–406.

Wallander, H., Ekblad, A., Godbold, D. L., Johnson, D., Bahr, A., Baldrian, P., Rudawska, M. (2013). Evaluation of methods to estimate production, biomass and turnover of ectomycorrhizal mycelium in forests soils – A review. *Soil Biology and Biochemistry*, *57*, 1034–1047.

Walsh, T., & Groll, A. (1999). Emerging fungal pathogens: evolving challenges to immunocompromised patients for the twenty-first century. *Transplant Infectious Disease*, *1*(4): 247–261.

Walsh, T., Groll, A., Hiemenz, J., Fleming, R., Roilides, E., & Anaissie, E. (2004). Infections due to emerging and uncommon medically important fungal pathogens. *Clinical Microbiology and Infection*, *10*(s1): 48–66.

Wang, H., Qi, M., & Cutler, A. J. (1993). A simple method of preparing plant samples for PCR. *Nucleic Acids Research*, *21*(17): 4153–4.

Wang, H., Xiao, M., Kong, F., Chen, S., Dou, H. T., Sorrell, T., Xu, Y. C. (2011). Accurate and practical identification of 20 *Fusarium* species by seven-locus sequence analysis and reverse line blot hybridization, and an in vitro antifungal susceptibility study. *J Clin Microbiol*, *49*(5): 1890–8.

Wang, H., Xu, Z., Gao, L., & Hao, B. (2009). A fungal phylogeny based on 82 complete genomes using the composition vector method. *BMC Evolutionary Biology*, *9*(1): 195.

Wasylnka, J. A., & Moore, M. M. (2000). Adhesion of *Aspergillus* species to extracellular matrix proteins: evidence for involvement of negatively charged carbohydrates on the conidial surface. *Infection and Immunity*, *68*(6): 3377–3384.

Wasylnka, J. A., & Moore, M. M. (2002). Uptake of *Aspergillus fumigatus* Conidia by phagocytic and nonphagocytic cells in vitro: quantitation using strains expressing green fluorescent protein. *Infection and Immunity*, *70*(6): 3156–3163.

Wellington, M., Bliss, J. M., & Haidaris, C. G. (2003). Enhanced Phagocytosis of Candida Species Mediated by Opsonization with a Recombinant Human Antibody Single-Chain Variable Fragment. *Infection and Immunity*, *71*(12): 7228–7231.

Wellington, M., Dolan, K., & Haidaris, C. G. (2007). Monocyte responses to Candida albicans are enhanced by antibody in cooperation with

antibody-independent pathogen recognition. *FEMS Immunology & Medical Microbiology*, *51*(1): 70–83.

Wheat, L. J., Goldman, M., & Sarosi, G. A. (2002). Fungal Infections in the Immunocompromised Host. In Robert H Rubin, Lowell S Young (Eds.), *Clinical Approach to Infection in the Compromised Host* (pp. 215–247). Springer US.

Wheat, L. J., Hackett, E., Durkin, M., Connolly, P., Petraitiene, R., Walsh, T. J., Hage, C. (2007). Histoplasmosis-Associated Cross-Reactivity in the BioRad Platelia *Aspergillus* Enzyme Immunoassay. *Clinical and Vaccine Immunology*, *14*(5): 638–640.

White, T., Bruns, T., Lee, S., & Taylor, J. (1990). Amplification and direct sequencing of fungal ribosomal RNA genes for phylogenetics, 315 – 322.

Williams, A. E., Hussell, T., & Lloyd, C. (2012). *Immunology: mucosal and body surface defences* (1st ed.). A John Wiley & Sons, Ltd., Publication.

Wong, W. c., White, M., & Wright, I. g. (1988). Production of monoclonal antibodies to *Fusarium oxysporum* f.sp. *cubense* race 4. *Letters in Applied Microbiology*, *6*(3): 39–42.

Wüthrich, M., Deepe, G. S., & Klein, B. (2012). Adaptive Immunity to Fungi. *Annual Review of Immunology*, *30*, 115–148.

Xia, Y., Větvička, V., Yan, J., Hanikýřová, M., Mayadas, T., & Ross, G. D. (1999). The β-Glucan-Binding Lectin Site of Mouse CR3 (CD11b/CD18) and Its Function in Generating a Primed State of the Receptor That Mediates Cytotoxic Activation in Response to iC3b-Opsonized Target Cells. *The Journal of Immunology*, *162*(4): 2281–2290.

Yang, Z., Jaeckisch, S. M., & Mitchell, C. G. (2000). Enhanced binding of *Aspergillus fumigatus* spores to A549 epithelial cells and extracellular matrix proteins by a component from the spore surface and inhibition by rat lung lavage fluid. *Thorax, 55(7):* 579–584.

Yazar, S., & Omurtag, G. Z. (2008). Fumonisins, Trichothecenes and Zearalenone in Cereals. *International Journal of Molecular Sciences*, *9*(11): 2062–2090.

Yera, H., Bougnoux, M. E., Jeanrot, C., Baixench, M. T., De Pinieux, G., & Dupouy-Camet, J. (2003). Mycetoma of the Foot Caused by *Fusarium solani*: Identification of the Etiologic Agent by DNA Sequencing. *Journal of Clinical Microbiology*, *41*(4): 1805–1808.

Yli-Mattila, T. (2010). Ecology and evolution of toxigenic *Fusarium* species in cereals in northern europe and asia. *Journal of Plant Pathology*, *92*(1): 7–18.

Yli-Mattila, T., Mach, R. L., Alekhina, I. A., Bulat, S. A., Koskinen, S., Kullnig-Gradinger, C. M., Klemsdal, S. S. (2004). Phylogenetic relationship of *Fusarium langsethiae* to *Fusarium poae* and *Fusarium sporotrichioides* as inferred by IGS, ITS, β-tubulin sequences and UP-PCR hybridization analysis. *International Journal of Food Microbiology*, *95*(3), 267–285.

Yli-Mattila, T., Paavanen-Huhtala, S., Bulat, S. A., Alekhina, I. A., & Nirenberg, H. I. (2002). Molecular, morphological and phylogenetic analysis of the *Fusarium avenaceum/F. arthrosporioides/F. tricinctum* species complex –a polyphasic approach. *Mycological Research*, *106*(6): 655–669.

Yoshida, M. (2006). Usefulness of determination of beta-D-glucan in the diagnosis of deep mycosis - Experience in Japan. *Medical Mycology*, *44*, S185–S189.

Yoshida, M., Obayashi, T., Iwama, A., Ito, M., Tsunoda, S., Suzuki, T., Miura, Y. (1997). Detection of plasma (1 → 3)-β-d-glucan in patients with *Fusarium*, *Trichosporon*, *Saccharomyces* and *Acremonium fungaemias*. *Medical Mycology*, *35*(5): 371–374.

Zeng, J. S., Sutton, D. A., Fothergill, A. W., Rinaldi, M. G., Harrak, M. J., & de Hoog, G. S. (2007). Spectrum of Clinically Relevant *Exophiala* Species in the United States. *Journal of Clinical Microbiology*, *45*(11): 3713–3720.

Zhang, J., Bruton, B. D., & Biles, C. L. (1999). *Fusarium solani* endo-polygalacturonase from decayed muskmelon fruit: purification and characterization. *Physiological and Molecular Plant Pathology*, *54*(5-6) : 171–186.

Zhang, N., O'Donnell, K., Sutton, D. A., Nalim, F. A., Summerbell, R. C., Padhye, A. A., & Geiser, D. M. (2006). Members of the *Fusarium solani* Species Complex That Cause Infections in Both Humans and Plants Are Common in the Environment. *Journal of Clinical Microbiology*, *44*(6): 2186–2190.

Zhang, S., Zhao, B., Liu, X., Gao, Z., & Huang, X. (2013). Construction of a new GFP vector and its use for *Fusaruim oxysporum* transformation. *Saudi Journal of Biological Sciences*, *20*(1): 23–27.

APPENDIX 1: Agglutination Data

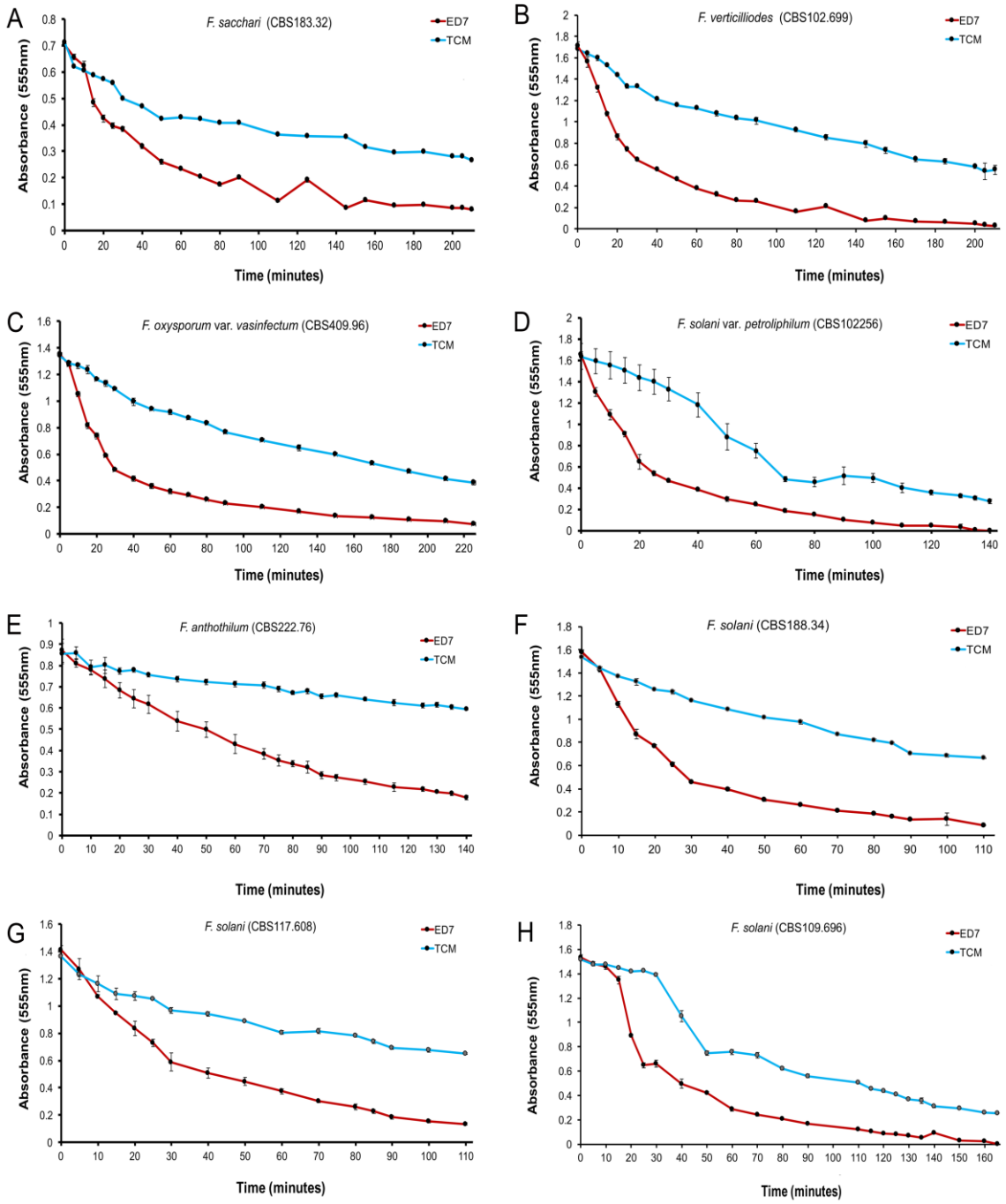


Figure 5.1. Agglutination of *Fusarium* species microconidia. (**A**) *F. sacchari* (CBS183.32), (**B**) *F. verticilliodes* (CBS102.699), (**C**) *F. oxysporum* var. *vasinfectum* (CBS409.96), (**D**) *F. solani* var. *petroliphilum* (CBS102256), (**E**) *F. anthothilum* (CBS222.76), (**F**) *F. solani* (CBS188.34), (**G**) *F. solani* (CBS117.608) and (**H**) *F. solani* (CBS109.696) spores suspended at 10^6 spore ml^{-1} in ED7 TCS and 10% TCM. Each point is the mean of 3 replicates ± S.E. Note rapid decrease in absorbance of light at 555nm due to agglutination of spores by mAb ED7.

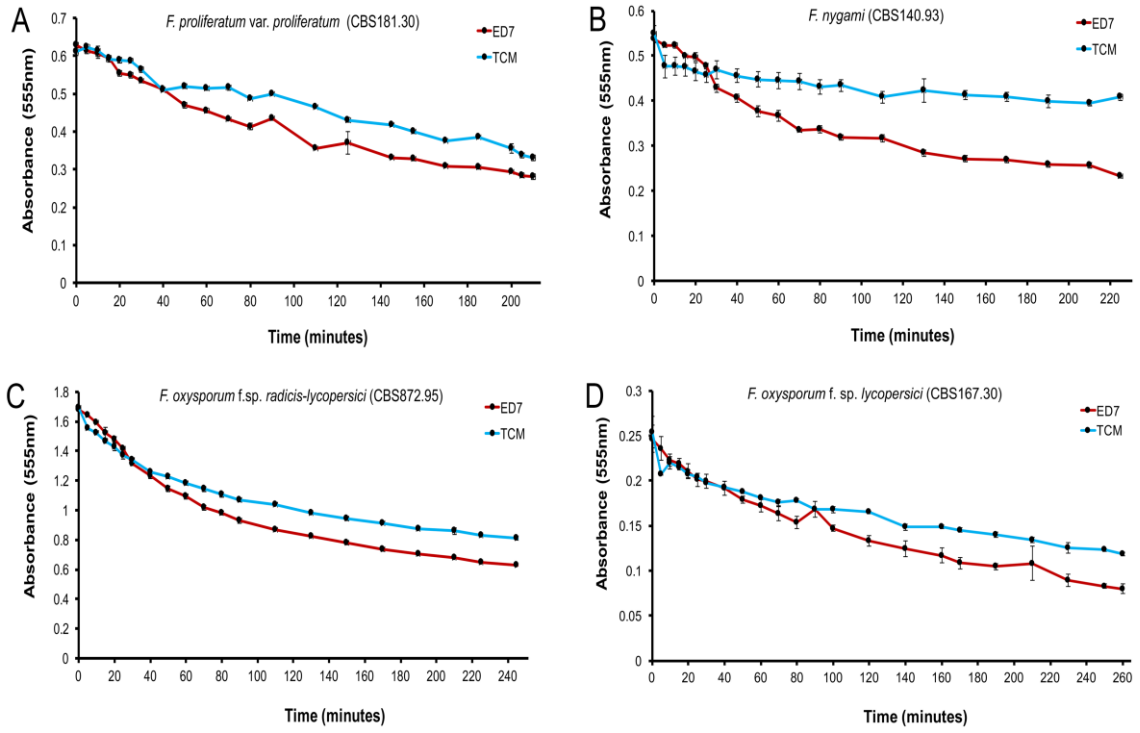


Figure 5.2. Agglutination of *Fusarium* species microconidia. (**A**) *F. proliferatum* var. *proliferatum* (CBS181.30), (**B**) *F. nygami* (CBS140.93), (**C**) *F. oxysporum* f.sp. *radicis-lycopersici* (CBS872.95) and (**D**) *F. oxysporum* f.sp. *lycopersici* (CBS167.30) spores suspended at 10^6 spore ml^{-1} in ED7 TCS and 10% TCM. Each point is the mean of 3 replicates ± S.E. Note weak agglutination of spores of these strains by mAb ED7 compared to above strains (**Figure 5.1**).

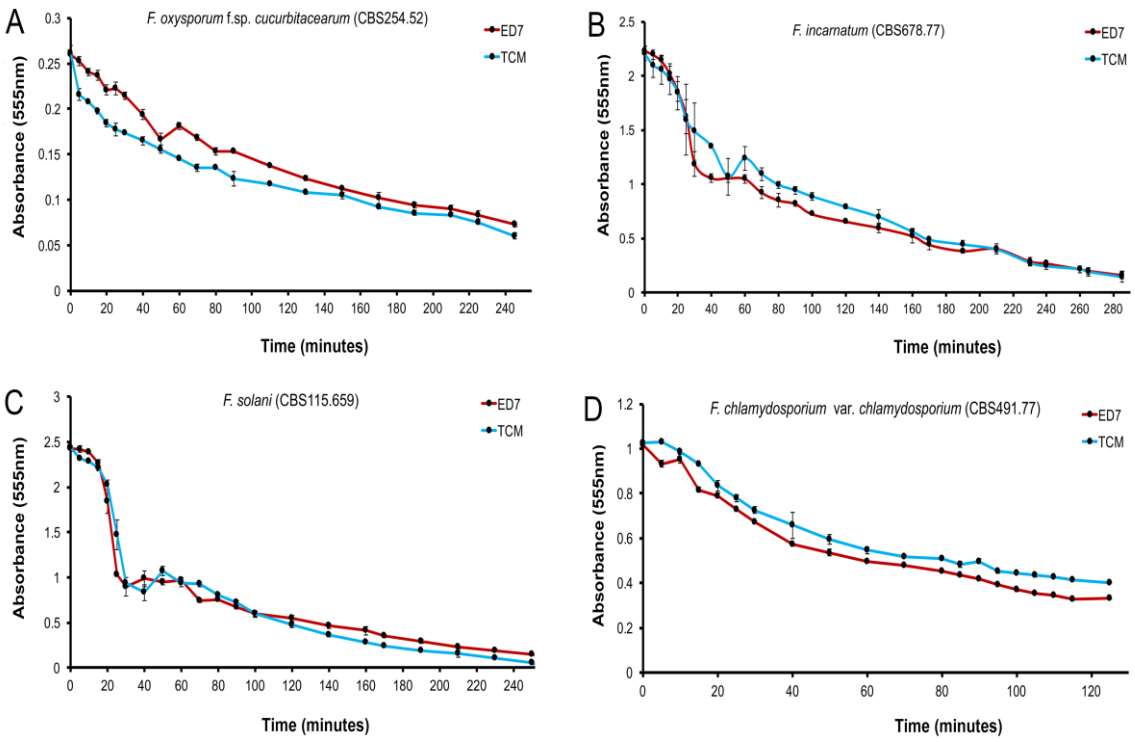
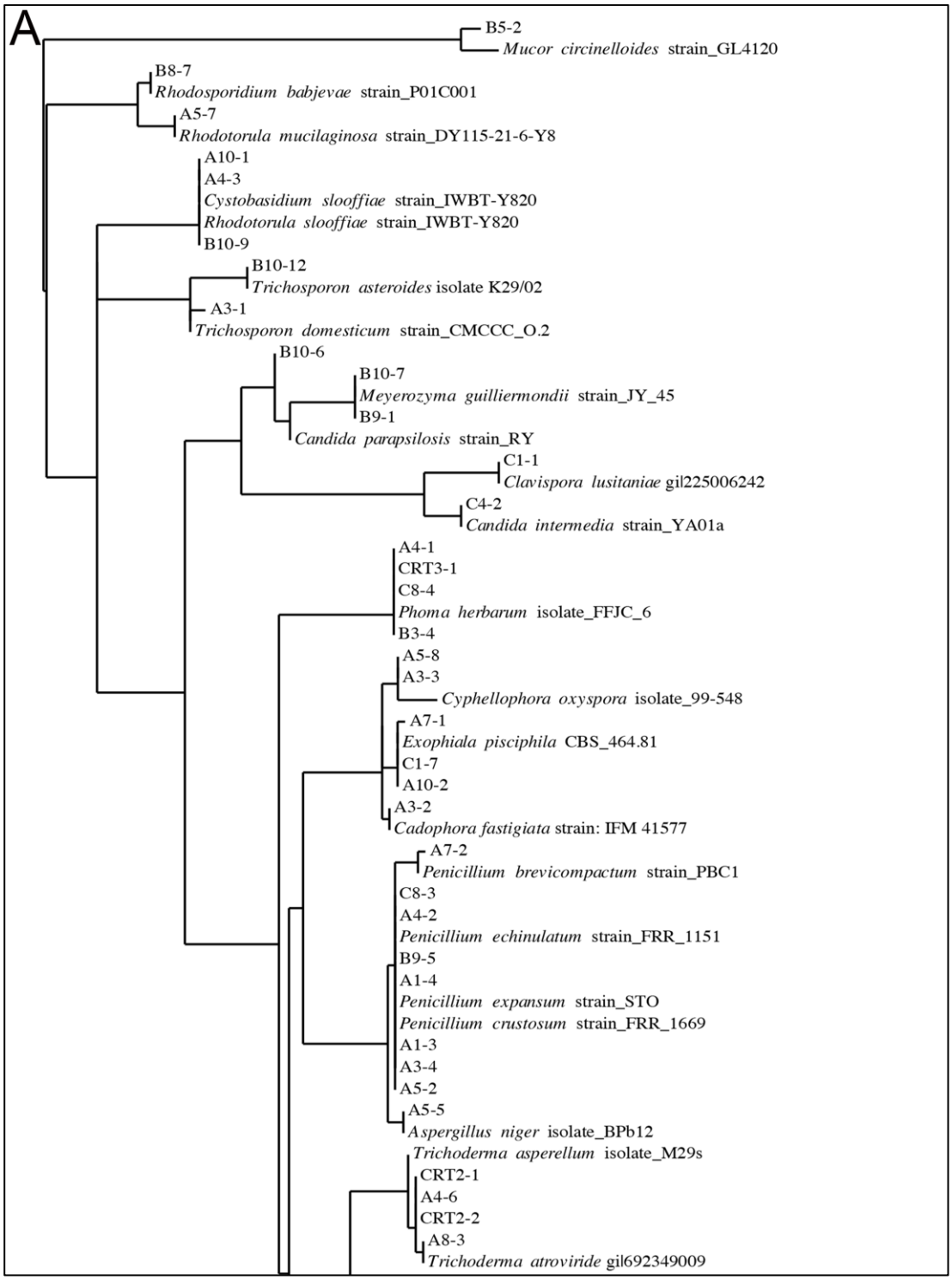
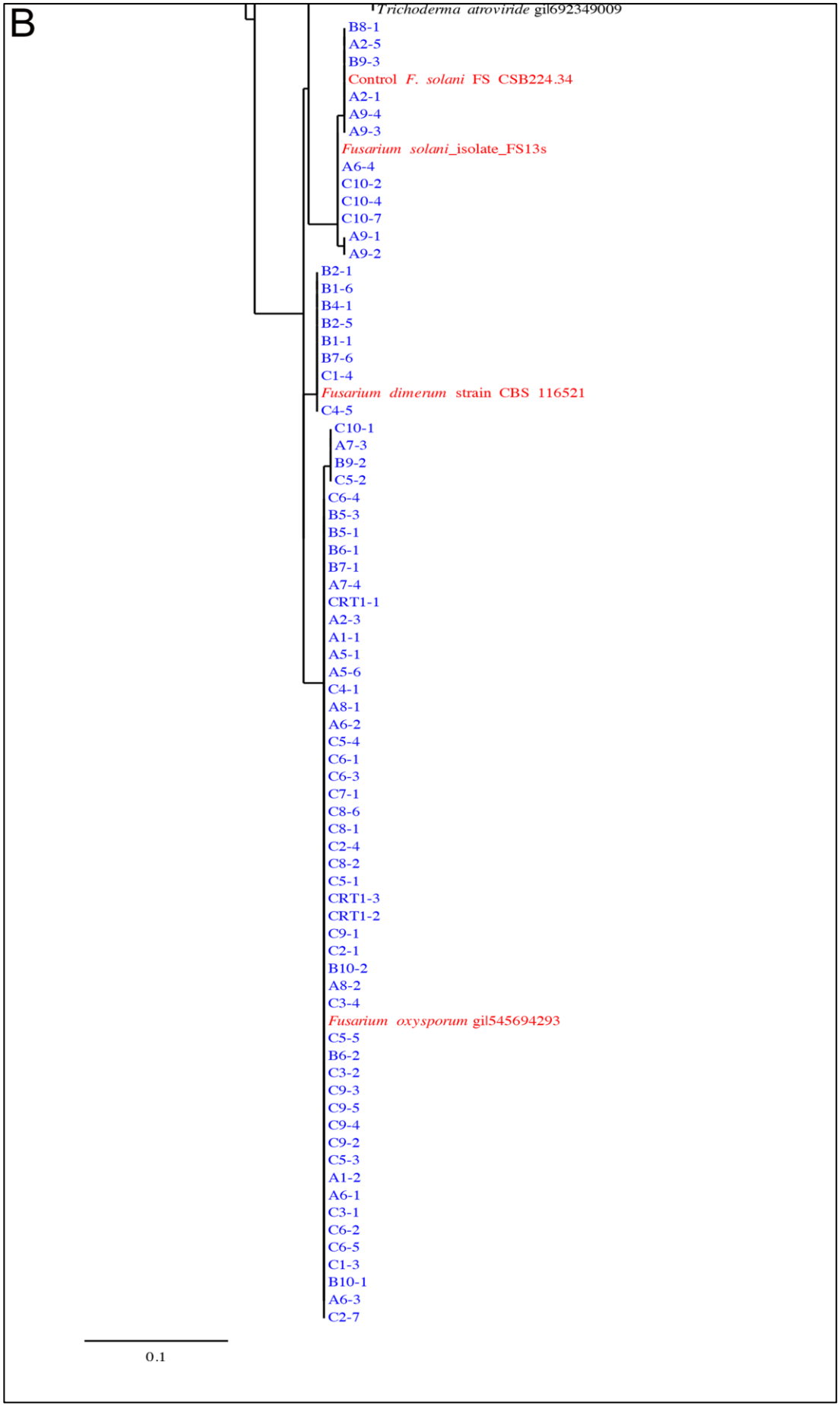


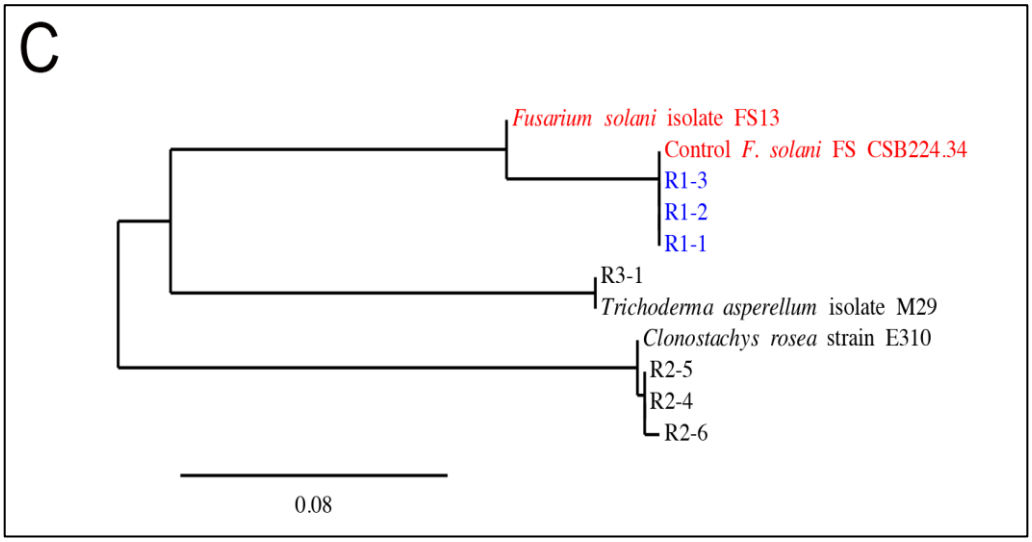
Figure 5.3. Agglutination of *Fusarium* species microconidia. (**A**) *F. oxysporum* f.sp. *cucurbitacearum* (CBS254.52), (**B**) *F. incarnatum* (CBS678.77), (**C**) *F. solani* (CBS115.659) and (**D**) *F. chlamydosporium* var. *chlamydosporium* (CBS491.77) spores suspended at 10^6 spore ml^{-1} in ED7 TCS and 10% TCM. Each point is the mean of 3 replicates ± S.E. Note lack of spore agglutination of these strains compared to *F. solani* (CBS224.34).

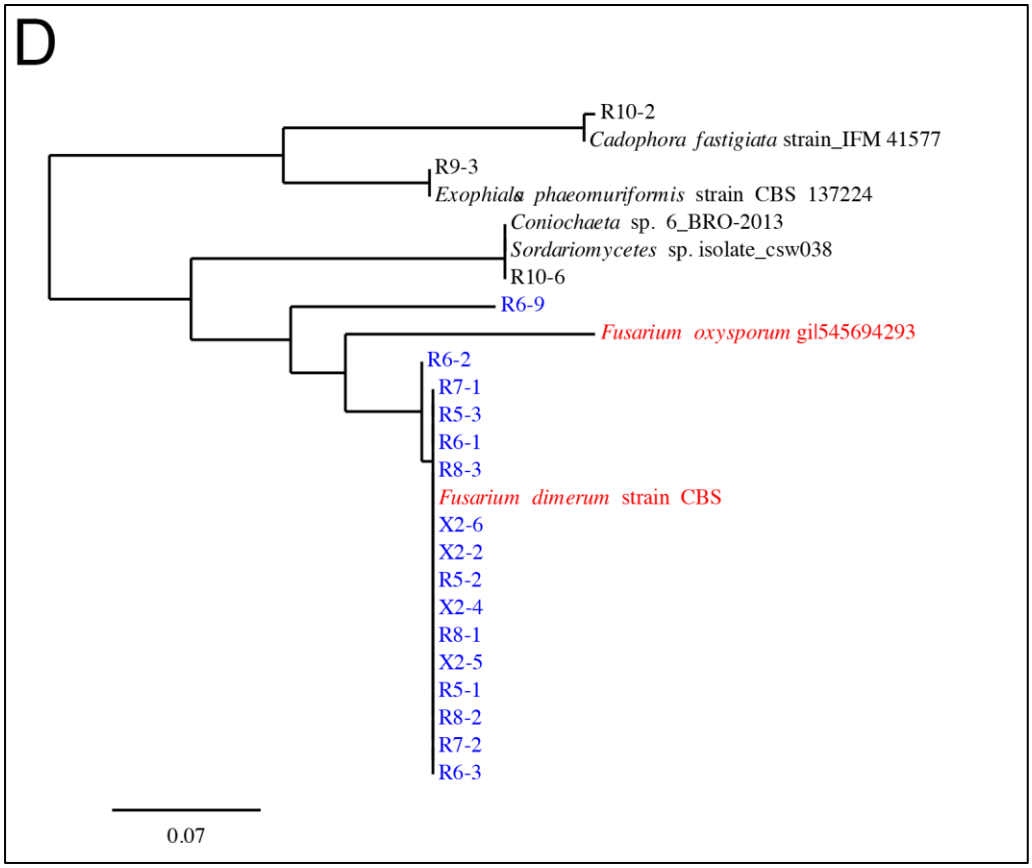
APPENDIX 2: Phylogeny Trees

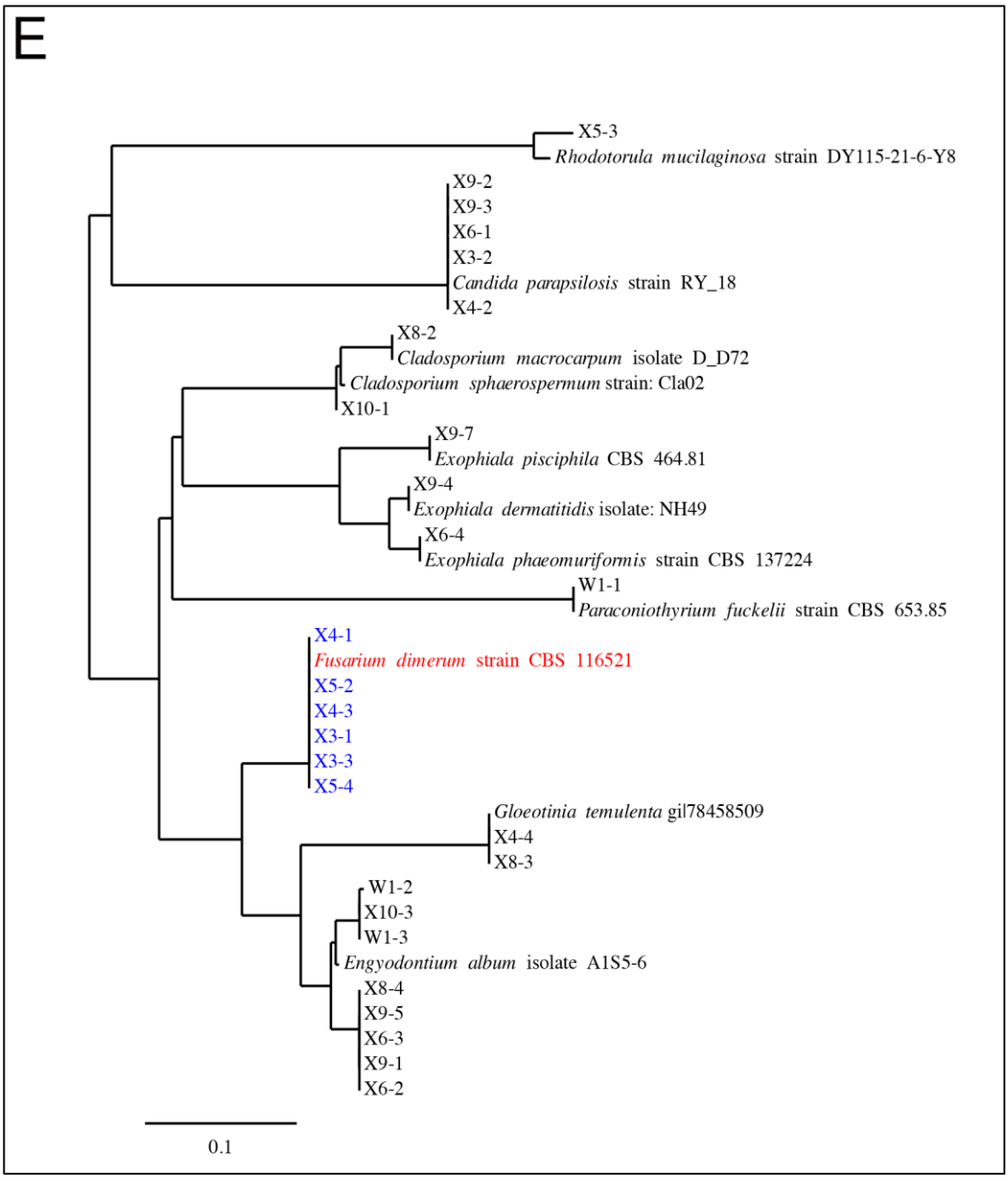


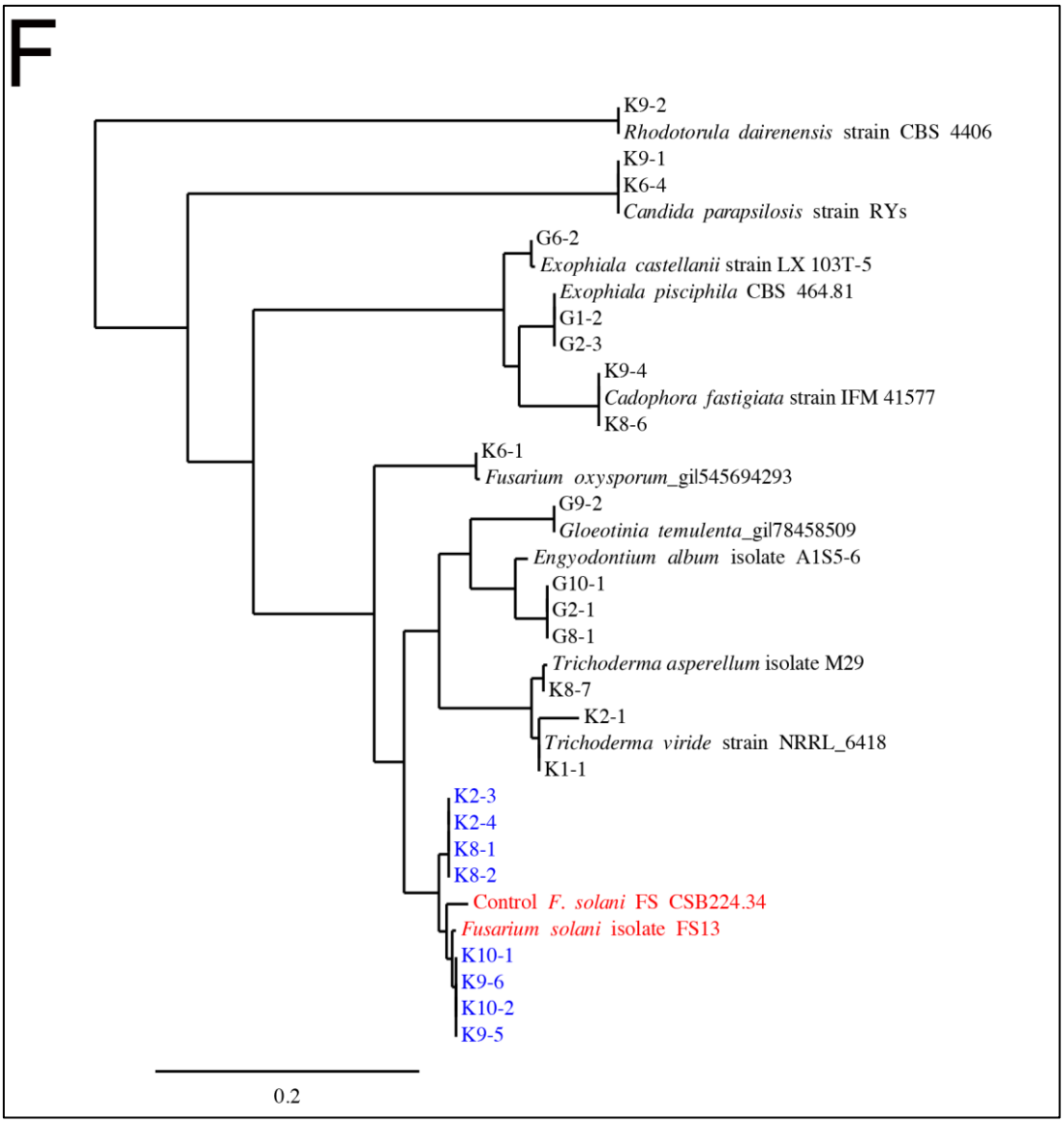
cont.


B
Trichoderma atroviride gil692349009
B8-1
A2-5
B9-3
Control F. solani FS CSB224.34
A2-1
A9-4
A9-3
Fusarium solani_isolate_FS13s
A6-4
C10-2
C10-4
C10-7
A9-1
A9-2
B2-1
B1-6
B4-1
B2-5
B1-1
B7-6
C1-4
Fusarium dimerum strain CBS 116521
C4-5
C10-1
A7-3
B9-2
C5-2
C6-4
B5-3
B5-1
B6-1
B7-1
A7-4
CRT1-1
A2-3
A1-1
A5-1
A5-6
C4-1
A8-1
A6-2
C5-4
C6-1
C6-3
C7-1
C8-6
C8-1
C2-4
C8-2
C5-1
CRT1-3
CRT1-2
C9-1
C2-1
B10-2
A8-2
C3-4
Fusarium oxysporum gil545694293
C5-5
B6-2
C3-2
C9-3
C9-5
C9-4
C9-2
C5-3
A1-2
A6-1
C3-1
C6-2
C6-5
C1-3
B10-1
A6-3
C2-7
0.1


C
Fusarium solani isolate FS13
Control F. solani FS CSB224.34
R1-3
R1-2
R1-1
R3-1
Trichoderma asperellum isolate M29
Clonostachys rosea strain E310
R2-5
R2-4
R2-6
0.08


D
R10-2
Cadophora fastigiata strain_IFM 41577
R9-3
Exophiala phaeomuriformis strain CBS 137224
Coniochaeta sp. 6_BRO-2013
Sordariomycetes sp. isolate_csw038
R10-6
R6-9
Fusarium oxysporum gil545694293
R6-2
R7-1
R5-3
R6-1
R8-3
Fusarium dimerum strain CBS
X2-6
X2-2
R5-2
X2-4
R8-1
X2-5
R5-1
R8-2
R7-2
R6-3
0.07


E
X5-3
Rhodotorula mucilaginosa strain DY115-21-6-Y8
X9-2
X9-3
X6-1
X3-2
Candida parapsilosis strain RY_18
X4-2
X8-2
Cladosporium macrocarpum isolate D_D72
Cladosporium sphaerospermum strain: Cla02
X10-1
X9-7
Exophiala pisciphila CBS 464.81
X9-4
Exophiala dermatitidis isolate: NH49
X6-4
Exophiala phaeomuriformis strain CBS 137224
W1-1
Paraconiothyrium fuckelii strain CBS 653.85
X4-1
Fusarium dimerum strain CBS 116521
X5-2
X4-3
X3-1
X3-3
X5-4
Gloeotinia temulenta gil78458509
X4-4
X8-3
W1-2
X10-3
W1-3
Engyodontium album isolate A1S5-6
X8-4
X9-5
X6-3
X9-1
X6-2
0.1


F
K9-2
Rhodotorula dairenensis strain CBS 4406
K9-1
K6-4
Candida parapsilosis strain RYs
G6-2
Exophiala castellanii strain LX 103T-5
Exophiala pisciphila CBS 464.81
G1-2
G2-3
K9-4
Cadophora fastigiata strain IFM 41577
K8-6
K6-1
Fusarium oxysporum_gil545694293
G9-2
Gloeotinia temulenta_gil78458509
Engyodontium album isolate A1S5-6
G10-1
G2-1
G8-1
Trichoderma asperellum isolate M29
K8-7
K2-1
Trichoderma viride strain NRRL_6418
K1-1
K2-3
K2-4
K8-1
K8-2
Control F. solani FS CSB224.34
Fusarium solani isolate FS13
K10-1
K9-6
K10-2
K9-5
0.2

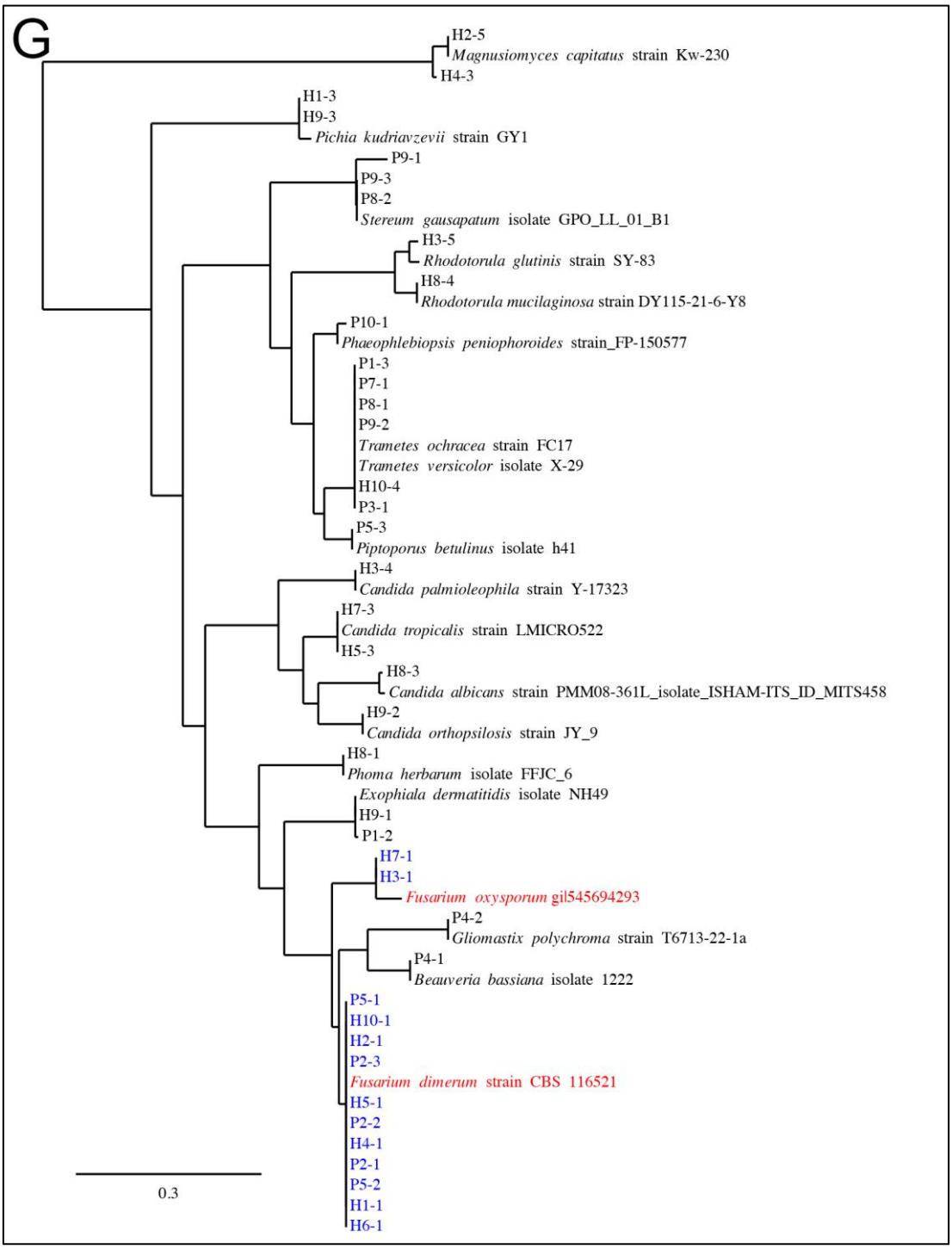


Figure 5. 4. Phylogram of the recovered fungal isolates from sink drains and water system based on ITS compare with reference isolates from NCBI. The trees are divided based on location of swab samples (**A** and **B**): University, (**C**): ICU, (**D**): ITU, (**E**): Ophthalmology Unit, (**F**): Haematology Unit and (**G**): Oncology Unit.

APPENDIX 3: ITS Sequences

>Seq1 [organism=*Candida parapsilosis*] [isolate X9-2] internal transcribed spacer 1, 5.8S ribosomal RNA gene, and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAACTTTGCTTTGGTAGGCCT
TCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAACCGATTATTT
AATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGAA
ATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCACATTGC
GCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCAAACCCTCGGG
TTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAGTATAAACTAATGGAT
AGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAATTCGACCTCAAATCAGG
TAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAAAAAG

>Seq2 [organism=*Candida parapsilosis*] [isolate X9-3] internal transcribed spacer 1, 5.8S ribosomal RNA gene, and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGGTAGGCC
TTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAACCGATTATT
TAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGA
AATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCACATTG
CGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCAAACCCTCGG
GTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAGTATAAACTAATGGA
TAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAATTCGACCTCAAATCAG
GTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAACCCGTGTC

>Seq3 [organism=*Candida parapsilosis*] [isolate X3-2] internal transcribed spacer 1, 5.8S ribosomal RNA gene, and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGGTAGGCC
TTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAACCGATTATT
TAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGA
AATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCACATTG
CGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCAAACCCTCGG
GTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAGTATAAACTAATGGA
TAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAATTCGACCTCAAATCAG
GTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAACTTTTTTT

>Seq4 [organism=*Candida parapsilosis*] [isolate X6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGGTAGG
CCTTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAACCGA
TTATTTAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCA
AACCCTCGGGTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAGTATA
AACTAATGGATAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAATTCG
ACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGGAAAAAGA
AAACCCCTT

>Seq5 [organism=*Fusarium dimerum*] [isolate X4-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TGCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGGTA
GGCCTTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAACC
GATTATTTAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCT
CAAACCCTCGGGTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAGTA
TAAACTAATGGATAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAATT
CGACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAA

>Seq6 [organism=*Rhodotorula mucilaginosa*] [isolate X5-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGGACGTCACTTAACTTGGAGTCCGAACTCTCACTTTCTAACCCTGTGCACTTGTTTGGG
ATAGTAACTCTCGCAAGAGAGCGAACTCCTATTCACTTATAAACACAAAGTCTATGAATG
TATTAAATTTTATAACAAAATAAAACTTTCAACAACGGATCTCTTGGCTCTCGCATCGAT
GAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAAT
CTTTGAACGCACCTTGCGCTCCATGGTATTCCGTGGAGCATGCCTGTTTGAGTGTCATGA
ATACTTCAACCCTCCTCTTTCTTAATGATTGAAGAGGTGTTTGGTTTCTGAGCGCTGCTG
GCCTTTACGGTCTAGCTCGTTCGTAATGCATTAGCATCCGCAATCGAACTTCGGATTGAC
TTGGCGTAATAGACTATTCGCTGAGGAATTCTAGTCTTCGGATTAGAGCCGGGTTGGGTT
AAAGGAAGCTTCTAATCAGAATGTCTACATTTTAAGATTAGATCTCAAATCAGGTAGGAC
TACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAAAAAAA

>Seq7 [organism=*Exophiala pisciphila*] [isolate X9-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TTATAGGCTCGACCTCCCACCCTTTGTTTACCCGACCCACGTTGCTTCGGCGAGCCCGTC
TCTCCCTCTCCGGGGGGGCAGACCGCCGGGGGGCCTTCAAACCGTCCTCTGGCCCGCGC
TCGTCGGTGGCCAACCTTTACAAATTCTTAATTAAACGTGTCTACTCTGAGTACTATTTTT
TAATTAAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCA
GCGAAATGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACG
CACATTGCGCCCTTTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCTCTC
AAGCCCCCCTTTTCTGGGCTTGGTGTTGGACGGCCTGGCGTCGGCGACGACCCCACCTC
CCAAAGACAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCCTCACCGAACAC
GTACTGGATCAAGGGTAGACGGAGCCCCGGTCGACCTCCCCTCACAGGGAGACACTTTTT
TACAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGA
GGGAAAAAGAAAAAAAA

>Seq8 [organism=*Exophiala dermatitidis*] [isolate X9-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTCTCAGGCCGACCTCCCAACCCTTTGTTTACCCGACCCATGTTGCTTCGGCGGGCCCGC
CGTTTCGACGGCCGCCGGAGGACCGCCTATTCAGGTCCTCTGGCCCGCGCCCGCCGGTA
GCCAATTCTACCAAACTCTTGAATCAAATCGTGTCCAATGTCTGAGTATATTACAAAATAA
AAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAAT
GCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTG
CGCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCC
CCGGCTTGGTGTTGGACGGTCTGGTCGAGCGTTTCCGCGCGACCCCTCCCAAAGACAATG
ACGGCGGCCTGGTTGGACCCCCGGTACACGGAGCTTCTTCACTGAGCACGTATCGGTTTC
AAGGTGTCCCCGGGACCCGGTCGACCTCTCTTGCTCCCCTGCGGGAGTGGGAGAGAACC
CCCCTTTTTATCAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATC
AATAAGCGAGGGAAAAA

>Seq9 [organism=*Exophiala phaeomuriformis*] [isolate X6-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TCAGGCCGACCTCCCAACCCTTTGTCTATTGAACCCTTGTTGCTTCGGCGGGCCCGTCGT
TATACGGCCGCCGGAGGACCGACTCCAGGTCCTCTGGCCCGCGCCCGCCGGTAGCCAAA
ATCACCAAACTCTTGAATAATCGTGTCTATGATGTCTGAGTACTTTTATTAAAATTAAGCA
AAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGAT
AAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTGCGCCC
TTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCTCCGGC
TTGGTGTTGGACGGTCTGGTCGAGCTGCTCGACCCCTCCCAAAGACAATGACGGCGGCCT
GGTTGGACCCCCGGTACATGGAGCTTCTTGATTGAGCACGTATCGGTTTCAAGGCGTCCC

CGGGACCCGGTCTCACTCTCTCCCCTCGCGGGAGGGAACCCCCCTTTTTTCTAAGGTTGA
CCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAACGAAGG

>Seq10 [organism=*Coniothyrium fuckelii*] [isolate W1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGGTGCGGCCCCCGGGGGCGTGGGCGTCTTCTGGCGTCCTCTCTCCCGGGCGGTAGGG
GTAACACCCTCACGCGCCACATTTCTGAATCCTTTTTTTACGAGCACCTTTCGTTCTCCTTC
GGTGGGGCAACCTGCCGCTGGAACCTATCAAACTCTTCTGCATCTAGCATTACCTGTTCT
GATACAAACAATCGTTACAACTTTCAACAATGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAGTGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCCTTGGTATTCCATGGGGCATGCCTGTTCGAGCGTCATCTACACC
CTCAAGCTCTGCTTGGTGTTGGGCGTCTGTCCCGCCTTTGCGCGTGGACTCGCCCCAAAT
TCATTGGCAGCGGTCCTTGCCTCCTCTCGCGCAGCACATTTGCGCTTCTCGAGGTGCGCG
GCCCGCGTCCACGAAGCAACATTACCGTCTTTGACCTCGGATCAGGTAGGGATACCCGCT
GAACTTAAGCATATCAATAAGCGAGGGAAAAAAAAAAA

>Seq11 [organism=*Cladosporium macrocarpum*] [isolate X8-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CGACGCCGGGCTTCGGCCTGGTTATTCATAACCCTTTGTTGTCCGACTCTGTTGCCTCCG
GGGCGACCCTGCCTTCGGGCGGGGGCTCCGGGTGGACACTTCAAACTCTTGCGTAACTTT
GCAGTCTGAGTAAACTTAATTAATAAATTAAAACTTTTAACAACGGATCTCTTGGTTCTG
GCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAAT
CATCGAATCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGGGGCATGCCTGTTCGAG
CGTCATTTCACCACTCAAGCCTCGCTTGGTATTGGGCAACGCGGTCCGCCGCGTGCCTCA
AATCGTCCGGCTGGGTCTTCTGTCCCCTAAGCGTTGTGGAAACTATTCGCTAAAGGGTGT
TCGGGAGGCTACGCCGTAAAACAACCCCATTTCTAAGGTTGACCTCGGATCAGGTAGGGA
TACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAAGAAATGTCCTAA

>Seq12 [organism=*Cladosporium sphaerospermum*] [isolate X10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CGGCCCTCGGGCCGGGATGTTCACAACCCTTTGTTGTCCGACTCTGTTGCCTCCGGGGCG
ACCCTGCCTCCGGGCGGGGGCCCCGGGTGGACATCTCAAACTCTTGCGTAACTTTGCAGT
CTGAGTAAATTTAATTAATAAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATC
GATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCG
AATCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGGGGCATGCCTGTTCGAGCGTCA
TTTCACCACTCAAGCCTCGCTTGGTATTGGGCGACGCGGTCCGCCGCGCGCCTCAAATCG
ACCGGCTGGGTCTTTCGTCCCCTCAGCGTTGTGGAAACTATTCGCTAAAGGGTGCCGCGG
GAGGCCACGCCGTAAAACAACCCCATTTCTAAGGTTGACCTCGGATCAGGTAGGGATACC

CGCTGAACTTAAGCATATCAATAAGCGGGGGAAAA

>Seq13 [organism=*Candida parapsilosis*] [isolate X4-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTGTGGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAACACGGGAGCG
GCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGAAAACACAATC
AATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAAT
GCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGC
GCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCTCAAGCCCCTT
CGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCCAAATACAGTG
GCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGACGGAGCGTA
GGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCC
GCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAACCAAA

>Seq14 [organism=*Fusarium dimerum*] [isolate X3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GATACAACTCCCAACCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCC
CGTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCT
GAGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATG
AAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATC
TTTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTAC
ATCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCG
TGCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCA
CTGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAACCCA

>Seq15 [organism=*Fusarium dimerum*] [isolate X5-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq16 [organism=*Fusarium dimerum*] [isolate X4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GATCACTCCAACCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCGT
AACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAG
TGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATC
CCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGC
CCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTG
GGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCA
GGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAACGAC

>Seq17 [organism=*Fusarium dimerum*] [isolate X3-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GATCACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCC
GTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTG
AGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGA
AGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCT
TTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACA
TCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGT
GCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCAC
TGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAACCCC

>Seq18 [organism=*Fusarium dimerum*] [isolate X5-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CATCACTCCAACCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGT
AACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAG
TGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATC
CCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGC
CCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTG
GGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCA
GGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAACCCC

>Seq19 [organism=*Gloeotinia temulenta*] [isolate X8-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TATCAACTCCCCAACCCTATGTGACTTACCACTTGTTGCTTCGGCGCGCGCTTCGGCGCC
GCCGAGGACACTTAAACTCTTTGTTTTTATAAGCATTTCTGAGTGGCCGAAAGGCAAAAA
TACAAATGAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCGCCAGCATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCG
AGCCCGACTCTTTTGAGGACGGCTCGGCGTTGGGGGACGGCAGACTCCCCCCGGCATCT
CGCCGGGGACGCCGCCCCCGAAATCTAGTGGCGGCCCTCCGAGGCGACCTCTGCGTAGT
AACTTGTTCTCGCACCGGAAAGCTCAGAGCGGCCACGCCGTAAAACCCCAACTTTTTTCAG
GTTGACCTCGAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAAGGAA
AAGAAAAGTTA

>Seq20 [organism=*Gloeotinia temulenta*] [isolate X4-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ATCAACTCCCAACCCTATGTGACTTACCACTTGTTGCTTCGGCGCGCGCTTCGGCGCCGC
CGAGGACACTTAAACTCTTTGTTTTTATAAGCATTTCTGAGTGGCCGAAAGGCAAAAATA
CAAATGAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAG
CGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCA
CATTGCGCCCGCCAGCATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGAG
CCCGACTCTTTTGAGGACGGCTCGGCGTTGGGGGACGGCAGACTCCCCCCGGCATCTCG
CCGGGGACGCCGCCCCCGAAATCTAGTGGCGGCCCTCCGAGGCGACCTCTGCGTAGTAA
CTTGTTCTCGCACCGGAAAGCTCAGAGCGGCCACGCCGTAAAACCCCAACTTTTTTCAGGT
TGACCTCGAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAA
GAAAACCC

>Seq21 [organism=*Engyodontium album*] [isolate W1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AATACTCCAACCCCATGTGACATTACCTCTATGTTGCTTCGGCGGAGCCGCCCCGGCGCC
CGGAACCCCCTGCGGTCCCGCGGCCCGGAACCAAGGCGCCCGCCGGAGGCCACAAACT
CTTCTGTTTTTACAGTTTCTTCTGAGTGTGCCGCAAGGCAGTATACAAATGAATCAAAACTT
TCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAA
TGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAA
TTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGAGCTCCCCTTTCGGGGGGA
GCCCGGCGTTGGGGACCCGGCGCTAACAACGCCGGCCCCGAAATGGAGTGGCGGCCCG
TCCGCGGCGACCTCTGCGTAGTAACAACCACTCGCACCGGGACCCGGGCGCGGCCACGC
CGTAAAACACCCCACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAAACTTCT

>Seq22 [organism=*Engyodontium album*] [isolate W1-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACAACTCCAACCCATGTGACATTACCTCTATGTTGCTTCGGCGGAGCCGCCCCGGCGCCC
GGAACCCCCTGCGGTCCCGCGGCCCGGAACCAAGGCGCCCGCCGGAGGCCACAAACTC
TTCTGTTTTTACAGTTTCTTCTGAGTGTGCCGCAAGGCAGTATACAAATGAGTCAAAACTTT
CAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAAT
GTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAAT
TCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGAGCTCCCCTTTCGGGGGGAG
CCCGGCGTTGGGGACCCGGCGCTAACAACGCCGGCCCCGAAATGGAGTGGCGGCCCGT
CCGCGGCGACCTCTGCGTAGTAACAACCACTCGCACCGGGACCCGGGCGCGGCCACGC
CGTAAAACACCCCACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAAACCCTG

>Seq23 [organism=*Engyodontium album*] [isolate X10-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GATAAACTCCAACCCATGTGACATTACCTCTATGTTGCTTCGGCGGAGCCGCCCCGGCGC
CCGGAACCCCCTGCGGTCCCGCGGCCCGGAACCAAGGCGCCCGCCGGAGGCCACAAAC
TCTTCTGTTTTTACAGTTTCTTCTGAGTGTGCCGCAAGGCAGTATACAAATGAATCAAAACT
TTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTA
ATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGA
ATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGAGCTCCCCTTTCGGGGGG
AGCCCGGCGTTGGGGACCCGGCGCTAACAACGCCGGCCCCGAAATGGAGTGGCGGCCC
GTCCGCGGCGACCTCTGCGTAGTAACAACCACTCGCACCGGGACCCGGGCGCGGCCACG
CCGTAAAACACCCCACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGAGGGAAAAGAAAATAACC

>Seq24 [organism=*Engyodontium album*] [isolate X9-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TACAACTCCCAACCCCATGTGACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGC
CCGGAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTG
TATTTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAA
CAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTG
AATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCT
GGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGG
CGTTGGGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGG
CGACCTCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAAC

CCCCACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATC
AATAAGCGGAGGGAAAAGAAAATAGA

>Seq25 [organism=*Engyodontium album*] [isolate X9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ATTACTCCAACCCATGTGACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCCG
GAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTAT
TTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACAA
CGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAAT
TGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGGC
GGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGTT
GGGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGAC
CTCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCCC
ACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATA
AGCGGAGGGAAAAGAAAACCGTT

>Seq26 [organism=*Engyodontium album*] [isolate X6-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTCACTCCAACCCCATGTGACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCC
GGAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTA
TTTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACA
ACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAA
TTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGG
CGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGT
TGGGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGA
CCTCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCC
CACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAAT
AAGCGAGGGAAAAAGAAAAGACC

>Seq27 [organism=*Engyodontium album*] [isolate X6-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CATGTGACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCCGGAACGTCTGTTT
CGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTATTTACAGTATCTT
CTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACAACGGATCTCTTGG
TTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAG
TGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGGCGGGCATGCCTGT
TCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGTTGGGGACCGGCA
CTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGACCTCTGCGTAGTA

AATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCCCACCTTCCGAATG
TTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAA
GAAAATCTCT

>Seq28 [organism=*Engyodontium album*] [isolate X8-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTACTCCCAACCCATGTGACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCCG
GAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTAT
TTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACAA
CGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAAT
TGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGGC
GGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGTT
GGGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGAC
CTCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCCC
ACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATA
AGCGGAGGAAAAGAAACCTTC

>Seq29 [organism=*Candida parapsilosis*] [isolate K6-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTGCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGG
TAGGCCTTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAA
CCGATTATTTAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCC
CTCAAACCCTCGGGTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAG
TATAAACTAATGGATAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAA
TTCGACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGA
AAAAGAAA

>Seq30 [organism=*Candida parapsilosis*] [isolate K9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGTGCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGG
TAGGCCTTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAA
CCGATTATTTAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCC
CTCAAACCCTCGGGTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAG
TATAAACTAATGGATAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAA

TTCGACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAA
AAGAAAATCCCCGT

>Seq31 [organism=*Rhodotorula dairenensis*] [isolate K9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GACAGGACGTCCACTTAACTTGGAGTCCGAACTCTCACTTTCTAACCCTGTGCATTTGTTT
GGGATAGTAACTCTCGCAAGAGAGCGAACTCCTATTCACTTATAAACACAAAGTCTATGAAT
GTATTTAATTTTATAACAAAATAAAACTTTCAACAACGGATCTCTTGGCTCTCGCATCGATGA
AGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACCTTGCGCTCCATGGTATTCCGTGGAGCATGCCTGTTTGAGTGTCATGAATAC
TTCAACCCTCCTCTTTCTTAATGATTGAAGAGGTGTTTGGATTCTGAGCGCTGCTGGCCTTA
CGGTCTAGCTCGTTCGTAATGCATTAGCATCCGCAATCGAACTTCGGATTGACTTGGCGTA
ATAGACTATTCGCTGAGGAATTCTAATCTTCGGATTAGAGCCGGGTTAGGTTAAAGGAAGC
TTCTAATCAGAATGTCTACATTTTAAGATTAGATCTCAAATCAGGTAGGACTACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq32 [organism=*Exophiala castellanii*] [isolate G7-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACGGTCTTCTAGGCCCGACCTCCCAACCCTATGTTTAATCGACCTGATGTTGCTTCGGC
GAGCCCGTCTCAAAAGGACCGCCGGAGGACCGTAACACGTCCTCTGGTCCGCGCTCGCC
GGTGGCCAAATCAAATTCTTTATTAAATGTATCATCTAAGTCAAAAATTTAATTAAAGCAA
AACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATA
AGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTGCGCCCT
TTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCCCCTTCT
TAATTGAAACCGGCTTGGTGTTGGACGGTTTGGTGTCGGCAACGTCACCCCTCCCAAAGA
CAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCCTCACCGAACACGTACTGG
ATCAAGGGTAGACGGGGCCCCGGTCGACCTCCCTTCACGGGGAGATTTATTTTTCAGGTT
GACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq33 [organism=*Exophiala castellanii*] [isolate G6-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGTCTTCTAGGCCCGACCTCCCAACCCTATGTTTAATCGACCTGATGTTGCTTCGGCGAG
CCCGTCTCAAAAGGACCGCCGGAGGACCGTAACACGTCCTCTGGTCCGCGCTCGCCGGT
GGCCAAATCAAATTCTTTATTAAATGTATCATCTAAGTCAAAAATTTAATTAAAGCAAAAC
TTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGT
AATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTGCGCCCTTTG
GCATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCCCCTTCTTAA
TTGAAACCGGCTTGGTGTTGGACGGTTTGGTGTCGGCAACGTCACCCCTCCCAAAGACAA

TGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCCTCACCGAACACGTACTGGATC
AAGGGTAGACGGGGCCCCGGTCGACCTCCCTTCACGGGGAGATTTATTTTTCAGGTTGAC
CTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGA

>Seq34 [organism=*Exophiala pisciphila*] [isolate G1-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTAGGGTCTTTTATAGGCTCGACCTCCCAACCCTTTGTTTACCCGACCCACGTTGCTTCG
GCGAGCCCGTCTCTCCCTCTCCGGGGGGGCAGACCGCCGGGGGGCCTTCAAACCGTCCT
CTGGCCCGCGCTCGTCGGTGGCCAACCTTTACAAATTCTTAATTAAACGTGTCTACTCTGA
GTACTATTTTTTAATTAAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGA
TGAAGAACGCAGCGAAATGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGA
ATCTTTGAACGCACATTGCGCCCTTTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTCAT
TATCACCTCTCAAGCCCCCCTTTTCTGGGCTTGGTGTTGGACGGCCTGGCGTCGGCGACG
ACCCCACCTCCCAAAGACAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCCT
CACCGAACACGTACTGGATCAAGGGTAGACGGAGCCCCGGTCGACCTCCCCTCACAGGG
AGACACTTTTTTACAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATA
TCAATAAGCGGAGGGAAAAGAAAA

>Seq35 [organism=*Exophiala pisciphila*] [isolate G2-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTACGGTCTTTTATAGGCTCGACCTCCCAACCCTTTGTTTACCCGACCCACGTTGCTTCG
GCGAGCCCGTCTCTCCCTCTCCGGGGGGGCAGACCGCCGGGGGGCCTTCAAACCGTCCT
CTGGCCCGCGCTCGTCGGTGGCCAACCTTTACAAATTCTTAATTAAACGTGTCTACTCTGA
GTACTATTTTTTAATTAAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGA
TGAAGAACGCAGCGAAATGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGA
ATCTTTGAACGCACATTGCGCCCTTTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTCAT
TATCACCTCTCAAGCCCCCCTTTTCTGGGCTTGGTGTTGGACGGCCTGGCGTCGGCGACG
ACCCCACCTCCCAAAGACAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCCT
CACCGAACACGTACTGGATCAAGGGTAGACGGAGCCCCGGTCGACCTCCCCTCACAGGG
AGACACTTTTTTACAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATA
TCAATAAGCGGAGGGAAAAGAAAAAAAT

>Seq36 [organism=*Cadophora fastigiata*] [isolate K8-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACGGTCTCTCTGGCCCGACCTCCCAACCCTATGTCTACTTGACCATCGTTGCTTCGGCG
AGCCCGTCCTCACGGACCGCCGGAGGGACCTTCACCGGCCCTCTGGTCCGCGCTCGTCG
GTAGCCCAACCATTAAAATCTTTAACCAAACGTGCCTTAATCTAAGTACAATTATTAAATA
AAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAA

TGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATT
GCGCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCC
CTGGCTTGGTGTTGGACGGTTTGGTGGAGGCCCCCTCGGGGGCTCCTGCCCCTCCCAAA
GACAATGACGGCGGCCTCGTTGGACCCCCGGTACACTGAGTTCTTCACGGGACACGTATC
GGATACATGGGTTTACGGGACACGGTCTGCCTCCCCTCAGGGAGAATCTTTCTAAGGTTG
ACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAAA
AAAA

>Seq37 [organism=*Cadophora fastigiata*] [isolate K9-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACGGTCTCTCTGGCCCGACCTCCCAACCCTATGTCTACTTGACCATCGTTGCTTCGGCG
AGCCCGTCCTCACGGACCGCCGGAGGGACCTTCACCGGCCCTCTGGTCCGCGCTCGTCG
GTAGCCCAACCATTAAAATCTTTAACCAAACGTGCCTTAATCTAAGTACAATTATTAAATA
AAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAA
TGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATT
GCGCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCC
CTGGCTTGGTGTTGGACGGTTTGGTGGAGGCCCCCTCGGGGGCTCCTGCCCCTCCCAAA
GACAATGACGGCGGCCTCGTTGGACCCCCGGTACACTGAGTTCTTCACGGGACACGTATC
GGATACATGGGTTTACGGGACACGGTCTGCCTCCCCTCAGGGAGAATCTTTCTAAGGTTG
ACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAAG
AAAAAAG

>Seq38 [organism=*Trichoderma viride*] [isolate K2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAACCCAATGTGAACCATACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGTGC
GTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGGACCAACCAAACTCTTTCTGTAGTCC
CCTCGCGGACGTTATTTTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCAAC
AACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGA
ATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTCTG
GCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGGTCGGCGTT
GGGGACTTCGGGAACCCCTAAGACGGGATCCCGGCCCCGAAATACAGTGGCGGTCTCGC
CGCAGCCTCTCATGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACG
TCCGTAAAACACCCAACTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAAC
TTAAGCATATCAATAAGCGAGGGAAAAAGAAAACCCT

>Seq39 [organism=*Trichoderma asperellum*] [isolate K1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACCTCCCAACCCAATGTGAACGTTACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGT

GCGTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGAACCAACCAAACTCTTTCTGTAGT
CCCCTCGCGGACGTATTTCTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCA
ACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGT
GAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTC
TGGCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGATCGGCG
TTGGGGATCGGGACCCCTCACACGGGTGCCGGCCCCGAAATACAGTGGCGGTCTCGCCG
CAGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACGTC
CGTAAAACACCCAACTTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGGGGAAAAAGAAA

>Seq40 [organism=*Trichoderma asperellum*] [isolate K8-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAACCCAATGTGAACGTTACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGTGC
GTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGAACCAACCAAACTCTTTCTGTAGTCC
CCTCGCGGACGTATTTCTTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCAA
CAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTG
AATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTCT
GGCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGATCGGCGT
TGGGGATCGGGACCCCTCACACGGGTGCCGGCCCCTAAATACAGTGGCGGTCTCGCCGC
AGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACGTCC
GTAAAACACCCAACTTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGAGGGAAAAAAAAAAT

>Seq41 [organism=*Gloeotinia temulenta*] [isolate G4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCCAACCCTATGTGAACTTACCACTTGTTGCTTCGGCGCGCGCTTCGGCGCCGCCGAGGA
CACTTAAACTCTTTGTTTTTATAAGCATTTCTGAGTGGCCGAAAGGCAAAAATACAAATG
AATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAAT
GCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGC
GCCCGCCAGCATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGAGCCCGAC
TCTTTTGAGGACGGCTCGGCGTTGGGGGACGGCAGACTCCCCCCGGCATCTCGCCGGGG
ACGCCGCCCCCGAAATCTAGTGGCGGCCCTCCGAGGCGACCTCTGCGTAGTAACTTGTTC
TCGCACCGGAAAGCTCAGAGCGGCCACGCCGTAAAACCCCAACTTTTTTCAGGTTGACCT
CGAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAA
CGGA

>Seq42 [organism=*Gloeotinia temulenta*] [isolate G9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTCCCAACCCTATGTGAACTTACCACTTGTTGCTTCGGCGCGCGCTTCGGCGCCGCCGAG
GACACTTAAACTCTTTGTTTTTATAAGCATTTCTGAGTGGCCGAAAGGCAAAAATACAAA
TGAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAA
ATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATT
GCGCCCGCCAGCATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGAGCCC
GACTCTTTTGAGGACGGCTCGGCGTTGGGGGACGGCAGACTCCCCCCGGCATCTCGCCG
GGGACGCCGCCCCCGAAATCTAGTGGCGGCCCTCCGAGGCGACCTCTGCGTAGTAACTT
GTTCTCGCACCGGAAAGCTCAGAGCGGCCACGCCGTAAAACCCCAACTTTTTTCAGGTTG
ACCTCGAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGA
AAAGATTT

>Seq43 [organism=*Engyodontium album*] [isolate G8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCATGTGAACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCCG
GAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTAT
TTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACAA
CGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAAT
TGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGGC
GGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGTT
GGGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGAC
CTCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCCC
ACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATA
AGCGGAGGAAAAA

>Seq44 [organism=*Engyodontium album*] [isolate G10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACCTCCCAACCCATGTGAACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCCG
GAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTAT
TTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACAA
CGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAAT
TGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGGC
GGGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGTT
GGGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGAC
CTCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCCC
ACCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATA
AGCGGGGGAAAAAGAAAA

>Seq45 [organism=*Engyodontium album*] [isolate G2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCCCAACCCATGTGAACATACCTACAAGTTGCTTCGGCGGAGCCGCCCCGGCGCCCG
GAACGTCTGTTTCGCGGCCCGGAACCAGGTGCCCGCCGGAGACCACAAACTCTTTTGTAT
TTACAGTATCTTCTGAGTGTGCCGCAAGGCAACAAACAAATGAATCAAAACTTTCAACAAC
GGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATT
GCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGAATTCTGGCG
GGCATGCCTGTTCGAGCGTCATTTCAACCCTCGGTCTCCCTCCGGGAGAGACCGGCGTTG
GGGACCGGCACTTACCCCGCCGGCCCCGAAATGGAGTGGCGGCCCGTCCGCGGCGACC
TCTGCGTAGTAAATCCACTCGCACCGGGACCCGGGCGCGGCCACGCCGTAAAACCCCCA
CCTTCCGAATGTTGACCTCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAA
GCGGAGGGAAAAGAA

>Seq46 [organism=*Fusarium oxysporum*] [isolate K6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAAAAGAAA

>Seq47 [organism=*Fusarium solani*] [isolate K2-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CCTGTGAACATACCTAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAACACGGGCCG
CCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATAAATGTTTTTTCTGAGTAAACAAGCAA
ATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAACGCAGCGA
AATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACAT
TGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCCTCAGGCCC
CCGGGCCTGGCGTTGGGGATCGGCGGAGCCCCCCGTGGGCACACGCCGTCCTCCAAATA
CAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGCAACTGGAGAGC
GGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAATCAGGTAGGAAT
ACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAACCGAA

>Seq48 [organism=*Fusarium solani*] [isolate K2-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCTCACCCTGTGAACATACCTAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAAC
ACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATAAATGTTTTTTCTGAGTA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC
CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCCCCCCGTGGGCACACGCCGT
CCTCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGCA
ACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAA

>Seq49 [organism=*Fusarium solani*] [isolate K10-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCTCACCCTGTGAACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAAC
ACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC
CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACACGCC
GTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCG
CAACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAA

>Seq50 [organism=*Fusarium solani*] [isolate K9-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCTCACCCTGTGAACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAAC
ACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC
CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACACGCC
GTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCG
CAACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGA

>Seq51 [organism=*Fusarium solani*] [isolate K10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCTCACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAACA
CGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGAGTAAA
ACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCC
TCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACACGCCG
TCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGC
AACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq52 [organism=*Fusarium solani*] [isolate K9-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCTCACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAACA
CGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGAGTAAA
ACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCC
TCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACACGCCG
TCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGC
AACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq53 [organism=*Trichoderma asperellum*] [isolate R3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GAATCACTCCAACCCATGTGACGTTACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGG
TGCGTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGAACCAACCAAACTCTTTCTGTAG
TCCCCTCGCGGACGTATTTCTTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTT
CAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAAT
GTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTAT
TCTGGCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGATCGG
CGTTGGGGATCGGGACCCCTCACACGGGCGCCGGCCCCTAAATACAGTGGCGGTCTCGC
CGCAGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACG
TCCGTAAAACACCCAACTTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAAAAAA

>Seq54 [organism=*Fusarium solani*] [isolate R1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CACTCATCACCCTGTGACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAAA
ACGGGCCGCCCCCGCCAGAGGACTCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC
CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGCC
GTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCG
CGACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTCG
AATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAACAAA
GC

>Seq55 [organism=*Fusarium solani*] [isolate R1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ATCACTCATCACCCTGTGACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTA
AAACGGGCCGCCCCCGCCAGAGGACTCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGT
AAAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAA
CCCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACG
CCGTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCT
CGCGACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCT
CGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAAGAAA

>Seq56 [organism=*Fusarium solani*] [isolate R1-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TTTCACTCATCACCCTGTGACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGT
AAAACGGGCCGCCCCCGCCAGAGGACTCTTAACTCTGTTTCTTATAATGTTTCTTCTGAG
TAAAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGA
AGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCT
TTGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACA
ACCCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCAC
GCCGTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACC
TCGCGACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACC
TCGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq57 [organism=*Bionectria ochroleuca*] [isolate R2-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CCATGTGACATACCTACTGTTGCTTCGGCGGGATTGCCCCGGGCGCCTCGTGTGCCCCG
GATCAGGCGCCCGCCTAGGAAACTTAATTCTTGTTTTATTTTGGAATCTTCTGAGTAGTTT
TTACAAATAAATAAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTCTGAGCGTCATTTCAACCCTC
ATGCCCCTAGGGCGTGGTGTTGGGGATCGGCCAAAGCCCGCGAGGGACGGCCGGCCCC
TAAATCTAGTGGCGGACCCGTCGTGGCCTCCTCTGCGAAGTAGTGATATTCCGCATCGGA
GAGCGACGAGCCCCTGCCGTTAAACCCCCAACTTTCCAAGGTTGACCTCAGATCAGGTAG
GAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACCCTGC

>Seq58 [organism=*Bionectria ochroleuca*] [isolate R2-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CCATGTGACATACCTACTGTTGCTTCGGCGGGATTGCCCCGGGCGCCTCGTGTGCCCCG
GATCAGGCGCCCGCCTAGGAAACTTAATTCTTGTTTTATTTTGGAATCTTCTGAGTAGTTT
TTACAAATAAATAAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTCTGAGCGTCATTTCAACCCTC
ATGCCCCTAGGGCGTGGTGTTGGGGATCGGCCAAAGCCCGCGAGGGACGGCCGGCCCC
TAAATCTAGTGGCGGACCCGTCGTGGCCTCCTCTGCGAAGTAGTGATATTCCGCATCGGA
GAGCGACGAGCCCCTGCCGTTAAACCCCCAACTTTCCAAGGTTGACCTCAGATCAGGTAG
GAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACCCCCC

>Seq59 [organism=*Bionectria ochroleuca*] [isolat R2-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CCATGTGACATACCTACTGTTGCTTCGGCGGGATTGCCCCGGGCGCCTCGTGTGCCCCG
GATCAGGCGCCCGCCTAGGAAACTTAATTCTTGTTTTATTTTGGAATCTTCTGAGTAGTTT
TTACAAATAAATAAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTCTGAGCGTCATTTCAACCCTC
ATGCCCCTAGGGCGTGGTGTTGGGGATCGGCCAAAGCCCGCGAGGGACGGGCGGCCCC
TAAATCTAGTGGCGGACCCGTCGTGGCCTCCTCTGCGAAGTAGTGATATTCCGCATCGGA
TAGCGACGAGCCCCTGCCGTTAAACCCCCAACTTTCCAAGGTTGACCTCAGATCAG

>Seq60 [organism=*Exophiala phaeomuriformis*] [isolate R9-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTCAGGCCGACCTCCCACCCTTTGTCTATTGAACCCTTGTTGCTTCGGCGGGCCCGTCGT
TATACGGCCGCCGGAGGACCGACTCCAGGTCCTCTGGCCCGCGCCCGCCGGTAGCCAAA
ATCACCAAACTCTTGAATAATCGTGTCTATGATGTCTGAGTACTTTTATTAAAATTAAGCA
AAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGAT
AAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTGCGCCC
TTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCTCCGGC
TTGGTGTTGGACGGTCTGGTCGAGCTGCTCGACCCCTCCCAAAGACAATGACGGCGGCCT
GGTTGGACCCCCGGTACATGGAGCTTCTTGATTGAGCACGTATCGGTTTCAAGGCGTCCC
CGGGACCCGGTCTCACTCTCTCCCCTCGCGGGAGGGAACCCCCCTTTTTTCTAAGGTTGA
CCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAAGAA
AAGGG

>Seq61 [organism=*Cadophora fastigiata*] [isolate R10-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TAGTCTCTCTGGTCGACCTCCCACCCTTTGTCTACTTGACCATCGTTGCTTCGGCGAGCC
CGTCCTCACGGACCGCCGGAGGGACCTTCACCGGCCCTCTGGTCCGCGCTCGTCGGTAG
CCCAACCATTAAAATCTTTAACCAAACGTGCCTTAATCTAAGTACAATTATTAAATAAAAG
CAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCG
ATAAGTAATGCTAATTGCAG

>Seq62 [organism=*Fusarium oxysporum*] [isolate R6-9] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTACACTCATCACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCG
TAACACGGGCCGCCCCCGCCAGAGGACCCCTAACTCTGTTTATATTATGTTTTTTCTGAG
TAAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAA
CCCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAAAGCCCCCTGTGGGCATACG
CCGTCCCCTAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCT
CGCAACTGGAGAGCGGCGCGGCCAAGCCGTAAAACCCCCAACTTCTGAATGTTGACCTCG
AATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAAAAAAAAA
TTGA

>Seq63 [organism=*Fusarium dimerum*] [isolate R8-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CATTCACTCCAACCCCCTGTGAAACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCC
CCGTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTC
TGAGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGAT

GAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAAT
CTTTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTA
CATCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCC
GTGCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGC
ACTGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGG
ATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAAGAAAACCCC
C

>Seq64 [organism=*Fusarium dimerum*] [isolate X2-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAACACGGGAG
CGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGAAAACACAA
TCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAA
ATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATT
GCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCTCAAGCCCC
TTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCCAAATACAG
TGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGACGGAGCG
TAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGTAGGAATAC
CCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACCCCCCC

>Seq65 [organism=*Fusarium dimerum*] [isolate X2-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA
CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC
TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG
ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACCG

>Seq66 [organism=*Fusarium dimerum*] [isolate R5-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TCACTCAAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG

AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAAGAAAACCCCC

>Seq67 [organism=*Fusarium dimerum*] [isolate R6-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTACTCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAATTTCC

>Seq68 [organism=*Fusarium dimerum*] [isolate R6-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACAATAACTCCCCAACCCCTGTGACATACCTAAACGTTGCCTCGGCGGACCCCCGCCTCC
CCGTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTC
TGAGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGAT
GAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAAT
CTTTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTA
CATCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCC
GTGCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGC
ACTGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGG
ATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAAATGGA

>Seq69 [organism=*Fusarium dimerum*] [isolate R7-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAACA
CGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGAA
AACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCTC

AAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCCA
AATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGAC
GGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGTA
GGAATACCCGCTGAACTTAAGCAA

>Seq70 [organism=*Fusarium dimerum*] [isolate R5-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTAACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCG
TAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGA
GTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAT
CCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTG
CCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACT
GGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq71 [organism=*Fusarium dimerum*] [isolate R8-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTTTACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCC
CGTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCT
GAGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATG
AAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATC
TTTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTAC
ATCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCG
TGCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCA
CTGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAATGTTAT

>Seq72 [organism=*Fusarium dimerum*] [isolate R5-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA
CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC
TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG

ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq73 [organism=*Fusarium dimerum*] [isolate R8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ATTTACTCCCAACCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCG
TAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGA
GTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAT
CCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTG
CCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACT
GGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAAGTTATT

>Seq74 [organism=*Fusarium dimerum*] [isolate X2-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTTACTCCCAACCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCG
TAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGA
GTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAT
CCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTG
CCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACT
GGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAATGTTT

>Seq75 [organism=*Fusarium dimerum*] [isolate R7-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTAAACTCCAACCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCG
TAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGA
GTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAT
CCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTG
CCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACT
GGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACTGTTG

>Seq76 [organism=*Fusarium dimerum*] [isolate X2-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAACACGGGA
GCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGAAAACACA
ATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGA
AATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACAT
TGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCTCAAGCCC
CTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCCAAATACA
GTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGACGGAGC
GTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGTAGGAATA
CCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACGGGTTT

>Seq77 [organism=*Fusarium dimerum*] [isolate R6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCCAACCCCTGTGACATACCTACACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAACA
CGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGAA
AACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCTC
AAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCCA
AATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGAC
GGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGTA
GGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACGTTTT

>Seq78 [organism=*Candida palmioleophila*] [isolate H3-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ATTGCAGCGCTTATTGCGCGGCGATTAAACCTTACACACAATGTTTTTCTTTATTAGAAA
CTATTGCTTTGGCTTGGCTAAGAAATTAGTCGGGCCAGAGGTTTACACAAACTTCAATTT
TTAATTGAATTGTTATTTAATACTTTGTCAATTTGTTGATTAAATTCAAACAATCTTCAA
AACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGAAATGCGATA
AGTAATATGAATTGCAGATTTTCGTGAATCATCGAATCTTTGAACGCACATTGCGCCCTC
TGGTATTCCAGAGGGCATGCCTGTTTGAGCGTCATTTCTCTCTCAAACCCTTGGGTTTGG
TATTGAGTGATACTCTTAGTCGAACTAGGCGTTTGCTTGAAATTTATTGGCATGAGTGAC
GCTGAGAAGTGCATTCAGGAAATATCAATGTATTAGGTTTATCCAACTCGTTGACAATTC
TTGGTTGTGAATTTTTGGTGTTAGGCTTTGCCTTAAAAAACAACAAACAAGTTTGACCTC
AAATCAGGTAGGATTACCCGCTGAACTTAAGCATATCAATAAGCGGGAGA

>Seq79 [organism=*Candida tropicalis*] [isolate H5-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTGCACCACATGTGTTTTTTATTGAACAAATTTCTTTGGTGGCGGGAGCAATCCTACCGC
CAGAGGTTATAACTAAACCAAACTTTTTATTTACAGTCAAACTTGATTTATTATTACAAT
AGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGAAAT
GCGATACGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCACATTGC
GCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCAAACCCCCGG
GTTTGGTGTTGAGCAATACGCTAGGTTTGTTTGAAAGAATTTAACGTGGAAACTTATTTT
AAGCGACTTAGGTTTATCCAAAACGCTTATTTTGCTAGTGGCCACCACAATTTATTTCAT
AACTTTGACCTCAAATCAGGTAGGACT

>Seq80 [organism=*Candida tropicalis*] [isolate H7-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTGCACCACATGTGTTTTTTATTGAACAAATTTCTTTGGTGGCGGGAGCAATCCTACCGC
CAGAGGTTATAACTAAACCAAACTTTTTATTTACAGTCAAACTTGATTTATTATTACAAT
AGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGAAAT
GCGATACGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCACATTGC
GCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCAAACCCCCGG
GTTTGGTGTTGAGCAATACGCTAGGTTTGTTTGAAAGAATTTAACGTGGAAACTTATTTT
AAGCGACTTAGGTTTATCCAAAACGCTTATTTTGCTAGTGGCCACCACAATTTATTTCAT
AACTTTGACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGG
AAAAAGAAAA

>Seq81 [organism=*Candida orthopsilosis*] [isolate H9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTGCTTACTGCATTTTTTTACACATGTGTTTTTCTTTTTTTTTGAAAACTTTGCTTTGG
TGGGCCCATGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAAGTCAACTGATTAA
CTAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGC
GAAATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCAC
ATTGCGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCCCTCAAACC
TTCGGGTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAGTATAAACT
AATGGATAGGTTTTTTTCCACTCATTGGTACAAACTCCAAAATTCTTCCAAATTCGACCT
CAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq82 [organism=*Candida albicans*] [isolate H8-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTGCACCACATGTGTTTTTCTTTGAAACAAACTTGCTTTGGCGGTGGGCCCAGCCTGCCG

CCAGAGGTCTAAACTTACAACCAATTTTTTATCAACTTGTCACACCAGATTATTACTAAT
AGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGAAAT
GCGATACGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTTGAACGCACATTGC
GCCCTCTGGTATTCCGGAGGGCATGCCTGTTTGAGCGTCGTTTCTCCCTCAAACCGCTGG
GTTTGGTGTTGAGCAATACGACTTGGGTTTGCTTGAAAGACGGTAGTGGTAAGGCGGGAT
CGCTTTGACAATGGCTTAGGTCTAACCAAAAACATTGCTTGCGGCGGTAACGTCCACCAC
GTATATCTTCAAACTTTGACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATC
AATAAGCGGAGGGAAAAGAATTTTT

>Seq83 [organism=*Pichia kudriavzevii*] [isolate H1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACTACCTGCGTGAGCGGAACGAAAACAACAACACCTAAAATGTGGAATATAGCATATAG
TCGACAAGAGAAATCTACGAAAAAACAAACAAAACTTTCAACAACGGATCTCTTGGTTCT
CGCATCGATGAAGAGCGCAGCGAAATGCGATACCTAGTGTGAATTGCAGCCATCGTGAAT
CATCGAGTTCTTGAACGCACATTGCGCCCCTCGGCATTCCGGGGGGCATGCCTGTTTGAG
CGTCGTTTCCATCTTGCGCGTGCGCAGAGTTGGGGGAGCGGAGCGGACGACGTGTAAAG
AGCGTCGGAGCTGCGACTCGCCTGAAAGGGAGCGAAGCTGGCCGAGCGAACTAGACTTT
TTTTCAGGGACGCTTGGCGGCCGAGAGCGAGTGTTGCGAGACAACAAAAAGCTCGACCTC
AAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAAGAA

>Seq84 [organism=*Pichia kudriavzevii*] [isolate H9-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTACTACCTGCGTGAGCGGACGAAAACAACAACACCTAAAATGTGGAATATAGCATATAG
TCGACAAGAGAAATCTACGAAAAAACAAACAAAACTTTCAACAACGGATCTCTTGGTTCT
CGCATCGATGAAGAGCGCAGCGAAATGCGATACCTAGTGTGAATTGCAGCCATCGTGAAT
CATCGAGTTCTTGAACGCACATTGCGCCCCTCGGCATTCCGGGGGGCATGCCTGTTTGAG
CGTCGTTTCCATCTTGCGCGTGCGCAGAGTTGGGGGAGCGGAGCGGACGACGTGTAAAG
AGCGTCGGAGCTGCGACTCGCCTGAAAGGGAGCGAAGCTGGCCGAGCGAACTAGACTTT
TTTTCAGGGACGCTTGGCGGCCGAGAGCGAGTGTTGCGAGACAACAAAAAGCTCGACCTC
AAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq85 [organism=*Phoma herbarum*] [isolate H8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GAGTTGTGGGCTTTGCCTGCTATCTCTTACCCATGTCTTTTGAGTACTTACGTTTCCTCG
GTGGGTTCGCCCGCCGATTGGACAATTTAAACCCTTTGCAGTTGCAATCAGCGTCTGAAA
AACATAATAGTTACAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCGAAATGCGATAAGTAGTGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCCTTGGTATTCCATGGGGCATGCCTGTTCGAGCGTCATTTGTACCTTCA

AGCATTGCTTGGTGTTGGGTGTTTGTCTCGCCTTTGCGTGTAGACTCGCCTTAAAACAAT
TGGCAGCCGGCGTATTGATTTCGGAGCGCAGTACATCTCGCGCTTTGCACTCATAACGAC
GACGTCCAAAAGTACATTTTAACACTCTTGACCTCGGATCAGGTAGGGATACCCGCTGAA
CTTAAGCATATCAATAAGCGGGGGAAA

>Seq86 [organism=*Exophiala dermatitidis*] [isolate H9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TCTAGGCCCGACCTCCCAACCCTTTGTTTACCCGACCCATGTTGCTTCGGCGGGCCCGCC
GTTTCGACGGCCGCCGGAGGACCGCCTATTCAGGTCCTCTGGCCCGCGCCCGCCGGTAG
CCAATTCTACCAAACTCTTGAATCAAATCGTGTCCAATGTCTGAGTATATTACAAAATAAA
AGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATG
CGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTGC
GCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCCC
CGGCTTGGTGTTGGACGGTCTGGTCGAGCGTTTCCGCGCGACCCCTCCCAAAGACAATGA
CGGCGGCCTGGTTGGACCCCCGGTACACGGAGCTTCTTCACTGAGCACGTATCGGTTTCA
AGGTGTCCCCGGGACCCGGTCGACCTCTCTTGCTCCCCTGCGGGAGTGGGAGAGAACCC
CCCTTTTTATCAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCA
ATAAGCGGGGGAAA

>Seq87 [organism=*Exophiala dermatitidis*] [isolate P1-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TAGGCCCGACCTCCCAACCCTTTGTTTACCCGACCCATGTTGCTTCGGCGGGCCCGCCGT
TTCGACGGCCGCCGGAGGACCGCCTATTCAGGTCCTCTGGCCCGCGCCCGCCGGTAGCC
AATTCTACCAAACTCTCGAATCAAATCGTGTCCAATGTTCTGAGTATATTCCAAAATAAAA
GCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGC
GATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACATTGCG
CCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCCCCCC
GGCTTGGTGTTGGACGGTCTGGTCGAGCGTTTCCGCGCGACCCCTCCCAAAGACAATGAC
GGCGGCCTGGTTGGACCCCCGGTACACGGAGCTTCTTCACTGAGCACGTATCGGTTTCAA
GGTGTCCCCGGGACCCGGTCGACCTCTCTTGCTCCCCTGCGGGAGTGGGAGAGAACCCC
CCTTTTTATCAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAA
TAAGCGGGGGA

>Seq88 [organism=*Fusarium oxysporum*] [isolate H7-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG

CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGGGGAAAAAGAA

>Seq89 [organism=*Fusarium oxysporum*] [isolate H3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGGGTAAA

>Seq90 [organism=*Gliomastix polychroma*] [isolate P4-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCACTGTGAACCTATACCACTGTTGCTTCGGCGGATACGCCCCGGGCGCA
CCCCCTCCAGGGGGTTGTGCCCCGGAACCAGGCGCCCGCCGGGGGACCGAAACCTCTG
TATTTACCGTTTGAGTACTCTGAGTGTGATTTACAAAATCAAAATTAAAACTTTCAACAACG
GATCTCTTGGCTCTAGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGCGAATTG
CAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTCTGGCGG
GCATGCCTGTCTGAGCGTCGTTTCGACCCTCGCGCCCGGCTTCTGTCGGGGGCGGTGTT
GGGGATCGGCCACACCCACTCCGGTGGGAGGCCGGCCCCTAAATCCAGTGGCGACCACG
CTGTAGCCTCCCCTGCGTAGTACTAAAACCACCTCGCAGGCGGAGAGCGGTGCGGCCCG
CCGTAAAACCCCCCAACTTTTACAAGGTCGACCTCAGATCAGGTAGGAATACCCGCTGAAC
TTAAGCATATCAATAAGCGGGGGGAAAAGAAA

>Seq91 [organism=*Beauveria bassiana*] [isolate P4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAACCCTTATGTGAACCTACCTATCGTTGCTTCGGCGGACTCGCCCCAGCCGGACG
CGGACTGGACCAGCGGCCGCCGGGGACCATCAAACTCTTGTATTATCAGCATCTTCTGAA
TACGCCGCAAGGCAAAACAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGC
ATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATCCAGTGAATCA

TCGAATCTTTGAACGCACATTGCGCCCGCCAGCATTCTGGCGGGCATGCCTGTTCGAGCG
TCATTTCAACCCTCGACCTCCCTTTGGGGAAGTCGGCGTTGGGGACCGGCAGCACACCGC
CGGCCCTGAAATGGAGTGGCGGCCCGTCCGCGGCGACCTCTGCGTAGTAAACCAACTCG
CACCGGAACCCCGACGTGGCCACGCCGTAAAACACCCAACTTCTGAACGTTGACCTCGAA
TCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq92 [organism=*Fusarium dimerum*] [isolate P2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAAGAAA

>Seq93 [organism=*Fusarium dimerum*] [isolate P2-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAAC
ACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGA
AAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCT
CAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCC
AAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGA
CGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGT
AGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGGAA

>Seq94 [organism=*Fusarium dimerum*] [isolate H10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA
CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC

TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG
ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq95 [organism=*Fusarium dimerum*] [isolate H2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA
CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC
TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG
ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGGAA

>Seq96 [organism=*Fusarium dimerum*] [isolate P5-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAGAAAACGGAGA

>Seq97 [organism=*Fusarium dimerum*] [isolate P5-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAAC
ACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTGA
AAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCCT
CAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCCC
AAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGGA

CGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGGT
A
GGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGAAAAAGAA

>Seq98 [organism=*Fusarium dimerum*] [isolate H5-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA
CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC
TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG
ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAGAAA

>Seq99 [organism=*Fusarium dimerum*] [isolate H6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq100 [organism=*Fusarium dimerum*] [isolate H1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG

GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq101 [organism=*Fusarium dimerum*] [isolate H4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA
CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC
TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG
ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAAGAAA

>Seq102 [organism=*Fusarium dimerum*] [isolate P2-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq103 [organism=*Rhodotorula glutinis*] [isolate H3-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GACGTCCACTTACTTGGAGTCCGAACTCTCACTTTCTAACCCTGTGCATCTGTTTTAAAA
TTGGCCAGTAGCTCTTCGGAGCGAACCACCATTTTTCACTTATACAAACACAAAGTCTAT
GAATGTAAACAAATTTATAACAAAACAAAACTTTCAACAACGGATCTCTTGGCTCTCGCA
TCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAATTGCAGAATTCAGTGAATCAT
CGAATCTTTGAACGCACCTTGCGCTCCTTGGTATTCCGAGGAGCATGCCTGTTTGAGTGT
CATGAAATCTTCAACCCACCTCTTTCTTAGTGAATCTGGTGGTGCTTGGTTTCTGAGCGC
TGCTCTGCTTCGGCTTAGCTCGTTCGTAATGCATTAGCATCCGCAACCGAAACTTCGGAT
TGACTTGGCGTAATAGACTATTCGCTGAGGATTCCAGACTTGTTCTGGAGCCGAGTTGGG
TTAAAGGAAGCTTCTAATCCTAAAGTCTATTTTTTGATTAGATCTCAAATCAGGTAGGAC

TACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAATAA

>Seq104 [organism=*Rhodotorula mucilaginosa*] [isolate H8-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGACGTCCACTTACTTGGAGTCCGAACTCTCACTTTCTAACCCTGTGCACTTGTTTGGGA
TAGTAACTCTCGCAAGAGAGCGAACTCCTATTCACTTATAAACACAAAGTCTATGAATGT
ATTAAATTTTATAACAAAATAAAACTTTCAACAACGGATCTCTTGGCTCTCGCATCGATG
AAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATC
TTTGAACGCACCTTGCGCTCCATGGTATTCCGTGGAGCATGCCTGTTTGAGTGTCATGAA
TACTTCAACCCTCCTCTTTCTTAATGATTGAAGAGGTGTTTGGTTTCTGAGCGCTGCTGG
CCTTTACGGTCTAGCTCGTTCGTAATGCATTAGCATCCGCAATCGAACTTCGGATTGACT
TGGCGTAATAGACTATTCGCTGAGGAATTCTAGTCTTCGGACTAGAGCCGGGTTGGGTTA
AAGGAAGCTTCTAATCAGAATGTCTACATTTTAAGATTAGATCTCAAATCAGGTAGGACT
ACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAAGGGG

>Seq105 [organism=*Stereum gausapatum*] [isolate P9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TATGACTGGAGTTGTAGCTGGCCTTTAAAACGGCATGTGCACGCTCCTTTCACTAATCCA
CACACACCTGTGCACCTTCGCGGGGGTCTCTTCGTTAACTCGAAGAGGCTCGCGTCCCTT
TACACACCCTTTGTATGTCTTAAGAATGTCTACTCGATGTAATAAAACGCATCTAATACA
ACTTTCAACAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAA
GTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCCCTTT
GGTATTCCGAAGGGCACACCTGTTTGAGTGTCGTGAAATTCTCAACCCTCTTCACTTTTG
TGAACGTAGTGGATTGGACTTGGAGGCTTTGCCGGGCTTCACCGCTCGGCTCCTCTCAAA
TGCATTAGTGCGTCTTGTTGCGACGTGCGCCTCGGTGTGATAATTATCTACGCTGTGGTG
TGCTTGCTTCTGTGGAGACGCGCTTTCTAACCGTCCGAAAGGACAGCTTTCATCGAACTT
TGACCTCAAATCAGGTGGGACTACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAA
AGAAAAAA

>Seq106 [organism=*Stereum gausapatum*] [isolate P8-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ATTATGACTGGAGTTGTAGCTGGCCTTTAAAAACGGCATGTGCACGCTCCTTTCACTAAT
CCACACACACCTGTGCACCTTCGCGGGGGTCTCTTCGTTAACTCGAAGAGGCTCGCGTCC
CTTTACACACCCTTTGTATGTCTTAAGAATGTCTACTCGATGTAATAAAACGCATCTAAT
ACAACTTTCAACAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGA
TAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCCC
TTTGGTATTCCGAAGGGCACACCTGTTTGAGTGTCGTGAAATTCTCAACCCTCTTCACTT
TTGTGAACGTAGTGGATTGGACTTGGAGGCTTTGCCGGGCTTCACCGCTCGGCTCCTCTC

AAATGCATTAGTGCGTCTTGTTGCGACGTGCGCCTCGGTGTGATAATTATCTACGCTGTG
GTGTGCTTGCTTCTGTGGAGACGCGCTTTCTAACCGTCCGAAAGGACAGCTTTCATCGAAC
TTTGACCTCAAATCAGGTGGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq107 [organism=*Stereum gausapatum*] [isolate P9-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGACTGGAGTTGTAGCTGGCCTTTAAAAACGGCATGTGCACGCTCCTTTCACTAATCCAC
ACACACCTGTGCACCTTCGCGGGGGTCTCTTTGTTAAACTCGAAGAGGCTCGCGTCCCTT
TACACACCCTTTGTATGTCTTAAGAATGTCTACTCGATGTAATAAAACGCATCTAATACA
ACTTTCAACAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAA
GTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCCCTTT
GGTATTCCGAAGGGCACACCTGTTTGAGTGTCGTGAAATTCTCAACCCTCTTCACTTTTG
TGAACGTAGTGGATTGGACTTGGAGGCTTTGCCGGGCTTCACCGCTCGGCTCCTCTCAAA
TGCATTAGTGCGTCTTGTTGCGACGTGCGCCTCGGTGTGATAATTATCTACGCTGTGGTG
TGCTTGCTTCTGTGGAGACGCGCTTTCTAACCGTCCGAAAGGACAGCTTTCATCGAACTT
TGACCTCAAATCAGGTGGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAA
AGAAAATTG

>Seq108 [organism=*Trametes versicolor*] [isolate P1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GACGAGTTGTAGCTGGCCTTCCGAGGCATGTGCACGCTCTGCTCATCCACTCTACCCCTG
TGCACTTACTGTAGGTTGGCGTGGGCTCCTTAACGGGAGCATTCTGCCGGCCTATGTATA
CTACAAACACTTTAAAGTATCAGAATGTAAACGCGTCTAACGCATCTATAATACAACTTT
TAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAAT
GTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCTCCTTGGTAT
TCCGAGGAGCATGCCTGTTTGAGTGTCATGGAATTCTCAACTTATAAATCCTTGTGATCT
ATAAGCTTGGACTTGGAGGCTTGCTGGCCCTTGCGGTCGGCTCCTCTTGAATGCATTAGC
TCGATTCCGTACGGATCGGCTCTCAGTGTGATAATTGTCTACGCTGTGACCGTGAAGTGT
TTTGGCGAGCTTCTAATCGTCCATTAGGACAACTTTTTAACATCTGACCTCAAATCAGGT
AGGACTACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAA

>Seq109 [organism=*Trametes versicolor*] [isolate P9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACGAGTTGTAGCTGGCCTTCCGAGGCATGTGCACGCTCTGCTCATCCACTCTACCCCTG
TGCACTTACTGTAGGTTGGCGTGGGCTCCTTAACGGGAGCATTCTGCCGGCCTATGTATA
CTACAAACACTTTAAAGTATCAGAATGTAAACGCGTCTAACGCATCTATAATACAACTTT
TAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAAT
GTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCTCCTTGGTAT

TCCGAGGAGCATGCCTGTTTGAGTGTCATGGAATTCTCAACTTATAAATCCTTGTGATCT
ATAAGCTTGGACTTGGAGGCTTGCTGGCCCTTGTTGGTCGGCTCCTCTTGAATGCATTAG
CTCGATTCCGTACGGATCGGCTCTCAGTGTGATAATTGTCTACGCTGTGACCGTGAAGTG
TTTTGGCGAGCTTCTAACCGTCCATTAGGACAACTTTTTAACATCTGACCTCAAATCAGG
TAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAA

>Seq110 [organism=*Trametes versicolor*] [isolate P7-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTTGACGAGTTGTAGCTGGCCTTCCGAGGCATGTGCACGCTCTGCTCATCCACTCTACCC
CTGTGCACTTACTGTAGGTTGGCGTGGGCTCCTTAACGGGAGCATTCTGCCGGCCTATGT
ATACTACAAACACTTTAAAGTATCAGAATGTAAACGCGTCTAACGCATCTATAATACAAC
TTTTAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGT
AATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCTCCTTGG
TATTCCGAGGAGCATGCCTGTTTGAGTGTCATGGAATTCTCAACTTATAAATCCTTGTGA
TCTATAAGCTTGGACTTGGAGGCTTGCTGGCCCTTGTTGGTCGGCTCCTCTTGAATGCAT
TAGCTCGATTCCGTACGGATCGGCTCTCAGTGTGATAATTGTCTACGCTGTGACCGTGAA
GTGTTTTGGCGAGCTTCTAACCGTCCATTAGGACAATTTTTTAACATCTGACCTCAAATC
AGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAA

>Seq111 [organism=*Trametes versicolor*] [isolate P8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTGACGAGTTGTAGCTGGCCTTCCGAGGCATGTGCACGCTCTGCTCATCCACTCTACCCC
TGTGCACTTACTGTAGGTTGGCGTGGGCTCCTTAACGGGAGCATTCTGCCGGCCTATGTA
TACTACAAACACTTTAAAGTATCAGAATGTAAACGCGTCTAACGCATCTATAATACAACT
TTTAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTA
ATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCTCCTTGGT
ATTCCGAGGAGCATGCCTGTTTGAGTGTCATGGAATTCTCAACTTATAAATCCTTGTGAT
CTATAAGCTTGGACTTGGAGGCTTGCTGGCCCTTGTTGGTCGGCTCCTCTTGAATGCATT
AGCTCGATTCCGTACGGATCGGCTCTCAGTGTGATAATTGTCTACGCTGTGACCGTGAAG
TGTTTTGGCGAGCTTCTAACCGTCCATTAGGACAATTTTTTAACATCTGACCTCAAATCA
GGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAAGA

>Seq112 [organism=*Trametes ochracea*] [isolate P3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGACGAGTTGTAGCTGGCCTTCCGAGGCATGTGCACGCTCTGCTCATCCACTCTACCCCT
GTGCACTTACTGTAGGTTGGCGTGGGCTCCTTAACGGGAGCATTCTGCCGGCCTATGTAT
ACTACAAACACTTTAAAGTATCAGAATGTAAACGCGTCTAACGCATCTATAATACAACTT
TTAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAA

TGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCTCCTTGGTA
TTCCGAGGAGCATGCCTGTTTGAGTGTCATGGAATTCTCAACTTATAAATCCTTGTGATC
TATAAGCTTGGACTTGGAGGCTTGCTGGCCCTCGTTGGTCGGCTCCTCTTGAATGCATTA
GCTCGATTCCGTACGGATCGGCTCTCAGTGTGATAATTGTCTACGCTGTGACCGTGAAGT
GTTTTGGCGAGCTTCTAACCGTCCATTAGGACAACTTTTTAACATCTGACCTCAAATCAG
GTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAAGA

>Seq113 [organism=*Trametes ochracea*] [isolate H10-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGACGAGTTGTAGCTGGCCTTCCGAGGCATGTGCACGCTCTGCTCATCCACTCTACCCCT
GTGCACTTACTGTAGGTTGGCGTGGGCTCCTTAACGGGAGCATTCTGCCGGCCTATGTAT
ACTACAAACACTTTAAAGTATCAGAATGTAAACGCGTCTAACGCATCTATAATACAACTT
TTAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAA
TGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTGCGCTCCTTGGTA
TTCCGAGGAGCATGCCTGTTTGAGTGTCATGGAATTCTCAACTTATAAATCCTTGTGATC
TATAAGCTTGGACTTGGAGGCTTGCTGGCCCTCGTTGGTCGGCTCCTCTTGAATGCATTA
GCTCGATTCCGTACGGATCGGCTCTCAGTGTGATAATTGTCTACGCTGTGACCGTGAAGT
GTTTTGGCGAGCTTCTAACCGTCCATTAGGACAACTTTTTAACATCTGACCTCAAATCAG
GTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAA

>Seq114 [organism=*Piptoporus betulinus*] [isolate P5-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGATGGGGTTGTCGCTGGCTGTTAGCAGCATGTGCACGCTCTGATCATTATCCATCTTAC
ACACCTGTGCACACACTGTAAGTCGGCTTTTGATGCAAAGTAAGGGTCTTCATTGACTCT
GCTTCAAATTGGGAGCCTTCTTATGTTTTATCACACACTACTTCAGTTTAAAGAATGTCA
AATCGCGTTTAACGCATTTAAATACAACTTTCAGCAACGGATCTCTTGGCTCTCGCATCG
ATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGA
ATCTTTGAACGCACCTTGCGCTCCTTGGTATTCCGAGGAGCATGCCTGTTTGAGTGTCAT
GGAATCATCAACTCTATTTACTTTTGTGAATAGGGCTTGGACTTGGAGGTTTTGCCGGTA
CTTGTGATCGGCTCCTCTTGAATGCATTAGCTTGAACCTTTGTGGATCAGCTTATCGGTG
TGATAATTGTCTACGCCGTTACTGTGAAGCATATATTAAAGGGCTCGGCTTCTAATCGTC
CTTTACGGGACAATAACTTTGACCTTTGACCTCAAATCAGGTAGGATTACCCGCTGAACT
TAAGCATATCAATAAGCGAGGGAAAAAGAAAAAAAA

>Seq115 [organism=*Phaeophlebiopsis peniophoroides*] [isolate P10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GAGGGGTTGTAGCTGGTCTCAATTAAGGGACATGTGCACACCTCACTCATCCACTCTCAA
ACCTCTGTGAACTTATTGTAGGTCGGTGAAAGGCTTGGTTTTAATTAATCAGGTTGGAAG

CCTTCCTATGTTTTTACTACAAACGCTTTAGTTATAGAATGTCATACGCGTATAACGCAT
TTATATACAACTTTCAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAA
ATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTT
GCGCTCCCTGGTATTCCGGGGAGCATGCCTGTTTGAGTATCATGGAATTCTCAACCTTCA
ATACTTTTTTAAAAAGAGTGTCGAAGGCTTGGACTTGGAGGCTTTGTGCTGGCTCTGTAT
TGAGTCGGCTCCTCTGAAATGTATTAGCGTGAATCACTATGGATCGCTTCGGTGTGATAA
TTATCTGCGCCGTGGTCGTGAAGTATTAATTCAAGTTCGCGCTTCTAATCGTCCTTCATG
GGACAATTACATTGAACTTTTGATCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCA
TATCAATAAGCGGGGGG

>Seq116 [organism=*Magnusiomyces capitatus*] [isolate H2-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ATATATTTTATATTACTTTGTGGAACATTTGGTTGAATTTACATGTTTTATTACAAAAAA
TTAATTATAATTAAAAAAATAATTTTAAGAAAACCTCCAACAACGGATCTCTTGGTTCCC
AGATCGATGAAGAGCGCAGCGAATTGCGAAATGTGATGTGTATTGCAGTGAATCATCAAT
TCTTGAACGCACATGGCACCCCTTTTAGGGGTATGCTTGTATGAGGGTGTTTAATATGAA
ATTGCTTTGGCTTTTTTTAAATAAATGGTTTTTCAAATTGTTACTAATAGACGAAAGAAT
CAGTGCAACAAGCTGTGTTGAATCTTTCATTAAATCTTTTAGTTAACTACTTTAACTATT
TGCACCTCATATCAAGCAAGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAA
AGAAAAGCCTTGT

>Seq117 [organism=*Magnusiomyces capitatus*] [isolate H4-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TATTTTATATTACTTTGTGGACATTTGGTTGAATTTACATGTTTTATTACAAAAAATTAA
TTATAATTAAAAAAATAATTTTAAGAAAACCTCCAACAACGGATCTCTTGGTTCCCAGAT
CGATGAAGAGCGCAGCGAATTGCGAAATGTGATGTGTATTGCAGTGAATCATCAATTCTT
GAACGCACATGGCACCCCTTTTAGGGGTATGCTTGTATGAGGGTGTTTAATATGAAATTG
CTTTGGCTTTTTTTAAATAAATGGTTTTTCAAATTGTTACTAATAGACGAAAGAATCAGT
GCAACAAGCTGTGTTGAATCTTTCATTAAATCTTTTAGTTAACTACTTTAACTATTTGCA
CCTCATATCAAGCAAGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGA
AGAAACCTTG

>Seq118 [organism=*Galactomyces candidum*] [isolate H7-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AATCCTCCGTTTTTGAACGCCCATTGGACTTTGGGGTATCCCCCAAAGTATACTTGGTTG
AACGTTGTTTCTCTCTTGGAATTGCTTTGCTCTTCTAAAATTTCCAATCAAATTCCTTTG
AAAAACAACACTATTCAACCTCCGAA

>Seq119 [organism=*Cyphellophora oxyspora*] [isolate A3-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGGGTGTCAACTTATGCGCCCGACCTCCAACCCTTTGTCTACTTGACCTTATGTTGCTTC
GGCAGGCCCGCTGGTGTCCCTTTGGGGCCCGGCCGCCGGGGGTCCTCGTGGCCCCGGG
TCCGCGCCTGCCGATGGCCCCTTCTTAAACTATTGTTAAAACGTGTCGTCTGAGTTTATGA
TTAAACAATCAAAACAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCGAAATGCGATAAGTAATGCGAATTGCAGAATCCGTGAGTCATCGAATCTTTGAAC
GCACATTGCGCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCT
CAAGCCCGGCTTGTAGTTGGTTGCGGCGGCCCCCACCGCCCTTCCCAAAGATAATGACGG
CGTCTGTGAGGACTCCTGTACACTGAGCTTTCGGGCACGTACACGGCTAGAAATCCAGGC
CCGGTCGCCCTCGGCCCCCCTCGTTTTCGAGGGACCGAGAACCCCCTTTATATCAAGGTT
GACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAG
AAA

>Seq120 [organism=*Cyphellophora oxyspora*] [isolate A5-8] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GGTGTCAACTTATGCGCCCGACCTCCACCCTTTGTCTACTTGACCTTATGTTGCTTCGGC
AGGCCCGCTGGTGTCCCTTTGGGGCCCGGCCGCCGGGGGTCCTCGTGGCCCCGGGTCC
GCGCCTGCCGATGGCCCCTTCTTAAACTATTGTTAAAACGTGTCGTCTGAGTTTATGATTAA
ACAATCAAAACAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCA
GCGAAATGCGATAAGTAATGCGAATTGCAGAATCCGTGAGTCATCGAATCTTTGAACGCA
CATTGCGCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAA
GCCCGGCTTGTAGTTGGTTGCGGCGGCCCCCACCGCCCTTCCCAAAGATAATGACGGCG
TCTGTGAGGACTCCTGTACACTGAGCTTTCGGGCACGTACACGGCTAGAAATCCAGGCCC
GGTCGCCCTCGGCCCCCCTCGTTTTCGAGGGACCGAGAACCCCCTTTATATCAAGGTTGA
CCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGA
AAATTC

>Seq121 [organism=*Cadophora fastigiata*] [isolate A3-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GTAGGGTCTCTCTGGCCCGACCTCCCAACCCTTTGTCTACTTGACCATCGTTGCTTCGGC
GAGCCCGTCCTCACGGACCGCCGGAGGGACCTTCACCGGCCCTCTGGTCCGCGCTCGTC
GGTAGCCCAACCATTAAAATCTTTAACCAAACGTGCCTTAATCTAAGTACAATTATTAAAT
AAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAA
ATGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCTTTGAACGCACAT
TGCGCCCTTTGGTATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATCACCCCTCAAGCC
CCTGGCTTGGTGTTGGACGGTTTGGTGGAGGCCCCCTCGGGGGCTCCTGCCCCTCCCAA
AGACAATGACGGCGGCCTCGTTGGACCCCCGGTACACTGAGTTCTTCACGGGACACGTAT

CGGATACATGGGTTTACGGGACACGGTCTGCCTCCCCTCAGGGAGAATCTTTCTAAGGTT
GACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAA
GAA

>Seq122 [organism=*Exophiala pisciphila*] [isolate A10-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGTCTTTTATAGGCTCGACCTCCCAACCCTTTGTTTACCCGACCCACGTTGCTTCGGCGA
GCCCGTCTCTCCCTCTCCGGGGGGGCAGACCGCCGGGGGGCCTTCAAACCGTCCTCTGG
CCCGCGCTCGTCGGTGGCCAACCTTTACAAATTCTTAATTAAACGTGTCTACTCTGAGTAC
TATTTTTTAATTAAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCGAATCT
TTGAACGCACATTGCGCCCTTTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTCATTATC
ACCTCTCAAGCCCCCCTTTTCTGGGCTTGGTGTTGGACGGCCTGGCGTCGGCGACGACCC
CACCTCCCAAAGACAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCCTCACC
GAACACGTACTGGATCAAGGGTAGACGGAGCCCCGGTCGACCTCCCCTCACAGGGAGAC
ACTTTTTTACAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAA
TAAGCGGAGGAAAAGAA

>Seq123 [organism=*Exophiala pisciphila*] [isolate C1-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGTACGGTCTTTTATAGGCTCGACCTCCCAACCCTTTGTTTACCCGACCCACGTTGCTTC
GGCGAGCCCGTCTCTCCCTCTCCGGGGGGGCAGACCGCCGGGGGGCCTTCAAACCGTC
CTCTGGCCCGCGCTCGTCGGTGGCCAACCTTTACAAATTCTTAATTAAACGTGTCTACTCT
GAGTACTATTTTTTAATTAAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCG
ATGAAGAACGCAGCGAAATGCGATAAGTAATGCGAATTGCAGAATTCCAGTGAGTCATCG
AATCTTTGAACGCACATTGCGCCCTTTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTCA
TTATCACCTCTCAAGCCCCCCTTTTCTGGGCTTGGTGTTGGACGGCCTGGCGTCGGCGAC
GACCCCACCTCCCAAAGACAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTCC
TCACCGAACACGTACTGGATCAAGGGTAGACGGAGCCCCGGTCGACCTCCCCTCACAGG
GAGACACTTTTTTACAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCAT
ATCAATAAGCGGAGGGAAAAGAAA

>Seq124 [organism=*Exophiala pisciphila*] [isolate A7-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTAGGGTCTTTTATAGGCTCGACCTCCCAACCCTTTGTTTACCCGACCCACGTTGCTTC
GGCGAGCCCGTCTCTCCCTCTCCGGGGGGGCAGACCGCCGGGGGGCCTTCAAACCGTC
CTCTGGCCCGCGCTCGTCGGTAGCCAACCTTTACAAATTCTTAATTAAACGTGTCTACTCT
GAGTACTATTTTTTAATTTAAAAGCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATC

GATGAAAAACGCAGCGAAATGCGATAAGTAATGCGAATTGCAAAATTCCAGTGAGTCATC
GAATCTTTGAACGCACATTGCGCCCTTTGGCATTCCGAAGGGCATGCCTGTTCGAGCGTC
ATTATCACCTCTCAAGCCCCCCTTTTCTGGGCTTGGTGTTGGACGGCCTGGCGTCGGCGA
CGACCCCACCTCCCAAAGACAATGACGGCGGCTTCGTGAGACCCCCGGTACACTGAGTTC
CTCACCGAACACGTACTGGATCAAGGGTAGACGGAGCCCCGGTCGACCTCCCCTCACAG
GGAGACACTTTTTTACAAGGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCA
TATCAATAAGCGGAGGGAAAAGAAA

>Seq125 [organism=*Trichoderma asperellum*] [isolate CRT2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTCACTCCCAACCCATGTGACGTTACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGT
GCGTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGAACCAACCAAACTCTTTCTGTAGT
CCCCTCGCGGACGTATTTCTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCA
ACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGT
GAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTC
TGGCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGATCGGCG
TTGGGGATCGGGACCCCTCACACGGGTGCCGGCCCCGAAATACAGTGGCGGTCTCGCCG
CAGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACGTC
CGTAAAACACCCAACTTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGAGGAAAAGAAAATTTTT

>Seq126 [organism=*Trichoderma asperellum*] [isolate A4-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCAATGTGAACGTTACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGT
GCGTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGAACCAACCAAACTCTTTCTGTAGT
CCCCTCGCGGACGTATTTCTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCA
ACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGT
GAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTC
TGGCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGATCGGCG
TTGGGGATCGGGACCCCTCACACGGGTGCCGGCCCCGAAATACAGTGGCGGTCTCGCCG
CAGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACGTC
CGTAAAACACCCAACTTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGAGGAAAAGAAATCCTCCT

>Seq127 [organism=*Trichoderma asperellum*] [isolate CRT2-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCAATGTGAACGTTACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGT

GCGTCGCAGCCCCGGAACCAGGCGCCCGCCGGAGGAACCAACCAAACTCTTTCTGTAGT
CCCCTCGCGGACGTATTTCTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCA
ACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGT
GAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTC
TGGCGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGATCGGCG
TTGGGGATCGGGACCCCTCACACGGGTGCCGGCCCCGAAATACAGTGGCGGTCTCGCCG
CAGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACGTC
CGTAAAACACCCAACTTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGAGGAAAAGAA

>Seq128 [organism=*Trichoderma atroviride*] [isolate A8-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CCAACCCAATGTGAACCATACCAAACTGTTGCCTCGGCGGGGTCACGCCCCGGGTGCGTC
GCAGCCCCGGAACCAGGCGCCCGCCGGAGGGACCAACCAAACTCTTTTCTGTAGTCCCC
TCGCGGACGTTATTTCTTACAGCTCTGAGCAAAAATTCAAAATGAATCAAAACTTTCAACA
ACGGATCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAA
TTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCCAGTATTCTGG
CGGGCATGCCTGTCCGAGCGTCATTTCAACCCTCGAACCCCTCCGGGGGGTCGGCGTTG
GGGACCTCGGGAGCCCCTAAGACGGGATCCCGGCCCCGAAATACAGTGGCGGTCTCGCC
GCAGCCTCTCCTGCGCAGTAGTTTGCACAACTCGCACCGGGAGCGCGGCGCGTCCACGT
CCGTAAAACACCCAACTTCTGAAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGAGGGAAAAGAAAGACGACGGC

>Seq129 [organism=*Fusarium dimerum*] [isolate B7-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq130 [organism=*Fusarium dimerum*] [isolate B2-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTAA

CACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGTG
AAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCCC
TCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCCC
CAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGGG
ACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAGG
TAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAACCTCTGTG

>Seq131 [organism=*Fusarium dimerum*] [isolate B1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TAACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGT
AACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAG
TGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATC
CCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGC
CCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTG
GGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCA
GGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq132 [organism=*Fusarium dimerum*] [isolate B2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TCATAAACTCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCC
CGTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCT
GAGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATG
AAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATC
TTTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTAC
ATCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCG
TGCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCA
CTGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAACCCC
C

>Seq133 [organism=*Fusarium dimerum*] [isolate B4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AGTCACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCC
GTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTG

AGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGA
AGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCT
TTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACA
TCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGT
GCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCAC
TGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAGAT

>Seq134 [organism=*Fusarium dimerum*] [isolate B1-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CATCACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCC
GTAACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTG
AGTGAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGA
AGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCT
TTGAACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACA
TCCCTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGT
GCCCCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCAC
TGGGACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAACCCCC

>Seq135 [organism=*Fusarium dimerum*] [isolate C1-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACCTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAAGAA

>Seq136 [organism=*Fusarium dimerum*] [isolate C4-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCTACAACGTTGCCTCGGCGGACCCCCGCCTCCCCGTA
ACACGGGAGCGGCCCGCCAGAGGACCCAACAAACCCTGTTATTTTCAGTATCTTCTGAGT
GAAAACACAATCAATTAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG

AACGCACATTGCGCCCGCCAGTACTCTGGCGGGCATGCCTGTTCGAGCGTCATTACATCC
CTCAAGCCCCTTCGGGCTTGGTGTTGGGCATCGGCCGTCCCTCCAGCGGCGGCCGTGCC
CCAAATACAGTGGCGGTCTCGCCCCCGGCTCCTCTGCGTAGTAGTAACATCTCGCACTGG
GACGGAGCGTAGGCCACGCCGTAAAACAACCCAACTTTCTGAATGTTGACCTCGGATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq137 [organism=*Fusarium solani*] [isolate A2-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCTCACCCTGTGACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAAAAC
GGGCCGCCCCCGCCAGAGGACCCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTAAAA
CAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAAC
GCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCCT
CAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGCCGT
CCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCGCG
ACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTCGAA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq138 [organism=*Fusarium solani*] [isolate A9-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCATCACCCTGTGACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAAAAC
GGGCCGCCCCCGCCAGAGGACCCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTAAAA
CAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAAC
GCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCCT
CAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGCCGT
CCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCGCG
ACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTCGAA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAATTTTA
TCATTACCTGTATTA

>Seq139 [organism=*Fusarium solani*] [isolate (CBS224.34)] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CACTCTCACCCTGTGAACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAAA
ACGGGCCGCCCCCGCCAGAGGACCCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC

CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGCC
GTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCG
CGACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTCG
AATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGGAAAAGAAA

>Seq140 [organism=*Fusarium solani*] [isolate B9-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCTCACCCTGTGAACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAAAA
CGGGCCGCCCCCGCCAGAGGACCCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTAAA
ACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCC
TCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGCCG
TCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCGC
GACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTCGA
ATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGGAAAAGAAA

>Seq141 [organism=*Fusarium solani*] [isolate A2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TCACTCATCACCCTGTGACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAA
AACGGGCCGCCCCCGCCAGAGGACCCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTA
AAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAAC
CCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGC
CGTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTC
GCGACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTC
GAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAC
GACA

>Seq142 [organism=*Fusarium solani*] [isolate A9-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACTCTCACCCTGTGAACTTACCTAAACGTTGCTTCGGCGGGAATAGACGGCCCTGTAAA
ACGGGCCGCCCCCGCCAGAGGACCCTTAACTCTGTTTCTTATAATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC

CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCTCGTGGGCGCACGCC
GTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCG
CGACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCCAACTCTTCTGAAGTTGACCTCG
AATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq143 [organism=*Fusarium solani*] [isolate B8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CACTCATCACCCCTGTGACATACCTAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTGA
AACGGGCCGCCCCCGCCAGAGGACCCCTAACTCTGTTTCTATAATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC
CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCCCCCCGTGGGCACACGCCGT
CCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATCGCGTAGTAGCTAACACCTCGCG
ACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTCTTCTGAAGTTGACCTCGAAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAACTCCGCC
A

>Seq144 [organism=*Fusarium solani*] [isolate A9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CACTCTCAACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCTGTAA
CAACGGGCCGCCCCCGCCAGAGGACCCCTAACTCTGTTTTTATAATGTTTTTCTGAGTAA
ACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCC
TCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAAGCCCCCTGTGGGCACACGCCGT
CCCTCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGCA
ACTGGAGAGCGGCGCGGCCATGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq145 [organism=*Fusarium solani*] [isolate A9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCATCACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCTGTAACA
ACGGGCCGCCCCCGCCAGAGGACCCCTAACTCTGTTTTTATAATGTTTTTCTGAGTAAAC
AAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCCTC

AGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAAGCCCCCTGTGGGCACACGCCGTCC
CTCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGCAAC
TGGAGAGCGGCGCGGCCATGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAATCAG
GTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGA

>Seq146 [organism=*Fusarium solani*] [isolate A6-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCTCACCCTGTGAACATACCTAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAACA
CGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTTTATAATGTTTTTTCTGAGTAAA
CAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAAC
GCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCCT
CAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCGCCCCCTGCGGGCACACGCCGT
CCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGCA
ACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAATC
AGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAA

>Seq147 [organism=*Fusarium solani*] [isolate C10-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTTAACTCATCACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCG
TAACACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGA
GTAAAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATG
AAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATC
TTTGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTAC
AACCCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACA
CGCCGTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACAC
CTCGCAACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCT
CGAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAAACT
GGAA

>Seq148 [organism=*Fusarium solani*] [isolate C10-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCATCACCCTGTGAACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAAC
ACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGAGTAA
AACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGA
ACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACC

CTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACACGCC
GTCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCG
CAACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAA

>Seq149 [organism=*Fusarium solani*] [isolate C10-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCATCACCCTGTGACATACCTAAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTAACA
CGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATTATGTTTCTTCTGAGTAAA
ACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAAGAA
CGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAACCC
TCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGAGGCGCCCCCTGCGGGCACACGCCG
TCCCCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCGC
AACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAAT
CAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq150 [organism=*Fusarium oxysporum*] [isolate C3-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq151 [organism=*Fusarium oxysporum*] [isolate C9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG

CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAAAAGAAAA

>Seq152 [organism=*Fusarium oxysporum*] [isolate C6-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAAAAGAAAAGGAGATA

>Seq153 [organism=*Fusarium oxysporum*] [isolate C9-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAAAAGAAA

>Seq154 [organism=*Fusarium oxysporum*] [isolate A6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGGGGAA

>Seq155 [organism=*Fusarium oxysporum*] [isolate C5-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGGGGAAAAGAA

>Seq156 [organism=*Fusarium oxysporum*] [isolate CRT1-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGAAAAGAAAAGATCTCG

>Seq157 [organism=*Fusarium oxysporum*] [isolate C9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAAA
CGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAAC
CATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCA
AGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCAC
GTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGCC
GTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAA
GCATATCAATAAGCGGAGGAAAAGAAATTA

>Seq158 [organism=*Fusarium oxysporum*] [isolate C5-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGAGGAAAAGAAA

>Seq159 [organism=*Fusarium oxysporum*] [isolate CRT1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGAGGAAAAGAAA

>Seq160 [organism=*Fusarium oxysporum*] [isolate A8-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq161 [organism=*Fusarium oxysporum*] [isolate C8-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACTCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA

CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGAGGAAAAGAAACACCCCC

>Seq162 [organism=*Fusarium oxysporum*] [isolate C5-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCTCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq163 [organism=*Fusarium oxysporum*] [isolate B9-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TACTCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ACGGGACGGCCCGCCAGAGGACCCCTAAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCTCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGAAAAGAAAATTCTCT

>Seq164 [organism=*Fusarium oxysporum*] [isolate C2-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA

CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGAAAAGAAAAC

>Seq165 [organism=*Fusarium oxysporum*] [isolate B10-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAAA
CGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAAC
CATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCA
AGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCAC
GTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGCC
GTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAA
GCATATCAATAAGCGGAGGAAAAGAAAACCTGGT

>Seq166 [organism=*Fusarium oxysporum*] [isolate C5-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAAGAAA

>Seq167 [organism=*Fusarium oxysporum*] [isolate B6-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTA
AAATGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAA
AACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCC
TCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGT

CACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCAC
GCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGGGGAAAAAAGAAA

>Seq168 [organism=*Fusarium oxysporum*] [isolate A1-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAA

>Seq169 [organism=*Fusarium oxysporum*] [isolate C9-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGGAAAAGAAA

>Seq170 [organism=*Fusarium oxysporum*] [isolate C9-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT

AAGCATATCAATAAGCGGGGGGA

>Seq171 [organism=*Fusarium oxysporum*] [isolate C6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq172 [organism=*Fusarium oxysporum*] [isolate C7-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq173 [organism=*Fusarium oxysporum*] [isolate A5-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TAACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGT
AAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTA
AAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACC
CTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGG
TCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCA
CGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAAC
TTAAGCATATCAATAAGCGGAGGGAAAAGAA

>Seq174 [organism=*Fusarium oxysporum*] [isolate A1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CATAACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGG
TAAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGT
AAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAG
AACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAAC
CCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCG
GTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCC
ACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGGAAAAGAAAATTTTTT

>Seq175 [organism=*Fusarium oxysporum*] [isolate C5-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq176 [organism=*Fusarium oxysporum*] [isolate C4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAA

>Seq177 [organism=*Fusarium oxysporum*] [isolate A7-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCACTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq178 [organism=*Fusarium oxysporum*] [isolate A8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GTAACTCCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGG
TAAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGT
AAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAG
AACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAAC
CCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCG
GTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCC
ACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGGAAAAGAAAATTTTA

>Seq179 [organism=*Fusarium oxysporum*] [isolate C6-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq180 [organism=*Fusarium oxysporum*] [isolate B5-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AATGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAA

>Seq181 [organism=*Fusarium oxysporum*] [isolate B7-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTA
AAATGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAA
AACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCC
TCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGT
CACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCAC
GCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq182 [organism=*Fusarium oxysporum*] [isolate B6-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAA
ATGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAA
CCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTC
AAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCA
CGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGC
CGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTA
AGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq183 [organism=*Fusarium oxysporum*] [isolate B5-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TCATACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGG
TAAAATGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGT
AAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAG
AACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAAC
CCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCG
GTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCC
ACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAATAAA

>Seq184 [organism=*Fusarium oxysporum*] [isolate A6-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq185 [organism=*Fusarium oxysporum*] [isolate C8-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq186 [organism=*Fusarium oxysporum*] [isolate B10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAAAAAGAAA

>Seq187 [organism=*Fusarium oxysporum*] [isolate C6-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAAA

>Seq188 [organism=*Fusarium oxysporum*] [isolate C1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TAACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGT
AAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTA
AAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGA
ACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTG
AACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACC
CTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGG
TCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCA
CGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAAC
TTAAGCATATCAATAAGCGGGGGAAAAAGAAAA

>Seq189 [organism=*Fusarium oxysporum*] [isolate C3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AACTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAA

>Seq190 [organism=*Fusarium oxysporum*] [isolate A7-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACTCCCAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCTCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGAGGGAAAAAGAA

>Seq191 [organism=*Fusarium oxysporum*] [isolate C8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ATTTAACTCCCAACCCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCC
GGTAAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGA
GTAAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGA
AGAACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCT
TTGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCA
ACCCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGG
CGGTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGG
CCACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTG
AACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAACCCCC

>Seq192 [organism=*Fusarium oxysporum*] [isolate C6-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAAAA
CGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAAAC
CATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCTCA
AGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTCAC
GTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACGCC
GTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTTAA
GCATATCAATAAGCGGGGGAAAAAGAAA

>Seq193 [organism=*Fusarium oxysporum*] [isolate C2-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGGGGAA

>Seq194 [organism=*Fusarium oxysporum*] [isolate A6-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CCTCCCAAACCCCTGTGAACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTA
AAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAA
AACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCC
TCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGT
CACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCAC
GCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGGGGAAAAAGAAA

>Seq195 [organism=*Fusarium oxysporum*] [isolate C10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTAATATGTAACTTCTGAGTAA
AACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAA
CGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGA
ACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCC
TCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCTCAAATTGATTGGCGGT
CACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCAC
GCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACT
TAAGCATATCAATAAGCGGGGGGAAAAGAAA

>Seq196 [organism=*Fusarium oxysporum*] [isolate A2-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq197 [organism=*Fusarium oxysporum*] [isolate CRT1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ACCTCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCGGTAA
AACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAGTAAA
ACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAAC
GCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAA
CGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAACCCT
CAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGCGGTC
ACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGCCACG
CCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq198 [organism=*Fusarium oxysporum*] [isolate A5-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GATACTCCCCAAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCG
GTAAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAG
TAAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAA
CCCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGC
GGTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGC
CACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGA
ACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAACCCCC

>Seq199 [organism=*Fusarium oxysporum*] [isolate C3-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CATCACTCCCCAACCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCCG
GTAAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGAG
TAAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAA
GAACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCAA
CCCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGGC
GGTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGGC
CACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTGA
ACTTAAGCATATCAATAAGCGAGGGAAAAGAAAATTCC

>Seq200 [organism=*Fusarium oxysporum*] [isolate C2-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGTCACTCCCCAACCCCCTGTGACATACCACTTGTTGCCTCGGCGGATCAGCCCGCTCCC
GGTAAAACGGGACGGCCCGCCAGAGGACCCCTAAACTCTGTTTCTATATGTAACTTCTGA
GTAAAACCATAAATAAATCAAAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGA
AGAACGCAGCAAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCT
TTGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTTCA
ACCCTCAAGCACAGCTTGGTGTTGGGACTCGCGTTAATTCGCGTTCCCCAAATTGATTGG
CGGTCACGTCGAGCTTCCATAGCGTAGTAGTAAAACCCTCGTTACTGGTAATCGTCGCGG
CCACGCCGTTAAACCCCAACTTCTGAATGTTGACCTCGGATCAGGTAGGAATACCCGCTG
AACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAATCCCT

>Seq201 [organism=*Penicillium brevicompactum*] [isolate A7-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CATGAGGCTCTGGGTCACCTCCCACCCGTGTTTATTTACCTTGTTGCTTCGGCGAGCCTG
CCTTTTGGCTGCCGGGGGACGTCAGTCCCCGGGTCCGTGCTCGCCGGAGACACCTTAGA
ACTCTGTCTGAAGATTGTAGTCTGAGATTAAATATAAATTATTTAAAACTTTCAACAACGG
ATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAATTGC
AGAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCTCTGGTATTCCGGAGGG
CATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCACGGCTTGTGTGTTGGGCCCCGTCCT
CCTTCCGGGGGACGGGTCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCAAGCGTAT
GGGGCTTTGTCACTCGCTTTGTAGGCCTGGCCGGCGCTTGCCGATCAACCAAACTTTTTAT
CAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAATAAGCGGAG
GGAAAAGAAAAAAAAA

>Seq202 [organism=*Phoma herbarum*] [isolate B3-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GAGTTGTGGGCTTTGCCTGCTATCTCTTACCCATGTCTTTTGAGTACTTACGTTTCCTCG
GTGGGTTCGCCCGCCGATTGGACAATTTAAACCCTTTGCAGTTGCAATCAGCGTCTGAAA
AACATAATAGTTACAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCGAAATGCGATAAGTAGTGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCCTTGGTATTCCATGGGGCATGCCTGTTCGAGCGTCATTTGTACCTTCA
AGCATTGCTTGGTGTTGGGTGTTTGTCTCGCCTTTGCGTGTAGACTCGCCTTAAAACAAT
TGGCAGCCGGCGTATTGATTTCGGAGCGCAGTACATCTCGCGCTTTGCACTCATAACGAC
GACGTCCAAAAGTACATTTTAACACTCTTGACCTCGGATCAGGTAGGGATACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGAAAAGAAAATGACCTC

>Seq203 [organism=*Phoma herbarum*] [isolate A4-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGTTTGTGGGCTTTGCCTGCTATCTCTTACCCATGTCTTTTGAGTACTTACGTTTCCTCG
GTGGGTTCGCCCGCCGATTGGACAATTTAAACCCTTTGCAGTTGCAATCAGCGTCTGAAA
AACATAATAGTTACAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCGAAATGCGATAAGTAGTGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCCTTGGTATTCCATGGGGCATGCCTGTTCGAGCGTCATTTGTACCTTCA
AGCATTGCTTGGTGTTGGGTGTTTGTCTCGCCTTTGCGTGTAGACTCGCCTTAAAACAAT
TGGCAGCCGGCGTATTGATTTCGGAGCGCAGTACATCTCGCGCTTTGCACTCATAACGAC
GACGTCCAAAAGTACATTTTAACACTCTTGACCTCGGATCAGGTAGGGATACCCGCTGAA
CTTAAGCATATCAATAAGCGGAGGAAAAGAAATGCTCATA

>Seq204 [organism=*Phoma herbarum*] [isolate C8-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

TGAGTGTGGGCTTTGCCTGCTATCTCTTACCCATGTCTTTTGAGTACTTACGTTTCCTCG
GTGGGTTCGCCCGCCGATTGGACAATTTAAACCCTTTGCAGTTGCAATCAGCGTCTGAAA
AACATAATAGTTACAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACGC
AGCGAAATGCGATAAGTAGTGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACG
CACATTGCGCCCCTTGGTATTCCATGGGGCATGCCTGTTCGAGCGTCATTTGTACCTTCA
AGCATTGCTTGGTGTTGGGTGTTTGTCTCGCCTTTGCGTGTAGACTCGCCTTAAAACAAT
TGGCAGCCGGCGTATTGATTTCGGAGCGCAGTACATCTCGCGCTTTGCACTCATAACGAC
GACGTCCAAAAGTACATTTTAACACTCTTGACCTCGGATCAGGTAGGGATACCCGCTGAA
CTTAAGCATATCAATAAGCGGGGGAAAAAGAA

>Seq205 [organism=*Phoma herbarum*] [isolate CRT3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGAGTTGTGGGCTTTGCCTGCTATCTCTTACCCATGTCTTTTGAGTACTTACGTTTCCTC
GGGGGGTTCGCCCGCCGATTGGACAATTTAAACCCTTTGCAGTTGCAATCAGCGTCTGAA
AAACATAATAGTTACAACTTTCAACAACGGATCTCTTGGTTCTGGCATCGATGAAGAACG
CAGCGAAATGCGATAAGTAGTGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAAC
GCACATTGCGCCCCTTGGTATTCCATGGGGCATGCCTGTTCGAGCGTCATTTGTACCTTC
AAGCATTGCTTGGTGTTGGGTGTTTGTCTCGCCTTTGCGTGTAGACTCGCCTTAAAACAA
TTGGCAGCCGGCGTATTGATTTCGGAGCGCAGTACATCTCGCGCTTTGCACTCATAACGA
CGACGTCCAAAAGTACATTTTAACACTCTTGACCTCGGATCAGGTAGGGATACCCGCTGA
ACTTAAGCATATCAATAAGCGGAGGGAAAAGAAA

>Seq206 [organism=*Rhodotorula mucilaginosa*] [isolate A5-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGGACGTCCACTTAACTTGGAGTCCGAACTCTCACTTTCTAACCCTGTGCACTTGTTTGG
GATAGTAACTCTCGCAAGAGAGCGAACTCCTATTCACTTATAAACACAAAGTCTATGAAT
GTATTAAATTTTATAACAAAATAAAACTTTCAACAACGGATCTCTTGGCTCTCGCATCGA
TGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAA
TCTTTGAACGCACCTTGCGCTCCATGGTATTCCGTGGAGCATGCCTGTTTGAGTGTCATG
AATACTTCAACCCTCCTCTTTCTTAATGATTGAAGAGGTGTTTGGTTTCTGAGCGCTGCT
GGCCTTTACGGTCTAGCTCGTTCGTAATGCATTAGCATCCGCAATCGAACTTCGGATTGA
CTTGGCGTAATAGACTATTCGCTGAGGAATTCTAGTCTTCGGATTAGAGCCGGGTTGGGT
TAAAGGAAGCTTCTAATCAGAATGTCTACATTTTAAGATTAGATCTCAAATCAGGTAGGA
CTACCCGCTGAACTTAAGCATATCAATAAGCGAGGGAAAAAGAAAATTTTTT

>Seq207 [organism=*Mucor circinelloides*] [isolate B5-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

CTTGTCCATTATTATCTATTTACTGTGAAATGTATTATTACTTGACGCTTGAGGGATGCT
CCACTGCTATAAGGATAGGCGGTGGGGATGCTAACCGAGTCATAATCAAGCTTAGGCTTG
GTATCCTATTATTATTTACCAAAAGAATTCAGAATTAATATTGTAACATAGACCTAAAAA
ATCTATAAAACAACTTTTAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGTAGC
AAAGTGCGATAACTAATGTGAATTGCATATTCAGTGAATCATCGAGTCTTTGAACGCAAC
TTGCGCTCATTGGTATTCCAATGAGCACGCCTGTTTCAGTATCAAAACAAACCCTCTATC
CAACATTTTGTTGAATAGGAATACTGAGAGTCTCTTGATCTATTCTGATCTCGAACCTCT
TGAAATGTACAAAGGCCTGATCTTGTTTGAATGCCTGAACTTTTTTTTAATATAAAGAGA
AGCTCTTGCGGTAAACTGTGCTGGGGCCTCCCAAATAATACTTTTTTTAAATTTGATCTGAA
ATCAGGCGGGATTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAATTG
G

>Seq208 [organism=*Aspergillus niger*] [isolate A5-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GCGGGTCTTTGGGCCCACCTCCCATCCGTGTCTATTATACCCTGTTGCTTCGGCGGGCCC
GCCGCTTGTCGGCCGCCGGGGGGGCGCCTTTGCCCCCCGGGCCCGTGCCCGCCGGAGA
CCCCAACACGAACACTGTCTGAAAGCGTGCAGTCTGAGTTGATTGAATGCAATCAGTTAAA
ACTTTCAACAATGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATAAC
TAATGTGAATTGCAGAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTG
GTATTCCGGGGGGCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGT
TGGGTCGCCGTCCCCCTCTCCGGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGT
CCGATCCTCGAGCGTATGGGGCTTTGTCACATGCTCTGTAGGATTGGCCGGCGCCTGCCG
ACGTTTTCCAACCATTTTTTCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTT
AAGCATATCAATAAGCGGAGGGAAAAGAA

>Seq209 [organism=*Candida parapsilosis*] [isolate B10-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

AGTGCTTACTGCATTTTTTCTTACACATGTGTTTTTCTTTTTTTGAAAACTTTGCTTTGA
TAGGCCTTCTATATGGGGCCTGCCAGAGATTAAACTCAACCAAATTTTATTTAATGTCAA
CCGATTATTTAATAGTCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAG
AACGCAGCGAAATGCGATAAGTAATATGAATTGCAGATATTCGTGAATCATCGAATCTTT
GAACGCACATTGCGCCCTTTGGTATTCCAAAGGGCATGCCTGTTTGAGCGTCATTTCTCC
CTCAAACCCTCGGGTTTGGTGTTGAGCGATACGCTGGGTTTGCTTGAAAGAAAGGCGGAG
TATAAACTAATGGATAGGTTTTTTCCACTCATTGGTACAAACTCCAAAACTTCTTCCAAA
TTCGACCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGA
AAAGAAA

>Seq210 [organism=*Rhodotorula slooffiae*] [isolate A4-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTAGGAGTTCTTTTTAGAGTCCGACCCTTTCATTTTCTTACACTGTGCACACACTTCTTT
TTTACACACACTTTTAACACCTTAGTATAAGAATGTAATAGTCTCTTAATTGAGCATAAA
TAAAAACAAAACTTTCAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGA
ATTGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCT
TGCACTCTTTGGTATTCCGAAGAGTATGTCTGTTTGAGTGTCATGAAACTCTCAACCCCC
CTATTTTGTAATGAGATGGGTGTGGGCTTGGATTATGGTTGTCTGTCGGCGTAATTGCCG
GCTCAACTGAAATACACGAGCAACCCTATTGAAATAAACGGTTTGACTTGGCGTAATAAT
TATTTCGCTAAGGACGTTTTCTTCAAATATAAGAGGTGCTTCTAATTCGCTTCTAATAGC
ATTTAAGCTTTAGACCTCAAATCAGTCAGGACTACCCGCTGAACTTAAGCATATCAATAA
GCGGAGGGAAAAGAAAAGGAGC

>Seq211 [organism=*Rhodotorula slooffiae*] [isolate A10-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTAGGAGTTCTTTTTAGAGTCCGACCCTTTCATTTTCTTACACTGTGCACACACTTCTTT
TTTACACACACTTTTAACACCTTAGTATAAGAATGTAATAGTCTCTTAATTGAGCATAAA
TAAAAACAAAACTTTCAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGA
ATTGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCT
TGCACTCTTTGGTATTCCGAAGAGTATGTCTGTTTGAGTGTCATGAAACTCTCAACCCCC
CTATTTTGTAATGAGATGGGTGTGGGCTTGGATTATGGTTGTCTGTCGGCGTAATTGCCG
GCTCAACTGAAATACACGAGCAACCCTATTGAAATAAACGGTTTGACTTGGCGTAATAAT
TATTTCGCTAAGGACGCTTTCTTCAAATATAAGAGGTGCTTCTAATTCGCTTCTAATAGC
ATTTAAGCTTTAGACCTCAAATCAGTCAGGACTACCCGCTGAACTTAAGCATATCAATAA
GCGAGGGAAAAAGAAAAAA

>Seq212 [organism=*Rhodosporidium babjevae*] [isolate B8-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGACGTCCACTTAACTTGGAGTCCGAACTCTCACTTTCTAACCCTGTGCATCTGTTAATT
GGAATAGTAGCTCTTCGGAGTGAACCACCATTCACTTATAAAACACAAAGTCTATGAATG
TATACAAATTTATAACAAAACAAAACTTTCAACAACGGATCTCTTGGCTCTCGCATCGAT
GAAGAACGCAGCGAAATGCGATACGTAATGTGAATTGCAGAATTCAGTGAATCATCGAAT
CTTTGAACGCACCTTGCGCTCCTTGGTATTCCGAGGAGCATGCCTGTTTGAGTGTCATGA
AATCTTCAACCCACCTCTTTCTTAGTGAATCTGGTGGTGCTTGGTTTCTGAGCGCTGCTC
TGCTTCGGCTTAGCTCGTTCGTAATGCATTAGCATCCGCAACCGAACTTCGGATTGACTT
GGCGTAATAGACTATTCGCTGAGGATTCTAGTTTACTAGAGCCGAGTTGGGTTAAAGGAA

GCTCCTAATCCTAAAGTCTATTTTTTGATTAGATCTCAAATCAGGTAGGACTACCCGCTG
AACTTAAGCATATCAATAAGCGGGGGGAA

>Seq213 [organism=*Meyerozyma guilliermondii*] [isolate B9-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TCTTTTGCAGCGCTTACTGCGCGGCGAAAAACCTTACACACAGTGTCTTTTTGATACAGA
ACTCTTGCTTTGGTTTGGCCTAGAGATAGGTTGGGCCAGAGGTTTAACAAAACACAATTT
AATTATTTTTACAGTTAGTCAAATTTTGAATTAATCTTCAAAACTTTCAACAACGGATCT
CTTGGTTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATATGAATTGCAGAT
TTTCGTGAATCATCGAATCTTTGAACGCACATTGCGCCCTCTGGTATTCCAGAGGGCATG
CCTGTTTGAGCGTCATTTCTCTCTCAAACCCCCGGGTTTGGTATTGAGTGATACTCTTAG
TCGGACTAGGCGTTTGCTTGAAAAGTATTGGCATGGGTAGTACTAGATAGTGCTGTCGAC
CTCTCAATGTATTAGGTTTATCCAACTCGTTGAATGGTGTGGCGGGATATTTCTGGTATT
GTTGGCCCGGCCTTACAACAACCAAACAAGTTTGACCTCAAATCAGGTAGGAATACCCGC
TGAACTTAAGCATATCAATAAGCGAAGGAAAAAA

>Seq214 [organism=*Clavispora lusitaniae*] [isolate C1-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TATTCGAATTTCTTAATATCAAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATG
AAGAACGCAGCGAATTGCGATACGTAGTATGACTTGCAGACGTGAATCATCGAATCTTTG
AACGCACATTGCGCCTCGAGGCATTCCTCGAGGCATGCCTGTTTGAGCGTCGCATCCCCT
CTAACCCCCGGTTAGGCGTTGCTCCGAAATATCAACCGCGCTGTCAAACACGTTTACAGC
ACGACATTTCGCCCTCAAATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGC
GAGGGAAAAGAAAAAACCC

>Seq215 [organism=*Candida intermedia*] [isolate C4-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTATTACTTGTTTTTGAGCAACACTAATAACCATTAATATTCAATAACCTAAAAAAAATC
AAAACTTTCAACAACGGATCTCTTGGTTCTCGCATCGATGAAGAACGCAGCGAATTGCGA
TACGTATCATGACTTGCAGACGTGAATCATCAATCTTTGAACGCACATTGCGCCTTGAGG
TATTCCTCAAGGCATGCCTGTTTGAGCGTCGGCTCCCTTCAAACCCCCGGGTTTGGTGTT
GCCTTCCGAAATATCACAGTTGTTGCAATACGTTACTTCAACTTTATTCTTTCGCCCTCA
AATCAGGTAGGACTACCCGCTGAACTTAAGCATATCAATAAGCGGGGGAAAAGAAA

>Seq216 [organism=*Penicillium echinulatum*] [isolate A4-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GATGAGGCTCTGGGTCACCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGCCC

GCCTTAACTGGCCGCCGGGGGGCTCACGCCCCCGGGCCCGCGCCCGCCGAAGACACCC
TCGAACTCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAACAA
CGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAAT
TGCAAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGG
GGCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCCGTC
CTCCGATTTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCGA
GCGTATGGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACCCAA
ATTTTTATCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAAT
AAGCGGAGGGAAAAGAAAATCTGT

>Seq217 [organism=*Meyerozyma guilliermondii*] [isolate B10-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TCTTTGCAGCGCTTACTGCGCGGCGAAAAACCTTACACACAGTGTCTTTTTGATACAGAA
CTCTTGCTTTGGTTTGGCCTAGAGATAGGTTGGGCCAGAGGTTTAACAAAACACAATTTA
ATTATTTTTACAGTTAGTCAAATTTTGAATTAATCTTCAAAACTTTCAACAACGGATCTC
TTGGTTCTCGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATATGAATTGCAGATT
TTCGTGAATCATCGAATCTTTGAACGCACATTGCGCCCTCTGGTATTCCAGAGGGCATGC
CTGTTTGAGCGTCATTTCTCTCTCAAACCCCCGGGTTTGGTATTGAGTGATACTCTTAGT
CGGACTAGGCGTTTGCTTGAAAAGTATTGGCATGGGTAGTACTAGATAGTGCTGTCGACC
TCTCAATGTATTAGGTTTATCCAACTCGTTGAATGGTGTGGCGGGATATTTCTGGTATTG
TTGGCCCGGCCTTACAACAACCAAACAAGTTTGACCTCAAATCAGGTAGGAATACCCGCT
GAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAAAAAAG

>Seq218 [organism=*Rhodotorula slooffiae*] [isolate B10-9] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GGACGTTCTTTTTAGAGTCCGACCCTTTCATTTTCTTACACTGTGCACACACTTCTTTTT
TACACACACTTTTAACACCTTAGTATAAGAATGTAATAGTCTCTTAATTGAGCATAAATA
AAAACAAAACTTTCAGCAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAAT
TGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCACCTTG
CACTCTTTGGTATTCCGAAGAGTATGTCTGTTTGAGTGTCATGAAACTCTCAACCCCCCT
ATTTTGTAATGAGATGGGTGTGGGCTTGGATTATGGTTGTCTGTCGGCGTAATTGCCGGC
TCAACTGAAATACACGAGCAACCCTATTGAAATAAACGGTTTGACTTGGCGTAATAATTA
TTTCGCTAAGGACGTTTTCTTCAAATATAAGAGGTGCTTCTAATTCGCTTCTAATAGCAT
TTAAGCTTTAGACCTCAAATCAGTCAGGACTACCCGCTGAACTTAAGCATATCAATAAGC
GGAGGAAAAGAAAAAAAA

>Seq219 [organism=*Trichosporon asteroides*] [isolate B10-12] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

ATGCCTTATTGGCTTATAACTATATCCACTTACACCTGTGAACTGTTCTACTACTTGACG
CAAGTCGAGTATTTTTACAAACAATGTGTAATGAACGTCGTTTTATTATAACAAAATAAA
ACTTTCAACAACGGATCTCTTGGCTCTCGCATCGATGAAGAACGCAGCGAATTGCGATAA
GTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTTTGAACGCAGCTTGCGCTCTCT
GGTATTCCGGAGAGCATGCCTGTTTCAGTGTCATGAAATCTCAACCACTAGGGTTTCCTA
ATGGATTGGATTTGGGCGTCTGCGATCTCTGATCGCTCGCCTTAAAAGAGTTAGCAAGTT
TGACATTAATGTCTGGTGTAATAAGTTTCACTGGGTCCATTGTGTTGAAGCGTGCTTCTA
ATCGTCCGCAAGGACAATTACTTTGACTCTGGCCTGAAATCAGGTAGGACTACCCGCTGA
ACTTAAGCATATCAATAAGCGGGGGAAA

>Seq220 [organism=*Penicillium crustosum*] [isolate A3-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGAGGGCTCTGGGTCCACCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGCCC
GCCTTAACTGGCCGCCGGGGGGCTTACGCCCCCGGGCCCGCGCCCGCCGAAGACACCC
TCGAACTCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAACAA
CGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAAT
TGCAAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGG
GGCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCCGTC
CCCCGATCTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCGA
GCGTATGGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACCCAA
ATTTTTATCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAAT
AAGCGGAGGAAAAGAAA

>Seq221 [organism=*Penicillium crustosum*] [isolate A5-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TGAGGGCTCTGGGTCCACCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGCCC
GCCTTAACTGGCCGCCGGGGGGCTTACGCCCCCGGGCCCGCGCCCGCCGAAGACACCC
TCGAACTCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAACAA
CGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAAT
TGCAAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGG
GGCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCCGTC
CCCCGATCTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCGA
GCGTATGGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACCCAA
ATTTTTATCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAAT
AAGCGGAGGGAAAAGAAA

>Seq222 [organism=*Ochroconis mirabilis*] [isolate C8-7] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence

GTGCTGTATTCGTGCTTCTCTTCGGAGACGCGAAGCTACCCGACCTGATGTCATTTTATC
CGAGGGCGCGCCCGCTTGAGAAGACAGCGGTTTGACGTTGTGGTGTTACCCGGACGAGG
CGCTTGTCTCGTAAGGGGAAGGGTCGG

>Seq223 [organism=*Penicillium expansum*] [isolate C8-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
ATCAGGGTCACCCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGCCCGCCTTA
ACTGGCCGCCGGGGGGCTTACGCCCCCGGGCCCGCGCCCGCCGAAGACACCCTCGAAC
TCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAACAACGGATC
TCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAATTGCAAA
TTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGGGGCATG
CCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCCGTCCCCCGA
TCTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCGAGCGTAT
GGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACCCAAATTTTTA
TCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAATAAGCGG
AGGAAAAGAAATCCTTC

>Seq224 [organism=*Trichosporon domesticum*] [isolate A3-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TTGCCTTATTGGCTTAACTATATCCATCTACACCTGTGAACTGTTTGATTGAATCTTCGG
ATTCGATTTTATACAAACATTGTGTAATGAACGTCATTATATTATAACAAAAAAAAAACT
TTCAACAACGGATCTCTTGGCTCTCGCATCGATGAAAAACGCAGCGAAATGCGATAAGTA
ATGTGAATTGCAAAATTCAGTGAATCATCGAATCTTTGAACGCAACTTGCGCTCTCTGGT
ATTCCGGAAAGCATGCCTGTTTGAGTGTCATGAAATCTCAACCATTAGGGTTTCTTAATG
GCTTGGATTTGGAGGTTTGCCAGTCTGACTGGCTCCTCTTAAAAAAGTTAGCAAGTTGAA
CTATTGCTATCTGGCGTAATAAGTTTCGCTGGAATGGTATTGTGAAGCGTGCTTCTAATC
GTCTTCGGACAATTTTTTGACTCTGGCCTCAAATCAGGTAGGACTACCCGCTGAACTTAA
GCATATCAATAAGCGGAG

>Seq225 [organism=*Penicillium expansum*] [isolate A1-4] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
CATGAGGCTCTGGGTCACCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGCCC
GCCTTAACTGGCCGCCGGGGGGCTTACGCCCCCGGGCCCGCGCCCGCCGAAGACACCC
TCGAACTCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAACAA
CGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAAT
TGCAAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGG
GGCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCCGTC

CCCCGATCTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCGA
GCGTATGGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACCCAA
ATTTTTATCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAAT
AAGCGGAGGAAAAGAAAATAAAGG

>Seq226 [organism=*Penicillium crustosum*] [isolate A1-3] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTGAGGGCCTCTGGGTCCACCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGC
CCGCCTTAACTGGCCGCCGGGGGGCTTACGCCCCCGGGCCCGCGCCCGCCGAAGACAC
CCTCGAACTCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAAC
AACGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGA
ATTGCAAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGG
GGGGCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCC
GTCCCCCGATCTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCT
CGAGCGTATGGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACC
CAAATTTTTATCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATC
AATAAGCGGAGGGAAAAGAAA

>Seq227 [organism=*Penicillium crustosum*] [isolate B9-5] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GTGAGGCCTCTGGGTCCACCTCCCACCCGTGTTTATTTTACCTTGTTGCTTCGGCGGGCC
CGCCTTAACTGGCCGCCGGGGGGCTTACGCCCCCGGGCCCGCGCCCGCCGAAGACACC
CTCGAACTCTGTCTGAAGATTGAAGTCTGAGTGAAAATATAAATTATTTAAAACTTTCAACAA
CGGATCTCTTGGTTCCGGCATCGATGAAGAACGCAGCGAAATGCGATACGTAATGTGAATT
GCAAATTCAGTGAATCATCGAGTCTTTGAACGCACATTGCGCCCCCTGGTATTCCGGGGG
GCATGCCTGTCCGAGCGTCATTGCTGCCCTCAAGCCCGGCTTGTGTGTTGGGCCCCGTCC
CCCGATCTCCGGGGGACGGGCCCGAAAGGCAGCGGCGGCACCGCGTCCGGTCCTCGAG
CGTATGGGGCTTTGTCACCCGCTCTGTAGGCCCGGCCGGCGCTTGCCGATCAACCCAAAT
TTTTATCCAGGTTGACCTCGGATCAGGTAGGGATACCCGCTGAACTTAAGCATATCAATAA
GCGAGGGA

>Seq228 [organism=*Lecythophora fasciculata*] [isolate R10-6] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
GCGAGGCTACTTAAACCATCGCGAACTCGTCCAAGTTGCTTCGGCGGCGCGGCCTCCCTC
ACGGGGGCGCCGCAGCCCCGCCTCTCCGGAGGTGTGGGGCGCCCGCCGGAGGTACGAA
ACTCTGTATTATAGTGGCATCTCTGAGTAAAAAACAAATAAGTTAAAACTTTCAACAACGGA
TCTCTTGGTTCTGGCATCGATGAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCA
GAATTCAGTGAATCATCGAATCTTTGAACGCACATTGCGCCCGCTAGTACTCTAGCGGGC

ATGCCTGTTCGAGCGTCATTTCAACCCTCAAGCCCTGCTTGGTGTTGGGGCCCTACGGCT
GCCGTAGGCCCTGAAAGGAAGTGGCGGGCTCGCTACAACTCCGAGCGTAGTAATTCATTA
TCTCGCTAGGGACGTTGCGGCGCGCTCCTGCCGTTAAAGACCATCTTTAACTCAAGGTTG
ACCTCGGATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAA
A

>Seq229 [organism=*Fusarium solani*] [isolate K8-1] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
TAAAACTCATCAACCCTGTGAACATACCTAAACGTTGCTTCGGCGGGAACAGACGGCCCC
GTAACACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATAAATGTTTTTTCT
GAGTAAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGAT
GAAGAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAAT
CTTTGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTA
CAACCCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCCCCCCGTGGGCACAC
GCCGTCCTCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACC
TCGCAACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTC
GAATCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGAAAAGAAA

>Seq230 [organism=*Fusarium solani*] [isolate K8-2] internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence; and 28S ribosomal RNA gene, partial sequence
AACTCATCAACCCTGTGAACATACCTAAACGTTGCTTCGGCGGGAACAGACGGCCCCGTA
ACACGGGCCGCCCCCGCCAGAGGACCCCCTAACTCTGTTTCTATAAATGTTTTTTCTGAG
TAAACAAGCAAATAAATTAAAACTTTCAACAACGGATCTCTTGGCTCTGGCATCGATGAA
GAACGCAGCGAAATGCGATAAGTAATGTGAATTGCAGAATTCAGTGAATCATCGAATCTT
TGAACGCACATTGCGCCCGCCAGTATTCTGGCGGGCATGCCTGTTCGAGCGTCATTACAA
CCCTCAGGCCCCCGGGCCTGGCGTTGGGGATCGGCGGAGCCCCCCGTGGGCACACGCC
GTCCTCCAAATACAGTGGCGGTCCCGCCGCAGCTTCCATTGCGTAGTAGCTAACACCTCG
CAACTGGAGAGCGGCGCGGCCACGCCGTAAAACACCCAACTTCTGAATGTTGACCTCGAA
TCAGGTAGGAATACCCGCTGAACTTAAGCATATCAATAAGCGGAGGGAAAAGAAAAGGTG
GAATGTGCTGTG

APPENDIX 4: TEF-1α Sequences

>Seq1 [organism=*Fusarium dimerum*] [isolate X4-2](X4-3) translation elongation factor 1 alpha (tef) gene, partial cds

1 alpha (tef) gene, partial cds

GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT
CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTAT
CAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGTCG

>Seq2 [organism=*Fusarium dimerum*] [isolate X3-1] translation elongation factor 1 alpha (tef) gene, partial cds

GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT
CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAGTCA
CTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCG

>Seq3 [organism=*Fusarium dimerum*] [isolate X5-2] translation elongation factor 1 alpha (tef) gene, partial cds

CGGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT

GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCT
ATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGA
TCCGGGGTAC

>Seq4 [organism=*Fusarium dimerum*] [isolate X3-3] translation elongation factor 1 alpha (tef) gene, partial cds

GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT
CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAGTCA
CTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGT

>Seq5 [organism=*Fusarium dimerum*] [isolate X5-4] translation elongation factor 1 alpha (tef) gene, partial cds

CCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAGTC
TTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCTAG
ACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGAG
AAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTCAC
GTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTGGT
GGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATTTT
CACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCAAT
AGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTCA
AGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCCCC
GCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAG
TCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq6 [organism=*Fusarium oxysporum*] [isolate K6-1] translation elongation factor 1 alpha (tef) gene, partial cds

AGTCGACGCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGCCATCGTCAATCCTGA
CCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCGTTTCACAGACCGGTC
ACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGAGAAGGTTA
GTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCCTACGACTCGAAACGT
ACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTAATTTTTTTGGTGGGG
CACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAACCATTCTCACAACCTC
AATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACAATAGGAAGCCGCTGA
GCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTCAAGGCCGAGCGTGA
GCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCTCGCTACTATGTCAC
CGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAACATATCACT
CAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGG

>Seq7 [organism=*Fusarium solani*] [isolate K2-4] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCCATCGCGATCTGCTTAT
CTCGGGTCGTGGAACCCCGCCTGGCATCACGGGCGGGGTACTCATCAGTCACTTCATGCT
GACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACC
ATCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCCCGATCGCGCCTTGCTATTCCACATC
GAATTCCCCGTCGAATTCCCTCCTCGCGACACGCTCTGCGCCCGCTTCTCTCGAGTTCCA
AAAATTTTGCGGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGTGA
CGTTGGACAAAGCCCTGATCCCTGCACACAAATAACACGAAACCCTCTTGGCGCGCATCA
TCACGTGGTTCACAACAGACGCTAACCGACTCAACAATAGGAAGCCGCTGAGCTCGGTAA
GGGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTAT
CACCATCGACATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGG
TATGTCGCTGTCACGTCTCTCACTCATGTCTCACCACTAACAGTCAACAGACGCCCCCGG
CCACCGTGACTTCATCAAGAACATGATACGGGGT

>Seq8 [organism=*Fusarium solani*] [isolate K2-3] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAGTCGACCACCGTAAGTCAAACCCTCCATCGCGATCTGCTTATC
TCGGGTCGTGGAACCCCGCCTGGCATCACGGGCGGGGTACTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCCCGATCGCGCCTTGCTATTCCACATCG
AATTCCCCGTCGAATTCCCTCCTCGCGACACGCTCTGCGCCCGCTTCTCTCGAGTTCCAA
AAATTTTGCGGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGTGAC
GTTGGACAAAGCCCTGATCCCTGCACACAAATAACACGAAACCCTCTTGGCGCGCATCAT

CACGTGGTTCACAACAGACGCTAACCGACTCAACAATAGGAAGCCGCTGAGCTCGGTAAG
GGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATC
ACCATCGACATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGT
ATGTCGCTGTCACGTCTCTCACTCATGTCTCACCACTAACAGTCAACAGACGCCCCCGGC
CACCGTGACTTCATCAAGAACATGATACGGGGG

>Seq9 [organism=*Fusarium solani*] [isolate K10-2] translation elongation factor 1 alpha (tef) gene, partial cds
GGCAACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTA
TCTCAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGC
TGACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAAC
CATCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCCTTGCTATTCCACAT
CGAATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGC
GGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACAA
AGCCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGTT
CACAACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTC
AAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGAC
ATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCTG
TCACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGAT
TTCATCAAGAACATGATCATGGGG

>Seq10 [organism=*Fusarium solani*] [isolate K9-6] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATC
TCAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCCTTGCTATTCCACATCG
AATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGCGG
TCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACAAAG
CCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGTTCA
CAACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAA
GTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGACAT
TGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCTGTC
ACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGATTT
CATCAAGAACATGATCCGGGGT

>Seq11 [organism=*Fusarium solani*] [isolate K10-1] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATCT
CAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTGA
CAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCAT
CGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCCTTGCTATTCCACATCGA
ATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGCGGT
CCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACAAAGC
CCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGTTCAC
AACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAG
TACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGACATT
GCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCTGTCA
CCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGATTTC
ATCAAGAACATGATCACTGGGTACTT

>Seq12 [organism=*Fusarium solani*] [isolate K9-5] translation elongation factor 1 alpha (tef) gene, partial cds

GCCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTAT
CTCAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCT
GACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACC
ATCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCCTTGCTATTCCACATC
GAATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGCG
GTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACAAA
GCCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGTTC
ACAACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCA
AGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGACA
TTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCTGT
CACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGATT
TCATCAAGAACATGATCATGG

>Seq13 [organism=*Fusarium solani*] [isolate R1-3] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTA
TCTTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGC
TGACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAAC
CATCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACAT
CGAATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCC
CAAAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGG

CGACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCAT
CACGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAG
GGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATC
ACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGT
ATGTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGC
CACCGTGATTTCATCAAGAACATGATCTG

>Seq14 [organism=*Fusarium solani*] [isolate R1-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATC
TTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACATCG
AATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCG
ACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCA
CGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGG
TTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCAC
CATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTAT
GTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGCCA
CCGTGATTTCATCAAGAACATGATCCGGGGGAC

>Seq15 [organism=*Fusarium solani*] [isolate R1-2] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATC
TTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACATCG
AATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCG
ACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCA
CGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGG
TTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCAC
CATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTAT
GTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGCCA
CCGTGATTTCATCAAGAACATGATCATGG

>Seq16 [organism=*Fusarium solani*] [isolate R6-9] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCCGTCGACTCTGGCAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTACTTATC
TCGGGTCGTGGAACCCCGCCTGGCATCTCGGGCGGGGTATTCATCATTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCCCGATCGCGCCTTGCTATTCCACATCG
AATCCCCTCCCTCGCGATACGCTCTGCGCCCGCTTCTCCCGAGTTCCAAAATTTTTGCGG
TCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACAAAG
CCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGTTCAC
AACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAG
TACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGACATT
GCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCTGTCA
CCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGACTTCAT
CAAGAACATGATCTG

>Seq17 [organism=*Fusarium dimerum*] [isolate R8-3] translation elongation factor 1 alpha (tef) gene, partial cds

GACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAG
TCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA
ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCT

>Seq18 [organism=*Fusarium dimerum*] [isolate X2-6] translation elongation factor 1 alpha (tef) gene, partial cds

GACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAG
TCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA

ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCG

>Seq19 [organism=*Fusarium dimerum*] [isolate X2-4] translation elongation factor 1 alpha (tef) gene, partial cds
TCGGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAG
TCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq20 [organism=*Fusarium dimerum*] [isolate R5-1] translation elongation factor 1 alpha (tef) gene, partial cds
AACGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAGT
CTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCTA
GACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGA
GAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTCA
CGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTGG
TGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATTT
TCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCAA
TAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTC
AAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCCC
CGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCA
GTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCG
GGGTAC

>Seq21 [organism=*Fusarium dimerum*] [isolate R6-3] translation elongation factor 1 alpha (tef) gene, partial cds
TCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA

CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT
GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCT
ATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGA
CCGGGGG

>Seq22 [organism=*Fusarium dimerum*] [isolate R6-2] translation elongation factor 1 alpha (tef) gene, partial cds

ATCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATC
TATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq23 [organism=*Fusarium dimerum*] [isolate R7-2] translation elongation factor 1 alpha (tef) gene, partial cds

GACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAG
TCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA
ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCA

TG

>Seq24 [organism=*Fusarium dimerum*] [isolate R5-2] translation elongation factor 1 alpha (tef) gene, partial cds

GACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAG
TCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA
ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGG

>Seq25 [organism=*Fusarium dimerum*] [isolate R8-2] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT
GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCT
ATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq26 [organism=*Fusarium dimerum*] [isolate R5-3] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT
GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA

CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCT
ATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq27 [organism=*Fusarium dimerum*] [isolate R8-1] translation elongation factor 1 alpha (tef) gene, partial cds

CGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATC
AGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACAC
CTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTT
CGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCT
TCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTG
TGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAA
TTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCAC
CAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACT
CCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTA
TCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq28 [organism=*Fusarium dimerum*] [isolate X2-5] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATC
TATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq29 [organism=*Fusarium dimerum*] [isolate R7-1] translation elongation factor 1 alpha (tef) gene, partial cds

ATCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG

TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATC
TATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATG
ATCATGG

>Seq30 [organism=*Fusarium dimerum*] [isolate X2-2] translation elongation factor 1 alpha (tef) gene, partial cds
GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT
CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTAT
CAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGG

>Seq31 [organism=*Fusarium dimerum*] [isolate R6-1] translation elongation factor 1 alpha (tef) gene, partial cds
GACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCGTGACTGCTCACTATCAG
GCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA
ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq32 [organism=*Fusarium dimerum*] [isolate H4-1] translation elongation factor 1 alpha (tef) gene, partial cds

ACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAGT
CTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCTA
GACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGA
GAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTCA
CGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTGG
TGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATTT
TCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCAA
TAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTC
AAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCCC
CGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCA
GTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGGGTA

>Seq33 [organism=*Fusarium oxysporum*] [isolate H7-1] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATC
TGCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACA
TCGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCG
AGAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTC
CCCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACC
GTAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTT
AACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCA
ACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGAC
TCCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCT
TCGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGACC

>Seq34 [organism=*Fusarium oxysporum*] [isolate H3-1] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC

TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCAGGGGGAC

>Seq35 [organism=*Fusarium dimerum*] [isolate P2-1] translation elongation factor 1 alpha (tef) gene, partial cds
TCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT
GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCCTGGGGTTCTTTGA
ACAAAGCCTCAA

>Seq36 [organism=*Fusarium dimerum*] [isolate P2-3] translation elongation factor 1 alpha (tef) gene, partial cds
ATCGGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACT
ATCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTA
CACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAA
GTTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTC
GCTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACA
TTGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAA
AAATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACC
CACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGAC
AAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAG
ACTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCAT
CTATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACAT
GACCGGGGGA

>Seq37 [organism=*Fusarium dimerum*] [isolate H10-1] translation elongation factor 1 alpha (tef) gene, partial cds
TCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT

GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCT
ATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGACATGTCA

>Seq38 [organism=*Fusarium dimerum*] [isolate H2-1] translation elongation factor 1 alpha (tef) gene, partial cds

ATCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATC
TATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq39 [organism=*Fusarium dimerum*] [isolate P5-1] translation elongation factor 1 alpha (tef) gene, partial cds

ACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAGT
CTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCTA
GACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGA
GAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTCA
CGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTGG
TGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATTT
TCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCAA
TAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTC
AAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCCC
CGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCA
GTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGTA

>Seq40 [organism=*Fusarium dimerum*] [isolate P5-2] translation elongation factor 1 alpha (tef) gene, partial cds

GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT
CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTAT
CAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq41 [organism=*Fusarium dimerum*] [isolate H1-1] translation elongation factor 1 alpha (tef) gene, partial cds

ATCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATC
TATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCT

>Seq42 [organism=*Fusarium dimerum*] [isolate H5-1] translation elongation factor 1 alpha (tef) gene, partial cds

ACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAGT
CTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCTA
GACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGA
GAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTCA
CGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTGG
TGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATTT
TCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCAA
TAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTC

AAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCCC
CGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAGTCACT
AACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGTAC

>Seq43 [organism=*Fusarium dimerum*] [isolate H6-1] translation elongation factor 1 alpha (tef) gene, partial cds
CCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT
GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAGT
CACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGG

>Seq44 [organism=*Fusarium dimerum*] [isolate P2-2] translation elongation factor 1 alpha (tef) gene, partial cds
CCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTAT
CAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACA
CCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGT
TCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGC
TTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATT
GTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAA
ATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCA
CCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGAC
TCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAGT
CACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCG

>Seq45 [organism=*Fusarium dimerum*] [isolate B7-6] translation elongation factor 1 alpha (tef) gene, partial cds
ACATTGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCA
CAAAAATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACA
ACCCACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTT
GACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTC

GAGACTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCAT
CATCTATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAA
CATGATCCGGGGTA

>Seq46 [organism=*Fusarium dimerum*] [isolate B2-5] translation elongation factor 1 alpha (tef) gene, partial cds
ATCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAG
TCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGG

>Seq47 [organism=*Fusarium dimerum*] [isolate B1-1] translation elongation factor 1 alpha (tef) gene, partial cds
CTCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAG
TCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGG

>Seq48 [organism=*Fusarium dimerum*] [isolate B2-1] translation elongation factor 1 alpha (tef) gene, partial cds
GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT

CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAGTCA
CTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATACGGGG

>Seq49 [organism=*Fusarium dimerum*] [isolate B4-1] translation elongation factor 1 alpha (tef) gene, partial cds
GGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCA
GTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACC
TAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTC
GAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTT
CACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGT
GGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAAT
TTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACC
AATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTC
CCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTAT
CAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTGTG

>Seq50 [organism=*Fusarium dimerum*] [isolate B1-6] translation elongation factor 1 alpha (tef) gene, partial cds
GAACGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAG
TCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA
ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGT

>Seq51 [organism=*Fusarium dimerum*] [isolate C1-4] translation elongation factor 1 alpha (tef) gene, partial cds

ATCGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATC
TATCAGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCAT

>Seq52 [organism=*Fusarium dimerum*] [isolate C4-5] translation elongation factor 1 alpha (tef) gene, partial cds

GACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTATCAG
TCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTACACCT
AGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCG
AGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCGCTTC
ACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACATTGTG
GTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAAAATT
TTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCCACCA
ATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGACTCC
CCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATC
AGTCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq53 [organism=*Fusarium solani*] [isolate A2-5] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATC
TTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACATCG
AATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCG
ACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCA
CGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGG

TTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCAC
CATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTAT
GTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGCCA
CCGTGATTTCATCAAGAACATGATCCGGGGTAC

>Seq54 [organism=*Fusarium solani*] [isolate A9-3] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTA
TCTTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGC
TGATAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATTGACAAGCGAAC
CATCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACAT
CGAATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCC
CAAAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGG
CGACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCAT
CACGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAG
GGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATC
ACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGT
ATGTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGC
CACCGTGATTTCATCAAGAACATGATCAT

>Seq55 [organism=*Fusarium solani*] [isolate B9-3] translation elongation factor 1 alpha (tef) gene, partial cds

CGTTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATCTT
GGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTGAC
AATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATC
GAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACATCGAA
TTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCCCAAA
AATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGAC
GTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCACG
TGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTT
CCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCA
TCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGT
CGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGCCACC
GTGATTTCATCAAGAACATGATCACTGGGTACCTCCA

>Seq56 [organism=*Fusarium solani*] [isolate A2-1] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTA
TCTTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGC
TGACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAAC
CATCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACAT
CGAATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCC
CAAAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGG
CGACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCAT
CACGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAG
GGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATC
ACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGT
ATGTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGC
CACCGTGATTTCATCAAGAACATGTCGGG

>Seq57 [organism=*Fusarium solani*] [isolate A9-4] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATC
TTGGGTCGTGGAACCCCACCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATATCTCCCGATCGCGCCTTGCTATTCCACATCG
AATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAAATTTTGCGGTTCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCG
ACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCA
CGTGGTTCACAACAGACACTGACTCGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGG
TTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCAC
CATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTAT
GTCGCCGTCATGTCTCTCACTCACGTCTCATCACTAACAATCCACAGACGCCCCCGGCCA
CCGTGATTTCATCAAGAACATGATCCGGGGGAC

>Seq58 [organism=*Fusarium solani*] [isolate B8-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAGCCCTCATCGCGATCTGCTTATC
TCGGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTACTCATCAGTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTCACATCTCCCCCGATCGCGCCTTGCTATCCCACATCG
AATTCCCCGTCGAATTCCCTCCTCCGCGACACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAAATTTTGCGGTTCGACCGTAAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCG

ACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCA
CGTGGTTCACAACAGACACTGACTGGTTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGG
TTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCAC
CATCGATATTGCTCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTAC
GTCGCCGTCATCTCTCACTCACGTCTCATCACTAACAATCAACAGACGCCCCCGGCCACC
GTGATTTCATCAAGAACATGACCGGGGG

>Seq59 [organism=*Fusarium solani*] [isolate A9-2] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATCT
CGGGTCGTGGAACCCCGCCTGGCATCTCGGGCGGGGTATTCATCATTCACTTCATGCTGA
CAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCAT
CGAGAAGTTCGAGAAGGTTGGTGACATCTGCCCCCGATCGCGCCTTGATATTCCACATCG
AATTCCCCGTCGAATTCCCTCCATCGCGATACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAATTTTTGCGGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGA
CGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCAT
CACGTGGTTCACGACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAG
GGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATC
ACCATCGACATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGT
ATGTTGCTGTCACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGC
CACCGTGACTTCATCAAGAACATGATCCGG

>Seq60 [organism=*Fusarium solani*] [isolate A9-1] translation elongation factor 1 alpha (tef) gene, partial cds
CAACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTATC
TCGGGTCGTGGAACCCCGCCTGGCATCTCGGGCGGGGTATTCATCATTCACTTCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATCTGCCCCCGATCGCGCCTTGATATTCCACATC
GAATTCCCCGTCGAATTCCCTCCATCGCGATACGCTCTGCGCCCGCTTCTCCCGAGTCCC
AAAATTTTTGCGGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCG
ACGTTGGACAAAGCCCTGATCCCTGCACACAAAAACACCAAACCCTCTTGGCGCGCATCA
TCACGTGGTTCACGACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAA
GGGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTAT
CACCATCGACATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGG
TATGTTGCTGTCACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGG
CCACCGTGACTTCATCAAGAACATGATCCGGGGGGGAA

>Seq61 [organism=*Fusarium solani*] [isolate A6-4] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTTCTTATC
TCGGGTCGTGGAACCCCGCCTGGCATCTCGGGCGGGGTATTCATCATTCACATCATGCTG
ACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCA
TCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCCCGATCGCGCCTTGCTATTCCAGATCG
AATTCCCCGTCGAATTCCCTCCCTCCCGATACGCTCTGCGCCCGCTTCTCCCGAGTCCCA
AAATTTTTGCGGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGA
CGTTGGACAAAGCCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCA
TCACGTGGTTCACAACAGACGCTAACTGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAA
GGGTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTAT
CACCATCGACATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGG
TATGTTGCTATCACCTCTCACACACATGTCTCACCACTAACAATCAACAGACGCCCCCGG
CCACCGTGATTTCATCAAGAACATGATCATGGGTACCTCCAAAAGGG

>Seq62 [organism=*Fusarium solani*] [isolate C10-4] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCCGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTAT
CTCAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGCT
GACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACC
ATCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCTTTGCTATTCCACATC
GAATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGCG
GTCCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACAA
AGCCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGTT
CACAACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTC
AAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGAC
ATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCTG
TCACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGAT
TTCATCAAGAACATGATCATGG

>Seq63 [organism=*Fusarium solani*] [isolate C10-2] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTA
TCTCAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGC
TGACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAAC
CATCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCTTTGCTATTCCACAT
CGAATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGC
GGTCCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACA

AAGCCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGT
TCACAACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTT
CAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGA
CATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCT
GTCACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGA
TTTCATCAAGAACATGATCATGG

>Seq64 [organism=*Fusarium solani*] [isolate C10-7] translation elongation factor 1 alpha (tef) gene, partial cds
CGGCACGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCATCGCGATCTGCTTA
TCTCAGGTCGTGGAACCCCGCCTGGTATCTCGGGCGGGGTATTCATCAGTCACTTCATGC
TGACAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAAC
CATCGAGAAGTTCGAGAAGGTTGGTGACATCTCCCTCGATCGCGCTTTGCTATTCCACAT
CGAATTCTCTCCCTCGCGATACGGTCTGCGCCCGCTTCTCCCGAGTCCCAAAATTTTTGC
GGTCCGACCGTAATTTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGCGACGTTGGACA
AAGCCCTGATCCCTGCACACAAAAAACACCAAACCCTCTTGGCGCGCATCATCACGTGGT
TCACAACAGACGCTAACCGGTCCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTT
CAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGA
CATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTTGCT
GTCACCTCTCTCACACATGTCTCACCACTAACAATCAACAGACGCCCCCGGCCACCGTGA
TTTCATCAAGAACATGATCT

>Seq65 [organism=*Fusarium oxysporum*] [isolate C3-4] translation elongation factor 1 alpha (tef) gene, partial cds
GGACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGG
GGAAC

>Seq66 [organism=*Fusarium oxysporum*] [isolate C9-2] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTG

>Seq67 [organism=*Fusarium oxysporum*] [isolate C6-5] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTGGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACATCCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGGGG

>Seq68 [organism=*Fusarium oxysporum*] [isolate C9-4] translation elongation factor 1 alpha (tef) gene, partial cds

GCCCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG

CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCAC
TGGGTACCTCCA

>Seq69 [organism=*Fusarium oxysporum*] [isolate A6-1] translation elongation factor 1 alpha (tef) gene, partial cds
GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGACCGGG
GGACCCTTC

>Seq70 [organism=*Fusarium oxysporum*] [isolate C5-3] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTGGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACATCCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGGAA

>Seq71 [organism=*Fusarium oxysporum*] [isolate CRT1-2] translation elongation factor 1 alpha (tef) gene, partial cds
CGGCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATC
TGCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACA
TCGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCG

AGAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTC
CCCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACC
GTAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTT
AACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCA
ACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGAC
TCCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTAC
TAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCTG

>Seq72 [organism=*Fusarium oxysporum*] [isolate C9-1] translation elongation factor 1 alpha (tef) gene, partial cds
CGGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACT
AACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGG

>Seq73 [organism=*Fusarium oxysporum*] [isolate C5-1] translation elongation factor 1 alpha (tef) gene, partial cds
GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCACT
GGGTACCTTCC

>Seq74 [organism=*Fusarium oxysporum*] [isolate CRT1-3] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATC
TGCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACA
TCGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCG
AGAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTC
CCCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACC
GTAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTT
AACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCA
ACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGAC
TCCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTAC
TAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGG

>Seq75 [organism=*Fusarium oxysporum*] [isolate A8-2] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTA
ACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGGAA

>Seq76 [organism=*Fusarium oxysporum*] [isolate C8-2] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG

CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCG

>Seq77 [organism=*Fusarium oxysporum*] [isolate C5-2] translation elongation factor 1 alpha (tef) gene, partial cds
CCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGCC
ATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCGAAGTCAACATACTGACATCGT
TTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAA
GTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCCT
ACGACTCGAAATGTGCCTGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTAA
TTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAACC
ATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACAA
TAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTC
AAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCT
CGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAAC
ATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCACTGGGTTA

>Seq78 [organism=*Fusarium oxysporum*] [isolate B9-2] translation elongation factor 1 alpha (tef) gene, partial cds
GCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCATATCTGC
CATCGTCAATCCCGACCAAGACCTGGCGGGGTATTTCTCAAAGTCAACATACTAACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCAATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTT

>Seq79 [organism=*Fusarium oxysporum*] [isolate C2-1] translation elongation factor 1 alpha (tef) gene, partial cds
GCCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC

TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAA
CATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGGAACCCT

>Seq80 [organism=*Fusarium oxysporum*] [isolate B10-2] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTGGG

>Seq81 [organism=*Fusarium oxysporum*] [isolate C5-5] translation elongation factor 1 alpha (tef) gene, partial cds
CTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGCCATCGTCAA
TCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCGTTTCACAGA
CCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGAGA
AGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCCTACGACTCG
AAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTAATTTTTTTG
GTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAACCATTCTCAC
AACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACAATAGGAAGC
CGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTCAAGGCCGA
GCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCTCGCTACTA
TGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAACAT
ATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCACTGGGTACTTCC

>Seq82 [organism=*Fusarium oxysporum*] [isolate B6-2] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGATAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACTGTA
ATTTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGTAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGG

>Seq83 [organism=*Fusarium oxysporum*] [isolate A1-2] translation elongation factor 1 alpha (tef) gene, partial cds
CCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCTATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTC

>Seq84 [organism=*Fusarium oxysporum*] [isolate C9-5] translation elongation factor 1 alpha (tef) gene, partial cds
CGGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCAT
GGGG

>Seq85 [organism=*Fusarium oxysporum*] [isolate C9-3] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCTG
GGGT

>Seq86 [organism=*Fusarium oxysporum*] [isolate C6-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGGGT

>Seq87 [organism=*Fusarium oxysporum*] [isolate C7-1] translation elongation factor 1 alpha (tef) gene, partial cds

CGGCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA

CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTG

>Seq88 [organism=*Fusarium oxysporum*] [isolate A5-6] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCG

>Seq89 [organism=*Fusarium oxysporum*] [isolate A1-1] translation elongation factor 1 alpha (tef) gene, partial cds
GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCACG
GGGTACCTCCA

>Seq90 [organism=*Fusarium oxysporum*] [isolate C5-4] translation elongation factor 1 alpha (tef) gene, partial cds
GCCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC

GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCACT
GGGTACCC

>Seq91 [organism=*Fusarium oxysporum*] [isolate C4-1] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTG

>Seq92 [organism=*Fusarium oxysporum*] [isolate A8-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTA
ACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGGAA

>Seq93 [organism=*Fusarium oxysporum*] [isolate C6-3] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTGGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACATCCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCAT

>Seq94 [organism=*Fusarium oxysporum*] [isolate B5-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGATAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACTGT
AATTTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGTAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTGG

>Seq95 [organism=*Fusarium oxysporum*] [isolate B7-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGATAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACTGT
AATTTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGTAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG

CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACT
AACATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGACCGGGGGA

>Seq96 [organism=*Fusarium oxysporum*] [isolate B6-1] translation elongation factor 1 alpha (tef) gene, partial cds
GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGATAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACTGT
AATTTTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGTAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACT
AACATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGGAA

>Seq97 [organism=*Fusarium oxysporum*] [isolate B5-3] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGATAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACTGT
AATTTTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGTAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGG
GGACCCCT

>Seq98 [organism=*Fusarium oxysporum*] [isolate A6-2] translation elongation factor 1 alpha (tef) gene, partial cds
GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG

AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTGG

>Seq99 [organism=*Fusarium oxysporum*] [isolate C8-6] translation elongation factor 1 alpha (tef) gene, partial cds
GTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCGTTTC
ACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTT
CGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCCTACG
ACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTAATTT
TTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAACCATT
CTCACATCCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACAATAG
GAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTCAAG
GCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCTCGC
TACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACT
AACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGGGTAC
CTCCAACCA

>Seq100 [organism=*Fusarium oxysporum*] [isolate B10-1] translation elongation factor 1 alpha (tef) gene, partial cds
GACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGCC
ATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCGT
TTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAA
GTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCCT
ACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTAA
TTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAACC
ATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACAA
TAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCTC
AAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCCT
CGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAAC
ATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGTA

>Seq101 [organism=*Fusarium oxysporum*] [isolate C6-2] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTGGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACATCCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAA
CATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCACGGGTT

>Seq102 [organism=*Fusarium oxysporum*] [isolate C1-3] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA
GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACT
AACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGTGG

>Seq103 [organism=*Fusarium oxysporum*] [isolate C3-1] translation elongation factor 1 alpha (tef) gene, partial cds

GGCCGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT

CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCG

>Seq104 [organism=*Fusarium oxysporum*] [isolate A7-3] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATACTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGG
GGTACCTCCA

>Seq105 [organism=*Fusarium oxysporum*] [isolate C8-1] translation elongation factor 1 alpha (tef) gene, partial cds
GGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCG

>Seq106 [organism=*Fusarium oxysporum*] [isolate C6-4] translation elongation factor 1 alpha (tef) gene, partial cds
CGGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA

GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACT
AACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATGCGG

>Seq107 [organism=*Fusarium oxysporum*] [isolate C2-7] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTAT
CTGCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGAC
ATCGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATC
GAGAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTT
CCCCTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGAC
CGTAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCT
TAACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTC
AACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGA
CTCCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTA
CTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCG

>Seq108 [organism=*Fusarium oxysporum*] [isolate A6-3] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTA
ACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCCGGGGGA

>Seq109 [organism=*Fusarium oxysporum*] [isolate C10-1] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGGGCTTATCTG
CCATCGTCAATCCCGACCAAGACCTGGCGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCACGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTGCCCGCTACCCCGCTCGAGACCAAAATTTTTGCAATACGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAC
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATGTCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG
GGTACCTCCAAAAA

>Seq110 [organism=*Fusarium oxysporum*] [isolate A2-3] translation elongation factor 1 alpha (tef) gene, partial cds

GCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTACTAA
CATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGACCGGGGGA

>Seq111 [organism=*Fusarium oxysporum*] [isolate CRT1-1] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATC
TGCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACA
TCGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCG
AGAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTC
CCCTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACC
GTAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTT
AACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCA

ACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAA
GCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGAC
TCCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCT
TCGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTG

>Seq112 [organism=*Fusarium oxysporum*] [isolate A5-1] translation elongation factor 1 alpha (tef) gene, partial cds
CACGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTGC
CATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATCG
TTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGA
AGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCCC
TACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGTA
ATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAAC
CATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAACA
ATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGCT
CAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTCC
TCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCG
TACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATGG
GTACCTCCAAATGTTT

>Seq113 [organism=*Fusarium oxysporum*] [isolate C3-2] translation elongation factor 1 alpha (tef) gene, partial cds
GGCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCTG
CCATCGTCAATCCCGGCCAAGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACATC
GTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAG
AAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCCC
CTACGACTCGAAACGTACCCGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGACCGT
AATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTAA
CCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAAC
AATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAGC
TCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACTC
CTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTC
GTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTG
>Seq114 [organism=*Fusarium oxysporum*] [isolate C2-4] translation elongation factor 1 alpha (tef) gene, partial cds
CGGCACGTCGACTCTGGCAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTATCT
GCCATCGTCAATCCTGACCAGGACCTGGTGGGGTATTTCTCAAAGTCAACATACTGACAT
CGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGA

GAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTTCC
CCTACGACTCGAAACGTACCCGCTACCCCCGCTCGAGACCAAAAATTTTGCAATATGACCG
TAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCTTA
ACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTCAA
CAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACAAG
CTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGACT
CCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTT
CGTACTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGACAG

>Seq115 [organism=*Fusarium solani*] [isolate K8-1] translation elongation factor 1 alpha (tef) gene, partial cds
CCGTCGACTCTGGCAAGTCGACCACCGTAAGTCAAACCCTCCATCGCGATCTGCTTATCT
CGGGTCGTGGAACCCCGCCTGGCATCACGGGCGGGGTACTCATCAGTCACTTCATGCTGA
CAATCATCTACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCAT
CGAGAAGTTCGAGAAGGTTGGTGACATCTCCCCCGATCGCGCCTTGCTATTCCACATCGA
ATTCCCCGTCGAATTCCCTCCTCGCGACACGCTCTGCGCCCGCTTCTCTCGAGTTCCAAA
AATTTTGCGGTCCGACCGTAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGTGACG
TTGGACAAAGCCCTGATCCCTGCACACAAATAACACGAAACCCTCTTGGCGCGCATCATC
ACGTGGTTCACAACAGACGCTAACCGACTCAACAATAGGAAGCCGCTGAGCTCGGTAAGG
GTTCCTTCAAGTACGCCTGGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCA
CCATCGACATTGCCCTCTGGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTA
TGTCGCTGTCACGTCTCTCACTCATGTCTCACCACTAACAGTCAACAGACGCCCCCGGCC
ACCGTGACTTCATCAAGAACATGATCACGGGGT

>Seq116 [organism=*Fusarium solani*] [isolate K8-2] translation elongation factor 1 alpha (tef) gene, partial cds
TACCACCGTAAAGTCAAACCCTCCATCGCGATCTGCTTATCTCGGGTCGTGGAACCCCGC
CTGGCATCACGGGCGGGGTACTCATCAGTCACTTCATGCTGACAATCATCTACAGACCGG
TCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAGTTCGAGAAGGT
TGGTGACATCTCCCCCGATCGCGCCTTGCTATTCCACATCGAATTCCCCGTCGAATTCCC
TCCTCGCGACACGCTCTGCGCCCGCTTCTCTCGAGTTCCAAAAATTTTGCGGTCCGACCG
TAATTTTTTTGGTGGGGCATTTACCCCGCCACTCGGGTGACGTTGGACAAAGCCCTGATC
CCTGCACACAAATAACACGAAACCCTCTTGGCGCGCATCATCACGTGGTTCACAACAGAC
GCTAACCGACTCAACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCT
GGGTCCTTGACAAGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGACATTGCCCTCT
GGAAGTTCGAGACTCCCCGCTACTATGTCACCGTCATTGGTATGTCCGCTGTCACGTCTC
TCACTCATGTCTCACCACTAACAGTCAACAGACGCCCCCGGCACCGTGACTTCATCAAGA
ACATTGATCACTGGTACCCTCCA

>Seq117 [organism=*Fusarium dimerum*] [isolate X4-1] translation elongation factor 1 alpha (tef) gene, partial cds

TCGGGACCGTCGACTCCGGAAGTCCACCACTGTAAGTTCTCACTCCATGACTGCTCACTA
TCAGTCTTACCCCGCCATCCTATCTGGTGGGAGTCCTTGCAACAGTATACTGACATCTAC
ACCTAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATCGAGAAG
TTCGAGAAGGTAGGTCACTTTTCCCTCCATTCCTGAGCCTTTCCCTACGCGATCGATTCG
CTTCACGTCCATCACCCCGCCCAACATCGATGATATTTTTTTTGAGTGGCTTTTTCACAT
TGTGGTGGGGGGTTCTTACCCCGCCGAACATGAGAAGCTGGCATTTTGCCCCACCACAAA
AATTTTCACATCCCTTCTCATGGCTCGTCACAAGCAATCAAGACATGATGCTAACAACCC
ACCAATAGGAAGCCGCCGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATCGCTCTCTGGAAGTTCGAGA
CTCCCCGCTACTATGTCACCGTCATTGGTAAGTTTTGATTGACTCATGCATGTCATCATCTATCAG
TCACTAACATTCATGATAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCATG

>Seq118 [organism=*Fusarium oxysporum*] [isolate A7-4] translation elongation factor 1 alpha (tef) gene, partial cds

CTCGGCCGTCGACTCTGGCAAGTCGACCACTGTGAGTACTCTCCTCGACAATGAGCTTAT
CTGCCATCGTCAATCCCGACCAAGACCTGGTGGGGTATTTCTCGAAGTCAACATACTGAC
ATCGTTTCACAGACCGGTCACTTGATCTACCAGTGCGGTGGTATCGACAAGCGAACCATC
GAGAAGTTCGAGAAGGTTAGTCACTTTCCCTTCGATCGCGCGTCCTTTGCCCATCGATTT
CCCCTACGACTCGAAATGTGCCTGCTACCCCGCTCGAGACCAAAAATTTTGCAATATGAC
CGTAATTTTTTTGGTGGGGCACTTACCCCGCCACTTGAGCGACGGGAGCGTTTGCCCTCT
TAACCATTCTCACAACCTCAATGAGTGCGTCGTCACGTGTCAAGCAGTCACTAACCATTC
AACAATAGGAAGCCGCTGAGCTCGGTAAGGGTTCCTTCAAGTACGCCTGGGTTCTTGACA
AGCTCAAGGCCGAGCGTGAGCGTGGTATCACCATCGATATTGCTCTCTGGAAGTTCGAGA
CTCCTCGCTACTATGTCACCGTCATTGGTATGTTGTCGCTCATGCTTCATTCTACTTCTCTTCGTA
CTAACATATCACTCAGACGCTCCCGGTCACCGTGATTTCATCAAGAACATGATCTGG

APPENDIX 5: Published Article

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Detection of human pathogenic *Fusarium* species in hospital and communal sink biofilms by using a highly specific monoclonal antibody

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Summary

The fungus *Fusarium* is well known as a plant pathogen, but has recently emerged as an opportunistic pathogen of humans. Habitats providing direct human exposure to infectious propagules are largely unknown, but there is growing evidence that plumbing systems are sources of human pathogenic strains in the *Fusarium solani* species complex (FSSC) and *Fusarium oxysporum* species complex (FOSC), the most common groups infecting humans. Here, a newly developed *Fusarium*-specific monoclonal antibody (mAb ED7) was used to track FSSC and FOSC strains in sink drain biofilms by detecting its target antigen, an extracellular 200 kDa carbohydrate, in saline swabs. The antigen was detectable in 52% of swab samples collected from sinks across a University campus and a tertiary care hospital. The mAb was 100% accurate in detecting FSSC, FOSC, and *F. dimerum* species complex (FDSC) strains that were present, as mixed fungal communities, in 83% of sink drain biofilms. Specificity of the ELISA was confirmed by sequencing of the internally transcribed spacer 1 (ITS1)-5.8S-ITS2 rRNA-encoding regions of culturable yeasts and moulds that were recovered using mycological culture, while transla-tion elongation factor (TEF)-1α analysis of *Fusarium* isolates included FSSC 1-a, FOSC 33, and FDSC ET-gr, the most common clinical patho-types in each group.

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Introduction

Species in the fungal genus *Fusarium* are ubiquitous environmental moulds, and pathogens of both plants and animals (Zhang et al., 2006; Thornton and Wills, 2015). In immunocompromised humans, such as patients with hematological malignancies and hematopoietic stem cell and solid organ transplant recipients, *Fusarium* species are significant emerging pathogens, causing a frequently fatal disseminated disease known as fusariosis with an associated mortality rate of 50%–75% (Boutati and Anaissie, 1997; Girmenia et al., 2000; Musa et al., 2000; Dignani and Anaissie, 2004; Jensen et al., 2004; Nucci and Anaissie, 2007). In some tertiary cancer cen-ters, *Fusarium* has emerged as the second most common mould pathogen after *Aspergillus* (Walsh and Groll, 1999; Muhammed et al., 2011). Regardless of human immune status, *Fusarium* species can cause localized nail infections (onychomycosis) (Arrese et al., 1996), bone and joint infections (Koehler et al., 2014), infections of burn wounds (Latenser, 2003), skin infec-tions (Nucci and Anaissie, 2002; Gurusidappa and Mamatha, 2011) and are the most frequent cause of mycotic eye infections known as fungal keratitis (Jurkunas et al., 2009), leading to progressive corneal destruction and endophthalmitis, with loss of vision or even loss of the affected eye (Dursun et al., 2003; Edelstein et al., 2012).

A recent multistate outbreak of fungal keratitis in the United States and in Singapore and Hong Kong was associated with contact lens solution contaminated with multiple strains of *Fusarium* and which led to visual loss in many patients and the need for corneal transplantation (Chang et al., 2006). While such outbreaks are rare, disseminated *Fusarium* infections and keratomycoses have increased in frequency over the past decade (Koehler et al., 2014) and an increasing body of evidence suggests that the main environmental sources of human pathogenic *Fusarium* species are contaminated water systems (Doggett, 2000; Anaissie et al., 2002; Mehl and Epstein, 2008; Anaissie et al., 2011). A number of studies have recovered pathogenic

Fusarium species from plumbing fixtures and it is hypothesized that microbial biofilms on fixtures may serve as important reservoirs of infectious *Fusarium* propagules in hospitals and homes (Mehl and Epstein, 2008; Short et al., 2011).

Identification of environmental reservoirs of human pathogenic moulds including *Fusarium* has typically relied on nucleic acid-based technologies following recovery of fungi using selective media (Anaissie et al., 2002; Mehl and Epstein, 2008; Short et al., 2011; Anaissie et al., 2011; Rougeron et al., 2014). Recently, highly specific monoclo-nal antibodies (mAb) have been used to identify pathogenic species or species complexes in environmental samples containing mixed populations of yeasts and moulds (Thornton, 2009; Davies and Thornton, 2014; Thornton et al., 2015). While mAb-based approaches similarly rely on culture for recovery of fungi from environmental samples, detection of diagnostic antigens in crude culture extracts using genus- or species-specific mAbs offers an attractive alternative approach to pathogen detection, particularly when combined with unsophisticated diagnostic modalities such as lateral-flow technology (Thornton, 2008, 2012).

In this study, we set out to determine whether a newly developed *Fusarium*-specific mAb (ED7) could be used to track the fungus by detecting a water-soluble diagnostic antigen in swabs of communal and hospital sinks. By using the mAb in an enzyme-linked immunsorbent assay (ELISA), we show that it can differentiate *Fusarium* spe-cies from other unrelated yeast and mould pathogens of humans present in mixed fungal communities encountered in sink biofilms. The ELISA represents a simple method for specific detection of *Fusarium* species in environmental reservoirs and for identifying plumbing systems contami-nated with the fungus.

Results

Production of hybridoma cell lines, isotyping of mAb and specificity

A single fusion was performed and 389 hybridoma cell lines were screened for specificity against a range of clini-cally relevant yeasts and moulds (Supporting Information Table S1). The aim was to identify cell lines secreting mAbs specific to *Fusarium* that could be used to track the fungus in environmental samples containing mixed species of human pathogenic fungi. To this end, a single cell line, ED7, was identified that produced mAb belonging to the immunoglobulin class M (IgM), which was genus-specific, reacting in ELISA tests with antigens from *Fusarium* species and with the *F. solani* teleomorph *Haematonectria haematococca* only (Fig. 1A and B). It did not cross-react with antigens from a wide range of unrelated mould and yeast species (Fig. 1A).

Western blotting of the ED7 antigen and epitope characterization

Gel electrophoresis and Western blotting studies showed that ED7 binds to a major antigen with molecular weight of 200 kDa which is secreted extracellularly by both *F. solani* and *F. oxysporum* (Fig. 2A). *Fusarium solani* antigens were subjected to enzymatic (Fig. 2B and C), heat (Fig. 2D) and chemical (Fig. 2E) modifications in order to char-acterize the epitope bound by ED7. Reductions in mAb binding following treatment with pronase shows that its epi-tope consists of protein, while reductions with trypsin indicate a protein epitope containing positively charged lysine and arginine side chains. The lack of reduction in ED7 binding following digestion of immobilized antigen with trypsin (Fig. 2B) and pronase (Fig. 2C) shows that it does not bind to a protein epitope. Reductions in mAb binding following heat treatment shows that an epitope is heat labile. There was no significant reduction in ED7 bind-ing over 70 min of heating, showing that its epitope is heat stable (Fig. 2D). Reductions in mAb binding following chemical digestion of an antigen with periodate shows that its epitope is carbohydrate and contains vicinal hydroxyl groups. The pronounced reductions in ED7 binding follow-ing periodate oxidation shows that its epitope consists of carbohydrate residues (Fig. 2E). Taken together, these results indicate that ED7 binds to an extracellular antigen and that its epitope is a heat stable carbohydrate moeity containing vicinal hydroxyl groups.

Immunofluorescence and immunogold electron microscopy

Immuno-localization studies using IF showed that the ED7 antigen was present on the surface of spores and hyphae (Fig. 2F–I), while IEM showed that the antigen was present in the spore and hyphal cell wall and in an extracellular fibrillar matrix surrounding both (Fig. 2J–L). In the TEM image shown in Fig. 2J, 56% of gold particles were distrib-uted in the fibrillar matrix surrounding the cell, while 40% and 4% of gold particles were distributed in the cell wall and cytoplasm respectively. This shows that the ED7 anti-gen is predominantly extracellular or located within the cell wall.

Immunodetection of *Fusarium* species in sink swabs and identification of fungi by analysis of the ITS regions of the rRNA-encoding gene unit and by translation elongation factor-1α PCR

Monoclonal antibody ED7 was highly specific for the three human pathogenic species of Fusarium, *F. solani*, *F. oxy-sporum* and *F. dimerum*, which were culturable from 75% of the sink swabs (Table 1 and Supporting Information Table S2). ELISA tests of the saline sink swabs showed

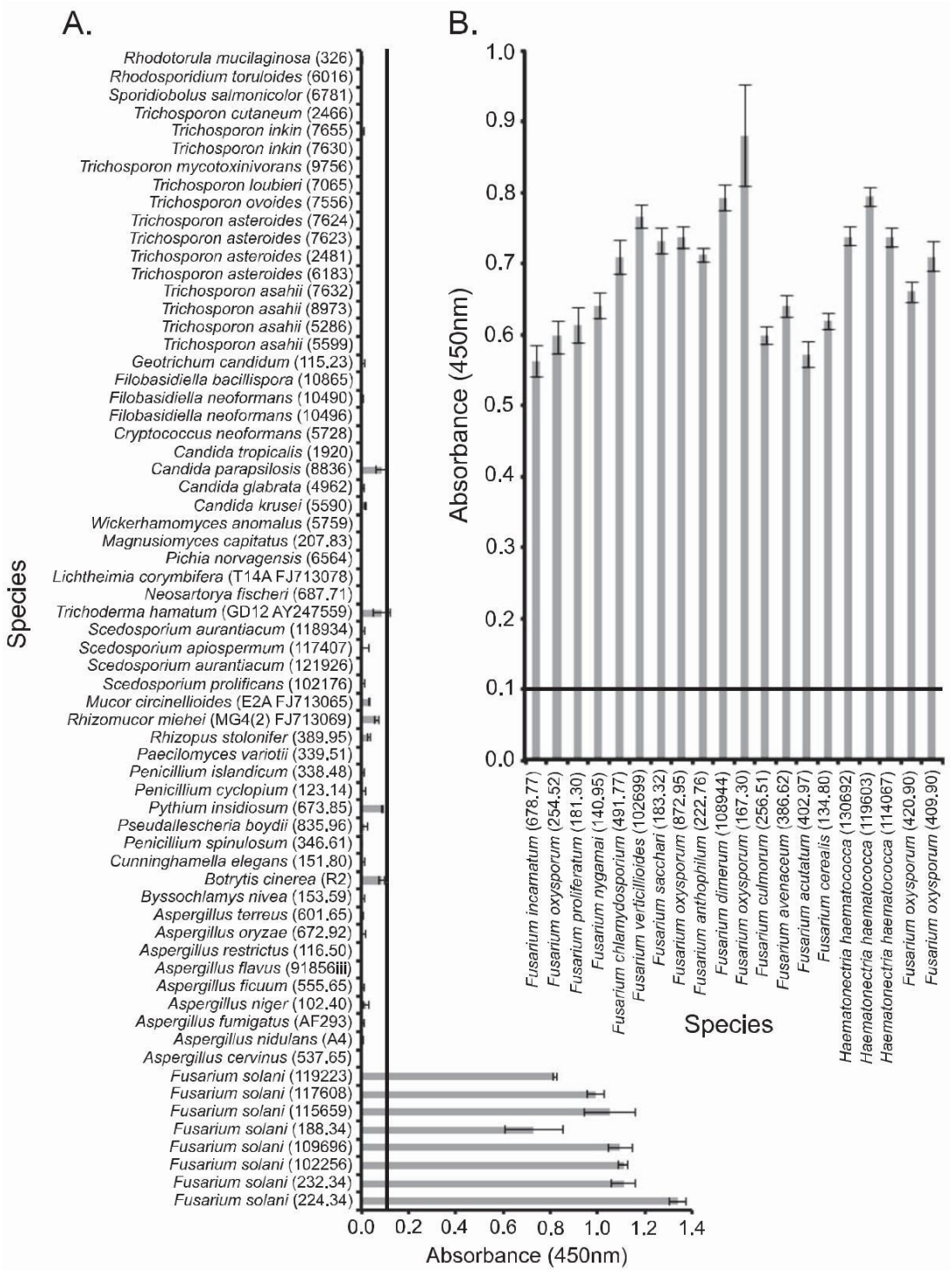
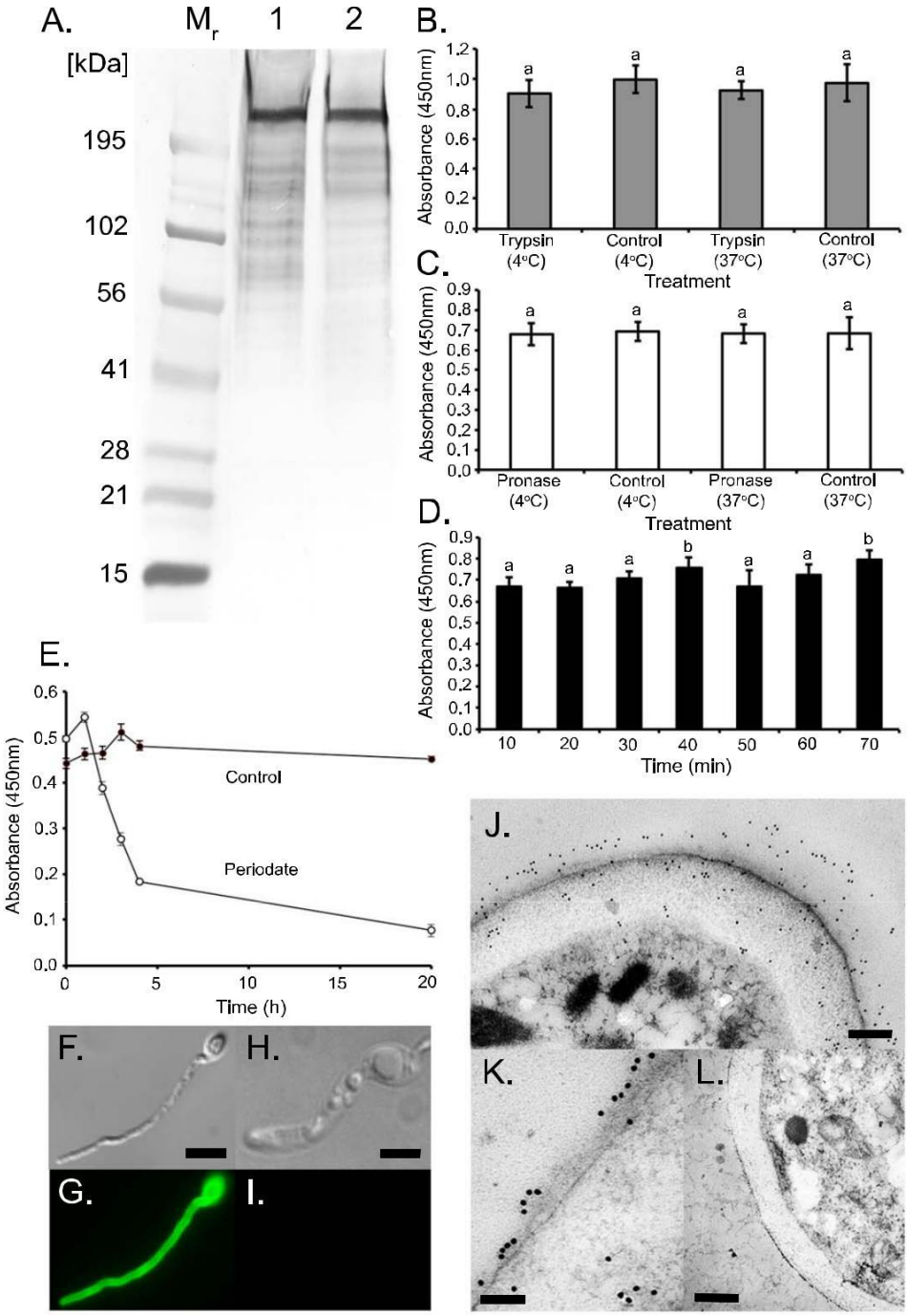


Fig. 1. Specificity of ED7 determined by ELISA tests of surface washings containing water-soluble antigens from *Fusarium* species and related and unrelated yeasts and moulds. (A) ELISA absorbance values at 450 nm for antigens from *F. solani* and unrelated yeasts and moulds and (B) for antigens from the *F. solani* teleomorph Haematonectria haematococca and related *Fusarium* species. Wells were coated with 60 lg protein ml^{21} buffer. Bars are the means of three biological replicates 6 standard errors and the threshold absorbance value for detection of antigen in ELISA is 0.100 (indicated by lines on graphs). Numbers in parentheses after species names denote strain numbers with further details of strains provided in Supporting Information Table S1.



that 52% contained detectable levels of *Fusarium* antigen (Table 1 and Supporting Information Table S2), with ELISA absorbance values in the range 0.100 (the threshold value for antigen detection) and up to 1.500. In four hospi-tal samples (samples S47, S48 and S49 from ophthalmology and sample S64 from oncology) *Fusarium* strains could not be recovered for identification by ITS sequencing despite detection of the diagnostic antigen in swab samples with absorbance values of 0.264, 0.530, 0.187 and 0.193 respectively (Table 1). This was likely due to the *Fusarium* isolates being outgrown in the mixed cul-ture plates by faster growing or more abundant unrelated fungi. Importantly, ED7 was shown not to cross-react with unrelated fungi (axenic culture absorbance values of 0.100 in all cases) including the human pathogenic yeast or yeast-like fungi *Candida*, *Exophiala*, *Meyerozyma*, *Rhodotorula*, *Trichosporon*, the human pathogenic hyaline or dematiaceous moulds *Aspergillus*, *Phialophora*, *Phoma*,

Fig. 2. Characterization of the ED7 antigen and its epitope and spatial distribution of the antigen in spores and hyphae.

(A) Western immunoblot with ED7 using culture fluid from 2-day-old PDB cultures of F. solani (CBS224.34) (lane 1) and F. oxysporum f.sp. lycopersici CBS167.30 (lane 2). Wells were loaded with 1.6 lg of protein. M_r denotes molecular weight in kDa. Note the major extracellular antigen with molecular weight of approximately 200 kDa.

(B) Absorbance values from ELISA tests with ED7 using surface washings containing water-soluble antigens immobilized to the wells of microtitre plates and treated with trypsin or PBS only (control) at 4°C and 378C. Bars are the means of three biological replicates 6 standard errors and bars with the same letter are not significantly different at $p < 0.001$ (ANOVA and Tukey–Kramer test).

(C) Absorbance values from ELISA tests with ED7 using surface washings containing water-soluble antigens immobilized to the wells of microtitre plates and treated with pronase or Milli-Q H_2O only (control) at 4°C and 378C. Bars are the means of three biological replicates 6 standard errors and bars with the same letter are not significantly different at $p < 0.001$ (ANOVA and Tukey–Kramer test).

(D) Stability of the water-soluble ED7 antigen following heating of surface washings at 1008C over a 70-minute period. Treated antigen was subsequently immobilized to the wells of microtitre plates and assayed by ELISA. Bars are the means of three biological replicates 6 standard errors and bars with the same letter are not significantly different at $p < 0.001$ (ANOVA and Tukey–Kramer test).

(E) Absorbance values from ELISA tests with ED7 using surface washings containing water-soluble antigens immobilized to the wells of microtitre plates and treated with periodate (open circles) or with acetate only control (closed circles) at 4°C over a 20-hour period. Each point is the mean of three biological replicates 6 standard errors.

(F to I) Photomicrographs of F. solani CBS224.34 immunostained with ED7 or TCM control and goat anti-mouse polyvalent Ig fluorescein isothiocyanate (FITC) conjugate. (F) Brightfield image of germinated conidium with hypha probed with ED7 followed by fluorochrome conjugate (scale bar 5 6 lm); (G) Same field of view as panel F but examined under epifluorescence. Note intense staining of the cell wall of microconidium and hypha; (H) Brightfield image of germinated conidium with hypha probed with TCM (negative control) followed by FITC conjugate (scale bar 5 3 lm); (I) Same field of view as panel H but examined under epifluorescence. Note lack of staining, demonstrating specific binding of ED7 to surface antigen.

(J to L) Immunogold labeling of sections of conidia and hyphae of *F. solani* CBS224.34. (J) Transverse section of conidium incubated with ED7 and anti-mouse immunoglobulin 20 nm gold particles, showing antigen in the cell wall and in an extracellular fimbrial matrix surrounding the spore (scale bar 5 250 nm); (K) Longitudinal section of hypha incubated with ED7 and anti-mouse immunoglobulin 20 nm gold particles, showing antigen in the cell wall and in an extracellular matrix surrounding the cell (scale bar 5 100 nm); (L) Transverse section of a conidium incubated with TCM (negative control) and anti-mouse immunoglobulin 20 nm gold particles, showing lack of staining by the secondary gold conjugate (scale bar 5 180 nm).

Trichoderma and the human pathogenic mucormycete *Mucor* (Table 1). The remaining 93% of samples positive for *Fusarium* antigen, either at the swab stage or following periods of biological amplification in mixed or axenic cultures (Supporting Information Table S2), yielded strains of the three *Fusarium* species. There was 100% concordance between *Fusarium* genus identification by ELISA and species identification by ITS sequencing (Table 1). The species of *F. solani* and *F. oxysporum* recovered from sink swabs were subsequently shown by TEF-1α PCR (Supporting Information Data Set 1) to belong to *F. solani* species complex (FSSC) 1-a, 1-c, 2-a, 2-v, 5-d, 5-k, 9-a, 15-a, 20-d and *F. oxysporum* (FOSC) species complexes 16, 33, 99, 111, 126, 134, 183 (Supporting Information Table S3). All of the recovered *F. dimerum* isolates belonged to the *F. dimerum* species complex (FDSC) ET-gr (Supporting Information Table S3). ITS analysis of axenic cultures (Table 1) showed that a number of sink samples (e.g., S2, S6, S17, S19, S21, S24, S30, S38) contained mixtures of *Fusarium* species, while ITS and TEF-PCR analysis (Table 1 and Supporting Information Table S3) showed that others contained mixtures of species complexes of the same species (e.g., S8, S9, S25, S28). Monoclonal antibody ED7 was able to detect all of the *Fusarium* species complexes recovered in this study.

In addition to drain swabs, water samples were collected from the taps of sinks in the hospital haematology and oncology units and from the main water tanks feeding the ophthalmology unit. The ED7 diagnostic antigen could not be detected in any of the water samples directly and, while all of the samples yielded fungi, only two of the tap samples (oncology W57 and W60) contained *Fusarium* strains that belonged to FDSC ET-gr. and which were detectable by ELISA at the mixed culture stage (Table 1). The sink biofilms corresponding to these water samples were also positive at the swab ELISA stage (Table 1).

Discussion

The genus *Fusarium* comprises ubiquitous environmental moulds capable of infecting plants and humans (Zhang et al., 2006). Unlike agriculture, where the most economically damaging pathogens are considered to be

F. graminearum and *F. oxysporum* (Dean et al., 2012), the species most commonly cited as human pathogens belong to the *Fusarium solani* species complex (FSSC), responsible for 50% of reported infections in humans, followed by strains in the FOSC (Torres and Kontoyiannis, 2011). The FDSC is less frequently reported as causing human dis-ease, but it is similarly capable of causing disseminated infections in immunocompromised patients (Bigley et al., 2004; Schroers et al., 2009).

While the natural habitats of plant pathogenic *Fusarium* strains are well characterized as soil and decaying plant material, habitats providing direct human exposure to infectious propagules are largely unexplored. The increasing frequency of opportunistic fungal infections in human means that improved surveillance methods are needed to identify environmental reservoirs of pathogens to limit the exposure of vulnerable individuals to potentially infective propagules. For *Fusarium*, there is a growing body of evidence to suggest that domestic and municipal water

Table 1. Locations and identities of sink swabs and water samples and results of ELISA tests and fungal identification based on ITS sequencing.

Sink ID	Location (Source)[a]	Swab ELISA abs (450 nm)[b]	Mixed culture ELISA abs (450 nm)[b]	Isolate no.	Axenic culture ELISA abs (450 nm)[b]	Identification based on ITS sequencing[c]	GenBank accession no.
A1	University (S1)	0.0763	0.0575	A1-1	1.3736	*Fusarium oxysporum*	KT876668
	University (S1)			A1-2	0.7646	*Fusarium oxysporum*	KT876662
	University (S1)			A1-3	0.0196	*Penicillium crustosum*	KT876719
	University (S1)			A1-4	0.0139	*Penicillium expansum*	KT876718
A2	University (S2)	0.5472	0.0045	A2-1	1.5723	*Fusarium solani*	KT876635
	University (S2)			A2-3	0.6554	*Fusarium oxysporum*	KT876690
	University (S2)			A2-5	1.1312	*Fusarium solani*	KT876631
A3	University (S3)	0.0053	0.0077	A3-1	0.0082	*Trichosporon domesticum*	KT876717
	University (S3)			A3-2	0.0162	*Cadophora fastigiata*	KT876615
	University (S3)			A3-3	0.0014	*Cyphellophora oxyspora*	KT876613
	University (S3)			A3-4	0.0093	*Penicillium crustosum*	KT876714
A4	University (S4)	0.0297	0.0049	A4-1	0.0105	*Phoma herbarum*	KT876697
	University (S4)			A4-2	0.0079	*Penicillium echinulatum*	KT876710
	University (S4)			A4-3	0.0029	*Cytobasidium slooffiae*	KT876704
	University (S4)			A4-6	0.0060	*Trichoderma asperellum*	KT876620
A5	University (S5)	0.0206	0.0039	A5-1	1.1815	*Fusarium oxysporum*	KT876692
	University (S5)			A5-2	0.0034	*Penicillium crustosum*	KT876715
	University (S5)			A5-5	0.0088	*Aspergillus niger*	KT876702
	University (S5)			A5-6	1.0630	*Fusarium oxysporum*	KT876667
	University (S5)			A5-7	0.0037	*Rhodotorula mucilaginosa*	KT876700
	University (S5)			A5-8	0.0088	*Cyphellophora oxyspora*	KT876614
A6	University (S6)	0.0251	0.0412	A6-1	1.1130	*Fusarium oxysporum*	KT876648
	University (S6)			A6-2	0.9410	*Fusarium oxysporum*	KT876678
	University (S6)			A6-3	0.6377	*Fusarium oxysporum*	KT876688
	University (S6)			A6-4	1.0020	*Fusarium solani*	KT876640
A7	University (S7)	0.1716	0.0082	A7-1	0.0098	*Exophiala pisciphila*	KT876618
	University (S7)			A7-2	0.0096	*Penicillium brevicompactum*	KT876695
	University (S7)			A7-3	1.0556	*Fusarium oxysporum*	KT876684
	University (S7)			A7-4	1.0077	*Fusarium oxysporum*	KT876671
A8	University (S8)	0.0431	0.0008	A8-1	1.1045	*Fusarium oxysporum*	KT876672
	University (S8)			A8-2	0.9707	*Fusarium oxysporum*	KT876654
	University (S8)			A8-3	0.0091	*Trichoderma atroviride*	KT876622
A9	University (S9)	0.3806	0.0052	A9-1	0.9087	*Fusarium solani*	KT876639
	University (S9)			A9-2	0.8849	*Fusarium solani*	KT876638
	University (S9)			A9-3	0.9004	*Fusarium solani*	KT876632
	University (S9)			A9-4	0.8093	*Fusarium solani*	KT876636
A10	University (S10)	0.0034	0.0028	A10-1	0.0188	*Rhodotorula slooffiae*	KT876705
	University (S10)			A10-2	0.0020	*Exophiala pisciphila*	KT876616
B1	University (S11)	0.0231	0.0005	B1-1	0.6064	*Fusarium dimerum*	KT876625
	University (S11)			B1-6	0.8138	*Fusarium dimerum*	KT876628
B2	University (S12)	0.0200	0.0020	B2-1	0.5678	*Fusarium dimerum*	KT876626
	University (S12)			B2-5	0.4827	*Fusarium dimerum*	KT876624
B3	University (S13)	0.0091	0.0023	B3-4	0.0026	*Phoma herbarum*	KT876696
B4	University (S14)	0.0163	0.0008	B4-1	0.6992	*Fusarium dimerum*	KT876627
B5	University (S15)	0.0132	0.0003	B5-1	0.8008	*Fusarium oxysporum*	KT876674
	University (S15)			B5-2	0.0048	*Mucor circinelloides*	KT876701
	University (S15)			B5-3	0.8851	*Fusarium oxysporum*	KT876677
B6	University (S16)	0.0229	0.0002	B6-1	0.8193	*Fusarium oxysporum*	KT876676
	University (S16)			B6-2	0.7582	*Fusarium oxysporum*	KT876661
B7	University (S17)	0.0395	0.0014	B7-1	0.8201	*Fusarium oxysporum*	KT876675
	University (S17)			B7-6	0.7758	*Fusarium dimerum*	KT876623
B8	University (S18)	0.0133	0.0011	B8-1	0.7347	*Fusarium solani*	KT876637
	University (S18)			B8-7	0.0095	*Rhodosporidium babjevae*	KT876706
B9	University (S19)	0.0212	0.0048	B9-1	0.0051	*Meyerozyma guilliermondii*	KT876707
	University (S19)			B9-2	0.8104	*Fusarium oxysporum*	KT876657

Table 1. cont.

Sink ID	Location (Source)[a]	Swab ELISA abs (450 nm)[b]	Mixed culture ELISA abs (450 nm)[b]	Isolate no.	Axenic culture ELISA abs (450 nm)[b]	Identification based on ITS sequencing[c]	GenBank accession no.
	University (S19)			B9-3	0.6302	*Fusarium solani*	KT876634
	University (S19)			B9-5	0.0017	*Penicillium crustosum*	KT876720
B10	University (S20)	0.5467	0.0394	B10-1	0.7467	*Fusarium oxysporum*	KT876680
	University (S20)			B10-2	0.7667	*Fusarium oxysporum*	KT876659
	University (S20)			B10-6	0.0071	*Candida parapsilosis*	KT876703
	University (S20)			B10-7	0.0251	*Meyerozyma guilliermondii*	KT876711
	University (S20)			B10-9	0.0031	*Cystobasidium slooffiae*	KT876712
	University (S20)			B10-12	0.0251	*Trichosporon asteroides*	KT876713
C1	University (S21)	0.0083	0.0269	C1-1	0.0044	*Clavispora lusitaniae*	KT876708
	University (S21)			C1-3	0.8875	*Fusarium oxysporum*	KT876682
	University (S21)			C1-4	0.8884	*Fusarium dimerum*	KT876629
	University (S21)			C1-7	0.0003	*Exophiala pisciphila*	KT876617
C2	University (S22)	0.2977	0.0047	C2-1	0.9443	*Fusarium oxysporum*	KT876658
	University (S22)			C2-4	0.9825	*Fusarium oxysporum*	KT876694
	University (S22)			C2-7	0.8677	*Fusarium oxysporum*	KT876687
C3	University (S23)	0.0808	0.0116	C3-1	0.9853	*Fusarium oxysporum*	KT876683
	University (S23)			C3-2	1.0160	*Fusarium oxysporum*	KT876693
	University (S23)			C3-4	0.7485	*Fusarium oxysporum*	KT876644
C4	University (S24)	0.1113	0.0202	C4-1	0.8930	*Fusarium oxysporum*	KT876670
	University (S24)			C4-2	0.0028	*Candida intermedia*	KT876709
	University (S24)			C4-5	0.9008	*Fusarium dimerum*	KT876630
C5	University (S25)	0.5741	0.0097	C5-1	0.9874	*Fusarium oxysporum*	KT876652
	University (S25)			C5-2	1.0996	*Fusarium oxysporum*	KT876656
	University (S25)			C5-3	1.1236	*Fusarium oxysporum*	KT876649
	University (S25)			C5-4	1.0355	*Fusarium oxysporum*	KT876669
	University (S25)			C5-5	1.0672	*Fusarium oxysporum*	KT876660
C6	University (S26)	0.0603	0.0348	C6-1	1.0135	*Fusarium oxysporum*	KT876665
	University (S26)			C6-2	0.9362	*Fusarium oxysporum*	KT876681
	University (S26)			C6-3	0.8898	*Fusarium oxysporum*	KT876673
	University (S26)			C6-4	1.0577	*Fusarium oxysporum*	KT876686
	University (S26)			C6-5	0.9445	*Fusarium oxysporum*	KT876646
C7	University (S27)	0.1279	0.0113	C7-1	0.9724	*Fusarium oxysporum*	KT876666
C8	University (S28)	0.0607	0.0035	C8-1	0.8694	*Fusarium oxysporum*	KT876685
	University (S28)			C8-2	0.9599	*Fusarium oxysporum*	KT876655
	University (S28)			C8-3	0.0031	*Penicillium expansum*	KT876716
	University (S28)			C8-4	0.0049	*Phoma herbarum*	KT876698
	University (S28)			C8-6	0.9003	*Fusarium oxysporum*	KT876679
C9	University (S29)	0.0310	0.0001	C9-1	1.0488	*Fusarium oxysporum*	KT876651
	University (S29)			C9-2	0.9254	*Fusarium oxysporum*	KT876645
	University (S29)			C9-3	0.9711	*Fusarium oxysporum*	KT876664
	University (S29)			C9-4	1.035	*Fusarium oxysporum*	KT876647
	University (S29)			C9-5	0.9604	*Fusarium oxysporum*	KT876663
C10	University (S30)	0.1002	0.0333	C10-1	0.9254	*Fusarium oxysporum*	KT876689
	University (S30)			C10-2	1.0198	*Fusarium solani*	KT876642
	University (S30)			C10-4	0.9007	*Fusarium solani*	KT876641
	University (S30)			C10-7	1.0697	*Fusarium solani*	KT876643
CRT1	University (S31)	0.6175	0.1129	CRT1-1	0.4639	*Fusarium oxysporum*	KT876691
	University (S31)			CRT1-2	0.9086	*Fusarium oxysporum*	KT876650
	University (S31)			CRT1-3	0.7503	*Fusarium oxysporum*	KT876652
CRT2	University (S32)	0.0303	0.0021	CRT2-1	0.0040	*Trichoderma asperellum*	KT876619
	University (S32)			CRT2-2	0.0417	*Trichoderma asperellum*	KT876621
CRT3	University (S33)	0.0663	0.0028	CRT3-1	0.0256	*Phoma herbarum*	KT876699
R1	ICU (S34)	0.0285	0.3620	R1-1	1.5057	*Fusarium solani*	KT876550
	ICU (S34)			R1-2	1.5154	*Fusarium solani*	KT876551
	ICU (S34)			R1-3	1.5555	*Fusarium solani*	KT876549

Table 1. cont.

Sink ID	Location (Source)[a]	Swab ELISA abs (450 nm)[b]	Mixed culture ELISA abs (450 nm)[b]	Isolate no.	Axenic culture ELISA abs (450 nm)[b]	Identification based on ITS sequencing[c]	GenBank accession no.
R2	ICU (S35)	0.0071	0.0147	R2-4	0.0096	*Clonostachys rosea*	KT876552
	ICU (S35)			R2-5	0.0018	*Clonostachys rosea*	KT876553
	ICU (S35)			R2-6	0.0035	*Clonostachys rosea*	KT876554
R3	ICU (S36)	0.0391	0.045	R3-1	0.0006	*Trichoderma asperellum*	KT876548
R5	ITU (S37)	0.7291	0.7724	R5-1	1.5606	*Fusarium dimerum*	KT876561
	ITU (S37)			R5-2	1.3481	*Fusarium dimerum*	KT876565
	ITU (S37)			R5-3	1.4990	*Fusarium dimerum*	KT876567
R6	ITU (S38)	1.2086	0.3691	R6-1	1.3388	*Fusarium dimerum*	KT876572
	ITU (S38)			R6-2	1.4734	*Fusarium dimerum*	KT876563
	ITU (S38)			R6-3	1.4312	*Fusarium dimerum*	KT876562
	ITU (S38)			R6-9	1.2648	*Fusarium oxysporum*	KT876557
R7	ITU (S39)	0.1121	0.8186	R7-1	1.3266	*Fusarium dimerum*	KT876570
	ITU (S39)			R7-2	1.2352	*Fusarium dimerum*	KT876564
R8	ITU (S40)	1.0157	0.3612	R8-1	1.2399	*Fusarium dimerum*	KT876568
	ITU (S40)			R8-2	1.1552	*Fusarium dimerum*	KT876566
	ITU (S40)			R8-3	1.1856	*Fusarium dimerum*	KT876558
R9	ITU (S41)	0.0391	0.0477	R9-3	0.0113	*Exophiala phaeomuriformis*	KT876555
R10	ITU (S42)	0.0549	0.0097	R10-2	0.0072	*Cadophora fastigiata*	KT876556
	ITU (S42)			R10-6	0.0124	*Coniochaeta fasciculata*	KT876721
X2	ITU (S43)	0.0630	1.5751	X2-2	1.1492	*Fusarium dimerum*	KT876571
	ITU (S43)			X2-4	1.2505	*Fusarium dimerum*	KT876560
	ITU (S43)			X2-5	1.1397	*Fusarium dimerum*	KT876569
	ITU (S43)			X2-6	1.1716	*Fusarium dimerum*	KT876559
X3	Ophthalmology Unit (S44)	1.0313	1.5035	X3-1	1.1353	*Fusarium dimerum*	KT876509
	Ophthalmology Unit (S44)			X3-2	0.0224	*Candida parapsilosis*	KT876498
	Ophthalmology Unit (S44)			X3-3	1.1856	*Fusarium dimerum*	KT876512
X4	Ophthalmology Unit (S45)	1.4582	1.4049	X4-1	1.1322	*Fusarium dimerum*	KT876511
	Ophthalmology Unit (S45)			X4-2	1.1523	*Fusarium dimerum*	KT876500
	Ophthalmology Unit (S45)			X4-3	0.0622	*Candida parapsilosis*	KT876508
	Ophthalmology Unit (S45)			X4-4	0.1245	*Gloeotinia temulenta*	KT876515
X5	Ophthalmology Unit (S46)	0.3325	1.4466	X5-2	1.0654	*Fusarium dimerum*	KT876510
	Ophthalmology Unit (S46)			X5-3	0.0599	*Rhodotorula mucilaginosa*	KT876501
	Ophthalmology Unit (S46)			X5-4	1.0862	*Fusarium dimerum*	KT876513
X6	Ophthalmology Unit (S47)	0.2640	1.3729	X6-1	0.0616	*Candida parapsilosis*	KT876499
	Ophthalmology Unit (S47)			X6-2	0.063	*Engyodontium album*	KT876522
	Ophthalmology Unit (S47)			X6-3	0.0603	*Engyodontium album*	KT876521
	Ophthalmology Unit (S47)			X6-4	0.0657	*Exophiala phaeomuriformis*	KT876504
X8	Ophthalmology Unit (S48)	0.5299	0.0223	X8-2	0.0118	*Cladosporium macrocarpum*	KT876506
	Ophthalmology Unit (S48)			X8-3	0.0985	*Gloeotinia temulenta*	KT876514
	Ophthalmology Unit (S48)			X8-4	0.0662	*Engyodontium album*	KT876523
X9	Ophthalmology Unit (S49)	0.1872	0.0061	X9-1	0.0686	*Engyodontium album*	KT876520
	Ophthalmology Unit (S49)			X9-2	0.0611	*Candida parapsilosis*	KT876496
	Ophthalmology Unit (S49)			X9-3	0.0641	*Candida parapsilosis*	KT876497
	Ophthalmology Unit (S49)			X9-4	0.0677	*Exophiala dermatitidis*	KT876503
	Ophthalmology Unit (S49)			X9-5	0.0705	*Engyodontium album*	KT876519
	Ophthalmology Unit (S49)			X9-7	0.0660	*Exophiala pisciphila*	KT876502
X10	Ophthalmology Unit (T1)	0.0132	0.0159	X10-1	0.0868	*Cladosporium sphaerospermum*	KT876507
	Ophthalmology Unit (T1)			X10-3	0.0130	*Engyodontium album*	KT876518
W1	Ophthalmology Unit (T2)	0.0117	0.0883	W1-1	0.0114	*Paraconiothyrium fuckelii*	KT876505
	Ophthalmology Unit (T2)			W1-2	0.0139	*Engyodontium album*	KT876517
	Ophthalmology Unit (T2)			W1-3	0.0108	*Engyodontium album*	KT876516
K1	Haematology Unit (S50)	0.2694	0.0093	K1-1	0.0172	*Trichoderma asperellum*	KT876534
K2	Haematology Unit (S51)	0.7466	1.1746	K2-1	0.0144	*Trichoderma viride*	KT876533
	Haematology Unit (S51)			K2-3	1.1385	*Fusarium solani*	KT876543
	Haematology Unit (S51)			K2-4	1.2487	*Fusarium solani*	KT876542

Table 1. cont.

Sink ID	Location (Source)[a]	Swab ELISA abs (450 nm)[b]	Mixed culture ELISA abs (450 nm)[b]	Isolate no.	Axenic culture ELISA abs (450 nm)[b]	Identification based on ITS sequencing[c]	GenBank accession no.
K6	Haematology Unit (S52)	1.0621	1.3090	K6-1	1.1544	*Fusarium oxysporum*	KT876541
	Haematology Unit (S52)			K6-4	0.0071	*Candida parapsilosis*	KT876524
K8	Haematology Unit (S53)	1.0120	1.2628	K8-1	1.2376	*Fusarium solani*	KT876722
	Haematology Unit (S53)			K8-2	1.2983	*Fusarium solani*	KT876723
	Haematology Unit (S53)			K8-6	0.0084	*Cadophora fastigiata*	KT876531
	Haematology Unit (S53)			K8-7	0.0051	*Trichoderma asperellum*	KT876535
K9	Haematology Unit (S54)	0.9843	1.2903	K9-1	0.0046	*Candida parapsilosis*	KT876525
	Haematology Unit (S54)			K9-2	0.0090	*Rhodotorula dairenensis*	KT876526
	Haematology Unit (S54)			K9-4	0.0073	*Cadophora fastigiata*	KT876532
	Haematology Unit (S54)			K9-5	1.1652	*Fusarium solani*	KT876547
	Haematology Unit (S54)			K9-6	1.0316	*Fusarium solani*	KT876545
K10	Haematology Unit (S55)	0.8638	1.0753	K10-1	1.0684	*Fusarium solani*	KT876546
	Haematology Unit (S55)			K10-2	1.0386	*Fusarium solani*	KT876544
G1	Haematology Unit (W50)	0.0082	0.0451	G1-2	0.0097	*Exophiala pisciphila*	KT876529
G2	Haematology Unit (W51)	0.0581	0.0553	G2-1	0.0104	*Engyodontium album*	KT876540
	Haematology Unit (W51)			G2-3	0.0062	*Exophiala pisciphila*	KT876530
G6	Haematology Unit (W52)	0.0125	0.0118	G6-2	0.0161	*Exophiala castellanii*	KT876528
G8	Haematology Unit (W53)	0.0075	0.0219	G8-1	0.0065	*Engyodontium album*	KT876538
G9	Haematology Unit (W54)	0.0002	0.0164	G9-2	0.0005	*Gloeotinia temulenta*	KT876537
G10	Haematology Unit (W55)	0.0021	0.0182	G10-1	0.0051	*Engyodontium album*	KT876539
H1	Oncology Unit (S56)	0.4822	1.2287	H1-1	0.9022	*Fusarium dimerum*	KT876595
	Oncology Unit (S56)			H1-3	0.0151	*Pichia kudriavzevii*	KT876578
H2	Oncology Unit (S57)	1.0579	1.1650	H2-1	0.9312	*Fusarium dimerum*	KT876590
	Oncology Unit (S57)			H2-5	0.0061	*Magnusiomyces capitatus*	KT876611
H3	Oncology Unit (S58)	1.0938	1.2091	H3-1	0.8909	*Fusarium oxysporum*	KT876584
	Oncology Unit (S58)			H3-4	0.0075	*Candida palmioleophila*	KT876573
	Oncology Unit (S58)			H3-5	0.0140	*Rhodotorula glutinis*	KT876598
H4	Oncology Unit (S59)	0.0253	1.0856	H4-1	0.8884	*Fusarium dimerum*	KT876596
	Oncology Unit (S59)			H4-3	0.0004	*Magnusiomyces capitatus*	KT876612
H5	Oncology Unit (S60)	1.0856	1.0193	H5-1	1.1148	*Fusarium dimerum*	KT876593
	Oncology Unit (S60)			H5-3	0.005	*Candida tropicalis*	KT876574
H6	Oncology Unit (S61)	0.5256	1.1898	H6-1	1.1238	*Fusarium dimerum*	KT876594
H7	Oncology Unit (S62)	1.0789	1.1588	H7-1	1.1513	*Fusarium oxysporum*	KT876583
	Oncology Unit (S62)			H7-3	0.0045	*Candida tropicalis*	KT876575
H8	Oncology Unit (S63)	0.0953	0.0152	H8-1	0.0063	*Phoma herbarum*	KT876580
	Oncology Unit (S63)			H8-3	0.0126	*Candida albicans*	KT876577
	Oncology Unit (S63)			H8-4	0.0088	*Rhodotorula mucilaginosa*	KT876599
H9	Oncology Unit (S64)	0.1926	0.0162	H9-1	0.0152	*Exophiala dermatitidis*	KT876581
	Oncology Unit (S64)			H9-2	0.0049	*Candida orthopsilosis*	KT876576
	Oncology Unit (S64)			H9-3	0.0109	*Pichia kudriavzevii*	KT876579
H10	Oncology Unit (S65)	0.4961	1.1005	H10-1	0.9606	*Fusarium dimerum*	KT876589
	Oncology Unit (S65)			H10-4	0.0163	*Trametes ochracea*	KT876608
P1	Oncology Unit (W56)	0.0030	0.0247	P1-2	0.0082	*Exophiala dermatitidis*	KT876582
	Oncology Unit (W56)			P1-3	0.0024	*Trametes versicolor*	KT876603
P2	Oncology Unit (W57)	0.0061	1.0891	P2-1	0.8451	*Fusarium dimerum*	KT876587
	Oncology Unit (W57)			P2-2	0.9429	*Fusarium dimerum*	KT876597
	Oncology Unit (W57)			P2-3	0.8627	*Fusarium dimerum*	KT876588
P3	Oncology Unit (W58)	0.0016	0.0202	P3-1	0.0177	*Trametes ochracea*	KT876607
P4	Oncology Unit (W59)	0.0035	0.0125	P4-1	0.0164	*Beauveria bassiana*	KT876586
	Oncology Unit (W59)			P4-2	0.0084	*Gliomastix polychroma*	KT876584
P5	Oncology Unit (W60)	0.0025	1.1746	P5-1	0.8563	*Fusarium dimerum*	KT876591
	Oncology Unit (W60)			P5-2	0.8066	*Fusarium dimerum*	KT876592
	Oncology Unit (W60)			P5-3	0.0024	*Piptoporus betulinus*	KT876609
P7	Oncology Unit (W62)	0.0011	0.0417	P7-1	0.0097	*Trametes versicolor*	KT876605
P8	Oncology Unit (W63)	0.0044	0.0060	P8-1	0.0144	*Trametes versicolor*	KT876606
	Oncology Unit (W63)			P8-2	0.0106	*Stereum gausapatum*	KT876601

Table 1. cont.

Sink ID	Location (Source)[a]	Swab ELISA abs (450 nm)[b]	Mixed culture ELISA abs (450 nm)[b]	Isolate no.	Axenic culture ELISA abs (450 nm)[b]	Identification based on ITS sequencing[c]	GenBank accession no.
P9	Oncology Unit (W64)	0.0022	0.0163	P9-1	0.0101	*Stereum gausapatum*	KT876600
	Oncology Unit (W64)			P9-2	0.0068	*Trametes versicolor*	KT876604
	Oncology Unit (W64)			P9-3	0.0140	*Stereum gausapatum*	KT876602
P10	Oncology Unit (W65)	0.0051	0.0280	P10-1	0.0032	*Phaeophlebiopsis peniophoroides*	KT876610

a. S, sink; W, tap water from corresponding sink number; T, water sample from main tank.

b. Threshold absorbance value for detection in ELISA 0.100. Shading indicates earliest point in sampling process at which antigen was detectable in ELISA tests with ED7.

c. *Fusarium* strains in bold further characterized by TEF-1α PCR analysis (Table S3 and Supporting Information Data Set 1).

systems are potential reservoirs of human pathogenic strains in the FSSC, FOSC and FDSC groups (Short et al., 2011).

Accurate techniques that can be used to identify the fungus in environmental samples containing mixed populations of fungi are currently lacking and, while nucleic acid-based technologies have been developed for the differentiation of *Fusarium* from other human pathogenic species and to identify *Fusarium* species complexes, such techniques have typically been used in retrospective analysis of axenic cultures collected during human and environmental population studies (Bouchara et al., 2009; Steinmann et al., 2011; Lackner et al., 2012). Furthermore, these studies have often employed *Fusarium*-selective media that eliminate other fungi present in polymicrobial communities (Short et al., 2011). While monoclonal anti-bodies (mAbs) and antibody fragments have been developed for detecting and differentiating *Fusarium* species in vitro or in planta (Wong et al., 1988; Arie et al., 1991, 1995; Danks et al., 1996; Hayashi et al., 1998; Hu et al., 2012, 2013), no attempts have been made to use mAbs to track human pathogenic strains in environmental samples. Jensen et al. (2011) recently reported the development of *Fusarium*-specific mAbs for immunohistochemical diagnosis of fusariosis. The IgM mAbs, which recognize 51 and 63 kDa antigens, reacted strongly with fungal elements in both experimentally infected animals and biopsy samples from patients with fusariosis sepsis and dissemination to the skin.

In this prospective study, we set out to determine whether human pathogenic species of *Fusarium* could be identified in sink drains directly by using crude antigen extracts of biofilms and detection using a genus-specific immunoglobulin M (IgM) mAb, ED7, that binds to an extracellular approximately 200kDa carbohydrate antigen present on the surface of spores and hyphae. While the function of the antigen is currently unknown we were able, in Enzyme-Linked Immunosorbent Assay (ELISA) tests, to detect its presence in 52% of swab samples and, following biological amplification of biofilms on a non-selective mycological medium, were able to identify additional biofilm samples containing pathogenic strains of *Fusarium*. This is the first time, to our knowledge, that a mAb-based detec-tion method has been used to track *Fusarium* in environmental samples. The mAb was able to differentiate *Fusarium* from a wide spectrum of unrelated fungi, including the human pathogens *Aspergillus* (Thornton and Wills, 2015), *Candida*, *Geotrichum*, *Rhodotorula* and *Trichosporon* (Miceli et al., 2011; Davies and Thornton, 2014), *Cyphellophora* and *Phialophora* (Feng et al., 2014), *Exophiala* (Zeng et al., 2007), *Trichoderma* (Sandoval-Denis et al., 2014), *Engyodontium* (Macedo^ et al., 2007; Thamke et al., 2015) and *Mucor* (Petrikkos et al., 2012), several of which have been reported previously to inhabit biofilms in water distribution systems (Dogget, 2000). The 100% accuracy of the ED7 ELISA, confirmed by using ITS sequencing and TEF PCR analysis of recovered isolates, demonstrates its robustness in detecting potentially infec-tious *Fusarium* species in polymicrobial communities. Importantly, ED7 reacted with all of the species complex strains isolated including the most common clinical patho-types of *Fusarium*, FSSC 1-a, FOSC 33 and FDSC ET-gr (Schroers et al., 2009; Short et al., 2011).

While the ED7 ELISA was able to identify *Fusarium* to the level of genus only, the simplicity of the mAb-based approach to detection, even when combined with a standard mycological isolation procedure, means that a recognized environmental niche of this group of patho-genic fungi can be monitored readily. The widespread occurrence of human pathogenic *Fusarium* species in sinks of a tertiary care hospital and sinks of a heavily popu-lated university campus, show that indoor plumbing-associated biofilms and water sources are an unseen source of *Fusarium* infectious propagules for nosocomial and community-acquired infections of vulnerable individu-als, an observation consistent with previous studies (Annaisie et al., 2011; Short et al., 2011). While no cases of fusariosis were reported during the course of this study, the close proximity of the patients to hospital sinks colon-ized with both pathogenic fusaria and with other opportunistic fungal pathogens is a serious concern given the known vulnerability of immunocompromised individuals to invasive fungal infections.

Experimental procedures

Ethics statement

All animal work described in this study was conducted under a UK Home Office Project License, and was reviewed by the institution's Animal Welfare Ethical Review Board (AWERB) for approval. The work was carried out in accordance with The Animals (Scientific Procedures) Act 1986 Directive 2010/63/ EU, and followed all the Codes of Practice which reinforce this law, including all elements of housing, care, and euthanasia of the animals. Permission for sink sampling at the Royal Devon and Exeter Hospital was granted by the Director of Infection Prevention and Control.

Fungal culture

Fungi (Supporting Information Table S1) were routinely cultured on Potato Dextrose Agar (PDA: 70139; Sigma), Sabouraud Dextrose Agar (SDA: Sabouraud Dextrose Broth (SDB: S3306; Sigma) containing 2% (w/v) agar), Malt Yeast extract Agar (MYA: Y3127; Sigma), or Oatmeal Agar (OA: O3506; Sigma), sterilized by autoclaving at 121°C for 15 minutes. Cultures were grown at 26°C under a 16 hours fluo-rescent light regime.

Development of mAb, preparation of immunogen and immunization regime

BALB/c mice were immunized with soluble antigens prepared from lyophilized mycelium of a human pathogenic strain of

Fusarium solani species complex 1-a (CBS strain 224.34). Conidia were suspended in water after 10-day-old PDA slant cultures were flooded with 5 ml dH_2O and gently agitated with an inoculation loop. Conidial suspensions were then filtered through Miracloth to remove mycelium and transferred to 1.5 ml micro-centrifuge tubes. The conidia were washed three times with dH_2O by repeated vortexing and centrifugation at 14 462g for 5 min and finally suspended in dH_2O to give a concentration of 10^6 conidia ml^{-1} solution. Flasks containing 100 ml of sterilized Potato Dextrose Broth (PDB: P6685; Sigma) were inoculated with 200 µl of the conidial suspension and incubated with shaking (75 rpm) for 48 h at 26°C. Hyphal biomass was collected on Miracloth, snap frozen in liquid N_2, and lyophilized. Culture filtrates were retained for gel electrophoresis and Western blotting studies and stored at 22°C until required. About 1 mg of lyophilized biomass was sus-pended in 1 ml phosphate buffered saline (PBS: 0.8% NaCl; 0.02% KCl; 0.115% Na_2HPO_4; 0.02% KH_2PO_4; pH7.2) and the resultant suspension centrifuged for 5 min at 14 462g. The supernatant, containing solubilized antigens, was used as the immunogen and as a source of antigens for hybridoma screening assays. For immunization, 6-week-old BALB/c female white mice were given four intraperitoneal injections (300 µl per injection) of antigen extract containing 2.3 mg protein ml^{-1} PBS at 2-week intervals and a single booster injection 5 days before fusion.

Production and screening of hybridomas and determination of antibody specificity

Hybridoma cells were produced by the method described elsewhere (Thornton, 2001) and the supernatants were screened by Enzyme-Linked Immunosorbent Assay (ELISA) against antigens immobilized to the wells of Maxisorp microtiter plates (442404; Nunc) (50 µl per well). For antibody specificity tests, fungi were grown on replicate agar slopes and surface washings containing water-soluble antigens prepared as described in Thornton (2001). Protein concentrations, determined spectrophotometrically at 280 nm (Nanodrop, Agilent Technologies Limited, Berkshire, UK), were adjusted to 60 µg ml^{-1} buffer. Fifty µl volumes were then used to coat the wells of microtitre plates. After incubating overnight at 4°C, wells were washed four times with PBST (PBS containing Tween-20, 0.05% (v/v)), once each with PBS and dH_2O and then air-dried at 238C in a laminar flow hood. The plates were stored in sealed plastic bags at 4°C in preparation for screening of hybridoma superna-tants by ELISA as described below.

Enzyme-linked immunosorbent assay

Wells containing immobilized antigens were incubated successively with hybridoma tissue culture supernatant (TCS) for 1 hour, followed with goat anti-mouse polyvalent (immunoglobulin classes IgG, IgA and IgM) peroxidase conjugate (A-0412; Sigma Chemical Company, Poole, United Kingdom) diluted 1 in 1000 in PBST for a further hour. Bound antibody was visualized by incubating wells with tetramethyl benzidine (TMB: T-2885; Sigma) substrate solution (Thornton, 2001) for 30 minutes. The reactions were stopped by the addition of 3 M H_2SO_4 and absorbance values were determined at 450 nm with an MRX automated microplate reader (Dynex Technologies, Billingshurst, UK). Wells were given four 5-minute rinses with PBST between incubations and a final rinse with PBS before addition of the substrate solution. Working volumes were 50 µl per well and control wells were incubated with tissue culture medium (TCM) containing 10% (v/v) fetal bovine serum. All incubation steps were performed at 238C in sealed plastic bags. The threshold for detection of the antigen in ELISA was determined from control means (23 TCM absorbance values) (Sutula et al., 1986). These values were consistently in the range 0.050–0.100. Consequently, absorbance values > 0.100 were considered as positive for the detection of antigen.

Determination of Ig subclass and cloning procedure

The Ig class of mAbs was determined by using antigen-mediated ELISA. Wells of microtitre plates coated with *F. solani* CBS224.34 water-soluble antigens from surface washings were incubated successively with ED7 TCS for 1 hour, followed with goat anti-mouse IgG_1, IgG_{2a}, IgG_{2b}, IgG_3, IgM or IgA-specific antiserum (ISO-2; Sigma) diluted 1 in 3000 in PBST for 30 minutes and rabbit anti-goat peroxidase conju-gate diluted 1 in 1000 (A-5420; Sigma) for a further 30 minutes. Bound antibody was visualized with TMB substrat as described above. Hybridoma cells lines were sub-cloned three times by limiting dilution. They were preserved by slowly freezing in fetal bovine serum/dimethyl sulfoxide (92:8 [v/v]), and stored in liquid nitrogen.

Gel electrophoresis and Western blotting

For sodium-dodecyl-sulphate-polyacrylamide gel electrophoresis (SDS-PAGE), culture filtrates from 2-days-old PDB shake cultures of *F. solani* CBS224.34 and *F. oxysporum* f.sp. *lycopersici* CBS167.30, prepared as described, were diluted in Laemmli buffer (Laemmli, 1970) and were denatured by heating at 958C for 10 min. Antigens were separated in 4%– 20% (w/v) polyacrylamide gradient gels (161-1159; Bio-Rad) for 1.5 hours at 238C (165V) under denaturing conditions, and pre-stained broad range markers (161-0318; Bio-Rad) were used for molecular weight determinations. For Westerns, sep-arated antigens were transferred electrophoretically to a PVDF membrane (162-0175; Bio-Rad). The membranes were blocked for 16 hours at 4°C with PBS containing 1% (w/v) bovine serum albumin (BSA) and incubated with ED7 TCS diluted 1 in 2 with PBS containing 0.5% (w/v) BSA (PBSA) for 2 hours at 238C. After washing three times with PBS, mem-branes were incubated for 1 hour with goat anti-mouse IgM (l-chain specific) alkaline phosphatase conjugate (A-9688; Sigma), diluted 1 in 15 000 in PBSA. After the membranes were washed twice with PBS and once with PBST, the bound antibodies were visualized by incubation in BCIP/NBT sub-strate solution. Reactions were stopped by immersion in dH_2O and air-dried between sheets of Whatman filter paper.

Characterization of antigen by enzymatic and chemical modifications and by heating

Water-soluble antigens from surface washings of slopes of *F. solani* CBS224.34 were prepared as described. Heat stability studies were conducted by placing tubes of solubilized antigen in a boiling water bath. At 10 minutes intervals, samples were removed, centrifuged at 14 462g for 5 minutes, and antigens immobilized to the wells of microtitre plates for assay by ELISA

as described. For periodate oxidation, microtitre wells containing immobilized antigens from surface washings of the fungus were incubated with 50 ml of sodium meta-periodate solution (20 mM $NaIO_4$ in 50 mM sodium acetate buffer (pH4.5)) or acetate buffer only (control) at 4°C in sealed plastic bags. Plates were given four 3-minute PBS washes before processing by ELISA as described. For protease digestions, microtitre wells containing immobilized antigen were incubated with 50 ml of pronase (protease XIV; 9 mg ml^{-1} in PBS) or trypsin (1 mg ml^{-1} in Milli-Q H_2O) solution or Milli-Q H_2O or PBS only controls respectively for 4 hours at 378C or 4°C. Plates were given four 3-minute rinses with PBS and then assayed by ELISA with ED7 TCS as described.

Immunofluorescence and immunogold electron microscopy

For immunfluorescence (IF), sterilized slides were coated with a washed spore suspensions of *F. solani* CBS224.34 containing 1% (w/v) glucose solution and incubated at 26°C for 16 hours to allow spore germination and formation of germ tubes. After air-drying, the cells were fixed to the slides as described in Thornton (2001) and incubated with ED7 TCS or TCM only (negative control) for 1 hour, followed by three 5 minute PBS washes. Slides were then incubated with goat anti-mouse poly-valent fluorescein isothiocyanate (FITC) conjugate (diluted 1 in 40 in PBS) (F1010; Sigma) for 30 minutes. Slides were given three 5 minute washes with PBS and mounted in PBS-glycerol mounting medium (F4680; Sigma) before overlaying with cover-slips. All incubation steps were performed at 238C in a humid environment to prevent evaporation and slides were stored in the dark, at 4°C, prior to examination using an epifluorescence microscope (Olympus IX81) fitted with 495 nm (excitation) and 518 nm (emission) filters for FITC. For immunogold BSA (PBST-BSA) which had been sterile filtered through a 0.2 lm filter. The grids were washed three times (3 minutes each) in sterile filtered PBST and then incubated in ED7 TCS or TCM only (negative control) for 1 hour. Afterffour washes (3 minutes each) with sterile filtered PBST, the grids
were incubated for a further hour in PBST-BSA containing a 1:20 dilution of goat anti-mouse 20 nm gold conjugate (EM.GAF20; BBI Solutions). The grids were washed four times (3 minutes each) in electron microscopy (IEM) the method described in Thornton & Talbot (2006) was used. Spores and hyphae of *F. solani* were prepared by incubating washed conidia in 1% (w/v) glucose solution at 26°C for 16 hours to allow spore germination and for-mation of germ tubes. Cells were embedded in LR White resin (Agar Scientific Ltd.) and ultra thin sections prepared for immu-nolabeling. Sections immobilized to nickel grids were blocked by immersion in PBST containing 1% (w/v)

Statistical analysis

Unless otherwise stated, numerical data were analysed using the statistical programme Minitab (Minitab 16, Minitabv^R, Coventry, UK). Analysis of variance (ANOVA) was used to compare means of more than two data sets and Post-hoc Tukey–Kramer analysis was then performed to distinguish which sets were significantly different from one another.

Sampling from drains

A total of 65 sinks were swabbed, comprising 32 sinks across the ICU, ITU, hematology, oncology and ophthalmology units of the Royal Devon and Exeter tertiary care hospital (Exeter, Devon, UK) and 33 restroom sinks located around the University of Exeter campus (Exeter, Devon, UK). In addition, cold-water samples were collected from taps connected to the sinks in the hematology and oncology unit, and from the two main water tanks feeding the ophthalmology unit. To isolate fungi from sink biofilms, sterile cotton buds (Boots, UK) wetted with PBS were used to scour the inner surfaces of sink drain-pipes for approximately 10 seconds. Swabs with visible ditritus were immersed in 1.5-ml micro-centrifuge tubes containing 1 ml PBS to dislodge biofilm debris, and the sealed tubes transferred to the laboratory for processing by ELISA and mycological culture.

Immunodetection of *Fusarium* species in sink swabs and identification of fungi by analysis of the ITS regions of the rRNA-encoding gene unit and Translation elongation factor-1α PCR

Biofilm debris was pelleted by centrifugation at 14 462g for 5 minutes and 50 µl samples of supernatant transferred to the wells of microtitre plates for assay by ELISA (Table 1 and Supporting Information Table S2; Swab-ELISA) as described. The biofilm pellet was re-suspended in 1 ml dH_2O, 200 µl samples spread on the surface of PDA containing 1 µg ml^{-1} of the broad-spectrum antibiotic rifampicin, and the plates incubated for 2 days at 26°C under a 16 hours fluorescent light regime. Fungi in these mixed culture plates were separated on the basis of gross morphological characteristics and axenic slope cultures generated following sub-culture on PDA. Crude anti-gen extracts were prepared as surface washings from mixed cultures and from axenic cultures and assayed by ELISA (Table 1 and Supporting Information Table S2; Mixed culture-ELISA and Axenic culture-ELISA, respectively) as described.

Fungal DNA was extracted from axenic culture material by using the CTAB method (Chow & Kafer, 1993) and fungi were identified by sequencing of the ITS1-5.8S-ITS2 region of the rRNA-encoding gene unit (White et al., 1990) according to procedures described elsewhere (Thornton et al., 2002), using the primers ITS1ext (5`-GTAACAAGGTTTCCGTAGGTG-3`) and ITS4ext (5`-TTCTTTTCCTCCGCTTATTGATATGC-3`). Species identity was predicted based on > 95% sequence identity
(E-value 5 0.0) (Altschul et al. 1997) of the ITS1-5.8S-ITS2 region of recovered species to species recorded in Gen-Bank. *Fusarium* species were further identified to species complex level by using the forward primer ef-1 (5`-ATGGG-TAAGGA(A/G)GACAAGAC-3`) and reverse primer ef-2 (5` GGA(G/A)GTACCAGT (G/C)ATCATGTT-3`), which amplify an approximately 700 bp region of Translation Elongation Factor 1-alpha (TEF-1α), the single-locus identification tool in Fusar-ium (Geiser et al., 2004). PCR reactions were carried out in a total volume of 25 µl consisting of 1 µl DNA at a concentration of 30–75 ng $µl^{-1}$, 12.5 µl of GoTaqv^R Green Master Mix DNA polymerase (Promega, MF7112), 9.5 µl of nuclease free water (Promega) and 1 µl of each primer at 20 pmol. The following cycling parameters were used: an initial denaturation step at 958C for 8 minutes; 35 cycles of 15 seconds at 958C (denaturation); 20 seconds at 54°C (annealing), 1 minutes at 728C (extension) followed by a final 5 minutes extension step at 728C. Phylogenetic sub-groups of *Fusarium* species were determined by interrogation of the FUSARIUM-ID v. 1.0 database (http://isolate.fusariumdb.org) (O'Donnell et al., 2010), with

the newly acquired TEF-1α sequences (Supporting Infor-mation Data Set 1).

Nucleotide sequence accession numbers

Newly determined ITS sequences were submitted to GenBank and the ITS accession numbers KT876496 to KT876723 were

obtained. Species designations of recovered fungi are shown in Table 1.

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Conflicts of Interest

We declare that none of the authors involved in writing this article have any conflicts of interest with respect to the con-tent of this article.

References

Altschul, S.F., Madden, T.L., Schaffer, A.A., Zhang, Z., Miller, W., and Lipman, D.J. (1997) Gapped BLAST and PSI-BLAST: a new generation of protein database programs. Nucleic Acids Res 25: 3389–3402.

Anaissie, E.J., Penzak, S.R., and Dignani, M.C. (2002) The hospital water supply as a source of nosocomial infections: a plea for action. Arch Intern Med 162: 1483–1492.

Anaissie, E.J., Kuchar, R.T., Rex, J.H., Francesconi, A., Kasai, M., Muller, F.M., et al. (2011) Fusariosis associated with pathogenic *Fusarium* species colonization of a hospital water system: a new paradigm for the epidemiology of opportunistic mould pathogens. Clin Infect Dis 33: 1871–1878.

Arie, T., Hayashi, Y., Nagatani, A., Furuya, M., and Yamaguchi, I. (1991) Production and partial characterization of monoclonal antibodies against *Fusarium oxysporum* 860926a. Ann Phytopathol Soc Jpn 57: 696–701.

Arie, T., Hayashi, Y., Yoneyama K, Nagatani, A., Furuya, M., and Yamaguchi, I. (1995) Detection of *Fusarium* spp. in plants with monoclonal antibody. Ann Phytopathol Soc Jpn 61: 311–317.

Arrese, J.E., Pierard-Franchimont, C., and Pierard, G.E. (1996) Fatal hyalohyphomycosis following *Fusarium* ony-chomycosis in an immunocompromised patient. Am J Der-matopathol 18: 196–198.

Bigley, V.H., Duarte, R.F., Gosling, R.D., Kibbler, C.C., Seaton, S., and Potter, M. (2004) *Fusarium dimerum* infection in a stem cell transplant recipient treated successfully with vori-conazole. Bone Marrow Transplant 34: 815–817.

Bouchara, J-P., Hsieh, H.Y., Croquefer, S., Barton, R., Marchais, V., Pihet, M., et al. (2009) Development of an oli-gonucleotide array for direct detection of fungi in sputum samples from patients with cystic fibrosis. J Clin Microbiol 47: 142–152.

Boutati, E.I., and Anaissie, E.J. (1997) *Fusarium*, a significant emerging pathogen in patients with haematological malig-nancies: ten years experience at a cancer centre and impli-cations for management. Blood 90: 999–1008.

Chang, D.C., Grant, G.B., O'Donnell, K., Wannemuehler, K.A., Noble-Wang, J., Rao, C.Y., et al. (2006) Multistate out-break of *Fusarium* keratitis associated with use of a contact lens solution. JAMA 296: 953–963.

Chow, T.Y.K., and Kafer,€ E. (1993) A rapid method for isolation of total nucleic acids from *Aspergillus nidulans*. Fungal Genet Newsl 40: 25–27.

Danks, J.N., Rizvi, R.H., Barker, I., Turner, J.A., Rahman, S., and Northway, B.J. (1996) Specific monoclonal antibodies to *Fusarium* species and *Michrodochium nivale*. Food Agric Immunol 8: 249–268.

Davies, G., and Thornton, C.R. (2014) Differentiation of the emerging human pathogens *Trichosporon asahii* and *Trichosporon asteroides* from other pathogenic yeasts and moulds by using species-specific monoclonal antibodies. PLoS ONE 9: e84789.

Dean, R., Van Kan, J.A.L., Pretorius, Z.A., Hammond-Kosack, K.E., Di Pietro, A., Spanu, P.D., et al. (2012) The top 10 fun-gal pathogens in molecular plant pathology. Mol Plant Pathol 13: 414–430.

Dignani, M.C., and Anaissie, E. (2004) Human fusariosis. Clin Microbiol Infect 10: 67–75.

Doggett, M.S. (2000) Characterisation of fungal biofilms within a municipal water distribution system. Appl Env Microbiol 66: 1249–1251.

Dursun, D., Fernandez, V., and Miller, D. (2003) Advanced *Fusarium* keratitis progressing to endophthalmitis. Cornea 22: 300–303.

Edelstein, S.L., Akduman, L., Durham, B.H., Fothergill, A.W., and Hsu, H.Y. (2012) Resistant *Fusarium* keratitis progressing to endophthalmitis. Ophthalmol Contact Lens 38: 331–335.

Feng, P., Qiaoyun, L., Najafzadeh, M.J., Gerrits van den Ende, A.H.G., Sun, J., Li, R., et al. (2014) *Cyphellophora* and its relatives in *Phialophora*: biodiversity and possible role in human disease. Fungal Divers 65: 17–45.

Geiser, D.M., del Mar Jimenez-Gasco, M., Kang, S., Makalowska, I., Veeraraghavan, N., Ward, T.D., et al. (2004) FUSARIUM-ID v.1.0: A DNA sequence database for identifying *Fusarium*. Eur J Plant Pathol 110: 473–479.

Girmenia, C., Pagano, L., Corvatta, L., Mele, L., Del, A., Favero, P., et al. (2000) The epidemiology of fusarioses in patients with haematological diseases. Br J Haematol 111: 272–276.

Gurusidappa, S.B., and Mamatha, H.S. (2011) Fusarial skin lesion in immunocompromised. Ind J Cancer 48: 116–117.

Hayashi, Y., Arie, T., Yoneyama, K., and Yamaguchi, I. (1998) Characterisation of the antigenic determinant on *Fusarium oxysporum* recognized by a genus-specific monoclonal anti-body. J Gen Appl Microbiol 44: 43–47.

Hu, Z-Q., Liu, J-L., Li, H-P., Xing, S., Xue, S., Zhang, J-B., et al. (2012) Generation of a highly reactive chicken-derived single-chain variable fragment against *Fusarium verticillioides* by phage display. Int J Mol Sci 13: 7038–7056.

Hu, Z-Q., Li, H-P., Zhang, J-B., Huang, T., Liu, J-L., Xue, S., et al. (2013) A phage-displayed chicken single-chain anti-body fused to alkaline phosphatase detects *Fusarium* pathogens and their presence in cereal grains. Anal Chim Acta 764: 84–92.

Jensen, T.G., Gahrn-Hansen, B., Arendrup, M., and Bruun, B. (2004) *Fusarium* fungaemia in immunocompromised patients. Clin Microbiol Infect 10: 499–501.

Jensen, H.M.E., Aalbæk, B., Jungersen, G., Hartvig, T., Moser, C., Rozell, B.L., et al. (2011) Immunohistochemical diagnosis of fusariosis with monoclonal antibodies. Myco-ses 54: s54–s55.

Jurkunas, U., Behlau, I., and Colby, K. (2009) Fungal keratitis: changing pathogens and risk factors. Cornea 28: 638–643.

Koehler, P., Tacke, D., and Cornely, O.A. (2014) Bone and joint infections by Mucorales, *Scedosporium*, *Fusarium* and even rarer fungi. Crit Rev Microbiol 9: 1–14.

Lackner, M., Najafzadeh, M.J., Sun, J., Lu, Q., and de Hoog, G.S. (2012) Rapid identification of *Pseudallescheria* and *Scedosporium* strains by using rolling circle amplification. Appl Environ Microbiol 78: 126–133.

Laemmli, U.K. (1970) Cleavage of structural proteins during assembly of the head of bacteriophage T4. Nature 227: 680–695.

Latenser, B.A. (2003) *Fusarium* infections in burn patients: a case report and review of the literature. J Burn Care Rehabil 24: 285–288.

Macedo,^ D.P.C., Neves, R.P., de Souza-Motta, C.M., and Magalhaes,~ O.M.C. (2007) Engyodontium album fungaemia: the first reported case. Braz J Microbiol 38: 110–112.

Mehl, H.L., and Epstein, L. (2008) Sewage and community shower drains are environmental reservoirs of *Fusarium* species complex group 1, a human and plant pathogen. Env Microbiol 10: 219–227.

Miceli, M.H., Dıaz, J.A., and Lee, S.A. (2011) Emerging opportunistic yeast infections. Lancet Infect Dis 11: 142–151.

Muhammed, M., Carneiro, H., Coleman, J., and Mylonakis, E. (2011) The challenge of managing fusariosis. Virulence 2: 91–96.

Musa, H.O., Al Eisa, A., Halim, M., Sahovic, E., Gyger, M., Chaudhri, N., et al. (2000) The spectrum of *Fusarium* infection in immunocompromised patients with haematological malignancies and in non-immunocompromised patients; A single institution experience over 10 years. Br J Haematol 108: 544–548.

Nucci, M., and Anaissie, E. (2002) Cutaneous infection by *Fusarium* species in healthy and immunocompromised hosts: implications for diagnosis and management. Clin Infect Dis 35: 909–920.

Nucci, N., and Anaissie, E. (2007) *Fusarium* infections in immuncompromised patients. Clin Microbiol Rev 20: 695–704.

O'Donnell, K., Sutton, D.A., Rinaldi, M.G., Sarver, B.A.J., Arunmozhi Balajee, S., Schroers, H.-J., et al. (2010) Internet-accessible DNA sequence database for identifying Fusaria from human and animal infections. J Clin Microbiol 48: 3708–3718.

Petrikkos, G., Skiada, A., Lortholary, O., Roilides, E., Walsh, T.J., and Kontoyiannis, D.P. (2012) Epidemiology and clinical manifestations of mucormycosis. Clin Infect Dis 54: S23–S24.

Rougeron, A., Schuliar, G., Leto, J., Sitterle, E., Landry, D., Bougnoux, M.E., et al. (2014) Human-impacted areas of France are environmental reservoirs of the *Pseudallescheria boydii*/*Scedosporium apiospermum* species complex. Env Microbiol 17: 1039–1048.

Sandoval-Denis, M., Sutton, D.A., Cano-Lira, J.F., Gene, J., Fothergill, A.W., Wiederhold, N.P., et al. (2014) Phylogeny of the clinically relevant species of the emerging fungus *Trichoderma* and their antifungal susceptibilities. J Clin Micro-biol 52: 2112–2125.

Schroers, H. J., O'Donnell, K., Lamprecht, S.C., Kammophthalmologyr, P.L., Johnson, S., Sutton, D.A., et al. (2009). Taxonomy and phylogeny of the *Fusarium dimerum* species group. Mycologia 101:44-70.

Short, D.P.G., O'Donnell, K., Zhang, N., Juba, J.H., and Geiser, D.M. (2011) Widespread occurrence of diverse human pathogenic types of the fungus *Fusarium* detected in plumbing drains. J Clin Microbiol 49: 4264–4272.

Steinmann, J., Schmidt, D., Buer, J., and Rath, P-M. (2011) Discrimination of *Scedosporium* prolificans against *Pseudallescheria boydii* and *Scedosporium apiospermum* by semiautomated repetitive sequence-based PCR. Med Mycol 49: 475–483.

Sutula, C.L., Gillett, J.M., Morrisey, S.M., and Ramsdell, D.C. (1986) Interpreting ELISA data and establishing the positive-negative threshold. Plant Dis 70: 722–726.

Thamke, D.C., Mediratta, D.K., Dhabarde, A., and Shukla, A.K. (2015) Mycotic keratitis due to Engyodontium album: first case report from India. Indian J Med Microbiol 33: 303– 304.

Thornton, C.R. (2001) Immunological methods for fungi. In Molecular and Cellular Biology of Filamentous Fungi, A Practical Approach. Talbot, N.J. (ed.). Oxford, UK: University Press, pp. 227–257.

Thornton, C.R. (2008) Development of an immunochromatographic lateral flow device for rapid serodiagnosis of invasive aspergillosis. Clin Vacc Immunol 15: 1095–1105.

Thornton, C.R. (2009) Tracking the emerging human pathogen *Pseudallescheria boydii* by using highly specific monoclonal antibodies. Clin Vacc Immunol 16: 756–764.

Thornton, C.R. (2012) Serological techniques for diagnosis. In Fungal Plant Pathogens, Principles and Protocols. Lane, C., Beales, P., and Hughes, K. (eds.). Wallingford, UK: CABI, pp. 159–177.

Thornton, C.R., Pitt, D., Wakley, G.E., Talbot, N.J. (2002) Pro-duction of a monoclonal antibody specific to the genus *Trichoderma* and closely related fungi, and its use to detect Trichoderma spp. in naturally infested composts Microbiol-ogy 148: 1263–1279.

Thornton, C.R., and Wills, O.E. (2015) Immunodetection of fungal and oomycete pathogens: Established and emerging threats to human health, animal welfare, and global food security. Crit Rev Microbiol 41: 27–51.

Thornton, C.R., Ryder, L.S., Le Cocq, K., and Soanes, D.M. (2015) Identifying the emerging human pathogen *Scedosporium prolificans* by using a species-specific monoclonal antibody that binds to the melanin biosynthetic enzyme tetrahydroxynaphthalene reductase. Environ Microbiol 17: 1023–1048.

Thornton, C.R., Talbot, N.J. (2006) Immunofluorescence microscopy and immunogold EM for investigating fungal infections of plants. Nat Protocols 1: 2506–2511.

Torres, H.J., and Kontoyiannis, D. (2011) Hyalohyphomycosis (hyaline moulds). In Essentials of Clinical Mycology, Kauffman, C.A., Pappas, P.G., Sobel, J.D., and Dismukes, W.E. (eds.). New York: Springer Science1Business Media, pp. 281–304.

Walsh, T.J., and Groll, A.H. (1999) Emerging fungal pathogens: evolving challenges to immunocompromised patients for the twenty-first century. Transplant Infect Dis 1: 247–261.

White, T.J., Bruns, T., Lee, S., Taylor, J. (1990) Amplification and direct sequencing of fungal ribosomal RNA genes for phyloge-netics. In PCR Protocols: a Guide to Methods and Applica-tions. Innis, M.A., Gelfand, D.H., Sninsky, J.J., and White, T.J. (eds). San Diego, USA: Academic Press, pp. 315–322.

Wong, W.C., White, M., and Wright, I.G. (1988) Production of monoclonal antibodies to *Fusarium oxysporum* f.sp. *cubense* race 4. Lett Appl Microbiol 6: 39–42.

Zeng, J.S., Sutton, D.A., Fothergill, A.W., Rinaldi, M.G., Harrak, M.J., and de Hoog, G.S. (2007) Spectrum of clini-cally relevant *Exophiala* species in the Unites States. J Clin Microbiol 45: 3713–3720.

Zhang, N., O'Donnell, K., Sutton, D.A., Nalim, F.A., Summerbell, R.C., Padhye, A.A., et al. (2006) Members of the *Fusarium solani* species complex that cause infections in both humans and plants are common in the environment.
J Clin Microbiol 44: 2186–2190.

Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Supporting Data Set 1. Translation elongation factor 1 alpha (tef) gene sequences of *Fusarium* species.

Table S1. Details of fungi used in ELISA specificity tests of ED7.

Table S2. Summary of ELISA tests and mycological culture of sink swabs.

Table S3. Summary of species complex identities following Translation Elongation Factor-1α PCR analysis of *Fusarium* isolates recovered from sink.