International Scholars Journals

International Journal of Bacteriology and Mycology ISSN 2756-3669 Vol. 13 (1), pp. 001-005, May, 2025. Available online at www.internationalscholarsjournals.org © International Scholars Journals

Full Length Research Paper

Systemic Fungal Infection Microbiology

Midhuna K. and Kiran A.

Panjab University, Chandigarh, India.

Accepted 13 May, 2025

Over the last 20 years, the prevalence of systemic fungal infections has skyrocketed. In the past, systemic infections were known to be caused by pathogenic dimorphic fungus. However, opportunistic fungi began to cause more infections, particularly in immunocompromised hosts, in the 1960s. More recently, infections in immunocompromised hosts have been linked to fungal agents that are newer and less prevalent. Infections with dimorphic fungi, including Histoplasma capsulatum and Penicillium marneffei, are becoming more common among AIDS patients in India. While P. marneffei is still only found in the state of Manipur, H. capsulatum is found throughout the nation. While there are reports of both C. neoformans var. neoformans (serotypes A & D) and C. neoformans var. gattii (serotypes B & C) in India, serotype A accounts for the majority of cases. Every facility reports a higher prevalence of cryptococcosis since AIDS first appeared. In nosocomial settings, systemic infections caused by species of Aspergillus, Candida, and Zygomycetes are common, and outbreaks caused by uncommon fungi are occasionally documented from tertiary care facilities. The need to establish high-quality diagnostic mycology laboratories in this nation and identify this growing number of possible fungal pathogens has been highlighted by the worldwide shift in systemic fungal infections.

Key words: Aspergillus, Candida, disseminated fungal infections, fungi, opportunistic infections, systemic fungal infections, Zygomycetes

INTRODUCTION

Fungi-induced systemic infections are a significant public health issue in both industrialized and developing nations worldwide. In the past, medical microbiology began with the identification of the causal role that fungus play in disease. Pasteur and Koch's precursor, Agostino Bassi, established the theory of harmful germs. Beauvaria bassiana, a mold that caused terrible silkworm illness, was discovered by Bassi in 1835. The first findings of fungal diseases in humans, such as favus (discovered by Remark and Schoenlein in 1837 and 1842, respectively), candidiasis (discovered by Gruby in 1842), and aspergillosis (discovered by Sluyter in 1847), came shortly after.[1], [2]

The fact that fungi are so common in nature shows how well-suited they are for survival. Less than 150–200 of the estimated hundreds of thousands of fungal species were thought to be human diseases. However, fungi have been growing in popularity among humans in recent years. Both the number of fungi that cause systemic diseases and the number of fungi that induce systemic discomfort are on the rise. Invasive aspergillosis is present in up to 7% of patients who pass away in teaching hospitals.[3], [4] Candida species are the fourth most prevalent isolation of patients in intensive care units and are responsible for 8–15% of nosocomial bloodstream infections.[5] Fungal infections are extremely common in certain patient groups: 15% of recipients of allogenic hemopoietic stem-cell transplants have a fungal infection [6], 20% of lung transplant recipients are colonized and infected [7], 60% of AIDS patients have Pneumocystis carinii (jiroveci) pneumonia or 20% have esophageal candidiasis [8], 30% of AIDS patients in Africa and Southeast Asia have cryptococcal meningitis [9], and 30% of AIDS patients in South-east Asia have infections caused by Penicillium marneffei.[10]

Although India's diverse climate makes it suitable for a wide range of fungal infections, the facts on the prevalence of systemic fungal infections in this country are unclear. Nonetheless, a clear upward tendency has been identified. Table 1 lists the systemic fungal infections that have been reported in India.

The second part of the 1980s had an eleven-fold increase in candidemia from our center [11], and the first half of the 1990s saw an additional 18-fold increase.[12]

Over the course of 23 months, 379 neonates and children (4.2% of all admissions) were reported to have fungemia caused by the uncommon yeast Pichia anomala from the same center.[13] Between 1980 and 1993, 95 patients developed cerebral aspergillosis, demonstrating the devastating effects of systemic aspergillosis.[14] With the advent of Apophysomyces elegans infection in India, 129 cases of invasive zygomycosis were detected between 1990 and 1999[15], indicating an increasing tendency in the disease.[16] Compared to the pre-AIDS era, the annual incidence of cryptococcosis has grown by around 15 times.[17] All of these numbers show that many patients are currently suffering from systemic disease due to a wide variety of fungus. This significant rise in systemic fungal infections can be attributed to a number of factors, such as improved treatment of other immunosuppressive side effects, new and more aggressive immunosuppressive regimens, improved survival in critical care, a high frequency of catheterization and instrumentation, increased clinician awareness, improved diagnostic techniques, and a greater use of broad spectrum antibiotics.

The two main categories of endemic mycoses caused by genuine pathogenic fungi and opportunistic fungal infections caused by a wide variety of saprophytic fungi comprise the etiology of systemic fungal diseases.

True pathogenic fungi

Unlike mycelial forms in culture, which are produced at 25–30°C, true pathogenic fungi generate a distinct form in tissue or at 37°C. Known as dimorphic fungi, they include Histoplasma duboisii, Blastomyces dermatitidis, Coccidioides immitis, Paracoccidioides brasiliensis, Penicillium marneffei, Sporothrix schenckii, and Histoplasma capsulatum. These fungi are typically limited by geography. The geophilic mold C. immitis is unique to the New World and has evolved to thrive in the arid regions of North, Central, and South America.[18] P. marneffei is only found in southeast Asia, where it may still coexist with bamboo rats.[10], [19] Both B. dermatitidis and H. capsulatum are found all over the world. While penicilliosis marneffei is only found in Manipur state, histoplasmosis and blastomycosis have been recorded from various states in India. Systemic sporotrichosis caused by S. schenckii var. luriei has only been reported once, and it is the only record from an Asian country.[20] In India, reports of histoplasmosis are rising in tandem with the rise of AIDS.

Opportunistic fungi

Species belonging to the genera Candida, Aspergillus, Cryptococcus, and Zygomycetes started to be often linked to patients undergoing therapy for organ transplants, diabetes, sarcoidosis, and cancer in the 1960s. At that time, the "big four" opportunists were responsible for more pathology and researchers' attention than all other fungi put together. But things have changed during the past 30 to 40 years, and fresh infections are being identified, particularly with the rise of AIDS. A variety of fungi, including species under the genera Pneumocystis, Candida, Cryptococcus, Histoplasma, Coccidioides, Aspergillus, and zygomycetes, can occasionally cause concurrent and/or consecutive opportunistic systemic fungal infections. In addition, a variety of less common fungal infections are routinely isolated from clinical specimens. This makes it challenging to categorize and research this class of fungal illnesses. The term "phaeohyphomycosis" was proposed by Ajello et al. to address this issue, as it encompasses all "infections of a cutaneous, subcutaneous, and systemic nature caused by hyphomycetous fungi that develop in host tissues in the form of dark-walled dematiaceous septate mycelial elements" [21]. Ajello and McGinnis proposed the term "hyalohyphomycosis" to include mycotic infections where the tissue form of the etiological agents is septate hyphae.[22]

Candida spp

The rise of non-C. albicans Candida (NAC) species as colonizers and pathogens that cause nosocomial fungal blood stream infections (BSI) has contributed to the overall rise in candidemia in recent years. In a thorough analysis of all studies published between 1952 and 1992, Wingard discovered that 12 of them contained a proportionately larger (>50%) isolation of NAC species.[23] C. glabrata, C. krusei, C. tropicalis, and C. parapsilosis were the NAC species that were isolated. Occasionally, other species were isolated, including C. guilliermondii, C. lusitaniae, C. dubliniensis, C. kefyr, C. lipolytica, and C. pelliculosa. According to the CDC's 1992–1993 population-based surveillance research, Candida albicans was the most prevalent species, followed by Candida parapsilosis, Candida tropicalis, and Candida glabrata. The percentage of Candida BSI caused by NAC species increased, and in particular, the frequency of BSI caused by C. glabrata increased, according to subsequent surveillance programs.[5] On the other hand, surveillance data from other nations still show that C. parapsilosis is more significant than C. glabrata.[24]

It has also been shown that patient age has a significant role

in defining the rank order of the Candia species that cause BSI.[23] In newborn age groups, it has been noted that Candida albicans and Candida parapsilosis predominate whereas C. glabrata and other NAC species are absent. In contrast, as people age, C. glabrata becomes a more significant disease. Individual NAC species' contributions also differ according on the patient and diagnostic category. Candida caused by C. parapsilosis is typically linked to prosthetic devices, hyperalimentation, or catheters.[25] Additionally, the most frequent species of Candida found on the hands of medical personnel in intensive care units, particularly those using gloves, is C. parapsilosis.[26] Therefore, it is likely that healthcare personnel's hands are the source of the organism's contamination of prosthetic devices. Patients with solid tumors in oncology are often affected by C. glabrata.[27]

Because C. krusei is inherently resistant to fluconazole, fluconazole prophylaxis has been shown to promote infection with this organism in certain bone marrow transplant facilities.[28] Regardless of antifungal prophylaxis, C. krusei infections are more common in patients with hematological malignancies, while they are less common in patients with solid tumors and intensive care unit patients.[29]

Like in the west, tertiary care facilities in India have also seen an increase in the frequency of NAC species, with isolation rates ranging from 52 to 96%. It is peculiar in this setting, nonetheless, as C. tropicalis is isolated more frequently in all age groups in India than C. glabrata or C. parapsilosis.[12], [30]–[32] The information compiled in Table 2 demonstrates the global isolation of Candida species from candidemia patients.

Other yeasts

Since AIDS first appeared, reports of cryptococcosis have increased.[17] Because the expensive medications are not as affordable in poorer nations, there is no change in incidence following a tri-drug regimen, despite a decrease in incidence being noted in developed nations. In recent years, a number of other saprophytic yeasts have been implicated in systemic infections. Table 3 contains those.

Aspergillus spp. and other moniliaceous fungi

The second most prevalent invasive fungal infection is systemic aspergillosis. Up to 30% of patients in postmortem se¬ries[34] and 36% of patients with pneumonia in bone-marrow transplant units have been documented to have the illness in specific patient groupings, such as hematological malignancies.[35] According to data collected in a cross-section of hospitals across the United States between 1980 and 1990, Aspergillus species were responsible for 1.3% of all nosocomial infections.[36]

The most frequent cause of invasive aspergillosis is A. fumigatus. The second most prevalent species, A. flavus, is isolated from nasal sinus lesions and systemic aspergillosis in immunocompromised patients. Nonetheless, A. flavus is the most frequent cause of all types of aspergillosis in South Africa, Sudan, and India. The third most frequent cause of invasive aspergillosis is A. niger. A. nidulans, A. versicolor, A. candidus, A. oryzae, A. sydowii, A. terreus, A. clavatus, and more species are hardly documented.

Human systemic infection can be caused by the worldwide soil saprobe Fusarium species. Neutropenic patients with hematological malignancies, as well as those undergoing bone-marrow and solid-organ transplantation, have been observed to have an increased incidence, frequently with a fatal prognosis.[37] Fungi from the genus Scedosporium, Pseudallescheria, Acremonium, Lecythophora, Phialemonium, Phaeoacremonium, Paecilomyces, and Emmonsia have been linked to further uncommon systemic illnesses.

Zygomycetes

Zygomycetes are prevalent nosocomial pathogens that cause systemic zygomycosis, much like Aspergillus species. However, because antemortem diagnosis is challenging, it is hard to determine the precise incidence. Additionally, systemic zygomycosis can be acquired in the community, particularly in patients with burns, malignant hematological diseases, uncontrolled diabetes mellitus, and other types of metabolic acidosis.

Rhizopus arrhizus and Rhizopus microsporus var. rhizopodoformis are the most frequent causes of systemic zygomycosis, ranked in order of apparent incidence despite the involvement of numerous other zygomycetes. Other less common etiological agents that have been shown to play a significant pathogenic role in humans include Saksenaea vasiformis, Absidia corymbifera, Apophysomyces elegans, Cunninghamella bertholletiae, Mucor species, and Rhizomucor pusillus. These molds may be isolated in great quantities from soil or decomposing organic waste, including bread and fruit, and they are common and thermotolerant. Hospitals and outdoor air frequently contain the spores.

Dematiaceous fungi

Although some ascomycetes, basidiomycetes, and zygomycetes are also considered dematiaceous due to the presence of a brown or black hue in their cell wall, dematiaceous fungi are frequently assumed to be solely hyphomycetes. Some have argued that the term "dematiaceous" is misleading and that "phaeoid" should be used instead.[38]

The term "phaeohyphomycosis" encompasses all fungi that

have dematiaceous cells in infected tissue, regardless of the taxonomic classification of the etiological agent, even though Ajello et al. [21] proposed it to cover all infections caused by hyphomycetous fungi having dark-walled dematiaceous separate mycelial elements in tissue. Although systemic infections can be caused by more than 100 fungal species, the condition is still uncommon.

Pneumocystis sp.

Phylogenetic investigations based on nuclear small-subunit rRNA sequence alignments demonstrated definitively in the late 1980s that P. carinii belongs to the fungal kingdom.[39] It was previously believed that P. carinii represented a single zoonotic species. It is now evident, therefore, that the organism first recognized as "P. carinii" is actually a family of similar species that are host-specific for mammals. The original species of Pneumocystis for organisms derived from humans, P. carinii f. sp. hominis, has been replaced by P. jiroveci.[40]

One of the main causes of pneumonia in people with AIDS and other immunocompromised conditions is P. jiroveci. But it can also result in systemic infection. Postmortem studies of patients with pulmonary pneumocystis infection have shown a 1–3% occurrence of pneumocystis infection in non-lung locations.[41], [42] Because extra-pulmonary pneumocystosis is not suspected, this is probably an underestimate. In that cohort of 52 patients, the lymph nodes had the highest rate of extra-pulmonary infection (44%) followed by the liver, spleen, and bone marrow (33%). Additionally, it is found in the thyroid, ear, pancreas, eyes, skin, gastrointestinal tract, genitourinary tract, adrenal glands, and other places.[41]

Conclusion

The clinical mycology laboratory needs to be able to identify this growing number of possible infections, as this review highlights. Previously believed to be contaminants, these organisms are now known to be pathogens that cause systemic infection in patients with impaired immune systems. Furthermore, it is crucial to understand that, despite the fact that a particular isolate might not be listed in textbooks as a fungal pathogen, it must be regarded as a potential pathogen due to its isolation from a typically sterile region and its capacity to develop at 37°C.

References

1. Kisch B. Forgotten leaders in modern medicine: Valentin, Gruby, Remark, Auerback. Trans Am Philos Soc 1954;44:139-317.

2. Ajello L. Systemic mycoses in modern medicine. Contr Microbiol Immunol 1977;3:2-6.

3. Groll AH, Shah PM, Mentzel C,Schneider M, Just-Nuebling G, Huebner K. Trends in the postmortem epidemiology of fungal infections at a University hospital. J Infect 1996;33:23-32.

4. Vogesar M, Hass A, Aust D, Ruckdeschel G. Postmortem analysis of invasive aspergillosis in a tertiary care hospital. Eur J Clin Microbiol Inf Dis 1997;16:1- 6.

5. Pfaller MA, Diekema DJ. Role of sentinel surveillance of candidemia: Trends in species distribution and antifungal susceptibility. J Clin Microbiol 2002;40:3551-7.

6. Ribaud P, Chastang C, Latge JP, et al. Outcome and prognostic factors of invasive aspergillosis after allogenic bone marrow transplantation. Clin In- fect Dis 1999;28:322-30.

7. Denning DW. Invasive aspergillosis. Clin Infect Dis 1998;26:781-805.

8. Moore RD, Chaisson RE. Natural history of opportunistic disease in an HIV infected urban clinical cohort. Ann Intern Med 1996;124:633-42.

9. Grant AD, Djomand G, DeCock KM. Natural history and spectrum of disease in adults with HIV/AIDS in Africa. AIDS 1997;11:S43-54.

10. Supparatpinyo K, Khamwan C, Baosoung V, Nelson KE, Sirisanthana T. Dis- seminated Penicillium marneffei infection in southeast Asia. Lancet 1994;344:110-3.

11. Chakrabarti A, Chander J, Kasturi P, Panigrahi D. Candidemia: a 10-year study in an Indian teaching hospital. Mycoses 1992;35:47-51.

12. Chakrabarti A, Ghosh A, Batra R, Kaushal A, Roy P, Singh H. Antifungal sus- ceptibility pattern of non-albicans Candida species and distribution of spe- cies isolated from candidemia cases over a 5 year period. Indian J Med Res 1996;104:171-6.

13. Chakrabarti A, Singh K, Narang A, et al. Outbreak of Pichia anomala infection in the pediatric service of a tertiary care centre in northern India. J Clin Microbiol 2001;39:1702-6.

14. Sharma BS, Khosla VK, Kak VK, et al. Intra-cranial fungal granuloma. Surg Neurol 1997;47:489-97.

15. Chakrabarti A, Das A, Sharma A, et al. Ten years' experience in zygomycosis at a tertiary care centre in India. J Infect 2001;42:261-6.

16. Chakrabarti A, Ghosh A, Prasad GS, et al. Apophysomyces elegans: an emerg- ing zygomycetes in India. J Clin Microbiol 2003;41:783-8.

17. Chakrabarti A, Sharma A, Sood A, et al. Changing scenario of cryptococco- sis in a tertiary care hospital in north India. Indian J Med Res 2000;112:56- 60.

18. Ajello L. Comparative ecology of respiratory mycotic disease agents. Bact Rev 1967;31:6-24.

19. Ranjana KH, Priyokumar K, Singh TJ, et al. Disseminated Penicillium marneffei infection among HIV-infected patients in Manipur state, India. J Infect 2002;45:268-71.

20. Padhye AA, Kaufman L, Durry E, et al. Fatal pulmonary sporotrichosis caused by Sporothrix schenckii var. luriei in India. J Clin Microbiol 1992;30:2492-4.

21. Ajello L. The gamut of human infections caused by dematiaceous fungi. Jpn J Med Mycol 1981;22:1-5.

22. Ajello L, McGinnis MR. Nomenclature of human pathogenic fungi. In Grundlagen der Antiseptik: Part 4. Factoren der Mikrobiellen Kolonisation. Edited by A. P. Krasilnikow et al. Berlin: Verlag Volk und Gesundheit; 1984, p.363-77.

23. Wingard JR. Importance of Candida species other than C. albicans as patho- gens in oncology patients. Clin Infect Dis 1995;20:115-25.

24. Chryssanthon E. Trends in antifungal susceptibility among Swedish Candida species blood stream isolates from 1994-1998: comparison of the E-test and the sensitive yeast on colorimetric antifungal panel with NCCLS M27-A reference method. J Clin Microbiol 2001;39:4181-3.

25. Weem JJ Jr., Chamberland ME, Ward J, Willy M, Padhye AA, Solomon SL. Candida parapsiolosis fungemia associated with parenteral nutrition and contaminated blood pressure transducers. J Clin Microbiol 1987;25:1029- 32.

26. Rangel – Frausto MS, Wiblin T, Blumberg HM, et al. National epidemiology of mycoses survey (NEMIS): variation in rates of blood stream infections due to Candida species in seven surgical intensive care units and six neonatal intensive care units. Clin Infect Dis 1999;29:253-8.

27. Kno AS, Brnadt ME, Pruitt WR, et al. The epidemiology of candidemia in two United States cities: result of a population based active surveillance. Clin Infect Dis 1999;29:1164-70.

28. Marr KA, Seidel K, White TC, Bowden RA. Candidemia in allogenic blood and marrow transplant recipients: evaluation of risk factors after adoption of pro-phylactic fluconazole. J Infect Dis 2000;181:309-16.

29. Viscoli C, Girmenia C, Maxinus A, et al. Candidemia in cancer patients. A prospective, multicentre surveillance studies in Europe by the Invasive Fun- gal Infection Group (IFIG) of the European Organization for Research & Treat- ment of Cancer (EORTC). Clin Infect Dis 1999;28:1071-9.

30. Chakrabarti A, Mohan B, Shrivastava SK, Marak RS, Ghosh A, Ray P. Change in distribution and antifungal susceptibility of Candida species isolated from candidemia cases in a tertiary care center during 1996-2000. Indian J Med Res 2002;116:5-12.

31. Rani R, Mohapatra NP, Mehta G, Randhawa VS. Changing trends of Candida species in neonatal septicaemia in a tertiary north Indian hospital. Indian J Med Res 2002;116:42-4.

32. Verma AK, Prasad KN, Singh M, Dixit AK, Ayyagari A. Candidemia in patients of a tertiary health care hospital from north India. Indian J Med Res 2003;117:122-8.

33. Hobson RP. The global epidemiology of invasive Candida infection – is the tide turning? J Hosp Infect 2003;55:159-68.

34. Bodey G, Bueltmann B, Duguid W, et al. Fungal infections in cancer patients and international autopsy survey. Eur J Clin Microbiol 1992;11:99-109.

35. Pannuti CS, Gingrich RD, Pfaller MA, Wenzel RP. Nosocomial pneumonia in adult patients undergoing bone-marrow transplantation: a 9-year study. J Clin Oncol 1991;9:77-84.

36. Fridkin SK, Jarvis WR. Epidemiology of nosocomial fungal infections. Clin Microbiol Rev 1996;9:499-511.

37. Guarro J, Gene J. Opportunistic fusarial infections in human. Eur J Clin Microbiol Infect Dis 1995;14:741-54.

38. Pappagianis D, Ajello L. Dematiaceous – a mycologic misnomer? J Med Vet Mycol 1994;32:319-21.

39. Edman JC, Kovacs JA, Masur H, Edman U. Ribosomal RNA sequence shows

Pneumocystis carinii to be a member of the fungi. Nature 1988;334:519-22.

40. Frenkel JK. Pneumocystis pneumonia, an immunodeficiency dependent dis- ease (IDD): a critical historical overview (with a latin rediscription of Pneumocystis jiroveci of humans in conformance with the International code of Botanical Nomenclature). J Eucaryot Microbiol 1999;46:89S-92S.

41. Telzak EE, Armstrong D. Extrapulmonary infection and other unusual mani- festations of Pneumocystis carinii. In P.D. Walzer (Ed.) Pneumocystis carinii pneumonia. 2nd Edn. Marcel Dekker: New York, NY; 1994, p.361-80.

42. Ng VL, YajKo DM, Hadley WK. Extrapulmonary pnumocystosis. Clin Microbiol Rev 1997;10:401-18.

Table 1: Systemic fungal infection in India

Endemic mycoses	Opportunistic fungal infections
Histoplasmosis	Invasive candidiasis
Blastomycosis	Invasive aspergillosis
Penicilliosis marneffei	Cryptococcosis
Sporotrichosis	Invasive zygomycosis
	Pneumocystis carinii (*jiroveci*) infection
	Phaeohyphomycosis
	Infection due to miscellaneous fungi

Table 2: *Candida* species causing blood stream infection worldwide

Species	USA 1997	Canada 1997	Europe 1997	Hungary 1996–2000	Latin America 1997	The Netherlands 1995	Northern Ireland 1996–2000	India 1991–2000
C. albicans	56	53	53	77	41	60	53	14
C. parapsilosis	9	23	21	7	38	2	11	2
C. glabrata	19	11	12	3	2	17	27	3
C. tropicalis	7	8	6	4	12	4	6	38
C. guilliermondii	1	–	4	–	2	2	–	12
C. krusei	2	2	1	6	–	2	–	5
Other *Candida* species	6	3	3	3	5	13	3	26

Table 3: Other yeasts or yeast-like organisms causing systemic fungal infections

Genus	Species	Occurrence	Remarks
Cryptococcus	*C. neoformans* (*C. neoformans* var. neoformans	Common	Increased number of cases due to emergence of AIDS
	C. neoformans var. gattii)	Rare	Habitat – *C. neoformans* var. neoformans in bird droppings, *C. neoformans* var. gattii in *Eucalyptus* trees, but recently both varieties isolated from debris in hollows of several big trees (Jamun, Pipal, etc.)
	C. albidus, C. laurentii		
fleotrichum	*fl. candidum*	Very rare	Only in extremely debilitated individuals
Prototheca	*P. wickerhamii*	Extremely rare	
Blastoschizomyces	*B. capitatus*	Rare	In patients with leukemia, endocarditis and other immunosuppressive conditions
Pichia (Hansenula)	*P. anomala*	Outbreak	Three outbreaks reported mainly in pediatric units
	P. augusta	Rare	From mediastinal lymphadenitis
Malassezia	*M. furfur, M. globosa, M. pachydermatis, M. obtusa, M. restricta, M. slooffiae, M. sympoidalis*	Rare outbreak	Usually present on skin *M. furfur, M. pachydermatis* in neonatal intensive care unit Associated risk factor – parenteral lipid formulation
Rhodotorula	*R. rubra*	Extremely rare	From indwelling central venous catheters
Saccharomyces	*S. cerevisiae*	Extremely rare	Associated with health food, baking
Trichosporon	*T. asahii, T. asteroides, T. cutaneum, T. inkin, T. mucoides, T. ovides*	Rare	*T. asahii* and less often *T. mucoides* cause disseminated infection in patients with hematological malignancies or immunosuppression