Systematic Review

Global Epidemiology and Antifungal Resistance Trends of Candida Species (2000-2025): A Systematic Review of Species Distribution, Outbreaks, and Emerging Multidrug Resistance

Shaik Khaja Mohiddin[1], Bathala Nagasrilatha[2], C. N. Sowmya[1], P. Mounika[3], T. Mythili[4]

[1]Assistant Professor, Department of Microbiology, Government Medical College, Kadapa, Andhra Pradesh, India. [2]Professor and Head, Department of Microbiology, Government Medical College, Kadapa, Andhra Pradesh, India. [3]Final-year Postgraduate, Department of Microbiology, Government Medical College, Kadapa, Andhra Pradesh, India. [4]Second-year Postgraduate, Department of Microbiology, Government Medical College, Kadapa, Andhra Pradesh, India.

Abstract

Background: The objective is to synthesize data on the distribution, antifungal resistance trends of Candida spp., hospital outbreaks and emergence of multidrug resistance across the world from 2000 to 2025. **Material and Methods:** A systematic review was carried out following PRISMA 2020 guidelines. Systematic reviews, meta-analyses, multi-country surveillance reports, cohort studies, cross-sectional studies and outbreak investigations were identified via PubMed/MEDLINE, Embase, Web of Science and Scopus databases for Candida species-level identification from human clinical infections. Clinical specimen source, geographic distribution, antifungal susceptibility, mechanisms of antifungal resistance, outbreak characteristics and outcomes of death were retrieved. Due to the differences in the study platforms, breakpoints, clinical settings and surveillance techniques across regions, findings were summarized narratively and supplemented with pooled estimates from eligible published meta-analyses. **Results:** After duplicates were removed, 139 studies contributed to the qualitative synthesis from 2864 records of which 755 full-text articles were evaluated. Candida albicans remained important, but non-albicans Candida species collectively exceeded Candida albicans in several regions and clinical settings. Candida tropicalis, Candida parapsilosis, Candida glabrata, and Candida auris showed the most clinically relevant shifts. Fluconazole resistance was particularly high in Candida auris and increased in Candida tropicalis in parts of Asia. Echinocandin resistance was increasingly reported in Candida glabrata and Candida parapsilosis. Candida auris caused multiple healthcare-associated outbreaks because of environmental persistence, delayed identification, multidrug resistance, and inter-facility transmission. **Conclusion:** Candida epidemiology has changed substantially over the last 25 years. Species-level identification, standardized susceptibility testing, active surveillance, infection prevention, and antifungal stewardship are essential to reduce morbidity, mortality, and outbreak risk from resistant Candida infections.

Keywords: Candida; candidemia; antifungal resistance; Candida auris; systematic review.

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Introduction

Candida species are among the most important fungal pathogens in clinical medicine, producing disease that ranges from superficial mucocutaneous infection to life-threatening invasive candidiasis.[1] Historically, Candida albicans was the dominant etiological agent; however, surveillance from the last two decades demonstrates a gradual shift toward non-albicans Candida species, particularly C. tropicalis, C. parapsilosis, C. glabrata, and C. krusei.[2,3] This transition has been linked to broader antifungal exposure, larger immunocompromised populations, increased use of central venous catheters and other invasive devices, and prolonged survival of critically ill patients in intensive care units.[4,5]

The discovery of Candida auris in 2009 drastically altered the global mycological landscape.[6] Some of the most interesting characteristics of C. auris are its ability to develop resistance to fluconazole, resistance to echinocandins and amphotericin B, environmental persistence and efficient transmission in healthcare settings.[7,8] C. auris is known as a critical priority pathogenic fungus and its clinical importance and public health significance indicated by the World Health Organization classification.[9]

The mortality risk due to invasive candidiasis is still significant, especially for critically ill, immunosuppressed, neonatal, and postoperative patients.[10] Species susceptibility profile plays a key role in therapeutic decision, as it relies heavily on the accurate species identification. C. krusei is intrinsically resistant to fluconazole; C. glabrata also tends to have decreased azole susceptibility, plus emerging echinocandin susceptibility, and C.

Address for correspondence: Dr. Bathala Nagasrilatha, Professor and Head, Department of Microbiology, Government Medical College, Kadapa, Andhra Pradesh, India.
E-mail: lathabathala77@gmail.com

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parapsilosis can have decreased echinocandin susceptibility, as a result of FKS polymorphisms.[11-13] Differences between Clinical and Laboratory Standards Institute and European Committee on Antimicrobial Susceptibility Testing breakpoints, as well as variation in laboratory capacity, complicate cross-study comparisons.[14]

Reliable global estimation is also limited by underreporting from regions with restricted diagnostic access and weak mycology surveillance infrastructure.[15] An updated synthesis is therefore needed to integrate evidence from systematic reviews, surveillance networks, cohort studies, and outbreak reports. This systematic review summarizes global and regional species distribution, antifungal resistance trends, hospital outbreaks, and mortality patterns of clinically relevant Candida species from 2000 to 2025.

Materials and Methods

Protocol and reporting standards

This systematic review was prepared in accordance with the PRISMA 2020 statement.[16] A protocol defining the review question, eligibility criteria, search strategy, data extraction plan, and quality assessment approach was prepared before screening. The protocol was not registered.

Eligibility criteria

Eligible studies reported species-level identification of Candida isolates obtained from human clinical infections. Bloodstream, urinary, oral, esophageal, vulvovaginal, cutaneous, wound, device-associated, respiratory, central nervous system, and deep-seated infections were considered. Eligible designs included systematic reviews, meta-analyses, multicentric surveillance reports, large cohort studies, cross-sectional prevalence studies, and outbreak investigations. Single case reports were excluded except when they described rare species, early Candida auris detections, or sentinel events with clear epidemiological importance. Purely in vitro studies without clinical data were excluded.

Information sources and search strategy

PubMed/MEDLINE, Embase, Web of Science, and Scopus were searched for articles published between 1 January 2000 and 7 November 2025. Search terms included combinations of "Candida," "candidemia," "invasive candidiasis," "Candida auris," "non-albicans Candida," "antifungal resistance," "azole resistance," "echinocandin resistance," "systematic review," "meta-analysis," "surveillance," and "outbreak." Reference lists of eligible articles and major surveillance reports were screened to identify additional relevant records.[17-19]

Study selection and data extraction

Two reviewers independently screened titles and abstracts. Full texts were reviewed when records appeared relevant or when eligibility was uncertain. Disagreements were resolved by discussion and, when needed, by consultation with a third reviewer [20]. Extracted data included study year, country or region, study design, patient group, specimen type, identification method, Candida species distribution, antifungal susceptibility method, breakpoints used, resistance proportions, resistance mechanisms, outbreak setting, infection-control findings, and mortality outcomes.

Quality assessment

The quality of systematic reviews was evaluated using the AMSTAR 2.[21] Appraisal of observational studies was conducted on relevant domains of the Newcastle-Ottawa Scale and Joanna Briggs Institute checklists such as representativeness, sampling clarity, laboratory confirmation, ascertaining of outcome and completeness of reporting.[22,23] Case definition, species confirmation, environmental investigation, typing method, and description of control measures were evaluated in the outbreak reports.

Data synthesis

Due to the differences in geographical location, clinical setting, methods of diagnosis, susceptibility breakpoints, and reporting format of the included studies, no de novo meta-analysis was conducted. Rather, the results of the eligible published meta-analyses were collated and large surveillance and multicenter studies.[24–26] were included. Data has been sorted by clinical specimen type and species, by antifungal class, by resistance mechanism, by outbreak setting and by reported mortality.

Ethical considerations

Approval by an ethics committee was not warranted as this review was based on aggregate data that do not involve any contact with patients, patient data or animal experiments.

Results

Study selection: There were 3215 records found in the database search. Of the 2864 records screened by title and abstract after duplicate removal, 755 full-text articles were retrieved and assessed for eligibility. Of these, 616 were excluded for not meeting the eligibility criteria. Lastly, 139 studies provided data for the qualitative synthesis – systematic reviews, meta-analyses, surveillance reports, cohort studies and outbreak investigations. The PRISMA flow diagram is shown in Figure 1.

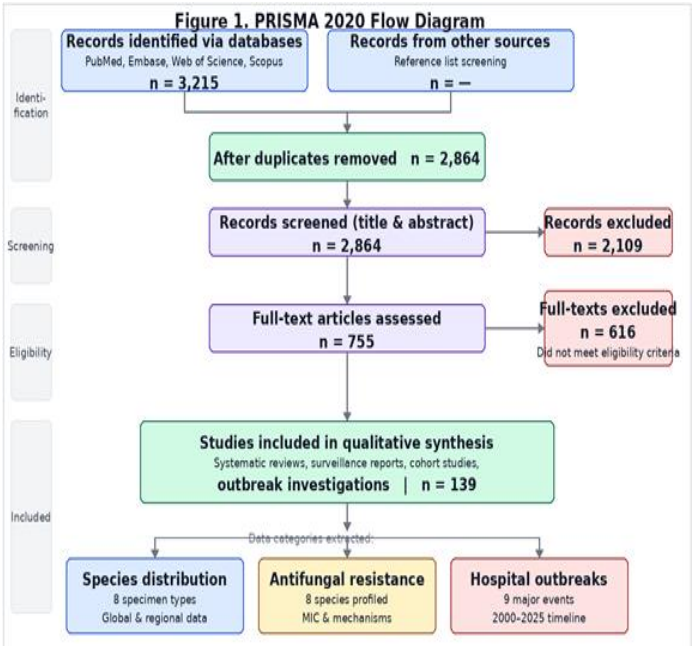

Figure 1: PRISMA 2020 flow diagram showing study selection for the systematic review of Candida epidemiology and antifungal resistance trends from 2000 to 2025.

Species distribution across clinical specimens

Species distribution varied by specimen type, patient population, and region. Bloodstream infections continued to be dominated by C. albicans, C. tropicalis, C. parapsilosis, and C. glabrata, with C. auris becoming increasingly important after 2010 in several countries.[27-30] A consistent shift toward non-albicans Candida species was evident, especially in Asia and South America.[30,31]

Urinary isolates were commonly represented by C. tropicalis and C. albicans, with C. tropicalis more frequently reported among patients with diabetes mellitus, catheterization, and prolonged hospitalization.[32] Vulvovaginal candidiasis remained predominantly associated with C. albicans, although C. glabrata was increasingly recognized in recurrent disease and after azole exposure.[33,34] Oral isolates consisted mainly of C. albicans, C. glabrata, C. tropicalis, and C. dubliniensis, with C. dubliniensis more frequently reported in immunosuppressed and human immunodeficiency virus-infected populations.[35]

Colonization was mostly associated with respiratory isolation, despite reports of invasive diseases among the most ill ventilated patients.[36] C. tropicalis and C. parapsilosis occurred frequently in burn wounds, postoperative tissue infections and device related infections.[37] Non-albicans Candida species like C. parapsilosis and C. guilliermondii contributed more to the problem of cutaneous and nail infections.[38] Central nervous system infections were rare but very serious, primarily in neonates and neurosurgical patients.[39]

Table 1: Distribution of major Candida species by clinical specimen type, 2000-2025

Sample type	Predominant species	Major epidemiological trend	Key references
Bloodstream infections	C. albicans, C. tropicalis, C. parapsilosis, C. glabrata, C. auris	Declining dominance of C. albicans with increasing non-albicans Candida; C. auris emerged as a multidrug-resistant bloodstream pathogen after 2010.	[27-30]
Urine	C. tropicalis, C. albicans, C. glabrata, C. krusei	C. tropicalis increased in catheterized, diabetic, and hospitalized patients; azole resistance was increasingly reported.	[32]
Vaginal swabs	C. albicans, C. glabrata, C. krusei, C. parapsilosis	C. albicans remained dominant; C. glabrata was associated with recurrent vulvovaginal candidiasis and prior azole exposure.	[33,34]
Oral cavity	C. albicans, C. glabrata, C. tropicalis, C. dubliniensis	C. dubliniensis was reported more frequently in immunosuppressed populations; C. glabrata and C. tropicalis increased among denture users and older adults.	[35]
Respiratory samples	C. albicans, C. tropicalis, C. glabrata	Most isolates represented colonization; invasive disease was reported mainly in critically ill and immunocompromised patients.	[36]
Wound and tissue	C. albicans, C. tropicalis, C. parapsilosis	C. tropicalis was prominent in burn wound infection; C. parapsilosis was frequent in postsurgical and catheter-associated infections.	[37]
Nail and skin	C. albicans, C. parapsilosis, C. guilliermondii	Non-albicans Candida species gained importance; topical azole exposure may contribute to resistance selection.	[38]
CNS/CSF	C. albicans, C. parapsilosis, C. glabrata	Rare but clinically severe; described mainly in neonates, neurosurgical patients, and immunocompromised hosts.	[39]

Abbreviations: CNS, central nervous system; ICU, intensive care unit; MIC, minimum inhibitory concentration; NAC, non-albicans Candida; NICU, neonatal intensive care unit.

Antifungal resistance trends

Species and geographical variability was observed in resistance pattern to fungicides. The global surveillance data showed low levels of resistance to fluconazole, echinocandin, and amphotericin B for C. albicans.[40] In contrast, C. tropicalis demonstrated an appreciable rise in fluconazole resistance in Asia, which was due to ERG11 mutations, efflux pump activity, biofilm formation, and antifungal selection pressure.[31,41]

A reduction in susceptibility to azoles and selection of echinocandin resistance due to FKS1 and FKS2 mutations were observed in C. glabrata.[42,43] C. parapsilosis was a growing concern due to decreasing echinocandin susceptibility and resistance signals reported in Europe and Latin America.[44] C. auris had the most alarming profile, with high fluconazole resistance; variable echinocandin resistance, and amphotericin B resistance dependent upon the clade.[28,29,45]

These patterns facilitate simple, routine species identification and susceptibility testing of clinically relevant isolates, with special focus on those from bloodstream infection, neonatal units, transplant services and intensive care units.[47]

Table 2: Antifungal resistance trends among major Candida species, 2000-2025

Species	Fluconazole resistance	Echinocandin resistance	Amphotericin B resistance	Major mechanisms and clinical implications	References
C. albicans	<10%; generally stable	<2%	<1%	ERG11 alteration and efflux pump activation in resistant isolates; resistance remains relatively uncommon in most surveillance datasets.	[40]
C. tropicalis	20-45% in several Asian reports; rising trend	<5%	<2%	ERG11 mutations, efflux pump activity, and biofilm-mediated tolerance; important in candidemia and candiduria.	[31,41]
C. glabrata	15-30%; reduced susceptibility and multidrug resistance	5-10%; mainly linked to FKS1/FKS2	<1%	Intrinsic azole tolerance with acquired resistance; echinocandin resistance threatens first-line therapy.	[42,43]

	increasingly reported	mutations			
C. parapsilosis	10-20%	5-12%; increasing in selected regions	<1%	FKS1 polymorphisms and biofilm formation on plastic devices; important in catheter-related and neonatal infection.	[44]
C. krusei	Intrinsic fluconazole resistance	<2%	1-4%	Altered azole target enzyme; species-level identification is essential before azole therapy.	[11,12]
C. auris	70-90%; frequently pan-azole resistant	5-10%; increasing in some clades and outbreaks	5-35%	ERG11 mutations, ERG pathway alterations, ABC transporter upregulation, environmental persistence, and clonal spread.	[28,29,45]
C. dubliniensis	<5%; uncommon	Rare	Rare	Closely related to C. albicans; resistance remains sporadic but should be monitored in recurrent infection.	[46]
C. guilliermondii	10-20%	2-5%	<2%	Reduced azole susceptibility and altered FKS gene signals reported in selected datasets.	[47]

Abbreviations: CNS, central nervous system; ICU, intensive care unit; MIC, minimum inhibitory concentration; NAC, non-albicans Candida; NICU, neonatal intensive care unit.

Hospital outbreaks

Infection outbreaks with Candida species were mainly associated with heavy device use, environmental contamination and susceptible hosts. Initial reports often embraced C. parapsilosis in neonatal and adult intensive care units due to hand transmission and catheter biofilms which persist.[48] In India, clusters of C. tropicalis have been reported in surgical and medical intensive care units with molecular evidence of clonal spread, as well as high level of fluconazole resistance.[49]

Since then, C. auris was the most problematic outbreak organism with large HA outbreaks reported in South Africa, UK, USA, India, Pakistan, Europe and Latin America (50-53). Some organisms have been identified only late, causing colonization to be slow to occur, survival in the environment and resistance to the commonly used antifungals. This allowed dissemination to be rapid between wards and institutions.[62-66]

Table 3: Major hospital outbreak patterns due to Candida species, 2000-2025

Period	Country/region	Species	Setting/source	Key outbreak findings	References
2000-2006	United States and Europe	C. parapsilosis	NICU/ICU; catheter-related infection	Hand transmission and catheter biofilms sustained outbreaks among neonates and critically ill patients.	[48]
2009-2013	India	C. tropicalis	Surgical and medical ICUs; urinary catheters	Fluconazole resistance and clonal spread reported in high-risk units.	[31,49]
2011-2016	South Africa	C. auris	ICUs and bloodstream infections	Large-scale healthcare-associated emergence with high azole resistance and significant mortality.	[50,63]
2012-2017	United Kingdom	C. auris	Critical care units	Environmental contamination, surface persistence, and fomite transmission documented.	[51,62]
2013-2019	United States	C. auris	Long-term care and acute-care hospitals	Multistate transmission with emerging echinocandin resistance signals.	[52,64]
2014-2020	Spain and Italy	C. parapsilosis	Surgical wards and catheter use	Echinocandin-resistant isolates and hand hygiene gaps reported.	[60,61]
2015-2023	India and Pakistan	C. auris	ICUs and ventilator-associated settings	Clade I dominance, hospital-to-hospital dissemination, and high mortality were reported.	[28,45,66]
2017-2022	Latin America	C. parapsilosis and C. glabrata	NICUs and surgical units	Persistent colonization and environmental reservoirs contributed to recurrent clusters.	[19]
2018-2025	Global	C. auris	Multicountry healthcare facilities	Global recognition as a critical-priority fungal pathogen with continued outbreaks despite control measures.	[9,53,77]

Abbreviations: CNS, central nervous system; ICU, intensive care unit; MIC, minimum inhibitory concentration; NAC, non-albicans Candida; NICU, neonatal intensive care unit.

Discussion

There is an evident shift in the epidemiology of Candida over the last two and a half decades as shown in this systematic review. C. albicans is still a significant pathogen, but there are now several datasets that indicate that non-albicans Candida species are now contributing a greater proportion, particularly C. tropicalis, C. parapsilosis, C. glabrata and C. auris.[54,55] Clinically significant because non-albicans Candida may be less susceptible to azoles, echinocandins or both.

The emergence of C. tropicalis in Asia is of particular importance in India and its neighbouring nations. It has been consistently reported to be associated with diabetes mellitus, catheterization, intensive care exposure and urinary or blood stream infection.[56] The high resistance rates reported in some regions, which are higher than 40%, indicate a potential for clonal expansion, ERG11-mediated resistance, biofilm tolerance, and extensive azole use creating the appearance of a high level of resistance. The results confirm local antibiogram-based antifungal policies as opposed to using global averages.

In many high-income environments, C. glabrata is a significant pathogen which wields a low susceptibility to azoles and ability to acquire echinocandin resistance. Clinically, the FKS mutations are important as the echinocandins are used as first-line treatment

for invasive candidiasis [58,59]. Likewise, C. parapsilosis has been considered a strong correlate to catheter related infection and neonatal sepsis. In Europe and Latin America, reports of C. parapsilosis isolates resistant to echinocandin drugs were particularly noteworthy because C. parapsilosis is a species that is known to be capable of forming biofilms on plastic surfaces and spread via hand-to-hand contact.[60,61] C. auris represents the most significant healthcare-associated threat among Candida species in recent decades. It is unique among Candida spp. for its ability to survive in the environment, to colonize without causing clinical symptoms, to be multi-drug resistant, and to be transmitted in healthcare settings.[62–66] Therapeutic uncertainty exists due to the high level of fluconazole resistance, and the relative lack of echinocandin resistance and the presence of clade-dependent amphotericin B resistance.[66] The features are the reasons why species identification, patient isolation, contact screening, environmental cleaning (with effective disinfectants) and molecular surveillance are all necessary in outbreak settings.[53,74-77]

Patterns also need to be considered which are specific to the type of specimen. Although C. albicans remains a prominent cause of oral candidiasis, other species of Candida, such as C. dubliniensis, C. glabrata and C. tropicalis are gaining significance in immunocompromised patients and individuals wearing dentures.[35,67] C. albicans is still the most common organism involved in RVC, with the other most common species occurring after exposure to azole antifungals, such as C. glabrata.[33,69] Increased Nail and Skin infection recovery of non-albicans Candida may be due to topical azole utilization and shifting environmental reservoirs.[70]

The review points to some critical surveillance gaps worldwide. Routine MALDI-TOF mass spectrometry, molecular identification and standardized susceptibility testing is not available in many LMIC areas. The delayed response of infection control measures may be caused by the confusion of the uncommon Candida species, C. auris and others, with other species.[75] Thus, investment in lab capacity, uniform break points for the AGPs, antifungal stewardship, and genomic epidemiology is necessary to identify resistant clones early and prevent spread between facilities.[76,77]

Strengths and limitations

This review's major limitation is that the evidence was synthesized across specimen types and regions, as well as across antifungal classes and outbreak situations, which likely introduces heterogeneity into the evidence. It combines with systematic reviews, large surveillance studies and outbreak investigations to give an overview of the changing epidemiology of Candida in a clinically useful way. Main limitations include: study design, laboratory methodology, susceptibility breakpoints and reporting quality. Certain areas are under-represented, and a number of resistance estimates were based on monitored networks rather than population-based networks. A de novo meta-analysis was not performed because of this heterogeneity.

Conclusion

There have been significant changes in Candida epidemiology between 2000 and 2025. However, non-albicans Candida species are becoming more common, and resistance to antifungals is growing in clinically relevant species like C. tropicalis, C. glabrata, C. parapsilosis and C. auris. Multidrug resistance, environmental stability and outbreak potential make C. auris a significant threat to healthcare. Specifics of species identification, standardized susceptibility testing, targeted antifungal stewardship and strong infection-prevention strategies form the basis of good outcomes. Increased surveillance and genomic epidemiology is required to track emergence of resistance and decrease the burden of invasive candidiasis worldwide.

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Conflicts of interest

There are no conflicts of interest.

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