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The Emerging Global Burden of Fungal Infections: Epidemiology, Climate Change, Antifungal Resistance and Public Health Challenges

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Abstract Fungal infections constitute an increasingly important global public health problem and are associated with substantial morbidity and mortality, particularly among immunocompromised and critically ill individuals. They range from highly prevalent superficial mycoses to invasive and disseminated infections with high case-fatality rates. Opportunistic pathogens, such as *Aspergillus*, *Candida*, *Cryptococcus*, *Rhodotorula*, *Geotrichum*, and *Trichosporon* cause diverse clinical syndromes, whereas endemic dimorphic fungi including *Blastomyces*, *Coccidioides*, *Histoplasma*, *Paracoccidioides*, and *Talaromyces* are strongly influenced by environmental and geographic exposure. Recent global estimates suggest that more than 6.5 million life-threatening invasive fungal infections occur annually, with approximately 3.8 million associated deaths. Climate change may alter fungal ecology, geographic distribution, and human exposure, while antifungal resistance increasingly compromises the effectiveness of the limited therapeutic options available. This review summarizes the epidemiology, major opportunistic and endemic mycoses, global morbidity and mortality, climate-related emergence, and antifungal resistance, and highlights the need for improved surveillance, laboratory diagnosis, antifungal stewardship, access to effective treatment, and coordinated One Health interventions.

Keywords: *Antifungal resistance, Climate change, Endemic mycoses, Fungal infections, Global burden, One Health, Opportunistic mycoses*

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1. Introduction

Fungi are eukaryotic organisms that occur as yeasts, moulds, or thermally dimorphic forms and are widely distributed in environmental and host-associated niches [1]. It is believed that around 5 million species of fungi are present in the world, out of which about 600 have been found to be implicated with various clinical disorders of humans as well as many species of animals [2]. Fungal infections have been encountered in both sexes, all age groups, and can occur in all seasons, and in urban and rural settings [1]. Human fungal diseases range from superficial and mucocutaneous infections to subcutaneous and invasive systemic mycoses. Superficial fungal infections are extremely common, whereas invasive fungal diseases are less frequent but are associated with substantial morbidity and mortality because diagnosis may be delayed and treatment options are limited [3]. The burden is particularly high among individuals with impaired host defenses, including patients with malignancy, human immunodeficiency virus (HIV) infection, organ transplantation, neutropenia, diabetes mellitus, and those receiving corticosteroids, cytotoxic drugs, or other immunosuppressive therapies [1]. It is important to mention that about 90% of invasive fungal infections have been described in immunocompromised subjects [2]. Important opportunistic pathogens include *Aspergillus*, *Candida*, *Cryptococcus*, *Geotrichum*, and *Trichosporon* species [1,4,5].

Endemic fungal infections are associated with defined

ecological niches and geographic regions and are commonly acquired from environmental reservoirs, particularly soil enriched with organic material or animal excreta [6]. Inhalation of infectious propagules can cause disease even in immunocompetent individuals. Major endemic or thermally dimorphic pathogens include *Blastomyces dermatitidis*, *Coccidioides immitis*, *Coccidioides posadasii*, *Histoplasma capsulatum*, *Paracoccidioides brasiliensis*, *Paracoccidioides lutzii*, and *Talaromyces marneffei*; *Sporothrix* species also cause important implantation and zoonotic mycoses [7,8]. Contemporary estimates indicate that life-threatening fungal diseases impose a much larger global burden than previously recognized [9]. Accordingly, this review delineates the epidemiology and public health importance of major opportunistic and endemic fungal infections, with particular emphasis on global burden, climate-related changes in fungal disease, and the emerging challenge of antifungal resistance.

2. Opportunistic Mycoses

2.1. Aspergillosis

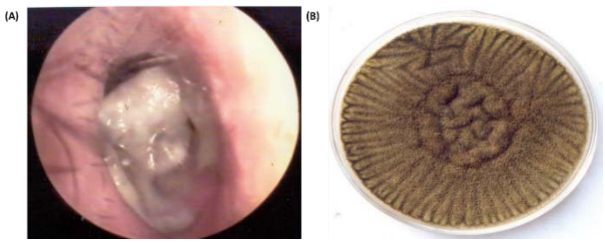

Figure 1. Otomycosis due to *Aspergillus niger*. (A) Greyish-black fungal material on endoscopic examination; (B) A. niger culture on Sabouraud dextrose agar with chloramphenicol. Source: [1]

Aspergillosis is caused predominantly by *Aspergillus fumigatus*, although several other species may be implicated, including *Aspergillus amstelodami*, *Aspergillus candidus*, *Aspergillus chevallieri*, *Aspergillus clavatus*, *Aspergillus deflectus*, *Aspergillus flavus*, *Aspergillus glaucus*, *Aspergillus nidulans*, *Aspergillus niger*, *Aspergillus ochraceous*, *Aspergillus restrictus*, *Aspergillus sydowii*, *Aspergillus tamari*, *Aspergillus terreus*, *Aspergillus udagawae*, *Aspergillus ustus*, and *Aspergillus versicolor* [1,10]. The disease can occur in sporadic as well as in epidemic form. Nosocomial outbreaks of aspergillosis have been documented in the patients who were residing near the construction work [1]. It is important to mention that the lungs were involved in about 75% of *Aspergillus* infection in HIV patients [1]. *Aspergillus* species are ubiquitous in soil, air, water, and decaying plant material, and infection is usually acquired by inhalation of airborne conidia; traumatic inoculation can also produce localized disease. *Aspergillus fumigatus* is the species most frequently associated with invasive disease in humans and animals [11]. Dave and Pal [12] additionally documented primary cutaneous aspergillosis caused by *A. fumigatus* in a 34-year-old immunocompetent poultry farmer following minor trauma while cleaning a poultry cage; direct microscopy and culture of a skin biopsy confirmed the infection, and environmental investigation recovered the organism from the patient's immediate surroundings. *Aspergillus niger* is an important cause of otomycosis, as illustrated in Figure 1.

2.2. Candidiasis

Candidiasis comprises mucosal, cutaneous, and invasive infections caused by *Candida* species [1]. *Candida* commonly colonizes the oral cavity, gastrointestinal tract, genital tract, and skin without causing disease; however, disruption of host defenses or local microbial balance can permit pathogenic overgrowth. Oropharyngeal candidiasis (thrush) typically presents with white oral plaques, soreness, and dysphagia, whereas vulvovaginal candidiasis produces genital symptoms and is common among women [13]. Mucosal candidiasis is especially frequent in patients with HIV infection or acquired immunodeficiency syndrome (AIDS), malignancy, or other forms of immunosuppression. A severe case of oropharyngeal candidiasis caused by *Candida albicans* is shown in Figure 2.

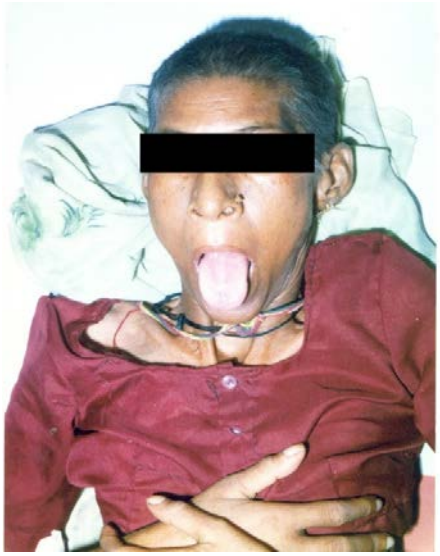

Figure 2. Severe oropharyngeal candidiasis due to *Candida albicans* in a woman with AIDS and tuberculosis; *C. albicans* was isolated from tongue scrapings on Pal sunflower seed medium

Candida albicans remains a major human pathogen, but non-albicans *Candida* species are also clinically important, particularly in invasive disease and in patients exposed to broad-spectrum antibiotics, corticosteroids, cytotoxic agents, indwelling devices, or prolonged hospitalization [4,14]. Of particular concern, *Candida auris* is a healthcare-associated pathogen capable of persistent colonization, nosocomial transmission, and resistance to multiple antifungal classes; the World Health Organization (WHO) includes *C. auris* among its critical-priority fungal pathogens [15,16]. Pal [17] was among the early investigators to report the isolation of *Candida tropicalis* from lung empyema in elderly, debilitated patient with diabetes, demonstrating the pathogenic potential of this opportunistic species. On Pal sunflower seed medium, *Candida* species produce white to cream colonies, whereas *Cryptococcus neoformans* develops a characteristic brown pigmentation, as shown in Figure 3.

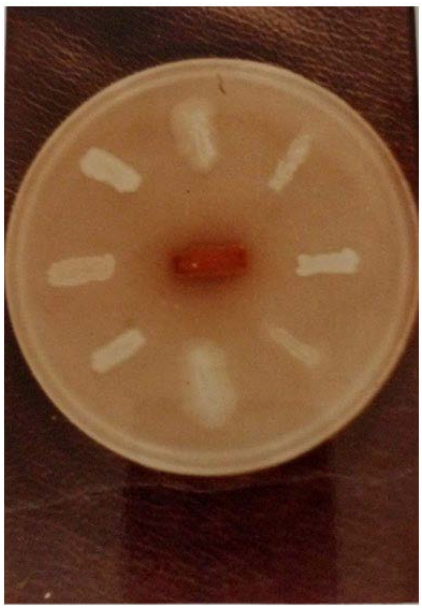

Figure 3. Colony appearance of *Candida* and *Cryptococcus neoformans* on Pal sunflower seed medium. *Candida* species form white-to-cream colonies, whereas *Cryptococcus neoformans* produces brown-pigmented colonies. Source: [18]

2.3. Cryptococcosis

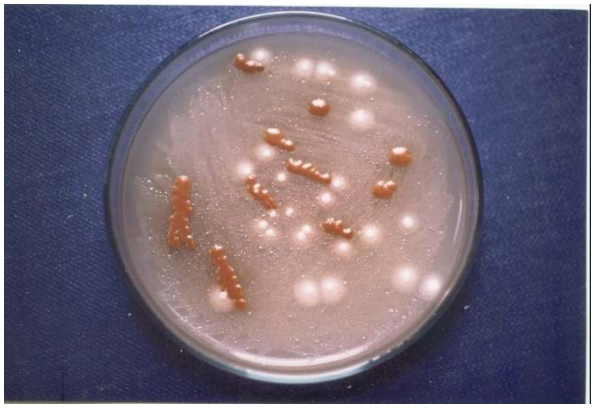

Figure 4. *Cryptococcus neoformans* isolated from clinical material of an immunocompromised patient on Pal sunflower seed medium. Source: [23]

Cryptococcosis is an environmentally acquired mycosis caused mainly by *Cryptococcus neoformans* and *Cryptococcus gattii* [19]. Infection most commonly follows inhalation of desiccated yeast cells or basidiospores from environmental reservoirs, and may remain confined to the lungs or disseminate, particularly to the central nervous system. It is reported that *C. neoformans* can survive for about 20 years in dry and old pigeon droppings that are not exposed to direct sunlight [20]. The burden remains substantial: a recent global analysis estimated approximately 194,000 cases of cryptococcal meningitis and 147,000 associated deaths annually [9], replacing older estimates that were largely derived from specific HIV-associated populations. Dave and Pal [21] reported primary cutaneous cryptococcosis in an immunocompetent parrot keeper who sustained minor trauma while cleaning a cage; *Cryptococcus neoformans* was isolated from the skin lesion and from the immediate avian environment, supporting environmental acquisition. Clinical manifestations vary with the site of infection and may include cough, fever, dyspnea, headache, neurological symptoms, and cutaneous lesions [22]. Pal sunflower seed medium developed by Pal in 1980 has been used in microbiology and public health laboratories for the rapid isolation and identification of *C. neoformans* from clinical as well as environmental specimens [1,20]. *Cryptococcus neoformans* imparts light to dark brown colored mucoid colonies on Pal sunflower seed medium. Figure 4 illustrates isolation of *Cryptococcus neoformans* from a clinical specimen from an immunocompromised patient on Pal sunflower seed medium.

2.4. Mucormycosis

Mucormycosis is an emerging and re-emerging fungal infection reported globally, including in countries like India. It is caused by filamentous fungi from the genera *Lichtheimia*, *Cunninghamella*, *Mucor*, *Rhizomucor*, and *Rhizopus*. These fungi are widely present in the environment and can invade various organs of the body, such as the sinuses, lungs, brain, heart, kidneys, and joints. The infection is exogenous, with the respiratory tract being the primary route of entry for the fungi. *Mucor*mycosis can manifest in both sporadic and epidemic forms, with most cases attributed to *Rhizopus oryzae* [24].

*Mucor*mycosis presents in several clinical forms, including cutaneous, subcutaneous, rhinocerebral, gastrointestinal, pulmonary, and systemic mucormycosis [1,25]. Less common forms include pyelonephritis, endocarditis, and osteomyelitis. Clinical manifestations of mucormycosis can include fever, headache, shortness of breath, chest pain, coughing, hemoptysis, eyelid edema, pain and redness around the eyes and nose, blurred or double vision, protruding eyes, sudden loss of vision, nasal discharge, stuffy nose, diminished sense of smell, facial paresthesia, skin lesions, toothaches, and altered mental status [1,25].

2.5. Rhodotoruliosis

Rhodotoruliosis is an opportunistic and emerging mycotic disease affecting both humans and animals [1]. It is caused by *Rhodotorula*, a basidiomycetous yeast that acts as a saprophyte in the environment. The disease can occur sporadically or in epidemic forms with exogenous sources of infection. The most frequently encountered *Rhodotorula* species include *Rhodotorula glutinis*, *Rhodotorula mucilaginosa* (formerly *Rhodotorula rubra*), and *Rhodotorula minuta*. These species can be isolated from a variety of sources, such as soil, air, plants, and household environments [1,26].

In humans, *Rhodotorula* has been associated with infections such as fungemia, meningitis, ventriculitis, peritonitis, endocarditis, keratitis, endophthalmitis, hydrosalpinx, oral ulcers, and lymphadenitis. Systemic infection occurs in patients who are immunocompromised [1]. In animals, it causes skin infections in chickens, sea lions, and cats; lung infections in sheep; epididymitis in dogs; and mastitis in cows and buffalo. *Rhodotorula* is an opportunistic pathogen that takes advantage of immunosuppressive conditions, indwelling devices, and antibiotic use [26].

2.6. Geotrichosis

Geotrichosis is an uncommon opportunistic mycosis caused predominantly by *Geotrichum candidum*, a ubiquitous saprophytic, yeast-like filamentous fungus that can occur in soil, water, air, food products, and as a commensal of the human digestive, respiratory, and cutaneous microbiota [1]. Disease is reported most often in individuals with impaired host defenses, including patients with malignancy, diabetes mellitus, HIV infection, leukosis, or organ transplantation, although clinically significant infection can also occur in immunocompetent hosts. Reported manifestations include oral, pulmonary, urinary, cutaneous, ocular, osteoarticular, and disseminated disease [27,28,29,30].

Clinical diagnosis requires careful correlation because *Geotrichum* may be recovered as a colonizer or contaminant. Direct microscopy demonstrating hyaline septate hyphae and rectangular arthroconidia, together with repeated isolation from appropriate clinical specimens, supports etiologic significance. Dave and Pal [29] demonstrated *G. candidum* in oral lesions of an immunocompromised patient; however, Ramya and et al. [27] reported pulmonary geotrichosis in an immunocompetent patient. Urinary infection due to *G. candidum* was described in an elderly patient with lung cancer [28]. A recent review of cutaneous geotrichosis noted that localized disease may follow trauma or burns, whereas disseminated cutaneous involvement occurs mainly in immunosuppressed patients; amphotericin B and voriconazole have been among the most frequently used systemic agents, although standardized clinical breakpoints for *Geotrichum* remain unavailable [30].

2.7. Trichosporonosis

*Trichosporon*osis is an opportunistic mycosis caused by basidiomycetous yeast-like fungi of the genus *Trichosporon*, which are widely distributed in nature and may colonize human skin, the gastrointestinal tract, and the respiratory tract [1]. *Trichosporon asahii* is the species most often associated with invasive disease, particularly in patients with hematological malignancies, neutropenia, organ transplantation, prolonged antibiotic exposure, or indwelling intravascular devices. Clinical presentations range from superficial white piedra to fungemia and disseminated infection with pulmonary or cutaneous involvement. Disseminated infection carries a grave prognosis, and hence, early diagnosis and prompt treatment are imperative [1]. *Trichosporon* species are intrinsically resistant to echinocandins and may show reduced susceptibility to amphotericin B; triazoles, particularly voriconazole, are generally preferred for invasive trichosporonosis [31].

3. Endemic Dimorphic Mycoses

3.1. Blastomycosis

Blastomycosis is caused by the thermally dimorphic fungus *Blastomyces dermatitidis*, which thrives in moist, acidic soil [1]. This species is the primary cause of infections in North America, especially around the Great Lakes, the St. Lawrence Seaway, and various south-central and southeastern states. Recently, other *Blastomyces* species have been identified as causing disease in different parts of the world [32].

Infection typically results from inhaling conidia from the mold phase of the fungus. Acute pulmonary infections can range from asymptomatic to typical community-acquired pneumonia, while chronic forms may present as mass-like lesions or cavitary pneumonia. In rare cases, pulmonary infections may progress to acute respiratory distress syndrome, which carries a high mortality rate. After an initial pulmonary infection, the yeast form of *Blastomyces* can spread hematogenously, often leading to cutaneous lesions. Additionally, it can involve the osteoarticular system, genitourinary tract, and central nervous system [32].

3.2. Coccidioidomycosis

Coccidioidomycosis, commonly known as Valley Fever [1], is an invasive dimorphic fungal infection caused by *Coccidioides immitis* and *Coccidioides posadasii*. These fungi are found in the arid desert soils of the southwestern United States, as well as in parts of Mexico, Central America, and South America [33]. Most *Coccidioides* infections are acquired by inhaling airborne arthroconidia. Occasionally, the infection can be acquired through primary cutaneous inoculation, leading to skin infections, or via organ transplantation from infected donor lungs, livers, or kidneys, resulting in disseminated disease [34,35]. Rare cases of neonatal transmission have also been reported, primarily due to the aspiration of infectious vaginal secretions during childbirth [36].

3.3. Histoplasmosis

Histoplasmosis is an environmentally acquired systemic mycosis caused by the thermally dimorphic fungus *Histoplasma capsulatum*. Infection usually follows inhalation of airborne microconidia from contaminated soil and most commonly begins in the lungs [37]. The majority of exposed immunocompetent individuals remain asymptomatic or develop a mild, self-limited respiratory illness; however, symptomatic pulmonary disease may present with fever, headache, non-productive cough, dyspnea, chest pain, and constitutional symptoms. In patients with impaired cell-mediated immunity or heavy exposure, infection may disseminate hematogenously and involve the skin, mucous membranes, lymphoreticular system, gastrointestinal tract, adrenal glands, or central nervous system. Because the clinical presentation can mimic tuberculosis and other systemic infections, laboratory confirmation is essential, particularly in endemic areas and among people living with HIV [6,37].

3.4. Paracoccidioidomycosis

Paracoccidioidomycosis is a chronic systemic endemic mycosis caused principally by *Paracoccidioides brasiliensis* and *Paracoccidioides lutzii* [38]. Infection is acquired mainly through inhalation of environmental propagules, producing a primary pulmonary infection that

may remain subclinical or progress to chronic respiratory disease. Hematogenous or lymphatic dissemination can involve the oral and nasal mucosa, skin, lymph nodes, gastrointestinal tract, and other internal organs, where painful ulcerative lesions and systemic manifestations may occur [38,39]. Natural infection has also been documented in animals, including dogs and several wild species. Canine cases may present with weight loss, generalized lymphadenopathy, hepatosplenomegaly, and disseminated disease, supporting the ecological and One Health relevance of *Paracoccidioides* in endemic areas [40,41].

3.5. Talaromycosis (formerly penicilliosis)

Talaromycosis is an endemic systemic mycosis caused by *Talaromyces marneffei*, a thermally dimorphic fungus formerly known as *Penicillium marneffei*. The disease is particularly important among people with advanced HIV infection and other forms of immunosuppression in endemic regions of Southeast Asia and southern China [42]. *Talaromyces marneffei* grows as a mould at approximately 25 °C and converts to a yeast-like form at 37 °C, a transition that is central to tissue invasion and pathogenesis [43]. Phylogenetic and taxonomic studies transferred the species from *Penicillium* to *Talaromyces*; therefore, the current disease name talaromycosis should be used, with penicilliosis retained only as a historical synonym [44,45].

Talaromycosis commonly presents as a disseminated febrile illness with weight loss, anemia, lymphadenopathy, respiratory symptoms, hepatosplenomegaly, and characteristic skin lesions, and it may clinically resemble histoplasmosis, cryptococcosis, or tuberculosis. Although infection is best recognized in humans, *T. marneffei* and related *Talaromyces* species have also been investigated in animals and environmental reservoirs, underscoring the importance of accurate species identification and ecological surveillance [46,47].

4. Global Morbidity and Mortality Due to Fungal Infections

Fungal diseases constitute a substantial but frequently underestimated cause of morbidity and mortality worldwide. Their clinical impact ranges from highly prevalent superficial and mucosal infections to life-threatening invasive and disseminated mycoses that may cause prolonged hospitalization, organ dysfunction, disability, chronic sequelae, and death. The burden is especially severe among patients with HIV/AIDS, malignancy, diabetes mellitus, neutropenia, organ transplantation, critical illness, and those receiving prolonged corticosteroid, antibiotic, cytotoxic, or other immunosuppressive therapies. Opportunistic fungi, such as *Aspergillus*, *Candida*, *Cryptococcus*, and *Pneumocystis* account for a major proportion of severe fungal disease [48].

More recent global estimates indicate that severe fungal disease is substantially more common than previously recognized. Denning [9] estimated approximately 6.5 million life-threatening invasive fungal infections annually and about 3.8 million associated deaths worldwide, of which roughly 2.5 million deaths were directly attributable to fungal disease. These estimates emphasize that invasive mycoses represent a major component of global infectious-disease mortality. The true burden is likely to remain underestimated because surveillance is limited, many infections are not diagnosed, access to mycology laboratories is uneven, and fungal disease may be recorded only as a complication of the underlying condition [9,49]. The principal current estimates are summarized in Table 1.

Table 1. Estimated global annual incidence and crude mortality of selected severe fungal diseases

Fungal disease / syndrome	Estimated annual cases	Estimated crude deaths
Invasive aspergillosis	2,113,000	1,801,000
Chronic pulmonary aspergillosis	1,837,272	340,000
Candida bloodstream infection/invasive candidiasis	1,565,000	995,000
Pneumocystis pneumonia	505,000	214,000
Cryptococcal meningitis	194,000	147,000
Disseminated histoplasmosis in AIDS	71,000	66,000
Talaromycosis	19,000	9,000
Mucormycosis	211,000	84,000
Coccidioidomycosis	30,000	2,000
All life-threatening fungal diseases (total estimate)	6,548,000	3,752,000

Note: Estimates are modeled global annual figures and should not be interpreted as complete surveillance counts. Disease definitions and confidence vary by syndrome. Source: [9]

The consequences of fungal disease extend beyond mortality. Survivors may experience chronic respiratory impairment, neurological disability, recurrent infection, visual loss, or other long-term sequelae depending on the pathogen and site of infection. In resource-limited settings, delayed diagnosis, restricted access to sensitive laboratory tests, and limited availability or affordability of antifungal medicines further increase morbidity and the risk of poor outcomes. Strengthening surveillance, diagnostic capacity, and access to effective treatment is therefore essential for accurately defining and reducing the global burden of mycoses [49].

5. Impact of Climate Change on Fungal Infections

Climate change is increasingly recognized as an important environmental factor capable of influencing the ecology, geographic distribution, and epidemiology of fungal pathogens. Changes in temperature, rainfall, humidity, drought patterns, and the frequency of extreme weather events can alter fungal growth, sporulation, survival, dispersal and human exposure. Warming temperatures and changing precipitation patterns may also increase the suitability of previously non-endemic areas for some soil-associated fungi, while environmental disturbances such as dust storms, floods, wildfires, deforestation, and land-use change can increase aerosolization or redistribution of infectious fungal propagules [50]. *Cryptococcus gattii* provides an important example of a fungal pathogen whose ecological

range may be influenced by environmental and climatic conditions. Traditionally associated with tropical and subtropical regions, *C. gattii* has also caused infections and outbreaks in temperate regions. Global warming has been proposed as one factor contributing to its emergence in some geographical areas, while temperature, humidity, urbanization, deforestation, and other environmental changes may influence the survival and dispersal of *Cryptococcus* species [19,23]. Similar climate-related shifts are being considered for endemic mycoses such as coccidioidomycosis and histoplasmosis, where changes in soil conditions, rainfall, drought, and temperature can modify habitat suitability and airborne exposure [50].

Rising environmental temperatures may also impose selective pressure on fungi, potentially favoring organisms that can tolerate temperatures closer to those of the human body. Climate-related changes have consequently been discussed in relation to the emergence of new fungal threats and to changes in antifungal resistance. However, the relationship between climate change and the emergence of individual fungal pathogens is complex, and climate change should be considered one of several interacting ecological, biological, and socioeconomic drivers rather than the sole cause of fungal emergence. Continued environmental surveillance integrating human, animal, and ecosystem health is needed to better predict how changing climatic conditions will influence future patterns of fungal disease [50].

6. Antifungal Resistance: An Emerging Public Health Challenge

Antifungal resistance has emerged as a major challenge in the prevention and treatment of invasive fungal diseases. Therapeutic options are intrinsically limited because systemic therapy relies primarily on a small number of drug classes, including azoles, polyenes, echinocandins, and flucytosine. Resistance can delay effective therapy, prolong hospitalization, increase healthcare costs, and contribute to treatment failure and mortality. In response to the growing threat, the World Health Organization published the first Fungal Priority Pathogens List in 2022, identifying 19 fungal pathogens and prioritizing actions in surveillance, laboratory capacity, research and development, and public health intervention [15].

Resistance is particularly important among *Candida* and *Aspergillus* species. *Candida auris* is a healthcare-associated pathogen with a marked capacity for nosocomial transmission, environmental persistence, and resistance to multiple antifungal classes, whereas azole-resistant *Aspergillus fumigatus* increasingly complicates the management of invasive aspergillosis. Other *Candida* species may show intrinsic or acquired resistance to specific antifungal agents, making species-level identification clinically important. Molecular mechanisms include alteration or overexpression of drug targets, activation of efflux pumps, changes in membrane sterol pathways, stress-response adaptation, aneuploidy and other genomic changes, and biofilm-associated tolerance. Alterations involving ERG11 in *Candida* and cyp51A in *A. fumigatus* are well-described contributors to azole resistance [51].

Antifungal resistance may emerge during prolonged or repeated clinical therapy, but environmental selection is also a major concern. Agricultural use of azole fungicides can select azole-resistant environmental strains of *A. fumigatus* because agricultural and medical azoles act on related fungal sterol pathways. Resistant conidia can subsequently be inhaled by individuals with no prior exposure to medical azoles, illustrating the interconnected human, agricultural, veterinary, and environmental dimensions of antifungal resistance. A One Health response should therefore combine antifungal stewardship, species-level identification, susceptibility testing when indicated, infection prevention and control, environmental and clinical surveillance, and responsible antifungal use across sectors [49,51].

An additional concern is the limited innovation and unequal access to fungal diagnostics and therapeutics. The WHO antifungal development landscape reported in 2025 that only four new antifungal drugs had received regulatory approval in the United States, European Union, or China during the preceding decade, while relatively few candidates had advanced to late-stage clinical development. Diagnostic gaps are also substantial, particularly in low- and middle-income countries, where delayed or unavailable species identification and susceptibility testing can promote inappropriate therapy. The 2026 WHO implementation blueprint consequently emphasizes coordinated national surveillance, strengthened mycology laboratory networks, equitable access to essential diagnostics and antifungals, stewardship, infection prevention, and sustained research investment as central components of the response to fungal disease and antifungal resistance [49,52].

7. Conclusions and Recommendations

Fungal infections are important causes of morbidity and mortality across all age groups and in both developing and developed countries of the world. Opportunistic mycoses such as aspergillosis, candidiasis, cryptococcosis, mucormycosis, rhodotoruliosis, geotrichosis, and trichosporonosis are especially consequential in immunocompromised and critically ill patients, whereas endemic mycoses including blastomycosis, coccidioidomycosis, histoplasmosis, paracoccidioidomycosis, and talaromycosis remain important in regions where environmental exposure occurs. The increasing global burden, changing ecology of fungal pathogens, and emergence of antifungal resistance reinforce the need for timely diagnosis, effective treatment, surveillance, antifungal stewardship, and integrated One Health prevention strategies.

Based on these conclusions, the following recommendations are proposed:

- Promote veterinary care and monitoring for fungal infections in domestic and wildlife animals to prevent zoonotic transmission.
- Encourage the use of advanced molecular and serological methods to differentiate between fungal species and strains.
- Increase awareness and education about fungal infections, focusing on risk factors, symptoms, and

preventive measures.

- Support research into new antifungal agents and treatment regimens to address resistant strains and improve patient outcomes.
- Implement strategies to reduce environmental exposure to fungal pathogens, particularly in areas where endemic fungi are prevalent.
- Further research work to elucidate the etiologic significance of opportunistic fungi in various clinical disorders of humans as well as animals may be rewarding.

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Contribution of Authors

All authors made significant contributions to the preparation and revision of this review.

Statement of Competing Interests

The authors have no competing interests.

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List of Abbreviations

WHO: World Health Organization;
HIV: Human immunodeficiency virus
AIDS: Acquired immunodeficiency syndrome

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