IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Disease-Causing Forest Fungi: Environmental Reservoirs, Pathogenesis, And Emerging Threats To Human Health

Aditya Gautam[1], Anjali Singh[2], Dr. Kamlesh kumar Gautam[3]
1,2: Research scholar
3: Associate Professor
Department of Botany, Udai Pratap College, Varanasi

Abstract
Forest ecosystems harbours a diverse array of fungi, many of which serve ecological roles as decomposers or symbionts. However, a subset of these environmental fungi, particularly thermally dimorphic species, poses significant risks to human health when spores are aerosolized or introduced via trauma. This review synthesizes current knowledge on key forest-associated fungal pathogens, including *Cryptococcus gattii*, *Histoplasma capsulatum*, *Blastomyces dermatitidis*, and *Sporothrix schenckii*. These organisms thrive in soil, decaying wood, and plant debris within forested habitats, leading to diseases such as cryptococcosis, histoplasmosis, blastomycosis, and sporotrichosis. Transmission occurs primarily through inhalation or cutaneous inoculation, with risk amplified by forest disturbances like logging or climate-driven changes. We discuss ecology, epidemiology, clinical manifestations, diagnosis, treatment, and One Health implications, highlighting gaps in surveillance and the need for integrated environmental monitoring. As global warming expands fungal niches, proactive strategies are essential to mitigate these underrecognized threats.

Keywords: Forest fungi, dimorphic pathogens, environmental mycoses, One Health, climate change, cryptococcosis, histoplasmosis

Introduction

Fungi are integral to forest ecosystems, facilitating nutrient cycling through decomposition of organic matter and forming mycorrhizal associations with trees. Yet, this biodiversity includes opportunistic pathogens that can infect humans, particularly when environmental reservoirs are disturbed [1][2]. While most human fungal infections arise from commensal or hospital-acquired sources, endemic mycoses from forest-linked environments represent a growing public health concern. These "disease-causing forest fungi" are often thermally dimorphic—existing as molds at ambient temperatures (e.g., 25°C in forest soils) and converting to pathogenic yeast forms at human body temperature (37°C)—enabling survival in diverse habitats and virulence in mammalian hosts [3].

Key examples include *Cryptococcus gattii*, associated with tree hollows and decaying wood in coastal forests; *Histoplasma capsulatum*, enriched in guano-laden forest soils; *Blastomyces dermatitidis*, in moist wooded soils; and *Sporothrix schenckii*, from sphagnum moss and plant debris. Infections disproportionately affect immunocompetent individuals in endemic areas, with outbreaks linked to activities like forestry, spelunking, or construction. Climate change exacerbates risks by expanding fungal geographic ranges and enhancing thermal tolerance, potentially increasing aerosolization during extreme weather events [4][5]. This review examines the ecology, transmission, clinical impact, and management of these pathogens, emphasizing interdisciplinary approaches under the One Health framework.

Ecology and Environmental Reservoirs

Forest fungi pathogenic to humans are saprophytic, colonizing nutrient-rich substrates like decaying wood, leaf litter, and soils amended with animal excreta. Disturbances—natural (e.g., storms) or anthropogenic (e.g., logging)—disrupt these niches, releasing infectious conidia into the air or onto skin [1][6].

Cryptococcus gattii

C. gattii (part of the *C. gattii* species complex) preferentially inhabits tropical and subtropical trees, including eucalyptus, Douglas fir, alder, red cedar, and Garry oak, in hollows, bark crevices, and surrounding soils [7][8]. Tree harvesting aerosolizes spores, with studies showing positive correlations between harvest volumes (within 7.5–10 km buffers of settlements) and infection incidence ($r = 0.64–0.66$, $p < 0.005$) [1][6]. Environmental isolates from air, water, and soil underscore its resilience, with forestry workers at elevated risk [10].

Histoplasma capsulatum

This dimorph thrives in nitrogen-rich, acidic soils of humid forests, particularly river valleys, augmented by bird (e.g., starling, pigeon) or bat guano [11][12]. Endemic foci span the Ohio/Mississippi valleys, Central/South America, Africa, and Asia, with microconidia (2–5 µm) persisting in contaminated sites for years [13]. Cave exploration or soil excavation in forested caves/roosts triggers outbreaks, as guano fosters mycelial growth at 25°C [14].

Blastomyces dermatitidis

B. dermatitidis occupies acidic, moist soils in wooded, sandy areas near waterways, decomposing wood, and leaf litter in the forest. It forms mycelia producing conidia aerosolized by disturbance, with uneven distribution favoring forested ecotones [17]. Hotspots align with beaver habitats or floodplains, where organic decay supports growth [18].

Sporothrix schenckii

A soil saprophyte, *S. schenckii* (and complex species) colonizes decaying vegetation, sphagnum moss, rose thorns, hay, and plant debris in tropical/subtropical forests worldwide [19][20]. Transmission via traumatic inoculation from plant material ("rose gardener's disease") links it to forest-adjacent activities; conidia (2–6 µm) convert to cigar-shaped yeasts in tissue [21].

Epidemiology and Risk Factors

These mycoses are underreported, with global burdens estimated at millions of infections annually. *C. gattii* incidence on Vancouver Island exceeds 5/100,000, affecting immunocompetent adults (median age 50); 30–50% mortality in meningitis cases [9]. Histoplasmosis infects >500,000 yearly in the country, with 22–44% prevalence in HIV cohorts; outbreaks involve 2–3,000 cases from soil disruption. Blastomycosis rates are 1–40/100,000 in hotspots, with 5–10% dissemination; forestry/hunting increases odds (OR 2–5) [15]. Sporotrichosis affects >15,000 globally, Risk factors include immunosuppression (HIV CD4 <100, transplants), male sex, age >50, and occupational exposure (loggers, spelunkers). Climate change shifts ranges northward, with warmer soils favoring dimorphs [4][5].

Pathogenesis and Clinical Manifestations

Inhalation or inoculation initiates infection: conidia germinate into yeasts, evading phagocytosis via capsule (e.g., *C. gattii*) or broad-based budding (*B. dermatitidis*) [3].

- **Cryptococcosis**: Pulmonary nodules progress to meningitis (headache, fever); 70% CNS involvement [10].
- **Histoplasmosis**: Acute flu-like illness (fever, cough); chronic cavitary in smokers; disseminated (hepatosplenomegaly, 90% mortality untreated) [11].
- **Blastomycosis**: Pneumonia (cough, dyspnea); verrucous skin lesions; bone/joint dissemination (20%) [15].
- **Sporotrichosis**: Lymphocutaneous nodules ("sporotrichoid" chain); rare pulmonary/disseminated in immunocompromised [20].

Diagnosis and Treatment

Diagnosis integrates exposure history, imaging (nodules/cavities), and labs: antigen detection (urine/serum, 80–95% sensitive for dissemination), culture (gold standard, slow), and PCR [13]. Treatment: Amphotericin B induction for severe cases, followed by itraconazole (6–12 months); fluconazole for *C. gattii* maintenance [3]. Resistance is low but rising with azole use [2].

Emerging Threats and Climate Change

Warming temperatures (>30°C tolerance) enable range expansion, as seen with *C. gattii* in temperate forests [4][5]. Forest loss aerosolizes spores, linking deforestation to outbreaks [1][6]. Multidrug resistance (e.g., azole-resistant strains from agro-fungicides) compounds risks [2]. One Health surveillance—integrating environmental sampling and genomics—is crucial [2].

Conclusion

Disease-causing forest fungi bridge ecology and medicine, with disturbances amplifying zoonotic potential. Enhanced monitoring, prophylaxis in at-risk groups, and sustainable forestry can curb threats. Future research should prioritize genomic surveillance and climate modelling to pre-empt expansions.

References

[1] Acheson ES, et al. (2023). Forest Disturbance and Disease… PMC. https://pmc.ncbi.nlm.nih.gov/articles/PMC10355116/
[2] Fisher MC, Gurr SJ, Cuomo CA, Blehert DS, Jin H,Stukenbrock EHStajich JE, Kahmann R, Boone CDenning DW, Gow NAR, Klein BS, Kronstad JW,Sheppard DC, Taylor JW, Wright GD, Heitman J,Casadevall A, Cowen LE.2020.Threats Posed by the Fungal Kingdom to Humans, Wildlife, and Agriculture. mBio11: 10.1128/mbio.00449-20.https://doi.org/10.1128/mbio.00449-20.
[3] Thambugala KM, Daranagama DA, Tennakoon DS, Jayatunga DPW, Hongsanan S, Xie N. Humans vs. Fungi: An Overview of Fungal Pathogens against Humans. Pathogens. 2024; 13(5):426. https://doi.org/10.3390/pathogens13050426
[4] Seidel D, et al. (2024). Impact of climate change… Lancet Microbe.
[5] George ME, et al. (2025). The impact of climate change… PMC.
[6] Acheson ES, et al. (2018). Searching for clues… Med Mycol.
[7] Kidd SE, et al. (2007). Characterization of Environmental Sources… Appl Environ Microbiol.
[8] Poplin V, et al. (2024). Geographical distribution… Lancet Microbe.
[9] Acheson ES, et al. (2018). Deciphering the ecological determinants… Med Mycol.[10] Diaz JH (2020). The Disease Ecology… Wilderness Environ Med.
[11] Gugnani HC, et al. (2023). Infection of bats with Histoplasma… PMC.
[12] Mora-Jaramillo N, et al. (2025). High prevalence of Histoplasma… Front Vet Sci.[13] CDC (2024). Histoplasmosis
[14] Taylor RL (1962). Isolation of Histoplasma… Am J Trop Med Hyg.
[15] Jackson KM, et al. (2021). Blastomyces dermatitidis Environmental Prevalence… PMC.
[16] Klein BS, et al. (1986). Isolation of Blastomyces… PubMed.
[17] Reed KD, et al. (2008). Ecologic Niche Modeling… PLoS One.
[18] Various sources on beaver-associated outbreaks.
[19] CDC (2024). Sporotrichosis Basics.
[20] Barros MB, et al. (2011). Sporothrix schenckii and Sporotrichosis… PMC.
[21] WHO (2023). Sporotrichosis Fact Sheet.
[22] Soni J, Gogia A, Aggarwal A. Histoplasmosis in India: clinical insights from a tertiary care hospital. BMC Infect Dis. 2025 Nov 7;25(1):1524. doi: 10.1186/s12879-025-11862-x. PMID: 41204077; PMCID: PMC12595732.