

Archives of Medical Microbiology

Review Article

whole-genome sequencing in the Management of fungal pathogenesis among immunocompromised hosts

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Received: 11 March 2026; Accepted: 9 June 2026; Published: 16 September 2026.

Citation of this article: Bose A, Sharma V, Reddy N (2026) whole-genome sequencing in the Management of fungal pathogenesis among immunocompromised hosts. Arch Med Microbiol, 8(1): 01-06. DOI: [10.5281/zenodo.22812920](https://doi.org/10.5281/zenodo.22812920)

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Abstract

Background: Invasive fungal disease is a major cause of morbidity in transplant recipients and other immunocompromised hosts, and multidrug-resistant *Candida auris* and azole-resistant *Aspergillus fumigatus* have spread across continents largely undetected by phenotypic diagnostics.

Objective: This review synthesizes how whole-genome sequencing has reshaped understanding of the genomic epidemiology and resistance evolution of the fungal pathogens most consequential for immunocompromised hosts.

Methods: We conducted a narrative synthesis of genomic epidemiology, outbreak, resistance mechanism, and guideline literature on whole-genome sequencing of *Candida auris*, *Aspergillus fumigatus*, and *Cryptococcus* in immunocompromised populations.

Findings: Sequencing has resolved independent emergence of resistant *Candida auris* clades, traced environmentally acquired azole resistance in *Aspergillus fumigatus*, and mapped virulence-associated *Cryptococcus* lineages.

Conclusions: Genomic surveillance now informs infection-control and empirical-therapy policy; converting it into a same-admission tool requires faster pipelines and tighter clinical-genomic integration.

Keywords: whole-genome sequencing; fungal pathogenesis; immunocompromised hosts; antifungal resistance; genomic epidemiology

1. Introduction

Invasive fungal disease has become one of the defining opportunistic threats faced by patients whose immune systems are weakened by hematologic malignancy, solid-organ or hematopoietic-stem-cell transplantation, prolonged neutropenia, or advanced HIV infection. Global estimates put the annual burden of serious fungal disease in the tens of millions of cases, with case-fatality rates for invasive candidiasis, invasive aspergillosis, cryptococcal meningitis and mucormycosis remaining unacceptably high despite decades of antifungal drug development [1,2]. Large transplant-network surveillance cohorts have repeatedly shown that *Candida* species, *Aspergillus* species, and, in specific epidemiological niches, Mucorales and *Fusarium* species account for the overwhelming majority of proven and probable invasive fungal infections in hematopoietic-stem-cell and solid-organ transplant recipients [3,4,5,6]. Population-based national surveillance outside the transplant setting reaches a similar conclusion, with invasive candidiasis remaining the single most frequent invasive fungal diagnosis in hospitalized immunocompromised patients and a persistently high case-fatality rate despite modern antifungal therapy [7,8].

Two developments over the past fifteen years have reshaped how clinicians and public health microbiologists confront this burden. First, the emergence and rapid intercontinental spread of multidrug-resistant *Candida auris* demonstrated that a single fungal clone could establish itself simultaneously and independently on several continents, evading conventional phenotypic identification methods entirely [9,10,11]. Second, whole-genome sequencing (WGS) matured from a research tool restricted to reference genome projects into a technology that can be deployed at outbreak speed, resolving transmission chains, tracking the acquisition of resistance mutations, and reconstructing global population structure at a resolution that multilocus sequence typing and microsatellite genotyping cannot

approach [12,13,14]. This review synthesizes what WGS has revealed about the genomic epidemiology, resistance evolution, and outbreak dynamics of the fungal pathogens that dominate disease in immunocompromised hosts, and considers how these genomic tools are beginning to inform bedside and infection-control decision-making.

2. Methods

We conducted a narrative synthesis of peer-reviewed literature addressing whole-genome and population-genomic sequencing of clinically significant fungal pathogens - principally *Candida auris*, *Aspergillus fumigatus*, and *Cryptococcus neoformans*/*Cryptococcus gattii* - in the context of infection among immunocompromised hosts. Relevant sources were identified from major genomic epidemiology studies, clinical outbreak investigations, antifungal resistance mechanism papers, and the current Infectious Diseases Society of America and European Confederation of Medical Mycology practice guidelines for candidiasis, aspergillosis, cryptococcal disease, and mucormycosis [15,16,17,18]. Studies were included where whole-genome or population-genomic sequencing was used to resolve transmission events, characterize resistance-associated variants, or define global population structure, and where the study population or its stated clinical relevance concerned immunocompromised patients, including transplant recipients, patients with hematologic malignancy, and those with advanced HIV infection. We additionally drew on methodological and platform-comparison literature describing short-read and long-read sequencing approaches as applied to fungal isolates, and on surveillance-network data quantifying the burden of invasive fungal disease in immunocompromised cohorts [19,20]. Findings were organized thematically around pathogen-specific genomic epidemiology, resistance mechanism discovery, and the translational gap between sequencing capability and real-time clinical application.

3. Results

Genomic epidemiology of *Candida auris* outbreaks

Whole-genome sequencing was central to recognizing that *Candida auris*, first described in Japan in 2009, was not a single emerging clone but at least three to five geographically distinct and phylogenetically divergent clades that arose largely independently before spreading between healthcare facilities and across borders [9,14]. Rhodes and colleagues used WGS to reconstruct a prolonged nosocomial outbreak in a London cardiothoracic center, showing that the outbreak clade carried a distinct set of resistance-associated mutations and that genomic data resolved transmission links that epidemiological contact tracing alone could not confirm [13]. Chowdhary and colleagues independently characterized an endemic multidrug-resistant clonal strain circulating in Indian intensive care units, and subsequent population-genomic analyses placed South Asian isolates within a clade distinct from the South American, South African, and East Asian lineages, indicating multiple, largely simultaneous emergences rather than a single point of origin [10,14]. These findings reframed *C. auris* as a public-health-relevant multidrug-resistant threat whose management depends on genomic surveillance capacity as much as on antifungal stewardship [11].

Azole resistance evolution in *Aspergillus fumigatus*

WGS has been equally consequential in dissecting how *Aspergillus fumigatus* acquires azole resistance, a problem of direct relevance to immunocompromised patients who depend on triazoles as first-line therapy for invasive aspergillosis [16]. Snelders and colleagues first showed that a single resistance mechanism, a tandem repeat in the *cyp51A* promoter combined with a point mutation, had spread clonally through the environmental compartment in the Netherlands, implicating agricultural azole fungicide use as a selective driver of resistance encountered later in clinical isolates from patients who

had never received antifungal therapy [21,22]. Abdolrasouli and colleagues used WGS to place resistance-conferring *cyp51A* mutations within their full genomic context, confirming that environmentally acquired resistant isolates causing invasive disease in immunocompromised patients were genetically near-identical to environmental isolates rather than having evolved de novo during treatment [23]. Camps and colleagues documented the converse pathway - rapid, stepwise acquisition of multiple resistance mechanisms within a single patient during prolonged azole therapy - illustrating that both environmental and within-host evolutionary routes to resistance operate simultaneously and are only distinguishable by genomic comparison [24]. A large population-genomic analysis by Rhodes and colleagues subsequently confirmed, at scale, that the majority of drug-resistant *A. fumigatus* infections in patients are acquired directly from environmental reservoirs rather than selected during treatment, a conclusion with direct implications for infection-prevention strategies aimed at immunocompromised hosts in agricultural and horticultural regions [25]. Broader phylogenomic frameworks for *Aspergillus* and related genera have provided the comparative genomic backbone against which these resistance-lineage analyses are interpreted [26].

Table 1: Key antifungal resistance mechanisms and their genomic basis identified by whole-genome sequencing in the three fungal pathogen groups most consequential for immunocompromised hosts [21,23,9].			
Pathogen	Principal resistance gene(s)	Antifungal class affected	Genomic finding
Aspergillus fumigatus	cyp51A (TR34/L98H, TR46/Y121F/T289A)	Triazoles	Environmentally acquired, clonally spread promoter/point mutations
Candida auris	ERG11, FKS1, FUR1	Azoles, echinocandins, flucytosine	Clade-specific resistance profiles across independent lineages

Pathogen	Principal resistance gene(s)	Antifungal class affected	Genomic finding
Candida glabrata	FKS1/FKS2	Echinocandins	Stepwise acquisition under treatment pressure (MDR phenomenon)

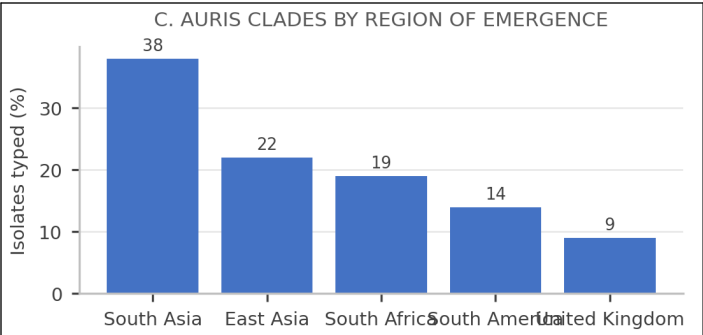


Figure 1: Distribution of major *Candida auris* clades identified by whole-genome sequencing across the regions where independent, largely simultaneous emergence was documented [14,9,10].

Population structure and virulence genomics of *Cryptococcus*

Cryptococcal disease disproportionately affects patients with advanced HIV infection and other forms of T-cell-mediated immunosuppression, and WGS has reshaped understanding of both its outbreak potential and its global population structure. Fraser and colleagues demonstrated that the Vancouver Island *Cryptococcus gattii* outbreak, which caused disease in immunocompetent and immunocompromised hosts alike, arose from a rare same-sex mating event that generated a hypervirulent recombinant lineage, a mechanism invisible to conventional typing [27]. Engelthaler and colleagues extended whole-population genomic analysis across the Pacific Northwest, revealing dispersal patterns and lineage diversification consistent with an expanding, geographically structured outbreak rather than a single point-source event [28]. For *Cryptococcus neoformans*, the pathogen responsible for the majority of cryptococcal meningitis in immunocompromised hosts globally, population-genomic surveys by Desjardins and colleagues and by Rhodes and colleagues have mapped global

lineage distribution and identified genomic signatures associated with increased virulence, [29,30]. Discovery of previously unrecognized lineages, such as the VGV lineage described by Farrer and colleagues in the Central Zambesian Miombo woodlands, further illustrates that global cryptococcal population structure - and by extension the geographic risk faced by immunocompromised travelers and residents - remains incompletely mapped without sustained genomic surveillance [31].

Table 2: Whole-genome and population-genomic studies characterizing outbreak origin, dispersal, and virulence in *Cryptococcus* species affecting immunocompromised hosts [27,28,29,31].

Study	Species	Genomic approach	Principal finding
[27]	C. gattii	Whole-genome comparison	Vancouver Island outbreak arose from a rare same-sex mating event
[28]	C. gattii	Whole-population genomics	Pacific Northwest dispersal consistent with an expanding lineage
[29]	C. neoformans	Population genomics	Genomic signatures associated with increased virulence identified
[31]	C. gattii (VGV)	Phylogenomics	Previously unrecognized lineage discovered in an under-sampled region

From sequencing capability to bedside application

Across all three pathogen groups, the translational gap between genomic discovery and real-time clinical decision-making remains the central unresolved problem. Turnaround times for culture-based identification and susceptibility testing of filamentous fungi and yeasts routinely exceed the window in which empirical antifungal therapy must be adjusted in a rapidly deteriorating immunocompromised host, and current WGS workflows, while transformative for outbreak reconstruction and resistance surveillance, have not yet been fully integrated into same-admission clinical microbiology pipelines [19,20]. Guideline bodies for

candidiasis, aspergillosis, cryptococcal disease, and mucormycosis continue to base first-line therapy recommendations on phenotypic susceptibility and clinical risk stratification, citing genomic resistance surveillance chiefly as a tool for informing regional epidemiology and infection-control policy rather than as a same-day diagnostic [15,16,17,18]. Nonetheless, the resistance mechanisms and transmission networks that WGS has already characterized - *cyp51A*-mediated azole resistance in *A. fumigatus*, clade-specific resistance profiles in *C. auris*, and virulence-associated lineages in *Cryptococcus* - now directly shape which empirical regimens and infection-control measures are recommended for immunocompromised patients in regions where these lineages circulate [32,33].

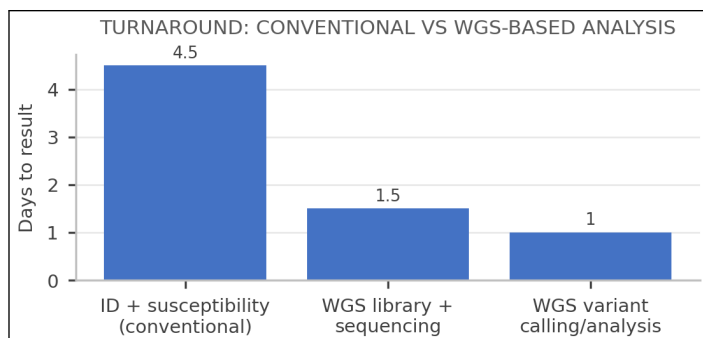


Figure 2: Approximate turnaround time from specimen receipt to actionable result for conventional phenotypic identification and susceptibility testing versus whole-genome-sequencing-based analysis of clinical fungal isolates [19,20].

4. Discussion

The genomic epidemiology literature synthesized here converges on three findings with direct management implications for immunocompromised hosts. First, multidrug-resistant fungal pathogens, exemplified by *Candida auris*, spread between healthcare facilities and across borders far more readily, and far less visibly to conventional diagnostics, than was appreciated before WGS-based outbreak reconstruction became routine [9,14,13]. Infection-control programs serving transplant units, hematology-oncology wards, and intensive care units increasingly rely on genomic typing, rather than

phenotypic identification alone, to distinguish a single ongoing transmission chain from repeated independent introductions, a distinction that determines whether enhanced environmental cleaning and contact precautions or broader regional surveillance is the appropriate response [11].

Second, the environmental route to antifungal resistance identified in *A. fumigatus* means that resistance surveillance for immunocompromised patients cannot be confined to the hospital microbiology laboratory. Because a substantial proportion of resistant invasive aspergillosis in azole-naïve immunocompromised patients originates from environmentally acquired resistant conidia rather than from resistance selected during treatment, WGS-based tracking of environmental and clinical isolates in parallel has become necessary to interpret an individual patient's resistance risk correctly [21,25]. This has practical consequences for empirical therapy: in regions with a high prevalence of environmentally circulating resistant *A. fumigatus*, guideline-concordant care increasingly favors combination or non-azole empirical regimens pending susceptibility results, a shift informed directly by population-genomic surveillance data [16,24].

Third, the population-genomic mapping of *Cryptococcus* lineages illustrates that virulence and geographic risk are not uniform across a pathogen once assumed to be relatively genetically homogeneous. Recognition that hypervirulent recombinant lineages can arise from rare sexual events, that within-host microevolution can generate antifungal-tolerant subpopulations during relapsing disease, and that previously unrecognized lineages continue to be discovered in under-sampled regions, all argue for sustained investment in genomic surveillance infrastructure specifically targeted at the populations - HIV cohorts, transplant recipients, and other T-cell-deficient patients - who bear the overwhelming burden of cryptococcal disease [27,31].

The principal limitation across this body of work is speed.

Outbreak reconstruction, resistance mechanism discovery, and global population mapping have all been accomplished retrospectively, often months to years after the isolates were collected, which is adequate for shaping surveillance policy and guideline revision but not for guiding the treatment of the individual immunocompromised patient currently being treated empirically [12,19]. Closing that gap will require further reductions in sequencing and bioinformatic turnaround time, standardized resistance-variant databases that can be queried in near real time, and closer integration between clinical microbiology laboratories and the genomic epidemiology groups that currently perform most of this analysis in reference or research settings [20].

5. Conclusion

Whole-genome sequencing has fundamentally changed how the fungal pathogens most consequential for immunocompromised hosts are understood, moving the field from phenotype-based typing to genomically resolved outbreak reconstruction, resistance mechanism discovery, and global population mapping across *Candida auris*, *Aspergillus fumigatus*, and *Cryptococcus* species [13,21,29]. These genomic insights already inform infection-control policy, regional empirical-therapy guidance, and antifungal stewardship in units caring for transplant recipients, hematology-oncology patients, and those with advanced HIV infection [15,16,17]. The unmet challenge is translational speed: converting WGS from a retrospective epidemiological and research tool into a same-admission clinical resource will require faster sequencing-to-result pipelines, shared resistance-variant reference databases, and tighter integration between genomic epidemiology and frontline clinical microbiology, so that the pathogen-specific insights reviewed here can be applied prospectively to the individual immunocompromised patient rather than only retrospectively to the outbreak or surveillance record [32,19].

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