Candidozyma auris (*Candida auris*) as a harbinger of novel fungal pathogens

Carla E. Schuh[1] Chaitanya S. Gokhale[1,2]

[1] *Chair for Computational and Theoretical Biology, University of Würzburg, Klara-Oppenheimer Weg 32, 97074, Würzburg, Germany*

[2] *Joint Institute for Individualisation in a Changing Environment (JICE), University of Münster and Bielefeld University, Germany*

Correspondence: carla.schuh@stud-mail.uni-wuerzburg.de, chaitanya.gokhale@uni-wuerzburg.de

Abstract

Mammals are protected from fungal pathogens by two evolutionary pillars: adaptive immunity and elevated body temperature. Anthropogenic climate change is eroding this thermal barrier, while a concurrent decline in human basal body temperature accelerates convergence from both directions. The emergence of *Candidozyma auris* (*Candida auris*), a thermotolerant, multidrug-resistant pathogen that arose independently as genetically distinct clades on multiple continents, may exemplify the consequences. Unlike its closest environmental relatives, which grow poorly at mammalian temperatures, *C. auris* thrives at 37 °C, tolerates up to 47 °C, persists on hospital surfaces, resists all three major antifungal classes, and causes invasive infections with 30–60% mortality. Its near-simultaneous, independent emergence implicates shared selective pressures, potentially rooted in global environmental change, rather than clonal spread. Here, we review the evolutionary biology, ecology, and clinical significance of *C. auris* within a framework of climate-driven pathogen emergence, evaluating current hypotheses for its origin and the molecular mechanisms underlying its thermotolerance and virulence. *C. auris* may represent only the first of many fungal species adapting to breach mammalian thermal defences, making predictive frameworks for anticipating fungal disease emergence an urgent priority.

Keywords: mycology, fungal diseases, pathogen evolution, climate change

1. Introduction

For most of mammalian evolutionary history, fungi have posed a negligible threat to mammals. While devastating to plants and ectothermic animals, driving worldwide amphibian declines and agricultural catastrophes, fungal pathogens have been largely unable to breach the defences of warm-blooded hosts (Rohr and Cohen, 2020). With a core temperature of approximately 37°C against a global mean temperature of roughly 15°C, mammals maintain a thermal gradient that only a fraction of the estimated 3–5 million fungal species can overcome (Casadevall et al., 2019; Maheshwari et al., 2000). A highly efficient adaptive immune system serves as a second line of defence. With these "twin pillars of protection" intact, mammals remain remarkably resistant to fungal disease (Casadevall, 2022). Of the approximately 148,000 described fungal species, merely 300–400 (0.3–0.4%) are currently capable of infecting humans, and most of these are opportunistic pathogens that threaten primarily immunocompromised individuals (Taylor et al., 2001; Thambugala et al., 2024).

This thermal barrier, however, is eroding. With a current gap between global mean temperature (15.08 ± 0.5 °C)(Nullis, 2026) and average human body temperature (37 °C) of approximately 22 °C, every degree Celsius rise in global mean temperature reduces the protective thermal gradient by approximately 5% (Garcia-Solache and Casadevall, 2010). In environmental fungi, meaning fungi that grow freely in the environment and not

inside a host organism, climate change is thought to be selecting for thermotolerance (Garcia-Solache and Casadevall, 2010). This trend is already evident among basidiomycetes (Robert et al., 2015). Compounding this pressure is a measurable decline in human basal body temperature over the past century (Protsiv et al., 2020). The convergence of warming environments and cooling hosts accelerates the collapse of thermal protection that has likely shielded mammals for millennia.

Consequences of this erosion may already be manifesting. In 2009, *Candida auris* was first identified in Japan (Satoh et al., 2009); a retrospective analysis identified the oldest sample, from 1996, in South Korea. Within a decade of its discovery, this thermotolerant pathogen emerged on five continents as genetically distinct clades, each independently acquiring multidrug resistance and causing persistent hospital outbreaks with mortality rates of 30–60% (Chakrabarti and Sood, 2021; Chybowska et al., 2020). Unlike its closest relatives, some of which grow only slowly at 37°C, *C. auris* displays exceptional thermotolerance (Casadevall et al., 2019; Heaney et al., 2020). The simultaneous and independent emergence of multiple thermotolerant, drug-resistant clades across disparate continents is unprecedented in medical mycology and points to a common selective pressure which could be rooted in global environmental change.

C. auris may represent the vanguard of a new era in infectious disease. Already, at least 6.5 million people suffer from invasive fungal infections each year, with 2.5 million deaths directly attributable, and these figures are almost certainly underestimates given uneven global surveillance and diagnostics (Denning, 2024). If climate change enables even a small fraction of environmental fungi to breach the thermal barrier, the consequences could be far larger—a scenario we quantify in *The Scale of the Problem*.

As the taxonomy of the polyphyletic *Candida* genus remains largely unresolved and many of its members are only distantly related, the correct classification is a subject of ongoing debate and research, further complicating the identification and documentation of *C. auris* cases (Francisco et al., 2025). Recent reclassifications of several members into the genus *Candidozyma* (Cendejas-Bueno et al., 2012; Francisco et al., 2025; Liu et al., 2024), including *Candida auris* (now *Candidozyma auris*), aim to better reflect their distant relation to *Candida spp* like *Candida albicans* and *Candida parapsilosis*(Liu et al., 2024). For continuity of nomenclature, *Candida auris* will be used throughout this review. It should be noted that our current knowledge about fungal evolution and phylogeny is severely limited (Rokas, 2026), mainly due to a lack of fungal fossil records and the broad diversity within the kingdom regarding morphology, lifestyles, genomic plasticity and more (Rokas, 2026).

This review examines *C. auris* as a case study for understanding how climate change may fundamentally alter the landscape of human fungal disease. We explore the underlying mechanisms of its rise, from molecular virulence factors to ecological hypotheses for its multi-continental emergence, and consider what it reveals about our vulnerability to future fungal threats. Unlike bacterial and viral pathogens, fungi have remained largely relegated to tropical medicine and immunocompromised populations. *C. auris* challenges this complacency, demonstrating that environmental fungi can rapidly evolve to cause severe disease while simultaneously acquiring resistance to our limited antifungal arsenal. Understanding the interwoven evolutionary and ecological forces that shaped *C. auris*, as visually summarized in **Figure** 1, is therefore essential preparation for a future in which the mammalian thermal barrier continues to erode.

2. The *C. auris* Case Study: Emergence, Spread, and Characteristics

2.1. Discovery and Delayed Recognition

In 2009 in Japan, Satoh et al. isolated an unusual yeast from a patient's ear canal and designated it *Candida auris* (Satoh et al., 2009). Retroactive analysis of archived *Candida* isolates later revealed that the organism had been circulating undetected in South Korea since at least 1996 (Lee et al., 2011), indicating years of cryptic evolution and dispersal before medical recognition. This delayed identification reflects a broader problem in clinical mycology. Phenotypic methods are slow and unreliable for distinguishing closely related species, and *C. auris* was absent from diagnostic databases for years after its description (Mizusawa et al., 2017). Many isolates were misidentified as *C. haemulonii* or related species, leading to systematic underestimation of its prevalence (Chakrabarti and Sood, 2021; Chatterjee et al., 2015; Govender et al., 2018). The problem is compounded

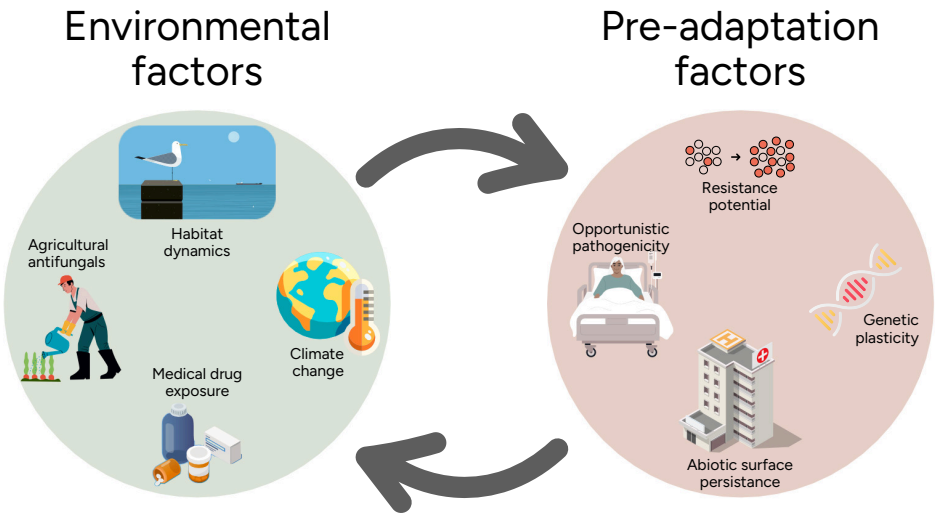

Figure 1. A visual summary of the hypothesised environmental influences that made *C. auris* into the pathogen we observe today. Factors in its natural environment (competition in a dynamic habitat, climate change, drug exposure in the medical context and agricultural antifungals, green circle) shaped a species with pre-adaptations (resistance potential to antifungals, genomic plasticity, ability to persist in hospitals, and being an opportunistic pathogenicity, brown circle). These factors influence one another, creating a feedback loop that produces a highly competent fungal pathogen with unprecedented characteristics.

in regions lacking diagnostic infrastructure: in sub-Saharan Africa, South America, and Eastern Europe, *C. auris* is almost certainly under-reported (Chybowska et al., 2020; Okoye et al., 2022; Ong et al., 2019). Where adequate surveillance does exist, the picture is striking. In Kenya, *C. auris* accounts for 38% of candidemia cases (Chybowska et al., 2020), suggesting that the true global burden is substantially higher than current estimates.

By 2016, medical institutions worldwide had been formally alerted to *C. auris* as an emerging pathogen. By 2019, it had been recovered from over 4,000 blood isolates across 40 countries (Chow et al., 2020; Chybowska et al., 2020), and from 2017 to 2023 reported case numbers increased 2- to 5-fold (Bing et al., 2024; De Gaetano et al., 2024; Lockhart et al., 2017; Lyman et al., 2023).

2.2. Multi-Continental Emergence and Clade Diversity

What makes *C. auris* unprecedented in medical mycology, is the pattern of its emergence: between 2009 and 2016, four major clades appeared independently in distinct geographic regions (see **Figure** 2), each exhibiting unique genetic signatures and resistance profiles (Chybowska et al., 2020; Lockhart et al., 2017). Since their initial discovery, two additional clades have been reported (Chow et al., 2019; Suphavilai et al., 2024). Despite sharing high sequence identity, these clades display 17-fold greater genetic diversity between them than within, confirming independent evolutionary trajectories rather than dispersal from a single source (Chow et al., 2020; Chybowska et al., 2020). Molecular clock estimates place the emergence of individual clades in the late 1990s or early 2000s, while their most recent common ancestor predates industrial-era warming (Chow et al., 2020). This parallel emergence of thermotolerant, drug-resistant lineages across disparate continents is the strongest argument for shared selective pressures consistent with global environmental change.

A critical transition appears to have occurred between 2013 and 2015, (see Fig. 2). Before this time period, *C. auris* cases were detected sporadically; afterward, epidemiological dynamics shifted. Isolated infections became prolonged nosocomial (hospital) outbreaks across an increasing number of countries (Chakrabarti and Sood, 2021). It is unclear whether this inflection reflects genuine changes in transmission and epidemiological dynamics or simply appears this way because of increased awareness and sampling efforts driving up the absolute number of detections. It is plausible that a certain threshold was crossed, allowing *C. auris* to spread widely, leading to more detections. It is also possible that *C. auris* was already present in more hospitals than previously assumed and only discovered after the WHO alert prompted researchers and medical professionals

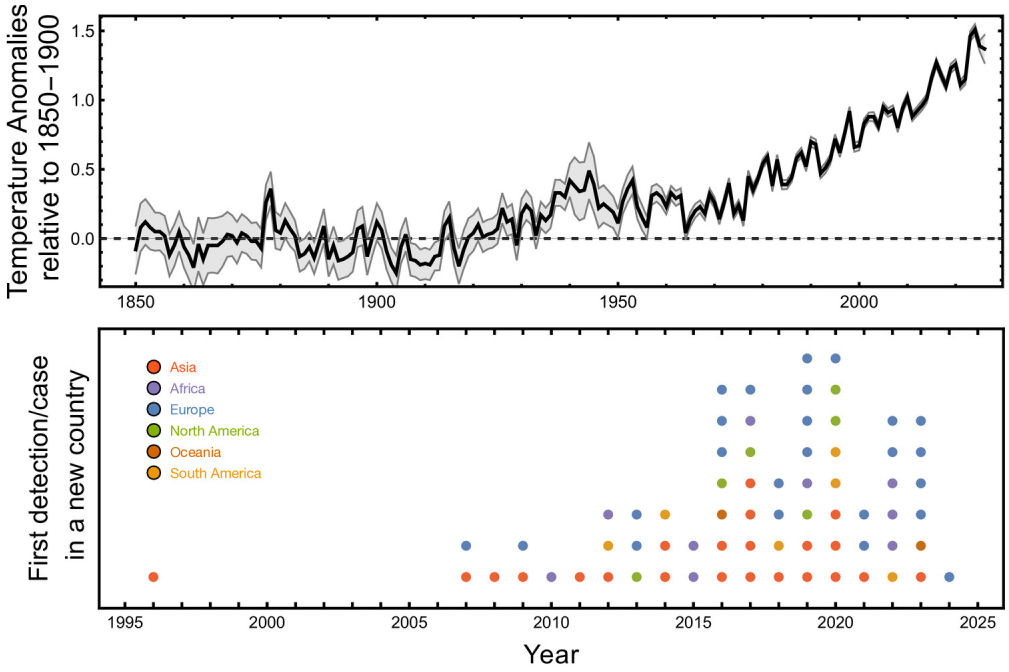

Figure 2. Plots of the global temperature anomalies and the first reported cases in countries classified per continent over time. *The top panel* depicts mean temperature anomalies, in an uncertainty band, relative to the reference period 1850-1900 (Morice et al., 2021). As per the Intergovernmental Panel on Climate Change (IPCC) standard Lai and Dzombak (2019), temperature anomalies are plotted rather than mean temperature, as the margin of uncertainty is lower for these datasets. Dataset anomalies are calculated relative to a 1981 to 2010 baseline and offset by 0.69 degrees Celsius, which is the best estimate of the difference for that period from the 1850-1900 average, as given in the IPCC Sixth Assessment Report. Temperature anomalies reflect the difference between current temperatures relative to an averaged historical baseline. *In the bottom panel*, each dot represents the earliest known sample of *C. auris* in a new country, either in the form of retroactively identified, historic isolates or new medical cases. They are sorted by the year the sample was taken, not by when it was discovered. The dots are colour-coded by continent. Multiple dots of the same colour in a year indicate detections in different countries within the same continent.

to actively look for it.

Molecular epidemiology reveals clusters of highly related isolates within individual facilities, indicating sustained local transmission, yet multiple countries harbour isolates from different clades, demonstrating repeated independent introductions (Chow et al., 2020). Tracking transmission routes is complicated by incomplete travel data: in the United States, only 7% of cases had *documented* healthcare exposure abroad, despite isolates spanning multiple continental clades (Chow et al., 2020). This circumstance most plausibly reflects unrecognised colonisation among transferred patients and healthcare workers, though cryptic environmental reservoirs cannot be excluded. Furthermore, the six clades show distinct profiles of antifungal resistance, virulence, and phenotype; we direct readers to detailed reviews of this complex topic (Chow et al., 2020; Chybowska et al., 2020; Santana et al., 2024). Fungal pathogens generally possess the capacity for long-distance dispersal through human travel and trade networks (Fisher et al., 2001; Simwami et al., 2011; Stukenbrock and McDonald, 2008), and *C. auris* seems to have fully exploited these anthropogenic pathways.

2.3. Clinical Significance

C. auris epidemiology has transformed from sporadic invasive infections to persistent nosocomial outbreaks, driven by its remarkable capacity for environmental persistence, whose mechanistic basis we detail in *Mechanistic Understanding* (Gómez-Gaviria et al., 2023; Zheng et al., 2025). The exact transmission patterns remain unclear, as relative contributions of environmental contamination and direct patient-to-patient spread vary between outbreaks (Chybowska et al., 2020), and transmission involves both symptomatic patients and asymptomatic carriers, including healthcare workers (Chybowska et al., 2020; Escandón et al., 2019). Whether *C. auris* can enter

a viable-but-non-culturable state, as demonstrated for other fungal pathogens, remains unclear (Chybowska et al., 2020).

Mortality rates for *C. auris* bloodstream infections range from 20% to 67%, with most countries reporting approximately 52% (Chowdhary et al., 2013; Gómez-Gaviria et al., 2023). In one European outbreak of 140 cases, 41% died within one month (Ruiz-Gaitán et al., 2018), reflecting both intrinsic virulence and the limited options available when standard antifungals fail. While *C. albicans* remains the most frequently isolated species in candidemia cases, *C. auris* and the broader *C. haemulonii* complex have emerged as significant opportunistic pathogens (Gómez-Gaviria et al., 2023), threatening immunocompromised individuals in particular (Gómez-Gaviria et al., 2023), but not exclusively—cases in immunocompetent patients are well documented (Das et al., 2018; Krishnasamy et al., 2021; Mohsin et al., 2020).

Risk factors mirror those for other invasive candida infections: immunosuppression (whether medically induced or arising from conditions such as cancer, diabetes, or AIDS), indwelling catheters, recent antifungal or antibiotic exposure, and prolonged hospitalisation or ICU stays (Du et al., 2020; Munshi et al., 2024; Wasylyshyn and Stoneman, 2024). Early presentations are often non-specific, and blood cultures frequently remain negative initially, delaying diagnosis and reducing survival probability (Ruiz-Gaitán et al., 2018). Survival rates increase dramatically when intervention occurs within 12 hours, yet diagnostic challenges often prevent such rapid action (Morrell et al., 2005).

Compounding the diagnostic problem is a constrained therapeutic landscape. As eukaryotes, fungi share substantial cellular machinery with their mammalian hosts, severely limiting the number of targets that can be exploited without unacceptable toxicity (Brown et al., 2017). The clinical antifungal arsenal reduces to three major drug classes—azoles, echinocandins, and polyenes—and *C. auris* has demonstrated that a single species can acquire resistance to all three (Ademe and Girma, 2020).

Current global guidelines for the treatment and prevention of candidiasis (Cornely et al., 2025) recommend azoles, specifically fluconazole, or echinocandin prophylaxis for patients in certain risk groups, such as patients undergoing abdominal surgery or chemotherapy. Should candidemia occur, the first-line treatment is echinocandins, followed by azoles. Yet because *C. auris* is already broadly resistant to azoles and echinocandins, with pan-resistant strains and clade-variable profiles documented (detailed in *Multidrug Resistance as Cross-Protection from Environmental Stress*), selecting an effective drug becomes increasingly problematic. This can severely impact patient outcomes as early intervention is crucial.

3. Mechanistic Understanding of the Success of *C. auris*

The near-simultaneous, multi-continental emergence of *C. auris* poses a fundamental question (Fig. 2): how did this organism successfully breach mammalian defences and spread globally when millions of other fungal species have not? The answer lies not in a single evolutionary innovation but in the convergence of three interconnected capabilities. Thermotolerance permits growth at mammalian body temperature. Its multidrug resistance defeats our limited antifungal arsenal and allows *C. auris* to thrive as a nosocomial pathogen, leading to high mortality rates and global spread within roughly one decade. Combined with its persistence mechanisms prolonging the survival outside the host, *C. auris* has the ability to drive outbreaks in medical facilities. Critically, these traits arise from conserved fungal stress response systems that may be latent in many environmental species, awaiting the appropriate selective pressures to be activated.

3.1. Thermotolerance Foundation

Perhaps the most striking characteristic of *C. auris*, and the one most directly relevant to the climate-emergence hypothesis, is its exceptional thermotolerance. While its closest phylogenetic relatives grow poorly at mammalian body temperature, *C. auris* thrives at 37°C, with some isolates surviving up to 47°C (Casadevall et al., 2019; Heaney et al., 2020). This thermal capacity exceeds that of most environmental fungi and surpasses many established human pathogens.

Understanding how *C. auris* acquired this thermotolerance is central not only to explaining this particular

pathogen's emergence but also to anticipating which other fungal species might follow similar evolutionary trajectories. If thermotolerance can evolve rapidly in environmental fungi, as experimental studies suggest (De Crecy et al., 2009), then a warming climate may be selecting for this trait across numerous lineages simultaneously—potentially generating multiple future pathogens in parallel.

The thermotolerance of *C. auris* derives not from novel molecular innovations but from stress-response pathways widespread across fungi (Casadevall et al., 2019). Current evidence gives no indication that these pathways function fundamentally differently in *C. auris* than in other fungi. While further research is needed on this topic, current findings suggest that the exact molecular mechanisms of HSP90 across species vary, but result in enhanced stress tolerance regardless (Fang et al., 2025). The capacity to endure thermal stress is latent in many lineages, and its activation under climate-driven selection is anticipatable rather than idiosyncratic. Central among these is Heat Shock Protein 90 (HSP90), an essential molecular chaperone that stabilises proteins involved in stress responses and is upregulated under thermal stress across diverse fungal taxa (Biebl and Buchner, 2019; Kim et al., 2019; Schopf et al., 2017). In *C. auris*, HSP90 depletion triggers morphological changes and compromises drug tolerance, while its overexpression may contribute to both thermotolerance and antifungal resistance (Gómez-Gaviria et al., 2023; Kim et al., 2019). Some lineages developed HSP90-independent drug resistance (Kim et al., 2019) through fixed mutations, suggesting an evolutionary trajectory consistent with a model in which HSP90 initially buffers (thermal) stress long enough to allow accumulation of adaptive genetic changes that eventually render the chaperone dispensable (Cowen and Lindquist, 2005). The exact mechanism remains unclear, but mutations affecting ERG11, specifically the Y132F variation, CDR1, TAC1, FUR1, FCY2 and FKS1 are thought to play a role in the emergence of HSP90-independent drug resistance (Chowdhary et al., 2018; Cowen and Lindquist, 2005; Hill et al., 2015; Kim et al., 2019; Opassathian et al., 2025; Phan-Canh et al., 2025b; Wang et al., 2025; White, 1997). Current research suggests that epimutations and epigenetic pathways confer drug tolerance in diverse fungi (Ma et al., 2026; Son et al., 2026; Torres-Garcia et al., 2020; Yaseen et al., 2022) and it is possible that similar mechanisms shape the seemingly strain-specific resistance profiles of *C. auris* (Xiao et al., 2025), which remains to be investigated further.

Additional stress protection is provided by Hog1, a stress-activated protein kinase with pleiotropic roles in osmotic and thermal stress responses. Deletion of Hog1 reduces virulence and alters cell morphology, underscoring its importance for pathogenesis (Day et al., 2018; Shivarathri et al., 2020), with strain-specific results (Shivarathri et al., 2020). That HSP90 and Hog1 are evolutionarily conserved across fungal taxa is a key point: the molecular machinery for thermotolerance exists widely and does not need to evolve *de novo*.

If the machinery is shared, why did *C. auris* emerge as a pathogen when its relatives did not? The distinction lies in the genomic and phenotypic context in which the conserved pathways operate. *C. auris* carries lineage-specific features that a stress-response account alone does not capture: copy-number variation and expanded families of drug transporters and lipases (Muñoz et al., 2018), clade-specific chromosomal rearrangements and a variable subtelomeric repertoire of cell-surface adhesins (Muñoz et al., 2021), clade-specific amplification of the azole target *ERG11* (Chow et al., 2020), and reversible, high-frequency phenotypic switching that generates standing variation in virulence traits (Phan-Canh et al., 2025a). These are not conserved stress machinery. They are contingent products of this lineage's genome architecture and plasticity. We therefore separate two claims that are easily conflated. The conserved machinery explains why the thermal barrier is *crossable*, and why its eventual crossing is predictable across fungi. The lineage-specific context explains why *C. auris* in particular crossed it and then succeeded as a nosocomial pathogen. While our first claim is surveyable, the second is contingent.

3.2. Multidrug Resistance as Cross-Protection from Environmental Stress

C. auris exhibits resistance to all three major antifungal classes: up to 90% of isolates are resistant to azoles, 30% to amphotericin B, and 2–8% to echinocandins (Ademe and Girma, 2020; Chowdhary et al., 2018; Lockhart et al., 2017), with pan-resistant isolates documented (Ademe and Girma, 2020). Resistance is facilitated through mutations in drug targets, altered ergosterol biosynthesis, amplification of efflux pumps and, as recently discovered, the conserved carbonic anyhdrase gene NCE103 and pyruvate regulating genes (Ahmed et al., 2025;

Chowdhary et al., 2014, 2023; Kim et al., 2019; Opassathian et al., 2025; Phan-Canh et al., 2026; Xiao et al., 2025), with profiles varying between clades and isolates (Abe et al., 2025; Ahmed et al., 2025). What makes this resistance particularly alarming is the speed at which it evolves: experimental populations developed high-level echinocandin resistance within three days (Carolus et al., 2021), raising the concern that novel antifungals may provide only a temporary reprieve.

A more fundamental insight, however, concerns the origin of this resistance. *C. auris* demonstrates tolerance to reactive oxygen species, high salinity, and cell wall stress, a profile consistent with survival in extreme environmental niches (Casadevall et al., 2019; Day et al., 2018). Fungal stress responses are often cross-protective: genes upregulated in response to one stressor commonly confer resistance to seemingly unrelated challenges (Brown et al., 2017). Selection for environmental stress tolerance may thus inadvertently co-select for drug resistance, explaining how *C. auris* arrived in clinical settings already equipped with multidrug resistance despite minimal prior exposure to antifungal agents.

This cross-protection hypothesis is reinforced by the elevated ergosterol and structural lipid content of *C. auris*, which simultaneously enhances membrane stability under environmental stress and reduces antifungal efficacy (Chybowska et al., 2020). The same HSP90 system further couples the two, stabilising stress-response proteins and enabling the rapid phenotypic plasticity that links thermal and drug tolerance (Kim et al., 2019). In other words, the mechanisms are not independent but facets of an integrated stress response architecture.

3.3. Persistence: The Hospital Reservoir

The third component of *C. auris*' success is its capacity for prolonged environmental persistence, which, despite adherence to standardized hygiene protocols, enabled it to claim hospitals as a novel reservoir (Chybowska et al., 2020; Gómez-Gaviria et al., 2023). This persistence stems from two interconnected strategies: biofilm formation and specialised survival structures.

Biofilms are the predominant mode of microbial life in natural environments (Lagree and Mitchell, 2017). In general, in the initial colonization phase, viable fungal cells or spores need to reach a suitable surface and attach to it. This is followed by the proliferation phase, during which the fungus induces rapid replication and expansion, enabling the formation of microcolonies. As the microcolonies mature, they form a biofilm with an increasingly complex 3D structure. During the subsequent dispersion phase, often induced by unfavourable conditions, the fungus disperses into the environment to ensure its survival. The exact mechanisms of biofilm formation vary widely among species and remain understudied at the time of writing this review. We refer readers to an insightful review for a detailed overview (Ramage et al., 2025). In *C. auris*, it is known that biofilms composed of blastoconidia and pseudohyphae are capable of reducing drug susceptibility through physical barriers and cellular aggregation (Gómez-Gaviria et al., 2023). Its ability to adhere to surfaces is conferred by adhesins Als4112, Scf1 (Santana et al., 2023; Wang et al., 2024) which are thought to enable aggregation as well, though the latter has only been observed in certain strains (Brown et al., 2020). Aggregation is associated with enhanced biofilm formation capabilities (Brown et al., 2020; Short et al., 2019; Vila et al., 2020; Willaert et al., 2021). Most concerning is *C. auris* ability to colonize surfaces in its environment just 4 hours after initial contamination (Sansom et al., 2024).

The second persistence strategy utilized by *C. auris* is a specialized survival structure. Under nutrient-poor conditions, *C. auris* has been observed to form giant lipid droplets (gLDs) under nutrient-poor conditions(Zheng et al., 2025). These gLDs enhance resistance to environmental stresses, elevated temperatures and antifungals, directly connecting nutrient storage, stress tolerance, and thermotolerance within a single cellular mechanism (Zheng et al., 2025).

Together, these persistence strategies point to *C.auris* being optimized for nutrient-poor, chronically stressful environments. Environmental fungi experience low-to-moderate but constant stress, and their survival systems have evolved accordingly (Brown et al., 2017). Laboratory conditions typically involve glucose-rich growth media and acute, high-dose stress exposures. What appears as virulence or drug resistance in clinical settings may therefore represent pre-existing adaptations for surviving harsh environmental niches.

3.4. Pre-adaptations Rather Than Novel Evolution

The mechanistic picture that emerges is not one of recent dramatic evolutionary innovation but rather the activation of a pre-existing adaptive toolkit—conserved stress-response pathways, robust membrane architecture, efficient efflux, and effective persistence strategies—that likely evolved for survival in variable, stressful environments rather than for human pathogenesis.

Climate change may not have created a pathogen *de novo* but rather shifted environmental conditions in a way that fungi with certain pre-existing traits suddenly found mammalian body temperature within their thermal tolerance range - a prime example of ecological opportunity. Through the same cross-protection, selection for environmental thermotolerance co-selects for drug resistance and persistence traits that surface as virulence in a clinical context—evolutionary baggage from environmental adaptation that coincidentally facilitates pathogenesis.

The most troubling concern is that these mechanisms are not unique to *C. auris*. Robert et al. proposed that thermotolerance may have been a common trait among ancestral fungi, subsequently lost in many lineages following a global temperature decline of approximately 5°C some 34 million years ago (Liu et al., 2009; Robert and Casadevall, 2009). If this is correct, the genetic architecture of thermotolerance may be more broadly conserved than currently appreciated and could re-emerge under sustained warming. HSP90 and stress-activated protein kinase pathways are conserved across fungal taxa; efflux pumps, biofilm formation, and lipid droplet accumulation represent widespread survival strategies. The natural capacity of fungi to degrade complex organic substrates has equipped them with diverse enzymatic arsenals that can contribute to virulence in a host context (Park et al., 2013). If *C. auris* succeeded by activating conserved systems rather than evolving novel ones, then many other environmental fungi possess similar latent capabilities. Identifying the selective pressures that activated these capabilities in *C. auris*, therefore, becomes critical for predicting which species might follow similar trajectories.

4. Evolutionary and Ecological Context

The previous section established that *C. auris* succeeded through conserved fungal mechanisms rather than novel evolutionary innovations. If the molecular toolkit for thermotolerance and stress resistance is widespread across fungi, why did *C. auris* emerge when and where it did? What does its emergence pattern reveal about selective pressures currently acting on environmental fungi worldwide?

4.1. The Missing Reservoir

Despite intense investigation, the environmental reservoir of *C. auris* remains unknown. Its closest phylogenetic relatives inhabit remarkably disparate ecological niches: some colonise flowers, others occur in human microbiomes, and still others reside in the gastrointestinal tracts of marine animals (Chybowska et al., 2020). Phylogenetic analysis confirms broad environmental flexibility, and the interspersion of environmental and clinical isolates among close relatives suggests previous transitions between free-living and host-associated states (Schutz et al., 2024). However, the substantial genetic distance separating *C. auris* from these relatives, many of which are incapable of growth at elevated temperatures (Casadevall et al., 2019), limits the utility of phylogenetic inference for reconstructing its ancestral ecology.

The lack of population genetic data from ancestral populations before the emergence of multidrug resistance further constrains our understanding of the evolutionary trajectory (Chybowska et al., 2020). Comparative genomics reveals changes in genes associated with drug resistance and virulence, suggestive of positive selection, but these analyses remain inconclusive without knowledge of ancestral population parameters (Chybowska et al., 2020). Discovery and sequencing of more closely related species could illuminate *C. auris*' evolutionary history, but such species remain elusive.

Physiology offers partial clues. *C. auris* tolerates high salinity (10% NaCl), suggesting adaptation to hypertonic environments (Casadevall et al., 2019; Day et al., 2018). Its obligate aerobic metabolism explains skin but not gut colonisation, implying a recent environmental origin rather than long-standing host association (Chybowska

et al., 2020). Taken together, the physiological profile points toward an environmental saprophyte adapted to oxidative, nutrient-limited conditions (Brown et al., 2017; Chybowska et al., 2020). Casadevall et al. proposed wetlands and marine environments as candidate ancestral habitats, where brackish conditions select for salt tolerance and sun-exposed shallow waters impose thermal stress, noting geographic overlap between *C. auris* emergence and wetland distributions (Casadevall et al., 2019). However, direct environmental isolations remain rare, and human introduction cannot be ruled out in cases where environmental samples have tested positive (Arora et al., 2021; White et al., 2024; Yadav et al., 2022, 2023).

4.2. Hypotheses for Pathogenic Emergence

Multiple non-exclusive hypotheses have been advanced to explain the transformation of an environmental saprophyte into a global pathogen, and we refer readers to detailed treatments of these elsewhere (Casadevall et al., 2019, 2021; Chakrabarti and Sood, 2021; Garcia-Bustos et al., 2023; Jackson et al., 2019). Despite their diversity, these hypotheses converge on a common theme: anthropogenic environmental change created conditions favouring thermotolerant, stress-resistant fungi. They differ principally in which selective pressures they foreground. The climate-driven selection hypothesis provides the most parsimonious explanation for the defining puzzle of *C. auris*: its simultaneous, independent emergence on multiple continents. Rising temperatures can directly select for thermotolerance in fungi, as demonstrated experimentally (De Crecy et al., 2009). If the most recent common ancestor (MRCA) of modern *C. auris* clades existed within the last 339 years, and individual clades emerged approximately a decade before detection; their origins coincide with accelerating anthropogenic warming (Chow et al., 2020). If *C. auris* possessed a latent capability to colonise mammals; progressively rising environmental temperatures would have favoured thermotolerant genotypes, eventually enabling the breach of the 37°C threshold. Because this selective pressure operates globally, it would act on geographically isolated populations in parallel, explaining the independent emergence of distinct clades across continents.

Several complementary mechanisms likely contributed. Agricultural azole fungicides may have pre-selected for drug resistance in environmental populations before clinical emergence (Fisher et al., 2018), though this alone does not account for resistance to non-azole antifungals, and azole use began decades before *C. auris* appeared without producing comparable emergence in other species (Casadevall et al., 2019). More plausibly, agricultural antifungals amplified pre-existing resistance mechanisms that were initially selected through cross-protection conferred by overlapping environmental stress responses. Anthropogenic habitat disruption from wetland drainage, mining, and land-use change may have increased human contact with ancestral *C. auris* populations while simultaneously fragmenting habitats in ways that created population bottlenecks, potentially visible in the low within-clade genetic diversity observed today.

More speculative proposals include avian reservoirs and horizontal gene transfer. The thermotolerance of *C. auris* (up to 47°C (Heaney et al., 2020)) is compatible with avian body temperatures (40 - 44°C), and migratory birds could in principle explain multi-continental presence (Casadevall et al., 2019). However, direct evidence of *C. auris* in wild bird populations is absent, and the genetic distinctiveness of continental clades argues against recent dispersal through a shared avian vector. Horizontal gene transfer from established pathogens such as *C. albicans* cannot be excluded (Casadevall et al., 2019; Fraser et al., 2005; Richards et al., 2011; Stukenbrock and McDonald, 2008), but virulence is typically a complex, polygenic trait that resists simple horizontal acquisition (Casadevall et al., 2019); genetic exchange might have fine-tuned existing capabilities rather than created them *de novo*.

4.3. Synthesis: A Convergent Eco-evolutionary Response

The evidence points to *C. auris* having emerged through the combined action of multiple mechanisms: climate-driven selection for thermotolerance, activating pre-existing stress response pathways; agricultural and environmental contamination pre-selecting for drug resistance through cross-protection; habitat disruption increasing human-fungal contact; and healthcare transmission dynamics amplifying adapted lineages (summarized in *Figure* 1). The species did not evolve novel pathogenic machinery, but it became compatible with mammalian hosts once environmental conditions shifted sufficiently.

The four major clades likely represent parallel evolutionary responses to similar selective pressures across different geographic regions. Their genetic distinctiveness argues against a single recent origin followed by subsequent dispersal; instead, environmental change triggered similar adaptive responses in geographically isolated populations that independently acquired the capacity to colonise mammals. That the MRCA predates the industrial era suggests the genetic potential for thermotolerance existed long before its expression became advantageous. This supports the notion of latent capabilities awaiting environmental triggers.

Critically, the mechanisms enabling *C. auris*' success: conserved stress response pathways, cross-protection between environmental and clinical stresses, and phenotypic plasticity are not unique to this species but represent fundamental features of fungal biology. If a single environmental fungus can breach mammalian thermal defences by activating conserved systems, the evolutionary barriers protecting mammals may be more permeable than previously assumed. The question shifts from whether climate change will enable additional fungi to overcome thermal barriers to how many, how soon, and whether we can predict which species pose the greatest risk.

5. Broader Implications: Beyond *C. auris*

The evolutionary trajectory of *C. auris* represents more than the emergence of a single problematic pathogen. It provides a case study in how systematic environmental change can transform the relationship between mammals and the fungal kingdom. If thermotolerance evolves through activation of conserved molecular pathways present across diverse fungal taxa, we face not an isolated threat but a fundamental shift in the selective landscape that could generate numerous *C. auris*-equivalent pathogens from the vast reservoir of environmental fungi.

5.1. The Scale of the Problem

Current estimates place the total number of fungal species at 3-5 million (Blackwell, 2011). Historically, fewer than 0.1% of described species could grow above ambient temperature. This thermal gap has been the principal barrier separating mammalian hosts from fungal disease (Brown et al., 2017).

As warming erodes this separation, consider a deliberately conservative scenario: if climate change confers thermotolerance on just 0.01% of an estimated 4.3 million fungal species, this would yield 430 newly thermotolerant species. If merely 1% of those become opportunistic pathogens, humanity faces at least four novel pathogenic fungi. More realistic estimates, accounting for the prevalence of conserved stress response pathways and cross-protection mechanisms, could be substantially higher. That humans inhale 1-10 fungal spores with every breath, and that fungi constitute the majority of airborne biomass (Fröhlich-Nowoisky et al., 2009), underscores that exposure is unavoidable and that, so far, temperature is a highly effective barrier.

5.2. Parallel Evolution Across Fungal Lineages

C. auris is not an isolated anomaly. Multiple fungal species have recently expanded host ranges, enhanced thermotolerance, or emerged as novel pathogens, suggesting that environmental change exerts parallel selective pressures across diverse fungal lineages.

Cryptococcus gattii has expanded from tropical regions into temperate zones, causing infections in previously unaffected areas (Chybowska et al., 2020). *Emergomyces africanus* emerged in South Africa as a cause of disseminated mycosis in immunocompromised individuals (Dukik et al., 2017). *Batrachochytrium salamandrivorans* has devastated amphibian populations globally (Chybowska et al., 2020; Rohr and Cohen, 2020). *Aspergillus fumigatus*, among the most prevalent fungal pathogens, is thought to have developed azole resistance through agricultural fungicide exposure (Gonzalez-Jimenez, 2021; Kang et al., 2022; Lockhart et al., 2023). The related *Candida haemulonii* complex shows increasing clinical relevance despite historically lacking thermotolerance (Casadevall et al., 2019; Gómez-Gaviria et al., 2023), and *Candida parapsilosis* has developed fluconazole resistance with a growing global case burden (Arastehfar et al., 2020; Daneshnia et al., 2023). *Trichophyton indotineae*, first classified in 2020 (Kano et al., 2020), is now the dominant cause of dermatophytosis (Ebert et al., 2020) , which affects 20–25% of the global population, with increasing drug resistance reported from India (Lockhart

et al., 2023).

Perhaps most striking is the recent case of *Rhodosporidiobolus fluvialis*, a marine yeast identified as the cause of fungemia in two unrelated patients in China. Incubation at 37°C induced rapid mutagenesis in *R. fluvialis*, generating phenotypes with increased virulence and resistance to fluconazole and caspofungin, a process attributed to reactive oxygen species accumulation during heat stress (Huang et al., 2024). This temperature-induced mutagenesis represents a mechanism through which rising environmental temperatures could drive similar evolutionary transitions in other species.

These examples share common features: recent emergence or range expansion, association with environmental change, and exploitation of previously inaccessible thermal or immunological niches. The pattern is unlikely to represent a series of independent evolutionary accidents; rather, it reflects the predictable outcome of environmental change systematically selecting for adaptations across fungal communities.

5.3. Structural Inequality and Disproportionate Burden

The threat posed by emerging fungal pathogens falls unevenly across human populations. Tropical and subtropical regions face the greatest risk: baseline environmental temperatures are already closer to mammalian body temperature, fungal biodiversity provides larger reservoirs of potential pathogens, and climate change impacts are more severe (Altizer et al., 2013; Rohr and Cohen, 2020). Yet these high-risk regions typically possess the least capacity to respond (Chybowska et al., 2020).

Healthcare infrastructure disparities amplify this asymmetry (Chybowska et al., 2020). Accurate identification of *C. auris* and related emerging species require molecular diagnostic capabilities often unavailable in resource-limited settings; effective therapy demands access to expensive antifungals that may be unaffordable; and infection control measures require resources that strained healthcare systems cannot provide. Paradoxically, the hospitals most likely to encounter emerging fungal pathogens may be least equipped to identify and control them.

Socioeconomic conditions further compound disease risk (Brown et al., 2017; Pfaller and Diekema, 2007). Immunocompromised populations, including individuals with HIV/AIDS, cancer patients, and transplant recipients, face the highest susceptibility yet are disproportionately represented in low-income communities with limited healthcare access (Brown et al., 2017; Pfaller and Diekema, 2007). Malnutrition, endemic infections, and chronic diseases further compromise immunity, expanding the pool of vulnerable individuals, while agricultural workers, miners, and others who disturb soil environments face increased exposure, often with inadequate protection (Brown et al., 2012).

The same regions experiencing the greatest anthropogenic environmental disruption, where intensive agricultural fungicide use and industrial activities impose novel selective pressures on fungal communities, may serve as evolutionary pools for emerging pathogens while lacking the resources to monitor or respond to threats they generate (Casadevall et al., 2019). Global health infrastructure has historically prioritised viral and bacterial threats, and while this is beginning to shift, the legacy of neglect manifests as limited surveillance, inadequate diagnostic capacity, and insufficient investment in antifungal development for diseases that primarily affect low-income populations (Brown et al., 2017).

5.4. The Limits of Traditional Control

Traditional approaches to emerging infectious diseases, such as surveillance, containment, and treatment, face fundamental challenges when applied to fungal pathogens (Brown et al., 2017). Unlike bacterial or viral diseases spreading through defined transmission chains, fungal infections arise primarily from environmental exposure, and controlling environmental reservoirs is effectively impossible: we cannot sterilise soil, eliminate airborne spores, or prevent human contact with the fungal world (Brown et al., 2012).

C. auris illustrates these limitations starkly. Despite rigorous cleaning protocols, it persists in healthcare environments and causes repeated outbreaks (Chybowska et al., 2020; Gómez-Gaviria et al., 2023). If a known pathogen cannot be effectively controlled within the relatively contained hospital environment, our capacity to manage

multiple emerging fungal species across diverse environmental reservoirs is severely limited.

The treatment-resistance cycle creates an additional challenge without clear resolution (Pfaller and Diekema, 2007). Widespread antifungal use selects for resistance, but withholding treatment condemns infected patients to high mortality. Unlike antibacterial stewardship programs, which can sometimes reverse resistance trends by reducing selection pressure, antifungal resistance can arise from environmental selection independent of clinical drug use (Brown et al., 2017; Casadevall et al., 2019; Fisher et al., 2018). The rate of resistance emergence has historically outpaced the development of new antifungal agents (Fisher et al., 2018), raising the prospect of encountering pathogens that arrive already resistant, leaving clinicians without effective interventions from the moment of emergence.

These limitations underscore a central point: if reactive control strategies are inadequate even for known pathogens, they will be overwhelmed by the emergence of multiple novel threats. A fundamentally different approach is required.

6. Future Directions: Toward Predictive Mycology

The emergence of *C. auris* reveals a pattern: conserved molecular mechanisms, climate-driven selection, and human-mediated dispersal converging to breach mammalian thermal defences, a pattern that is likely to recur across fungal lineages. Responding effectively requires transforming our approach from reactive crisis management to predictive mycology: integrating evolutionary theory, ecological surveillance, and computational modelling to forecast which fungi pose the greatest threats, where they will emerge, and how to intervene before they do.

6.1. Environmental Surveillance and Predictive Modelling

Mathematical models enable exploration of processes too slow to observe directly, span scales from molecules to ecosystems, and test interventions before deployment. Computational models of heat shock responses in *Candida albicans* have successfully predicted pathogen behaviour under clinically relevant conditions that are difficult to study experimentally (Leach et al., 2012). Laboratory evolution demonstrates that fungi can rapidly evolve thermotolerance through multiple genetic routes (De Crecy et al., 2009; Schwiesow et al., 2025), and population genetic approaches pioneered in plant pathogen research—using genome scans to identify signatures of adaptation—demonstrate feasibility for predicting evolutionary responses to environmental change (Grandaubert et al., 2019; Stukenbrock and Bataillon, 2012).

The critical limitation is the absence of ancestral population data to parametrise these models (Chybowska et al., 2020). We observe clinical isolates after their emergence but miss the selective episodes that precede detection. This necessitates prospective environmental surveillance: sampling environmental fungi now, characterising thermal-tolerance distributions, quantifying genetic variation, and measuring selection coefficients under warming scenarios to provide the empirical foundation for evolutionary forecasting. Metagenomic sequencing of hospital air, high-risk soils, and agricultural settings where antifungal exposure selects for resistance would create early warning systems for emerging thermotolerant fungi (Nnadi and Carter, 2021).

Ecological niche modelling demonstrates feasibility at geographic scales. Climate projections predict substantial range expansions for soil-borne pathogens such as *Coccidioides*, with infections potentially increasing 50% as endemic regions shift (Nnadi and Carter, 2021). Genomic offset modelling has forecast distribution shifts in forest fungal pathogens (Hessenauer et al., 2025) successfully. Extended to human pathogens, such approaches could map regions where environmental conditions will become permissive for thermotolerant fungi, overlaying disease risk onto population distributions to identify vulnerable communities before outbreaks occur.

The ultimate goal is integration across scales: linking molecular mechanisms to population dynamics to epidemiological outcomes. Agent-based models provide computational scaffolding for this integration, simulating individual fungal genotypes competing under climate selection and occasionally dispersing into healthcare settings, where epidemiological dynamics determine outbreak success. The framework exists; implementation requires sustained interdisciplinary collaboration and robust data infrastructure.

6.2. Technological Development and Capacity Building

Prediction alone is insufficient without the tools to act on it. Definitive identification of *C. auris* currently depends on MALDI-TOF mass spectrometry with a curated reference database or on sequencing of the ribosomal ITS and D1/D2 regions, while rapid screening relies on species-specific real-time PCR (Mizusawa et al., 2017; Zhu et al., 2020). Standard biochemical panels misidentify the species, and both mass spectrometry and sequencing need capital equipment and reference databases that many hospitals lack, so definitive identification can take weeks (Chybowska et al., 2020). Such delays can prove fatal for critically ill patients. Closing this gap means more than shipping instruments. It would entail placing validated PCR assays and reference spectra in regional laboratories, supplying the reagents and controls they consume, training staff to run and interpret them, and funding this durably rather than once. This is what we intend by technology transfer and capacity building.

Drug development must move beyond single-target paradigms that inevitably lead to resistance. Conserved stress-response pathways, such as Hog1, represent attractive targets for broad-spectrum antifungals effective against diverse emerging pathogens (Day et al., 2018; Shivarathri et al., 2020). Host-directed therapies that enhance immunity or disrupt virulence mechanisms could provide antifungal activity without selecting for resistance (Gómez-Gaviria et al., 2023). Novel agents currently in clinical trials may offer temporary relief (Chybowska et al., 2020), but given the speed at which resistance evolves, continuous development pipelines rather than one-off drug approvals are essential.

This effort must therefore prioritise the tropical and low-income settings that face the greatest risk yet hold the least capacity to respond (Nnadi and Carter, 2021; Ramos Irizarry et al., 2025), and local researchers should lead it rather than receiving interventions designed elsewhere; their knowledge of local ecology, transmission, and healthcare constraints is what makes solutions fit local needs. Bioinformatic tools for identifying essential gene sets directly in organisms of interest, rather than extrapolating from model species, represent a key priority (Gómez-Gaviria et al., 2023; Kim et al., 2019).

An effective response, therefore, demands coordination among medical mycology, evolutionary biology, climate science, and public health. The infrastructure for pathogen surveillance exists: influenza forecasting and vaccine strain selection demonstrate that pathogen prediction works when adequately resourced. However, equivalent systems for fungal pathogens require sustained investment and cross-sectoral coordination between medical, agricultural, and environmental domains.

Pre-determining likely candidate species would enable informed, anticipatory screening and expedited diagnostics, gaining crucial time for patients, healthcare facilities, and researchers. Open-access repositories for fungal genomes, phenotypes, and clinical outcomes would provide the raw material for computational prediction, while population genomic analyses integrating both polymorphism and divergence data can disentangle the types of selection acting along genomes and identify functionally important loci (Taliadoros et al., 2025). Understanding how evolutionary potential is maintained in pathogen populations despite strong directional selection requires close integration of evolutionary genomics with experimental studies (Stukenbrock and Bataillon, 2012).

7. Conclusion

The emergence of *C. auris* forces a recognition that mammals' historical freedom from fungal disease was not a permanent feature of our biology, but a contingent consequence of environmental conditions that are now changing. We evolved in a world where temperature separated us from most fungi; that world is disappearing. Ectothermic vertebrates already experience fungal disease at rates that would be catastrophic for human populations; the thermal immunity that distinguishes mammals from amphibians, reptiles, and fish is being systematically eroded. The future of mammalian immunity revolves around one central question: Why did *Candida auris* in particular emerge as a pathogen when others did not? As discussed in *Thermotolerance Foundation*, *Pre-adaptations Rather Than Novel Evolution* and throughout the article, many strategies *C. auris* utilizes are conserved and widespread among fungal taxa. It is reasonable to expect that we should currently be observing a rise of infections caused by other fungi, especially related *Candida spp*. A current rise of candidiasis

infections caused by other *Candida spp.*(Arastehfar et al., 2020; Daneshnia et al., 2023; Gómez-Gaviria et al., 2023; Harchand et al., 2025) in addition to the observations discussed in *Parallel Evolution Across Fungal Lineages* are reasons that we might currently be witnessing early stages of their emergence. However, many of these species are even less studied than *C. auris* and their documentation suffer from the same issues, like misidentifications and re-classifications into *Candidozyma*. With the analysis of the phylogeny of the *Candida* genus still ongoing and with spotty global case documentation, current research lacks sufficient evidence to draw any definite conclusions about other species not being established as pathogens in the near future.

A pragmatic agenda follows directly from the preceding sections—prospective environmental sampling and experimental-evolution studies, validated ecological niche models, continued antifungal development, and mycological capacity building where risk is highest—all converging on a single long-term goal: a functioning system of predictive mycology in which environmental surveillance feeds real-time data into models that generate probabilistic forecasts of emergence, guiding targeted interventions before outbreaks occur. This vision is ambitious but feasible, and the alternative, accepting repeated crises as thermotolerant, multidrug-resistant pathogens emerge one after another, is untenable.

The promise of predictive mycology lies not in eliminating uncertainty but in transforming how we engage with it. Rather than waiting for the next pathogen to appear, we can monitor the evolutionary landscape, identify species approaching thermal thresholds, and intervene before they cross over (Altizer et al., 2013; Rohr and Cohen, 2020). This requires treating fungal disease emergence not as a series of independent shocks but as a predictable consequence of systematic environmental change, one that theoretical biology, integrated with empirical surveillance and coordinated global action, can help us navigate.

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Research and Writing: C. S. C. G. All authors approved the final version.

Data & Code Availability

The data underlying **Figure** 2 is available at `https://github.com/tecoevo/c_auris`, and the review by Jones et al. Jones et al. (2024) has proved invaluable in compiling this data.

Conflict of Interest

The authors declare no conflict of interest.

References

M. Abe, A. Koizumi, T. Umeyama, H. Tomuro, Y. Muraosa, N. Nakyayama, S. Oiki, S. Shimada, Y. Murakami, M. Ota, I. Yoshimi, A. Takahashi, T. Ono, M. Sasaki, E. Uchida-Fujii, M. Nakashita, H. Kurosu, T. Yamagishi, T. Shinohara, Y. Hoshino, and Y. Miyazaki. Microbiological Characteristics and Drug Resistance Rates of Candida auris Isolates in Japan. *Japanese Journal of Infectious Diseases*, 78(6):179–185, 2025. doi: 10.7883/yoken.JJID.2025.025.

M. Ademe and F. Girma. Candida auris: From Multidrug Resistance to Pan-Resistant Strains. *Infection and Drug Resistance*, 13:1287–1294, May 2020. ISSN null. doi: 10.2147/IDR.S249864. URL `https://www.tandfonline.com/doi/abs/10.2147/IDR.S249864`. _eprint: https://www.tandfonline.com/doi/pdf/10.2147/IDR.S249864.

S. H. Ahmed, I. M. A. El-Kholy, A. A. El-Mehalawy, E. M. Mahmoud, and N. A. Elkady. Molecular characterization of some multidrug resistant Candida Auris in egypt. *Scientific Reports*, 15(1):4917, Feb. 2025. ISSN 2045-2322. doi: 10.1038/s41598-025-88656-3. URL `https://www.nature.com/articles/s41598-025-88656-3`.

S. Altizer, R. S. Ostfeld, P. T. J. Johnson, S. Kutz, and C. D. Harvell. Climate Change and Infectious Diseases: From Evidence to a Predictive Framework. *Science*, 341(6145):514–519, Aug. 2013. ISSN 0036-8075, 1095-9203. doi: 10.1126/science.1239401. URL https://www.science.org/doi/10.1126/science.1239401.

A. Arastehfar, C. Lass-Flörl, R. Garcia-Rubio, F. Daneshnia, M. Ilkit, T. Boekhout, T. Gabaldon, and D. S. Perlin. The Quiet and Underappreciated Rise of Drug-Resistant Invasive Fungal Pathogens. *Journal of Fungi*, 6(3):138, Sept. 2020. ISSN 2309-608X. doi: 10.3390/jof6030138. URL https://www.mdpi.com/2309-608X/6/3/138.

P. Arora, P. Singh, Y. Wang, A. Yadav, K. Pawar, A. Singh, G. Padmavati, J. Xu, and A. Chowdhary. Environmental Isolation of Candida auris from the Coastal Wetlands of Andaman Islands, India. *mBio*, 12(2):10.1128/mbio.03181–20, Mar. 2021. doi: 10.1128/mbio.03181-20. URL https://journals.asm.org/doi/full/10.1128/mbio.03181-20.

M. M. Biebl and J. Buchner. Structure, Function, and Regulation of the Hsp90 Machinery. *Cold Spring Harbor Perspectives in Biology*, 11(9):a034017, Sept. 2019. ISSN 1943-0264. doi: 10.1101/cshperspect.a034017. URL http://cshperspectives.cshlp.org/lookup/doi/10.1101/cshperspect.a034017.

J. Bing, H. Du, P. Guo, T. Hu, M. Xiao, S. Lu, C. J. Nobile, H. Chu, and G. Huang. Candida auris-associated hospitalizations and outbreaks, China, 2018–2023. *Emerging Microbes & Infections*, 13(1):2302843, Dec. 2024. ISSN null. doi: 10.1080/22221751.2024.2302843. URL https://doi.org/10.1080/22221751.2024.2302843. _eprint: https://doi.org/10.1080/22221751.2024.2302843.

M. Blackwell. The Fungi: 1, 2, 3 … 5.1 million species? *American Journal of Botany*, 98(3):426–438, 2011. ISSN 1537-2197. doi: 10.3732/ajb.1000298. URL https://onlinelibrary.wiley.com/doi/abs/10.3732/ajb.1000298. _eprint: https://bsapubs.onlinelibrary.wiley.com/doi/pdf/10.3732/ajb.1000298.

A. J. P. Brown, L. E. Cowen, A. di Pietro, and J. Quinn. Stress Adaptation. *Microbiology Spectrum*, 5(4): 10.1128/microbiolspec.funk–0048–2016, July 2017. doi: 10.1128/microbiolspec.funk-0048-2016. URL https://journals.asm.org/doi/10.1128/microbiolspec.funk-0048-2016.

G. D. Brown, D. W. Denning, N. A. R. Gow, S. M. Levitz, M. G. Netea, and T. C. White. Hidden Killers: Human Fungal Infections. *Science Translational Medicine*, 4(165), Dec. 2012. ISSN 1946-6234, 1946-6242. doi: 10.1126/scitranslmed.3004404. URL https://www.science.org/doi/10.1126/scitranslmed.3004404.

J. L. Brown, C. Delaney, B. Short, M. C. Butcher, E. McKloud, C. Williams, R. Kean, and G. Ramage. Candida auris Phenotypic Heterogeneity Determines Pathogenicity *In Vitro*. *mSphere*, 5(3):e00371–20, June 2020. ISSN 2379-5042. doi: 10.1128/mSphere.00371-20. URL https://journals.asm.org/doi/10.1128/mSphere.00371-20.

H. Carolus, S. Pierson, J. F. Muñoz, A. Subotić, R. B. Cruz, C. A. Cuomo, and P. V. Dijck. Genome-Wide Analysis of Experimentally Evolved Candida auris Reveals Multiple Novel Mechanisms of Multidrug Resistance. *mBio*, 12(2):10.1128/mbio.03333–20, 2021. doi: 10.1128/mbio.03333-20.

A. Casadevall. Immunity to Invasive Fungal Diseases. *Annual Review of Immunology*, 40(1):121–141, Apr. 2022. ISSN 0732-0582, 1545-3278. doi: 10.1146/annurev-immunol-101220-034306. URL https://www.annualreviews.org/doi/10.1146/annurev-immunol-101220-034306.

A. Casadevall, D. P. Kontoyiannis, and V. Robert. On the Emergence of Candida auris: Climate Change, Azoles, Swamps, and Birds. *mBio*, 10(4):e01397–19, Aug. 2019. ISSN 2161-2129, 2150-7511. doi: 10.1128/mBio.01397-19. URL https://journals.asm.org/doi/10.1128/mBio.01397-19.

A. Casadevall, D. P. Kontoyiannis, and V. Robert. Environmental Candida auris and the Global Warming Emergence Hypothesis. *mBio*, 12(2):10.1128/mbio.00360–21, Mar. 2021. doi: 10.1128/mbio.00360-21. URL https://journals.asm.org/doi/full/10.1128/mbio.00360-21.

E. Cendejas-Bueno, A. Kolecka, A. Alastruey-Izquierdo, B. Theelen, M. Groenewald, M. Kostrzewa, M. Cuenca-Estrella, A. Gómez-López, and T. Boekhout. Reclassification of the Candida haemulonii Complex as Candida haemulonii (C. haemulonii Group I), C. duobushaemulonii sp. nov. (C. haemulonii Group II), and C. haemulonii var. vulnera var. nov.: Three Multiresistant Human Pathogenic Yeasts. *Journal of Clinical Microbiology*, 50(11):3641–3651, Nov. 2012. ISSN 0095-1137, 1098-660X. doi: 10.1128/JCM.02248-12. URL https://journals.asm.org/doi/10.1128/JCM.02248-12.

A. Chakrabarti and P. Sood. On the emergence, spread and resistance of Candida auris: host, pathogen and environmental tipping points. *Journal of Medical Microbiology*, 70(3):001318, 2021. ISSN 1473-5644. doi: 10.1099/jmm.0.001318. URL https://www.microbiologyresearch.org/content/journal/jmm/10.1099/jmm.0.001318.

S. Chatterjee, S. V. Alampalli, R. K. Nageshan, S. T. Chettiar, S. Joshi, and U. S. Tatu. Draft genome of a commonly misdiagnosed multidrug resistant pathogen Candida auris. *BMC Genomics*, 16(1):686, Dec. 2015. ISSN 1471-2164. doi: 10.1186/s12864-015-1863-z. URL http://www.biomedcentral.com/1471-2164/16/686.

N. A. Chow, T. De Groot, H. Badali, M. Abastabar, T. M. Chiller, and J. F. Meis. Potential Fifth Clade of *Candida auris,* Iran, 2018. *Emerging Infectious Diseases*, 25(9):1780–1781, Sept. 2019. ISSN 1080-6040, 1080-6059. doi: 10.3201/eid2509.190686.

649 URL http://wwwnc.cdc.gov/eid/article/25/9/19-0686_article.htm.

650 N. A. Chow, J. F. Muñoz, L. Gade, E. L. Berkow, X. Li, R. M. Welsh, K. Forsberg, S. R. Lockhart, R. Adam, A. Alanio, A. Alastruey-
651 Izquierdo, S. Althawadi, A. B. Araúz, R. Ben-Ami, A. Bharat, B. Calvo, M. Desnos-Ollivier, P. Escandón, D. Gardam,
652 R. Gunturu, C. H. Heath, O. Kurzai, R. Martin, A. P. Litvintseva, and C. A. Cuomo. Tracing the Evolutionary History and
653 Global Expansion of Candida auris Using Population Genomic Analyses. *mBio*, 11(2):10.1128/mbio.03364–19, Apr. 2020.
654 doi: 10.1128/mbio.03364-19. URL https://journals.asm.org/doi/10.1128/mbio.03364-19.

655 A. Chowdhary, C. Sharma, S. Duggal, K. Agarwal, A. Prakash, P. K. Singh, S. Jain, S. Kathuria, H. S. Randhawa, F. Hagen, and
656 J. F. Meis. New Clonal Strain of *Candida auris* , Delhi, India: New Clonal Strain of *Candida auris* , Delhi, India. *Emerging*
657 *Infectious Diseases*, 19(10):1670–1673, Oct. 2013. ISSN 1080-6040, 1080-6059. doi: 10.3201/eid1910.130393. URL
658 http://wwwnc.cdc.gov/eid/article/19/10/13-0393_article.htm.

659 A. Chowdhary, V. Anil Kumar, C. Sharma, A. Prakash, K. Agarwal, R. Babu, K. R. Dinesh, S. Karim, S. K. Singh, F. Hagen, and
660 J. F. Meis. Multidrug-resistant endemic clonal strain of Candida auris in India. *European Journal of Clinical Microbiology*
661 *& Infectious Diseases*, 33(6):919–926, June 2014. ISSN 0934-9723, 1435-4373. doi: 10.1007/s10096-013-2027-1. URL
662 http://link.springer.com/10.1007/s10096-013-2027-1.

663 A. Chowdhary, A. Prakash, C. Sharma, M. Kordalewska, A. Kumar, S. Sarma, B. Tarai, A. Singh, G. Upadhyaya, S. Upadhyay,
664 P. Yadav, P. K. Singh, V. Khillan, N. Sachdeva, D. S. Perlin, and J. F. Meis. A multicentre study of antifungal susceptibility
665 patterns among 350 Candida auris isolates (2009–17) in India: role of the ERG11 and FKS1 genes in azole and echinocandin
666 resistance. *Journal of Antimicrobial Chemotherapy*, 73(4):891–899, Apr. 2018. ISSN 0305-7453, 1460-2091. doi: 10.1093/
667 jac/dkx480. URL https://academic.oup.com/jac/article/73/4/891/4794718.

668 A. Chowdhary, K. Jain, and N. Chauhan. Candida auris Genetics and Emergence. *Annual Review of Microbiology*, 77(Volume
669 77, 2023):583–602, Sept. 2023. ISSN 0066-4227, 1545-3251. doi: 10.1146/annurev-micro-032521-015858. URL
670 https://www.annualreviews.org/content/journals/10.1146/annurev-micro-032521-015858.

671 A. D. Chybowska, D. S. Childers, and R. A. Farrer. Nine Things Genomics Can Tell Us About Candida auris. *Frontiers in*
672 *Genetics*, 11:351, Apr. 2020. ISSN 1664-8021. doi: 10.3389/fgene.2020.00351. URL https://www.frontiersin.
673 org/article/10.3389/fgene.2020.00351/full.

674 O. A. Cornely, R. Sprute, M. Bassetti, S. C.-A. Chen, A. H. Groll, O. Kurzai, C. Lass-Flörl, L. Ostrosky-Zeichner, R. Rautemaa-
675 Richardson, G. Revathi, M. E. Santolaya, P. L. White, A. Alastruey-Izquierdo, M. C. Arendrup, J. Baddley, A. Barac, R. Ben-
676 Ami, A. J. Brink, J. H. Grothe, J. Guinea, F. Hagen, B. Hochhegger, M. Hoenigl, S. Husain, K. Jabeen, H. E. Jensen, S. S.
677 Kanj, P. Koehler, T. Lehrnbecher, R. E. Lewis, J. F. Meis, M. H. Nguyen, Z. D. Pana, P.-M. Rath, I. Reinhold, D. Seidel,
678 T. Takazono, D. C. Vinh, S. X. Zhang, J. Afeltra, A. M. S. Al-Hatmi, A. Arastehfar, S. Arikan-Akdagli, F. Bongomin, F. Carlesse,
679 M. Chayakulkeeree, L. Y. A. Chai, L. Chamani-Tabriz, T. Chiller, A. Chowdhary, C. J. Clancy, A. L. Colombo, A. Cortegiani, D. E.
680 Corzo Leon, L. Drgona, A. Dudakova, J. Farooqi, S. Gago, M. Ilkit, J. D. Jenks, N. Klimko, R. Krause, A. Kumar, K. Lagrou,
681 M. S. Lionakis, B. E. Lmimouni, M. K. Mansour, J. Meletiadis, S. C. Mellinghoff, M. Mer, M. Mikulska, P. Montravers, C. F.
682 Neoh, V. Ozenci, L. Pagano, P. Pappas, T. F. Patterson, P. Puerta-Alcalde, L. Rahimli, S. Rahn, E. Roilides, C. Rotstein,
683 T. Ruegamer, R. Sabino, J. Salmanton-García, I. S. Schwartz, E. Segal, N. Sidharthan, T. Singhal, J. Sinko, R. Soman,
684 A. Spec, J. Steinmann, J. Stemler, S. J. Taj-Aldeen, A. F. Talento, G. R. Thompson, C. Toebben, H. Villanueva-Lozano,
685 R. Wahyuningsih, B. Weinbergerová, N. Wiederhold, B. Willinger, P. C. Y. Woo, and L.-P. Zhu. Global guideline for
686 the diagnosis and management of candidiasis: an initiative of the ECMM in cooperation with ISHAM and ASM. *The*
687 *Lancet Infectious Diseases*, 25(5):e280–e293, May 2025. ISSN 14733099. doi: 10.1016/S1473-3099(24)00749-7. URL
688 https://linkinghub.elsevier.com/retrieve/pii/S1473309924007497.

689 L. E. Cowen and S. Lindquist. Hsp90 Potentiates the Rapid Evolution of New Traits: Drug Resistance in Diverse Fungi.
690 *Science*, 309(5744):2185–2189, 2005. doi: 10.1126/science.1118370.

691 F. Daneshnia, J. N. De Almeida Júnior, M. Ilkit, L. Lombardi, A. M. Perry, M. Gao, C. J. Nobile, M. Egger, D. S. Perlin, B. Zhai,
692 T. M. Hohl, T. Gabaldón, A. L. Colombo, M. Hoenigl, and A. Arastehfar. Worldwide emergence of fluconazole-resistant
693 Candida parapsilosis: current framework and future research roadmap. *The Lancet Microbe*, 4(6):e470–e480, June 2023.
694 ISSN 26665247. doi: 10.1016/S2666-5247(23)00067-8. URL https://linkinghub.elsevier.com/retrieve/
695 pii/S2666524723000678.

696 S. Das, R. Tigga, G. Rai, P. K. Singh, S. Datt, A. Tyagi, and N. P. Singh. Candida auris colonization in an immunocompetent
697 patient: A new threat in medical ICU. *Medical Mycology Case Reports*, 21:54–56, Sept. 2018. ISSN 22117539. doi: 10.
698 1016/j.mmcr.2018.05.004. URL https://linkinghub.elsevier.com/retrieve/pii/S2211753918300678.

699 A. M. Day, M. M. McNiff, A. Da Silva Dantas, N. A. R. Gow, and J. Quinn. Hog1 Regulates Stress Tolerance and Virulence in
700 the Emerging Fungal Pathogen Candida auris. *mSphere*, 3(5):e00506–18, Oct. 2018. ISSN 2379-5042. doi: 10.1128/
701 mSphere.00506-18. URL https://journals.asm.org/doi/10.1128/mSphere.00506-18.

702 E. De Crecy, S. Jaronski, B. Lyons, T. J. Lyons, and N. O. Keyhani. Directed evolution of a filamentous fungus for

thermotolerance. *BMC Biotechnology*, 9(1):74, Dec. 2009. ISSN 1472-6750. doi: 10.1186/1472-6750-9-74. URL https://bmcbiotechnol.biomedcentral.com/articles/10.1186/1472-6750-9-74.

S. De Gaetano, A. Midiri, G. Mancuso, M. G. Avola, and C. Biondo. Candida auris Outbreaks: Current Status and Future Perspectives. *Microorganisms*, 12(5):927, May 2024. ISSN 2076-2607. doi: 10.3390/microorganisms12050927. URL https://www.mdpi.com/2076-2607/12/5/927. Number: 5.

D. W. Denning. Global incidence and mortality of severe fungal disease. *The Lancet Infectious Diseases*, 24(7):e428–e438, July 2024. ISSN 14733099. doi: 10.1016/S1473-3099(23)00692-8. URL https://linkinghub.elsevier.com/retrieve/pii/S1473309923006928.

H. Du, J. Bing, T. Hu, C. L. Ennis, C. J. Nobile, and G. Huang. Candida auris: Epidemiology, biology, antifungal resistance, and virulence. *PLOS Pathogens*, 16(10):e1008921, Oct. 2020. ISSN 1553-7374. doi: 10.1371/journal.ppat.1008921. URL https://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1008921.

K. Dukik, J. F. Muñoz, Y. Jiang, P. Feng, L. Sigler, J. B. Stielow, J. Freeke, A. Jamalian, B. Gerrits van den Ende, J. G. McEwen, O. K. Clay, I. S. Schwartz, N. P. Govender, T. G. Maphanga, C. A. Cuomo, L. F. Moreno, C. Kenyon, A. M. Borman, and S. de Hoog. Novel taxa of thermally dimorphic systemic pathogens in the Ajellomycetaceae (Onygenales). *Mycoses*, 60(5):296–309, 2017. ISSN 1439-0507. doi: 10.1111/myc.12601. URL https://onlinelibrary.wiley.com/doi/abs/10.1111/myc.12601. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/myc.12601.

A. Ebert, M. Monod, K. Salamin, A. Burmester, S. Uhrlaß, C. Wiegand, U.-C. Hipler, C. Krüger, D. Koch, F. Wittig, S. B. Verma, A. Singal, S. Gupta, R. Vasani, A. Saraswat, R. Madhu, S. Panda, A. Das, M. M. Kura, A. Kumar, S. Poojary, S. Schirm, Y. Gräser, U. Paasch, and P. Nenoff. Alarming India-wide phenomenon of antifungal resistance in dermatophytes: A multicentre study. *Mycoses*, 63(7):717–728, July 2020. ISSN 1439-0507. doi: 10.1111/myc.13091.

P. Escandón, N. A. Chow, D. H. Caceres, L. Gade, E. L. Berkow, P. Armstrong, S. Rivera, E. Misas, C. Duarte, H. Moulton-Meissner, R. M. Welsh, C. Parra, L. A. Pescador, N. Villalobos, S. Salcedo, I. Berrio, C. Varón, A. Espinosa-Bode, S. R. Lockhart, B. R. Jackson, A. P. Litvintseva, M. Beltran, and T. M. Chiller. Molecular Epidemiology of Candida auris in Colombia Reveals a Highly Related, Countrywide Colonization With Regional Patterns in Amphotericin B Resistance. *Clinical Infectious Diseases*, 68(1):15–21, Jan. 2019. ISSN 1058-4838. doi: 10.1093/cid/ciy411. URL https://doi.org/10.1093/cid/ciy411.

T. Fang, H. Lu, and Y. Jiang. Extracellular fungal Hsp90 represents a promising therapeutic target for combating fungal infections. *European Journal of Pharmaceutical Sciences*, 207:107041, Apr. 2025. ISSN 09280987. doi: 10.1016/j.ejps.2025.107041. URL https://linkinghub.elsevier.com/retrieve/pii/S0928098725000405.

M. C. Fisher, G. L. Koenig, T. J. White, G. San-Blas, R. Negroni, I. G. Alvarez, B. Wanke, and J. W. Taylor. Biogeographic range expansion into South America by Coccidioides immitis mirrors New World patterns of human migration. *Proceedings of the National Academy of Sciences*, 98(8):4558–4562, 2001. ISSN 0027-8424. doi: 10.1073/pnas.071406098.

M. C. Fisher, N. J. Hawkins, D. Sanglard, and S. J. Gurr. Worldwide emergence of resistance to antifungal drugs challenges human health and food security. *Science*, 360(6390):739–742, May 2018. doi: 10.1126/science.aap7999. URL https://www.science.org/doi/10.1126/science.aap7999.

E. C. Francisco, D. H. Caceres, J. G. P. Brunelli, G. Garcia-Effron, A. Arastehfar, F. D. C. Ribeiro, M. N. De Almeida, S. S. Gonçalves, J. Nóbrega De Almeida, C. Lass-Flörl, T. Boekhout, and A. L. Colombo. An update on clinically relevant, rare, and emerging *Candida* and Saccharomycotina yeasts that have been recently reclassified from *Candida*. *Clinical Microbiology Reviews*, 38(4):e00064–23, Dec. 2025. ISSN 0893-8512, 1098-6618. doi: 10.1128/cmr.00064-23. URL https://journals.asm.org/doi/10.1128/cmr.00064-23.

J. A. Fraser, S. S. Giles, E. C. Wenink, S. G. Geunes-Boyer, J. R. Wright, S. Diezmann, A. Allen, J. E. Stajich, F. S. Dietrich, J. R. Perfect, and J. Heitman. Same-sex mating and the origin of the Vancouver Island Cryptococcus gattii outbreak. *Nature*, 437(7063):1360–1364, Oct. 2005. ISSN 1476-4687. doi: 10.1038/nature04220. URL https://www.nature.com/articles/nature04220.

J. Fröhlich-Nowoisky, D. A. Pickersgill, V. R. Després, and U. Pöschl. High diversity of fungi in air particulate matter. *Proceedings of the National Academy of Sciences*, 106(31):12814–12819, Aug. 2009. doi: 10.1073/pnas.0811003106. URL https://www.pnas.org/doi/abs/10.1073/pnas.0811003106.

V. Garcia-Bustos, M. D. Cabañero-Navalon, A. Ruiz-Gaitán, M. Salavert, M. Á. Tormo-Mas, and J. Pemán. Climate change, animals, and Candida auris: insights into the ecological niche of a new species from a One Health approach. *Clinical Microbiology and Infection*, 29(7):858–862, July 2023. ISSN 1198743X. doi: 10.1016/j.cmi.2023.03.016. URL https://linkinghub.elsevier.com/retrieve/pii/S1198743X23001325.

M. A. Garcia-Solache and A. Casadevall. Global Warming Will Bring New Fungal Diseases for Mammals. *mBio*, 1(1):e00061–10, May 2010. ISSN 2161-2129, 2150-7511. doi: 10.1128/mBio.00061-10. URL https://journals.asm.org/doi/10.1128/mBio.00061-10.

M. Gómez-Gaviria, J. A. Martínez-Álvarez, J. O. Chávez-Santiago, and H. M. Mora-Montes. Candida haemulonii Complex and Candida auris: Biology, Virulence Factors, Immune Response, and Multidrug Resistance. *Infection and Drug Resistance*, Volume 16:1455–1470, Mar. 2023. ISSN 1178-6973. doi: 10.2147/IDR.S402754. URL https://www.dovepress.com/candida-haemulonii-complex-and-candida-auris-biology-virulence-factors-peer-reviewed-fulltext-artic

Gonzalez-Jimenez. Are Point Mutations in HMG-CoA Reductases (Hmg1 and Hmg2) a Step towards Azole Resistance in Aspergillus fumigatus? *Molecules*, 2021. doi: 10.3390/molecules26195975. URL https://www.mdpi.com/1420-3049/26/19/5975.

N. P. Govender, R. E. Magobo, R. Mpembe, M. Mhlanga, P. Matlapeng, C. Corcoran, C. Govind, W. Lowman, M. Senekal, and J. Thomas. Candida auris in South Africa, 2012–2016. *Emerging Infectious Diseases*, 24(11):2036–2040, Nov. 2018. ISSN 1080-6040. doi: 10.3201/eid2411.180368. URL https://pmc.ncbi.nlm.nih.gov/articles/PMC6200016/.

J. Grandaubert, J. Y. Dutheil, and E. H. Stukenbrock. The genomic determinants of adaptive evolution in a fungal pathogen. *Evolution Letters*, 3(3):299–312, June 2019. ISSN 2056-3744. doi: 10.1002/evl3.117. URL https://pure.mpg.de/view/item_3049174_2.

R. Harchand, B. Spruijtenburg, E. F. J. Meijer, T. de Groot, S. M. Rudramurthy, and J. F. Meis. Candida vulturna, the Next Fungal Menace? A Narrative Review. *Mycoses*, 68(5):e70070, 2025. ISSN 1439-0507. doi: 10.1111/myc.70070. URL https://onlinelibrary.wiley.com/doi/abs/10.1111/myc.70070. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/myc.70070.

H. Heaney, J. Laing, L. Paterson, A. W. Walker, N. A. R. Gow, E. M. Johnson, D. M. MacCallum, and A. J. P. Brown. The environmental stress sensitivities of pathogenic Candida species, including Candida auris, and implications for their spread in the hospital setting. *Medical Mycology*, 58(6):744–755, Jan. 2020. ISSN 1369-3786. doi: 10.1093/mmy/myz127. URL https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7398771/.

P. Hessenauer, N. Feau, R. Heinzelmann, and R. C. Hamelin. Genomic Exploration of Climate-driven Evolution and Evolutionary Convergence in Forest Pathogens. *Genome Biology and Evolution*, 17(5):evaf069, May 2025. ISSN 1759-6653. doi: 10.1093/gbe/evaf069. URL https://doi.org/10.1093/gbe/evaf069.

J. A. Hill, T. R. O'Meara, and L. E. Cowen. Fitness Trade-Offs Associated with the Evolution of Resistance to Antifungal Drug Combinations. *Cell Reports*, 10(5):809–819, 2015. doi: 10.1016/j.celrep.2015.01.009.

J. Huang, P. Hu, L. Ye, Z. Shen, X. Chen, F. Liu, Y. Xie, J. Yu, X. Fan, M. Xiao, C. K. M. Tsui, W. Wang, Y. Li, G. Zhang, K. H. Wong, L. Cai, F.-y. Bai, Y. Xu, and L. Wang. Pan-drug resistance and hypervirulence in a human fungal pathogen are enabled by mutagenesis induced by mammalian body temperature. *Nature Microbiology*, 9(7):1686–1699, June 2024. ISSN 2058-5276. doi: 10.1038/s41564-024-01720-y. URL https://www.nature.com/articles/s41564-024-01720-y.

B. R. Jackson, N. Chow, K. Forsberg, A. P. Litvintseva, S. R. Lockhart, R. Welsh, S. Vallabhaneni, and T. Chiller. On the Origins of a Species: What Might Explain the Rise of Candida auris? *Journal of Fungi*, 5(3):58, July 2019. ISSN 2309-608X. doi: 10.3390/jof5030058. URL https://www.mdpi.com/2309-608X/5/3/58.

C. R. Jones, C. Neill, A. M. Borman, E. L. Budd, M. Cummins, C. Fry, R. L. Guy, K. Jeffery, E. M. Johnson, R. Manuel, M. Mirfendereseky, G. Moore, B. Patel, S. Schelenz, K. Staniforth, S. K. Taori, and C. S. Brown. The laboratory investigation, management, and infection prevention and control of Candida auris: a narrative review to inform the 2024 national guidance update in England. *Journal of Medical Microbiology*, 73(5):001820, 2024. ISSN 1473-5644. doi: 10.1099/jmm.0.001820. URL https://www.microbiologyresearch.org/content/journal/jmm/10.1099/jmm.0.001820.

S. E. Kang, L. G. Sumabat, T. Melie, B. Mangum, M. Momany, and M. T. Brewer. Evidence for the agricultural origin of resistance to multiple antimicrobials in Aspergillus fumigatus, a fungal pathogen of humans. *G3 Genes|Genomes|Genetics*, 12 (2):jkab427, Feb. 2022. ISSN 2160-1836. doi: 10.1093/g3journal/jkab427. URL https://doi.org/10.1093/g3journal/jkab427.

R. Kano, U. Kimura, M. Kakurai, J. Hiruma, H. Kamata, Y. Suga, and K. Harada. Trichophyton indotineae sp. nov.: A New Highly Terbinafine-Resistant Anthropophilic Dermatophyte Species. *Mycopathologia*, 185(6):947–958, Dec. 2020. ISSN 0301-486X. doi: 10.1007/s11046-020-00455-8. URL https://pure.teikyo.jp/en/publications/trichophyton-indotineae-sp-nov-a-new-highly-terbinafine-resistant/.

S. H. Kim, K. R. Iyer, L. Pardeshi, J. F. Muñoz, N. Robbins, C. A. Cuomo, K. H. Wong, and L. E. Cowen. Genetic Analysis of *Candida auris* Implicates Hsp90 in Morphogenesis and Azole Tolerance and Cdr1 in Azole Resistance. *mBio*, 10(1): e02529–18, 2019. doi: 10.1128/mBio.02529-18.

L. Krishnasamy, J. Senthilganesh, C. Saikumar, and P. Nithyanand. Biofilm-forming fluconazole-resistant Candida auris causing vulvovaginal candidiasis in an immunocompetent patient: A case report. *Asian Pacific Journal of Tropical Medicine*, 14(2):94, Feb. 2021. ISSN 1995-7645. doi: 10.4103/1995-7645.306768. URL https://journals.lww.com/aptm/fulltext/2021/14020/biofilm_forming_fluconazole_resistant_candida.7.aspx.

K. Lagree and A. P. Mitchell. Fungal Biofilms: Inside Out. In *The Fungal Kingdom*, pages 873–886.

John Wiley & Sons, Ltd, 2017. ISBN 978-1-68367-082-7. doi: 10.1128/9781555819583.ch42. URL https://onlinelibrary.wiley.com/doi/abs/10.1128/9781555819583.ch42. Section: 42 _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1128/9781555819583.ch42.

Y. Lai and D. A. Dzombak. Use of Historical Data to Assess Regional Climate Change Use of Historical Data to Assess Regional Climate Change. *Journal of Climate*, 32(14):4299–4320, 2019. ISSN 0894-8755. doi: 10.1175/jcli-d-18-0630.1.

M. D. Leach, K. Tyc, A. J. P. Brown, and E. Klipp. Modelling the regulation of thermal adaptation in Candida albicans, a major fungal pathogen of humans. *PloS ONE*, 7(3):e32467, Mar. 2012. ISSN 1932-6203. doi: 10.1371/journal.pone.0032467. URL https://abdn.elsevierpure.com/en/publications/modelling-the-regulation-of-thermal-adaptation-in-candida-albican/.

W. G. Lee, J. H. Shin, Y. Uh, M. G. Kang, S. H. Kim, K. H. Park, and H.-C. Jang. First Three Reported Cases of Nosocomial Fungemia Caused by Candida auris. *Journal of Clinical Microbiology*, 49(9):3139–3142, Sept. 2011. ISSN 0095-1137, 1098-660X. doi: 10.1128/JCM.00319-11. URL https://journals.asm.org/doi/10.1128/JCM.00319-11.

F. Liu, Z.-D. Hu, X.-M. Zhao, W.-N. Zhao, Z.-X. Feng, A. Yurkov, S. Alwasel, T. Boekhout, K. Bensch, F.-L. Hui, F.-Y. Bai, and Q.-M. Wang. Phylogenomic analysis of the Candida auris- Candida haemuli clade and related taxa in the Metschnikowiaceae, and proposal of thirteen new genera, fifty-five new combinations and nine new species. *Persoonia - Molecular Phylogeny and Evolution of Fungi*, 52(1):22–43, 2024. doi: 10.3767/persoonia.2024.52.02.

Z. Liu, M. Pagani, D. Zinniker, R. DeConto, M. Huber, H. Brinkhuis, S. R. Shah, R. M. Leckie, and A. Pearson. Global Cooling During the Eocene-Oligocene Climate Transition. *Science*, 323(5918):1187–1190, Feb. 2009. doi: 10.1126/science.1166368. URL https://www.science.org/doi/10.1126/science.1166368.

S. R. Lockhart, K. A. Etienne, S. Vallabhaneni, J. Farooqi, A. Chowdhary, N. P. Govender, A. L. Colombo, B. Calvo, C. A. Cuomo, C. A. Desjardins, E. L. Berkow, M. Castanheira, R. E. Magobo, K. Jabeen, R. J. Asghar, J. F. Meis, B. Jackson, T. Chiller, and A. P. Litvintseva. Simultaneous Emergence of Multidrug-Resistant *Candida auris* on 3 Continents Confirmed by Whole-Genome Sequencing and Epidemiological Analyses. *Clinical Infectious Diseases*, 64(2):134–140, Jan. 2017. ISSN 1058-4838, 1537-6591. doi: 10.1093/cid/ciw691. URL https://academic.oup.com/cid/article-lookup/doi/10.1093/cid/ciw691.

S. R. Lockhart, A. Chowdhary, and J. A. W. Gold. The rapid emergence of antifungal-resistant human-pathogenic fungi. *Nature Reviews Microbiology*, 21(12):818–832, Dec. 2023. ISSN 1740-1534. doi: 10.1038/s41579-023-00960-9. URL https://www.nature.com/articles/s41579-023-00960-9.

M. Lyman, K. Forsberg, D. J. Sexton, N. A. Chow, S. R. Lockhart, B. R. Jackson, and T. Chiller. Worsening Spread of Candida auris in the United States, 2019 to 2021. *Annals of Internal Medicine*, 176(4):489–495, Apr. 2023. ISSN 0003-4819. doi: 10.7326/M22-3469. URL https://www.acpjournals.org/doi/full/10.7326/M22-3469.

X. Ma, J. Cui, Y. Tao, G. Liao, and L. Wang. Emergence of traits in human fungal pathogens. *Trends in Microbiology*, 0(0), Jan. 2026. ISSN 0966-842X, 1878-4380. doi: 10.1016/j.tim.2025.12.005. URL https://www.cell.com/trends/microbiology/abstract/S0966-842X(25)00368-3.

R. Maheshwari, G. Bharadwaj, and M. K. Bhat. Thermophilic Fungi: Their Physiology and Enzymes. *Microbiology and Molecular Biology Reviews*, 64(3):461–488, Sept. 2000. doi: 10.1128/mmbr.64.3.461-488.2000. URL https://journals.asm.org/doi/10.1128/mmbr.64.3.461-488.2000.

M. Mizusawa, H. Miller, R. Green, R. Lee, M. Durante, R. Perkins, C. Hewitt, P. J. Simner, K. C. Carroll, R. T. Hayden, and S. X. Zhang. Can Multidrug-Resistant Candidaauris Be Reliably Identified in Clinical Microbiology Laboratories? *Journal of Clinical Microbiology*, 55(2):638–640, Jan. 2017. doi: 10.1128/jcm.02202-16. URL https://journals.asm.org/doi/full/10.1128/jcm.02202-16.

J. Mohsin, S. Weerakoon, S. Ahmed, Y. Puts, Z. Al Balushi, J. F. Meis, and A. M. Al-Hatmi. A Cluster of Candida auris Blood Stream Infections in a Tertiary Care Hospital in Oman from 2016 to 2019. *Antibiotics*, 9(10):638, Sept. 2020. ISSN 2079-6382. doi: 10.3390/antibiotics9100638. URL https://www.mdpi.com/2079-6382/9/10/638.

C. P. Morice, J. J. Kennedy, N. A. Rayner, J. P. Winn, E. Hogan, R. E. Killick, R. J. H. Dunn, T. J. Osborn, P. D. Jones, and I. R. Simpson. An Updated Assessment of Near-Surface Temperature Change From 1850: The HadCRUT5 Data Set. *Journal of Geophysical Research: Atmospheres*, 126(3), 2021. ISSN 2169-897X. doi: 10.1029/2019jd032361.

M. Morrell, V. J. Fraser, and M. H. Kollef. Delaying the Empiric Treatment of Candida Bloodstream Infection until Positive Blood Culture Results Are Obtained: a Potential Risk Factor for Hospital Mortality. *Antimicrobial Agents and Chemotherapy*, 49(9):3640–3645, Sept. 2005. doi: 10.1128/aac.49.9.3640-3645.2005. URL https://journals.asm.org/doi/10.1128/aac.49.9.3640-3645.2005.

J. F. Muñoz, L. Gade, N. A. Chow, V. N. Loparev, P. Juieng, E. L. Berkow, R. A. Farrer, A. P. Litvintseva, and C. A. Cuomo. Genomic insights into multidrug-resistance, mating and virulence in Candida auris and related emerging species. *Nature Communications*, 9(1):5346, Dec. 2018. ISSN 2041-1723. doi: 10.1038/s41467-018-07779-6. URL https:

//www.nature.com/articles/s41467-018-07779-6.

J. F. Muñoz, R. M. Welsh, T. Shea, D. Batra, L. Gade, D. Howard, L. A. Rowe, J. F. Meis, A. P. Litvintseva, and C. A. Cuomo. Clade-specific chromosomal rearrangements and loss of subtelomeric adhesins in *Candida auris*. *Genetics*, 218(1):iyab029, May 2021. ISSN 1943-2631. doi: 10.1093/genetics/iyab029. URL https://academic.oup.com/genetics/article/doi/10.1093/genetics/iyab029/6149093.

A. Munshi, F. Almadani, J. Ossenkopp, M. Alharbi, A. Althaqafi, A. Alsaedi, A. Al-Amri, and H. Almarhabi. Risk factors, antifungal susceptibility, complications, and outcome of *Candida auris* bloodstream infection in a tertiary care center in the western region of Saudi Arabia. *Journal of Infection and Public Health*, 17(1):182–188, Jan. 2024. ISSN 1876-0341. doi: 10.1016/j.jiph.2023.11.021. URL https://www.sciencedirect.com/science/article/pii/S1876034123004124.

N. E. Nnadi and D. A. Carter. Climate change and the emergence of fungal pathogens. *PLOS Pathogens*, 17(4):e1009503, Apr. 2021. ISSN 1553-7374. doi: 10.1371/journal.ppat.1009503. URL https://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1009503.

C. Nullis. WMO confirms 2025 was one of warmest years on record, 2026. URL https://wmo.int/news/media-centre/wmo-confirms-2025-was-one-of-warmest-years-record.

C. A. Okoye, E. Nweze, and C. Ibe. Invasive candidiasis in Africa, what is the current picture? *Pathogens and Disease*, 80(1): ftac012, June 2022. ISSN 2049-632X. doi: 10.1093/femspd/ftac012. URL https://academic.oup.com/femspd/article/doi/10.1093/femspd/ftac012/6572357.

C. W. Ong, S. C.-A. Chen, J. E. Clark, C. L. Halliday, S. E. Kidd, D. J. Marriott, C. L. Marshall, A. J. Morris, C. O. Morrissey, R. Roy, M. A. Slavin, A. J. Stewardson, L. J. Worth, C. H. Heath, and on behalf of the Australian and New Zealand Mycoses Interest Group (ANZMIG); and the Healthcare Infection Control Special Interest Group (HICSIG); both of the Australasian Society for Infectious Diseases (ASID). Diagnosis, management and prevention of Candida auris in hospitals: position statement of the Australasian Society for Infectious Diseases. *Internal Medicine Journal*, 49(10):1229–1243, 2019. ISSN 1445-5994. doi: 10.1111/imj.14612. URL https://onlinelibrary.wiley.com/doi/abs/10.1111/imj.14612. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/imj.14612.

K. Opassathian, N. Boonmee, S. Swangkaew, P. Jenjaroenpun, T. Wongsurawat, W. Kruasuwan, P. Ngamskulrungroj, and C. Mitrpant. A study of genomic complexity underlying multidrug resistance in Candida auris strains in Thailand. *Scientific Reports*, 15(1):41478, 2025. doi: 10.1038/s41598-025-25386-6.

M. Park, E. Do, and W. H. Jung. Lipolytic Enzymes Involved in the Virulence ofHuman Pathogenic Fungi. *Mycobiology*, 41(2):67–72, 2013. ISSN 1229-8093. URL https://www.kci.go.kr/kciportal/ci/sereArticleSearch/ciSereArtiView.kci?sereArticleSearchBean.artiId=ART001780420.

M. A. Pfaller and D. J. Diekema. Epidemiology of Invasive Candidiasis: a Persistent Public Health Problem. *Clinical Microbiology Reviews*, 20(1):133–163, Jan. 2007. doi: 10.1128/cmr.00029-06. URL https://journals.asm.org/doi/10.1128/cmr.00029-06.

T. Phan-Canh, S. Jenull, T. Bitencourt, S. Seiser, D. Moser, M. Motamedi, A. Chowdhary, A. Elbe-Bürger, and K. Kuchler. White-Brown switching controls phenotypic plasticity and virulence of Candida auris. *Cell Reports*, 44(7):115976, July 2025a. ISSN 22111247. doi: 10.1016/j.celrep.2025.115976. URL https://linkinghub.elsevier.com/retrieve/pii/S2211124725007478.

T. Phan-Canh, D.-M. Nguyen-Le, P.-L. Luu, N. Khunweeraphong, and K. Kuchler. Rapid *in vitro* evolution of flucytosine resistance in *Candida auris*. *mSphere*, 10(4):e00977–24, Apr. 2025b. ISSN 2379-5042. doi: 10.1128/msphere.00977-24. URL https://journals.asm.org/doi/10.1128/msphere.00977-24.

T. Phan-Canh, C. Coman, M. Lackner, N. Troppmair, C. Müller, D. Cerbu, S. Seiser, P. Penninger, I. Tsymala, N. Khunweeraphong, T. Bitencourt, A. Knarr, S. Jenull, H. Arzani, L.-M. Zenz, G. Ianiri, W. Chen, A. Chowdhary, H. L. T. Mobley, M. Hartl, D. Moser, R. Ahrends, A. Elbe-Bürger, and K. Kuchler. Candida auris skin tropism and antifungal resistance are mediated by carbonic anhydrase Nce103. *Nature Microbiology*, 11(2):461–475, Feb. 2026. ISSN 2058-5276. doi: 10.1038/s41564-025-02189-z. URL https://www.nature.com/articles/s41564-025-02189-z.

M. Protsiv, C. Ley, J. Lankester, T. Hastie, and J. Parsonnet. Decreasing human body temperature in the United States since the Industrial Revolution. *eLife*, 9:e49555, Jan. 2020. ISSN 2050-084X. doi: 10.7554/eLife.49555. URL https://doi.org/10.7554/eLife.49555.

G. Ramage, R. Kean, R. Rautemaa-Richardson, C. Williams, and J. L. Lopez-Ribot. Fungal biofilms in human health and disease. *Nature Reviews Microbiology*, 23(6):355–370, June 2025. ISSN 1740-1526, 1740-1534. doi: 10.1038/s41579-025-01147-0. URL https://www.nature.com/articles/s41579-025-01147-0.

P. Ramos Irizarry, D. F. Q. Smith, and A. Gusa. Climate Change Impacts on Environmental Fungi: Human Health and Fungal Disease. In A. C. Gerstein, R. S. Shapiro, and I. V. Ene, editors, *One Health and Mycology*, pages 67–101. Springer Nature

Switzerland, Cham, 2025. ISBN 978-3-032-07822-3. doi: 10.1007/82_2025_305. URL https://doi.org/10.1007/82_2025_305.

T. A. Richards, D. M. Soanes, M. D. M. Jones, O. Vasieva, G. Leonard, K. Paszkiewicz, P. G. Foster, N. Hall, and N. J. Talbot. Horizontal gene transfer facilitated the evolution of plant parasitic mechanisms in the oomycetes. *Proceedings of the National Academy of Sciences*, 108(37):15258–15263, Sept. 2011. doi: 10.1073/pnas.1105100108. URL https://www.pnas.org/doi/abs/10.1073/pnas.1105100108.

V. Robert, G. Cardinali, and A. Casadevall. Distribution and impact of yeast thermal tolerance permissive for mammalian infection. *BMC Biology*, 13(1):18, Feb. 2015. ISSN 1741-7007. doi: 10.1186/s12915-015-0127-3. URL https://doi.org/10.1186/s12915-015-0127-3.

V. A. Robert and A. Casadevall. Vertebrate Endothermy Restricts Most Fungi as Potential Pathogens. *The Journal of Infectious Diseases*, 200(10):1623–1626, Nov. 2009. ISSN 0022-1899. doi: 10.1086/644642. URL https://doi.org/10.1086/644642.

J. R. Rohr and J. M. Cohen. Understanding how temperature shifts could impact infectious disease. *PLOS Biology*, 18(11): e3000938, Nov. 2020. ISSN 1545-7885. doi: 10.1371/journal.pbio.3000938. URL https://dx.plos.org/10.1371/journal.pbio.3000938.

A. Rokas. The Origin and Early Evolution of Fungi: Challenges, Inferences, and Principles. *Annual review of cell and developmental biology*, 2026. doi: 10.1146/annurev-cellbio-111524-045246.

A. Ruiz-Gaitán, A. M. Moret, M. Tasias-Pitarch, A. I. Aleixandre-López, H. Martínez-Morel, E. Calabuig, M. Salavert-Lletí, P. Ramírez, J. L. López-Hontangas, F. Hagen, J. F. Meis, J. Mollar-Maseres, and J. Pemán. An outbreak due to Candida auris with prolonged colonisation and candidaemia in a tertiary care European hospital. *Mycoses*, 61(7):498–505, 2018. ISSN 1439-0507. doi: 10.1111/myc.12781. URL https://onlinelibrary.wiley.com/doi/abs/10.1111/myc.12781. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/myc.12781.

S. E. Sansom, G. M. Gussin, M. Schoeny, R. D. Singh, H. Adil, P. Bell, E. C. Benson, C. E. Bittencourt, S. Black, M. Del Mar Villanueva Guzman, M. C. Froilan, C. Fukuda, K. Barsegyan, E. Gough, M. Lyman, J. Makhija, S. Marron, L. Mikhail, J. Noble-Wang, M. Pacilli, R. Pedroza, R. Saavedra, D. J. Sexton, J. Shimabukuro, L. Thotapalli, M. Zahn, S. S. Huang, and M. K. Hayden. Rapid Environmental Contamination With *Candida auris* and Multidrug-Resistant Bacterial Pathogens Near Colonized Patients. *Clinical Infectious Diseases*, 78(5):1276–1284, May 2024. ISSN 1058-4838, 1537-6591. doi: 10.1093/cid/ciad752. URL https://academic.oup.com/cid/article/78/5/1276/7459886.

D. J. Santana, J. A. E. Anku, G. Zhao, R. Zarnowski, C. J. Johnson, H. Hautau, N. D. Visser, A. S. Ibrahim, D. Andes, J. E. Nett, S. Singh, and T. R. O'Meara. A Candida auris–specific adhesin, Scf1, governs surface association, colonization, and virulence. *Science*, 381(6665):1461–1467, Sept. 2023. doi: 10.1126/science.adf8972. URL https://www.science.org/doi/10.1126/science.adf8972.

D. J. Santana, G. Zhao, and T. R. O'Meara. The many faces of Candida auris: Phenotypic and strain variation in an emerging pathogen. *PLOS Pathogens*, 20(3):e1012011, Mar. 2024. ISSN 1553-7374. doi: 10.1371/journal.ppat.1012011. URL https://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1012011.

K. Satoh, K. Makimura, Y. Hasumi, Y. Nishiyama, K. Uchida, and H. Yamaguchi. Candida auris sp. nov., a novel ascomycetous yeast isolated from the external ear canal of an inpatient in a Japanese hospital. *Microbiology and Immunology*, 53(1):41–44, 2009. ISSN 1348-0421. doi: 10.1111/j.1348-0421.2008.00083.x. URL https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1348-0421.2008.00083.x. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1348-0421.2008.00083.x.

F. H. Schopf, M. M. Biebl, and J. Buchner. The HSP90 chaperone machinery. *Nature Reviews Molecular Cell Biology*, 18 (6):345–360, June 2017. ISSN 1471-0072, 1471-0080. doi: 10.1038/nrm.2017.20. URL https://www.nature.com/articles/nrm.2017.20.

K. Schutz, T. Melie, S. D. Smith, and C. A. Quandt. Patterns recovered in phylogenomic analysis of Candida auris and close relatives implicate broad environmental flexibility in Candida/Clavispora clade yeasts. *Microbial Genomics*, 10(4): 001233, 2024. ISSN 2057-5858. doi: 10.1099/mgen.0.001233. URL https://www.microbiologyresearch.org/content/journal/mgen/10.1099/mgen.0.001233.

M. J. W. Schwiesow, L. A. Farinella, M. Ruzic, J. T. Leinas, N. C. Elde, and Z. A. Hilbert. Distinct routes to thermotolerance in the fungal pathogen Cryptococcus neoformans. *Genetics*, 231(3):iyaf165, Nov. 2025. ISSN 1943-2631. doi: 10.1093/genetics/iyaf165.

R. Shivarathri, S. Jenull, A. Stoiber, M. Chauhan, R. Mazumdar, A. Singh, F. Nogueira, K. Kuchler, A. Chowdhary, and N. Chauhan. The Two-Component Response Regulator Ssk1 and the Mitogen-Activated Protein Kinase Hog1 Control Antifungal Drug Resistance and Cell Wall Architecture of Candida auris. *mSphere*, 5(5):e00973–20, Oct. 2020. ISSN 2379-5042. doi: 10.1128/mSphere.00973-20. URL https://journals.asm.org/doi/10.1128/mSphere.00973-20.

B. Short, J. Brown, C. Delaney, L. Sherry, C. Williams, G. Ramage, and R. Kean. Candida auris exhibits resilient biofilm characteristics in vitro: implications for environmental persistence. *Journal of Hospital Infection*, 103(1):92–96, Sept. 2019. ISSN 01956701. doi: 10.1016/j.jhin.2019.06.006. URL https://linkinghub.elsevier.com/retrieve/pii/S0195670119302609.

S. P. Simwami, K. Khayhan, D. A. Henk, D. M. Aanensen, T. Boekhout, F. Hagen, A. E. Brouwer, T. S. Harrison, C. A. Donnelly, and M. C. Fisher. Low Diversity Cryptococcus neoformans Variety grubii Multilocus Sequence Types from Thailand Are Consistent with an Ancestral African Origin. *PLoS Pathogens*, 7(4):e1001343, Apr. 2011. ISSN 1553-7374. doi: 10.1371/journal.ppat.1001343. URL https://dx.plos.org/10.1371/journal.ppat.1001343.

Y.-E. Son, C. Pérez-Arques, and J. Heitman. Epimutations driven by RNAi or heterochromatin evoke transient antimicrobial drug resistance in pathogenic Mucor fungi. *PLOS Biology*, 24(2):e3003598, Feb. 2026. ISSN 1545-7885. doi: 10.1371/journal.pbio.3003598. URL https://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.3003598.

E. H. Stukenbrock and T. Bataillon. A population genomics perspective on the emergence and adaptation of new plant pathogens in agro-ecosystems. *PLoS Pathogens*, 8(9):e1002893, 2012. ISSN 1553-7366. doi: 10.1371/journal.ppat.1002893.

E. H. Stukenbrock and B. A. McDonald. The Origins of Plant Pathogens in Agro-Ecosystems. *Annual Review of Phytopathology*, 46(1):75–100, Sept. 2008. ISSN 0066-4286, 1545-2107. doi: 10.1146/annurev.phyto.010708.154114. URL https://www.annualreviews.org/doi/10.1146/annurev.phyto.010708.154114.

C. Suphavilai, K. K. K. Ko, K. M. Lim, M. G. Tan, P. Boonsimma, J. J. K. Chu, S. S. Goh, P. Rajandran, L. C. Lee, K. Y. Tan, B. B. S. Ismail, M. K. Aung, Y. Yang, J. X. Y. Sim, I. Venkatachalam, B. P. Z. Cherng, B. Spruijtenburg, K. S. Chan, L. L. E. Oon, A. L. Tan, Y. E. Tan, L. Wijaya, B. H. Tan, M. L. Ling, T. H. Koh, J. F. Meis, C. K. M. Tsui, and N. Nagarajan. Detection and characterisation of a sixth Candida auris clade in Singapore: a genomic and phenotypic study. *The Lancet Microbe*, 5(9), Sept. 2024. ISSN 2666-5247. doi: 10.1016/S2666-5247(24)00101-0. URL https://www.thelancet.com/journals/lanmic/article/PIIS2666-5247(24)00101-0/fulltext.

D. Taliadoros, L. Potgieter, A. Dhiman, N. A. Wyatt, M. McMullan, C. Jung, M. D. Bolton, and E. H. Stukenbrock. Genome-wide Evidence of Host Specialization in Wild and Farmland Populations of the Fungal Leaf Spot Pathogen, Cercospora beticola. *Genome Biology and Evolution*, 17(4):evaf053, Apr. 2025. ISSN 1759-6653. doi: 10.1093/gbe/evaf053. URL https://doi.org/10.1093/gbe/evaf053.

L. H. Taylor, S. M. Latham, and M. E. Woolhouse. Risk factors for human disease emergence. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 356(1411):983–989, July 2001. ISSN 0962-8436, 1471-2970. doi: 10.1098/rstb.2001.0888. URL https://royalsocietypublishing.org/doi/10.1098/rstb.2001.0888.

K. M. Thambugala, D. A. Daranagama, D. S. Tennakoon, D. P. W. Jayatunga, S. Hongsanan, and N. Xie. Humans vs. Fungi: An Overview of Fungal Pathogens against Humans. *Pathogens*, 13(5):426, May 2024. ISSN 2076-0817. doi: 10.3390/pathogens13050426. URL https://pmc.ncbi.nlm.nih.gov/articles/PMC11124197/.

S. Torres-Garcia, I. Yaseen, M. Shukla, P. N. C. B. Audergon, S. A. White, A. L. Pidoux, and R. C. Allshire. Epigenetic gene silencing by heterochromatin primes fungal resistance. *Nature*, 585(7825):453–458, Sept. 2020. ISSN 0028-0836, 1476-4687. doi: 10.1038/s41586-020-2706-x. URL https://www.nature.com/articles/s41586-020-2706-x.

T. Vila, D. Montelongo-Jauregui, H. Ahmed, T. Puthran, A. S. Sultan, and M. A. Jabra-Rizk. Comparative Evaluations of the Pathogenesis of Candida auris Phenotypes and Candida albicans Using Clinically Relevant Murine Models of Infections. *mSphere*, 5(4):10.1128/msphere.00760–20, Aug. 2020. ISSN 2379-5042. doi: 10.1128/msphere.00760-20. URL https://journals.asm.org/doi/10.1128/msphere.00760-20.

T. W. Wang, D. Sofras, D. Montelongo-Jauregui, T. O. Paiva, H. Carolus, Y. F. Dufrêne, A. A. Alfaifi, C. McCracken, V. M. Bruno, P. Van Dijck, and M. A. Jabra-Rizk. Functional redundancy in Candida auris cell surface adhesins crucial for cell-cell interaction and aggregation. *Nature Communications*, 15(1):9212, Oct. 2024. ISSN 2041-1723. doi: 10.1038/s41467-024-53588-5. URL https://www.nature.com/articles/s41467-024-53588-5.

Y. Wang, L. , Li, L. , Jiahao, W. , Xiaoyue, Z. , Junmin, X. , Liyan, C. , Wenying, , and S. Lu. Epidemiological Trends and Antimicrobial Resistance of Candida auris: A Focus on 7 Cases in a Single Medical Institution of Southern China. *Infection and Drug Resistance*, 18:2557–2568, Dec. 2025. ISSN null. doi: 10.2147/IDR.S512301. URL https://www.tandfonline.com/doi/abs/10.2147/IDR.S512301. _eprint: https://www.tandfonline.com/doi/pdf/10.2147/IDR.S512301.

A. Wasylyshyn and E. K. Stoneman. Management of Candida auris. *JAMA*, 331(7):611–612, Feb. 2024. ISSN 0098-7484. doi: 10.1001/jama.2023.24921. URL https://doi.org/10.1001/jama.2023.24921.

T. C. White. Increased mRNA levels of ERG16, CDR, and MDR1 correlate with increases in azole resistance in Candida albicans isolates from a patient infected with human immunodeficiency virus. *Antimicrobial Agents and Chemotherapy*, 41(7):1482–1487, 1997. doi: 10.1128/AAC.41.7.1482.

T. C. White, B. D. Esquivel, E. M. Rouse Salcido, A. M. Schweiker, A. R. dos Santos, L. Gade, E. Petro, B. KuKanich, and K. S. KuKanich. Candida auris detected in the oral cavity of a dog in Kansas. *mBio*, 15(2):e03080–23, Jan. 2024. doi: 10.1128/mbio.03080-23. URL https://journals.asm.org/doi/full/10.1128/mbio.03080-23.

R. G. Willaert, Y. Kayacan, and B. Devreese. The Flo Adhesin Family. *Pathogens*, 10(11):1397, Oct. 2021. ISSN 2076-0817. doi: 10.3390/pathogens10111397. URL https://www.mdpi.com/2076-0817/10/11/1397.

W. Xiao, H. Zhou, J. Huang, C. Xin, J. Zhang, H. Wen, and Z. Song. Comparative analyses of the biological characteristics, fluconazole resistance, and heat adaptation mechanisms of Candida auris and members of the Candida haemulonii complex. *Applied and Environmental Microbiology*, 91(4):e02406–24, Mar. 2025. doi: 10.1128/aem.02406-24. URL https://journals.asm.org/doi/10.1128/aem.02406-24.

A. Yadav, K. Jain, Y. Wang, K. Pawar, H. Kaur, K. K. Sharma, V. Tripathy, A. Singh, J. Xu, and A. Chowdhary. Candida auris on Apples: Diversity and Clinical Significance. *mBio*, 13(2):e00518–22, Mar. 2022. doi: 10.1128/mbio.00518-22. URL https://journals.asm.org/doi/10.1128/mbio.00518-22.

A. Yadav, Y. Wang, K. Jain, V. A. R. Panwar, H. Kaur, V. Kasana, J. Xu, and A. Chowdhary. Candida auris in Dog Ears. *Journal of Fungi*, 9(7):720, July 2023. ISSN 2309-608X. doi: 10.3390/jof9070720. URL https://www.mdpi.com/2309-608X/9/7/720.

I. Yaseen, S. A. White, S. Torres-Garcia, C. Spanos, M. Lafos, E. Gaberdiel, R. Yeboah, M. El Karoui, J. Rappsilber, A. L. Pidoux, and R. C. Allshire. Proteasome-dependent truncation of the negative heterochromatin regulator Epe1 mediates antifungal resistance. *Nature Structural & Molecular Biology*, 29(8):745–758, Aug. 2022. ISSN 1545-9993, 1545-9985. doi: 10.1038/s41594-022-00801-y. URL https://www.nature.com/articles/s41594-022-00801-y.

Q. Zheng, C. Li, T. Hu, J. Bing, C. J. Nobile, H. Chu, and G. Huang. Candida auris cells form giant lipid droplets to survive in harsh environments. *Communications Biology*, 8(1):1–14, May 2025. ISSN 2399-3642. doi: 10.1038/s42003-025-08204-7. URL https://www.nature.com/articles/s42003-025-08204-7.

Y. Zhu, B. O'Brien, L. Leach, A. Clarke, M. Bates, E. Adams, B. Ostrowsky, M. Quinn, E. Dufort, K. Southwick, R. Erazo, V. B. Haley, C. Bucher, V. Chaturvedi, R. J. Limberger, D. Blog, E. Lutterloh, and S. Chaturvedi. Laboratory Analysis of an Outbreak of Candida auris in New York from 2016 to 2018: Impact and Lessons Learned. *Journal of Clinical Microbiology*, 58(4):e01503–19, Mar. 2020. ISSN 0095-1137, 1098-660X. doi: 10.1128/JCM.01503-19. URL https://journals.asm.org/doi/10.1128/JCM.01503-19.