Impact of climate change and natural disasters on fungal infections

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The effects of climate change and natural disasters on fungal pathogens and the risks for fungal diseases remain incompletely understood. In this literature review, we examined how fungi are adapting to an increase in the Earth's temperature and are becoming more thermotolerant, which is enhancing fungal fitness and virulence. Climate change is creating conditions conducive to the emergence of new fungal pathogens and is priming fungi to adapt to previously inhospitable environments, such as polluted habitats and urban areas, leading to the geographical spread of some fungi to traditionally non-endemic areas. Climate change is also contributing to increases in the frequency and severity of natural disasters, which can trigger outbreaks of fungal diseases and increase the spread of fungal pathogens. The populations mostly affected are the socially vulnerable. More awareness, research, funding, and policies on the part of key stakeholders are needed to mitigate the effects of climate change and disaster-related fungal diseases.

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Introduction

The impact of climate change on emerging and re-emerging infectious diseases is becoming increasingly recognised.[1] Climate change refers to long-term shifts in temperatures and weather patterns, which are disrupting ecologic systems worldwide, leading to shifts in the global distributions of pathogens, hosts, and disease reservoirs. Because climate change exacerbates inequities, populations most susceptible to infectious diseases, including the global poor and those with little access to quality health care, will bear the brunt of the adverse health effects of the changing climate.[1,2]

Climate change is also having a profound impact on invasive fungal diseases. Unlike humans, many pathogenic fungi are thriving as the Earth's temperature increases, quickly adapting to higher temperatures and becoming more virulent and potent.[3] These differences have been associated with substantial changes in fungal disease epidemiology and the emergence of new pathogens, such as *Candida auris*, which shows heat tolerance and has adapted to human body temperatures.[3–5] Climate change has also influenced the spread of endemic fungal diseases such as coccidioidomycoses and histoplasmosis[6,7] and the geographical ranges of other fungi, including those that affect non-human animals and plants. In addition, fungal plant pathogens are evolving with climate change and represent an increasing threat to global food security. A comprehensive understanding of the changing epidemiology of fungal pathogens requires a shift in the clinical index of suspicion. Without this adjustment, a persistent risk of underdiagnosis or misdiagnosis of fungal infections will remain.

In addition, as a detrimental byproduct of climate change, the world is facing increased risks for natural disasters that have in turn been associated with global fungal outbreaks.[8–10] To inform relevant worldwide initiatives and help to tackle some of those devastating consequences, we performed a detailed review of the literature examining how climate change and natural disasters are impacting the risks for fungal diseases and discuss intervention and remediation strategies.

Climate change: impact on fungal diseases

Although climate change exerts a substantial negative impact on human health,[11] some pathogenic fungi are benefiting from climate change, gradually adapting to higher temperatures and becoming more prevalent and possibly more virulent. In this section, we review fungal adaptation to heat stress, describe how climate change could influence underlying anthropogenic factors that affect fungal ecosystems and host susceptibility, and discuss the observed and expected impact of climate change on the epidemiology of fungal diseases.

Adaptation of pathogenic fungi to heat stress and climate change

Most fungal taxa are adapted to environmental conditions that are vastly different from the human body, and low thermal tolerance prevents the fungi from withstanding mammalian body temperatures.[12] Thermal adaptation is not the sole factor but represents a major prerequisite for rendering fungi capable of infecting humans or mammals. Rising environmental temperatures might provide an important avenue for the fungal stress adaptation machinery to adapt to high-temperature environments, potentially promoting their pathogenicity in humans. Details regarding the temperature-sensing mechanisms, heat stress and adaptation responses, and metabolic changes that occur in fungi in response to climate change are provided in figure 1 and the appendix (pp 7–10).

Impact of climate change and underlying anthropogenic factors on fungal ecosystems, resistance, and host susceptibility

Global warming and fungal heat adaptation are inter-related with many other determinants of climate change and its underlying anthropogenic factors that have pleiotropic

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See **Online** for appendix

effects on fungal ecosystems and host susceptibility (figure 2). Modelling data predicted that fungal communities will change on a broad spatial scale with a changing climate, favouring the expansion of saprotrophic fungi (appendix pp 11–12).[13]

The adaptation of pathogenic fungi to one environmental stressor also primes them to withstand other adverse environments. This observation extends to several anthropogenic factors associated with climate change. For instance, fungal melanin confers cross-protection against heat stress, pH stress, heavy metals, and radioactive isotopes, thereby contributing to the ability of some fungal taxa to proliferate in metal-polluted areas, acidic environments, and radioactively polluted wastelands after nuclear catastrophes.[14]

Contamination of rivers, lakes, and tap water with agricultural and industrial pollutants profoundly impacts the risks for acquiring fungal diseases. Pollution of water bodies and water supply systems has been associated with increased growth and diversity of fungi, including pathogenic species, as many pollutants (eg, nitrate or iron) can provide a favourable pH and nutritional environment for fungal growth.[15] Once contaminated, freshwater then presents a source of superficial and systemic fungal infections and poses a threat to health-care systems, even in industrialised countries.[2,16–18] At least 11 classes of fungi are known to degrade plastic, including fungi that can be pathogenic to humans, such as *Acremonium* spp, *Alternaria* spp, *Aspergillus* spp, *Cladosporium* spp, *Fusarium* spp, and several mucormycetes species.[19] Although the ability of some fungi to degrade plastics might offer a solution for mitigating the accumulation of plastics in landfills and microplastics in water, the ubiquitous nature of microplastics in global water supplies could also promote fungal growth and subsequent antifungal resistance.[20]

Rapid urbanisation affects both climate change and microbial ecosystems in various ways. A high urban population density and associated industrial and ecological factors, such as a reduced canopy cover, contribute to notably increased local air and soil temperatures and surrounding stream warming.[21] This urban heat-island effect exerts evolutionary pressure on microorganisms and has been associated with greater fungal stress adaptation than that observed with rural isolates from neighbouring geographical areas.[22]

Climate change affects not only fungal adaptation and exposure but also host susceptibility to pathogenic fungi. For example, increased ultraviolet light exposure has been linked to numerous adverse effects on human immunity and immune responses, including unfavourable T-cell polarisation, increased production of inhibitory cytokines, and altered complement activation.[23] As environmental temperatures have been rising, the average human body temperatures in the USA have decreased by 0·03°C per birth decade since the industrial revolution. This trend is most likely associated with economic development, improved standards of living, and lower resting metabolic rates due to reduced inflammation and chronic infections.[24] Continuation of the trend of decreasing human body temperatures and narrowing of the thermal exclusion gradient between fungi and humans could further lead to alignment between host and fungal temperature preferences and increase human susceptibility to environmental fungi.[25] Seasonal influences on meteorological conditions and changes in warming patterns, along with alterations in light–dark cycles, melatonin secretion, and potential disruptions in circadian rhythms can also affect host susceptibility,[26,27] including changes in the immune response, alterations in the expression of epithelial receptors, and changes in mucosal surface characteristics.[28]

Increased susceptibility to fungal infection and shifts in fungal epidemiology related to climate change are not limited to pathogenic fungi affecting humans, as fungal plant pathogens such as *Puccinia striiformis*[3] or *Fusarium graminearum* are evolving with climate change and threatening food security (appendix pp 11–12). Notably, food insecurity and nutrient deficiency are inter-related with various hallmarks of human immune impairment, especially in young children, and are considered key mediators of malnutrition-related immunosuppression induced by climate change, which increases the susceptibility of these individuals to fungal diseases.[29]

The threat of climate change to food security has contributed to adaptive agricultural practices (including increased land use, chemical treatments, and fungicides for farming) and cultivation of a small number of highly productive crops to optimise agricultural output.[30,31] Acquired azole resistance in *Aspergillus fumigatus* that is partly driven by the use of environmental fungicides is becoming increasingly problematic during treatment of aspergillosis in humans.[32] Fusariosis is an emerging mould infection caused by *Fusarium* spp that are frequently resistant to azoles and other antifungals,[33] and two outbreaks of multidrug-resistant CNS fusariosis were recently reported in Mexico.[34] Concurrently, some newer fungicides show mechanisms of action that are similar to those of novel antifungal candidates currently in late-stage development. Notable examples for such fungicides include ipflufenoquin, which shares its mode of action with olorofim,[35] and aminopyrifen, which shares its mode of action with fosmanogepix.[36] These findings suggest that these newer fungicides, with shared mechanisms of action, could inadvertently contribute to the spread of fungal resistance in the environment and disable urgently needed novel antifungal treatments even before they become available for clinical use.[35,37] Together, these changes could have detrimental effects on biodiversity and promote the development of antifungal resistance.

Impact of climate change on the epidemiology of fungal infections

Changes in the epidemiology of fungal infections due to climate change are multifactorial and mainly driven by a combination of new emerging species, a broader geographical dissemination of existing fungal species to

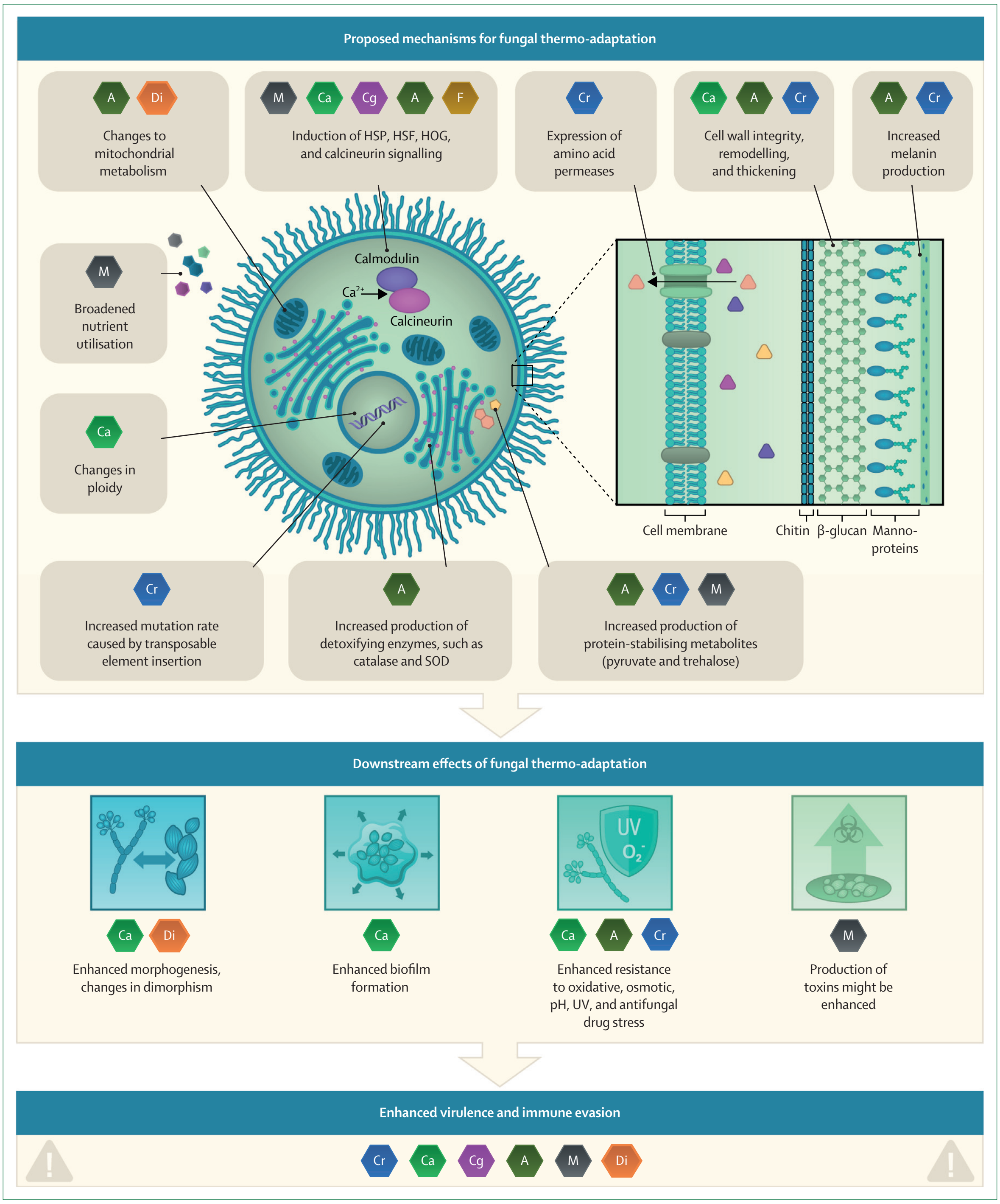

Figure 1: **Proposed mechanisms of fungal thermo-adaptation**
The proposed mechanism underlying fungal thermo-adaptation and downstream effects that could lead to enhanced virulence and immune evasion. A=*Aspergillus*. ATPase=adenosine triphosphatase. Ca=*Candida albicans*. Cg=*Candida glabrata*. Cr=*Cryptococcus*. Di=dimorphic fungi. F=*Fusarium*. HOG=high-osmolarity glycerol. HSF=heat shock factor. HSP=heat shock protein. M=Mucorales. SOD=superoxide dismutase. UV=ultraviolet.

larger endemic areas, and an increased dispersion of infectious fungal propagules.

As the world warms, fungi adapt to higher temperatures,[38] resulting in the emergence of new fungal species as human pathogens.[3,4] For instance, a link has been established between the frequent thermotolerance of ascomycetous yeasts and their prevalence among pathogenic fungi affecting humans, and basidiomycetous yeasts and some

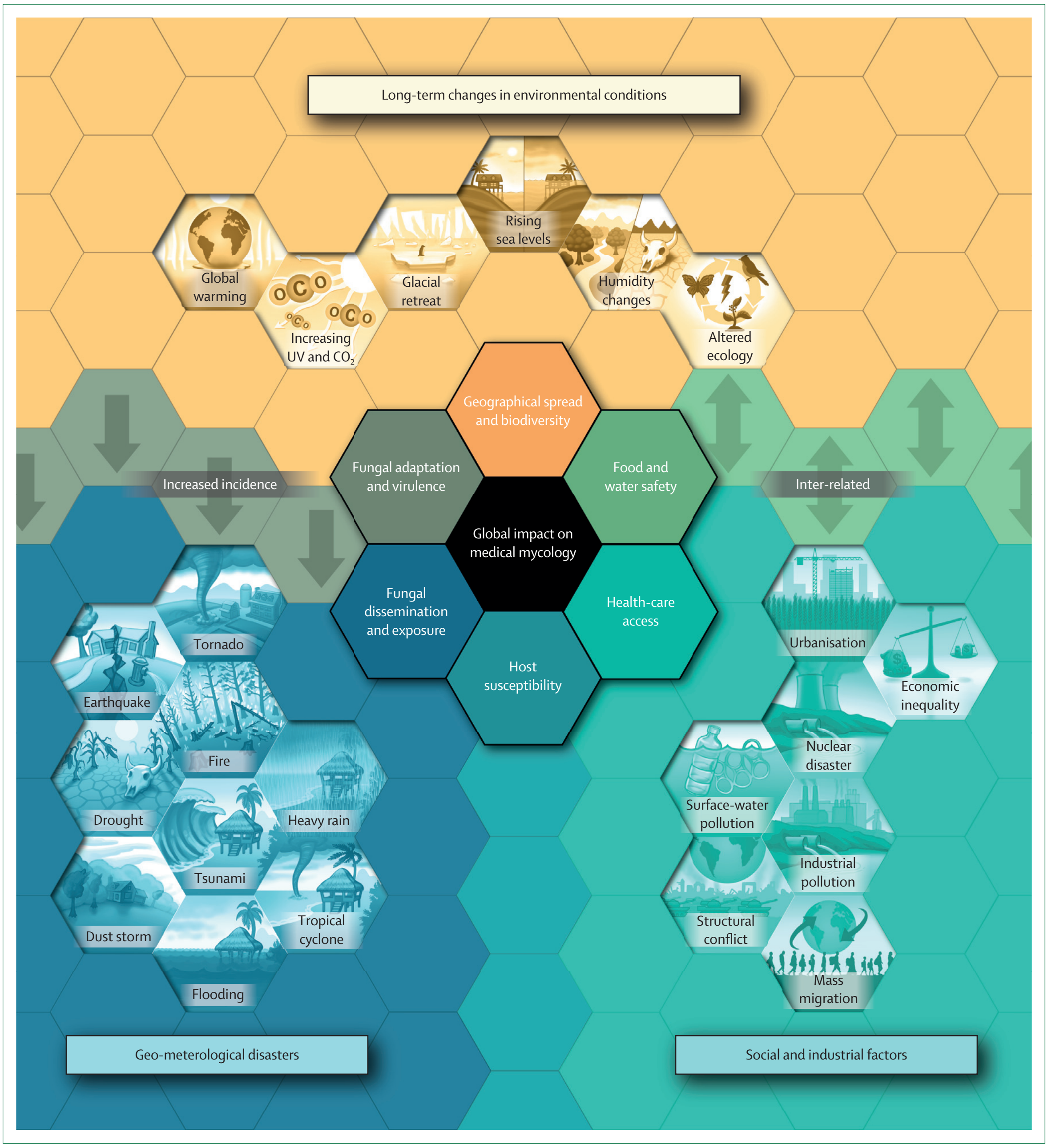

Figure 2: **Impact of climate change and natural disasters on fungi**
Long-term changes in environmental conditions are interrelated with social and industrial factors and lead to increased incidences of geo-meteorological disasters. All these factors have a global impact on medical mycology. CO$_2$=carbon dioxide. UV=ultraviolet.

previously less thermotolerant ascomycetous yeast taxa are rapidly adapting to a warming planet.[39] For example, the yeast *Candida orthopsilosis* is a human-associated opportunistic pathogen belonging to the *Candida parapsilosis* complex that originates from warm marine ecosystems, outlining the effect of warming ecosystems on the emergence of new fungal pathogens.[40] *Fusarium oxysporum* has historically been a banana pathogen but is now recognised as a human pathogen.[41,42] Therefore, many fungal species not yet associated with human disease could become emerging human pathogens.

The most prominent example of an emerging yeast pathogen is *C auris*, which is hypothesised to have evolved from a plant saprophyte to become a human pathogen after adapting to higher temperatures.[5] This hypothesis is strengthened by the finding that an environmental *C auris* isolate grew slower at mammalian temperatures than did clinical *C auris* strains, suggestive of a more recent adaptation of the ancestors of the clinical strains to mammalian temperatures.[43] Other factors that could contribute to *C auris* becoming a human pathogen include expanded farming practices, deforestation, and disruption of coastal ecosystems.[44] Details regarding *C auris*, which was recognised as one of the four fungal pathogens of crucial importance in WHO's recently published fungal pathogens priority list[45], and other human pathogens that appear to have emerged owing to climate change, namely, *Cryptococcus deuterogattii, F oxysporum,* and several *Bipolaris* and *Cladosporium* spp, are provided in the appendix (pp 12–14).

Climate change is also contributing to the geographical spread of so-called endemic mycoses such as coccidioidomycosis and histoplasmosis, necessitating continued redrawing of maps of endemic fungal infections.[6,7,46] Indeed, the term endemic mycoses is increasingly being recognised as a misnomer, given the frequency at which these dimorphic fungi are now being recognised outside of traditional endemic areas.[47,48] For instance, histoplasmosis was historically endemic to the Ohio and Michigan River Valleys in the USA and parts of Central and South America,[49] although autochthonous cases have been observed in several non-endemic states in the USA and non-endemic provinces in Canada.[6] Furthermore, modelling estimates have indicated that *Histoplasma capsulatum* is most likely to be now present throughout the upper midwest and eastern Atlantic coastal regions of the USA,[50] and *Histoplasma* spp are now recognised as endemic in much of Africa and Asia as well as in parts of Europe and Australia.[6] Coccidioidomycosis caused by *Coccidioides immitis* has historically been endemic to the Central Valley, CA, and Arizona, California, Texas, Utah, and New Mexico in the USA and that caused by *Coccidioides posadasii* has been historically endemic to central and South America. Nonetheless, autochthonous cases of coccidioidomycosis caused by *C immitis* have also been documented in Washington, USA.[51] Modelling suggests that the endemic range of coccidioidomycosis in the USA will more than double, expanding north into dry regions in Idaho, Wyoming, Nebraska, Montana, South Dakota, and North Dakota, with the current estimate of 34 460 cases projected to increase by 50% by the year 2100.[52] Globally, this modelling suggests that coccidioidomycosis might eventually spread to Canada, throughout Mexico, and parts of Central and South America, where the numbers of reported cases are progressively increasing.[52,53] *Blastomyces* spp, which cause blastomycosis, have historically been endemic to the southcentral, southeastern, and midwestern regions of the USA and a few provinces in Canada. Cases are increasingly being seen outside of these traditionally endemic areas,[54,55] and blastomycosis is now recognised to occur in parts of Africa and the Middle East.[56,57] Paracoccidioidomycosis, caused by *Paracoccidioides lutzii*, has historically been endemic to large parts of South America, central America, and Mexico but is expanding to new areas of Brazil, Ecuador, and Venezuela. The understanding of the epidemiology of *Emergomyces* spp is incomplete, with its climate sensitivity remaining scarcely studied.[6] Finally, although not directly shown, feline and zoonotic outbreaks of the dimorphic fungus *Sporothrix brasiliensis* in South America (especially Brazil) have been hypothesised to be related to gradual temperature increases that have enabled its adaptation to invasive yeast growth.[58]

The warming planet and other negative aspects of climate change can affect the airborne dispersal and, thus, the environmental burden of infectious propagules of pathogenic moulds. Experimental work has shown that fungi, not exclusively triggered by mechanical stressors, autonomously synchronise the ejection of thousands of spores in a single puff and create a flow of air that propagate these spores to atmospheric currents and new infection sites.[59] Climate change, weather patterns, and atmospheric conditions can influence the dispersal of fungal spores in the air. For instance, warmer temperatures can lead to increased turbulence in the atmosphere, affecting the vertical and horizontal movement of airborne spores. This altered dispersal can affect the geographical distribution and range expansion of fungal pathogens. Furthermore, temperature changes can lead to adaptations, potentially altering the species-specific synchronisation patterns and thus affecting the prevalence and spread of spores. The behaviour of fungal spores in manipulating a local fluid environment to reduce air resistance and enhance spore dispersal or regulate their own temperature through evapotranspiration is a fascinating and understudied aspect of fungal biology.[60]

In addition, under low-humidity conditions, fungal spores are naturally folded, which can improve their transportation through the air and enable them to travel longer distances, increasing their dispersal. Conidial counts of the common opportunistic mould genus *Aspergillus* increase with high temperature and low precipitation, with environmental spore counts being positively associated with incidence rates of invasive aspergillosis in humans.[61] Reduced spore size due to natural folding can also lead to deeper deposition within the alveoli in the lungs.[62]

Several fungal infections with predominantly cutaneous or ocular manifestations are also considered to be affected by climate change.[46] For instance, dermatophyte infections due to *Epidermophyton*, *Microsporum*, or *Trichophyton* spp as well as chromoblastomycosis and fungal eumycetoma have a predilection for moist and warm conditions, show considerable seasonality, and have been associated with climate changes (appendix pp 12–14).

Natural disasters and fungal outbreaks

Climate change is triggering profound long-term effects on fungal ecosystems, as discussed, and has been associated with an increasing frequency and intensity of devastating natural disasters, which in turn often trigger outbreaks of fungal diseases.[10,63]

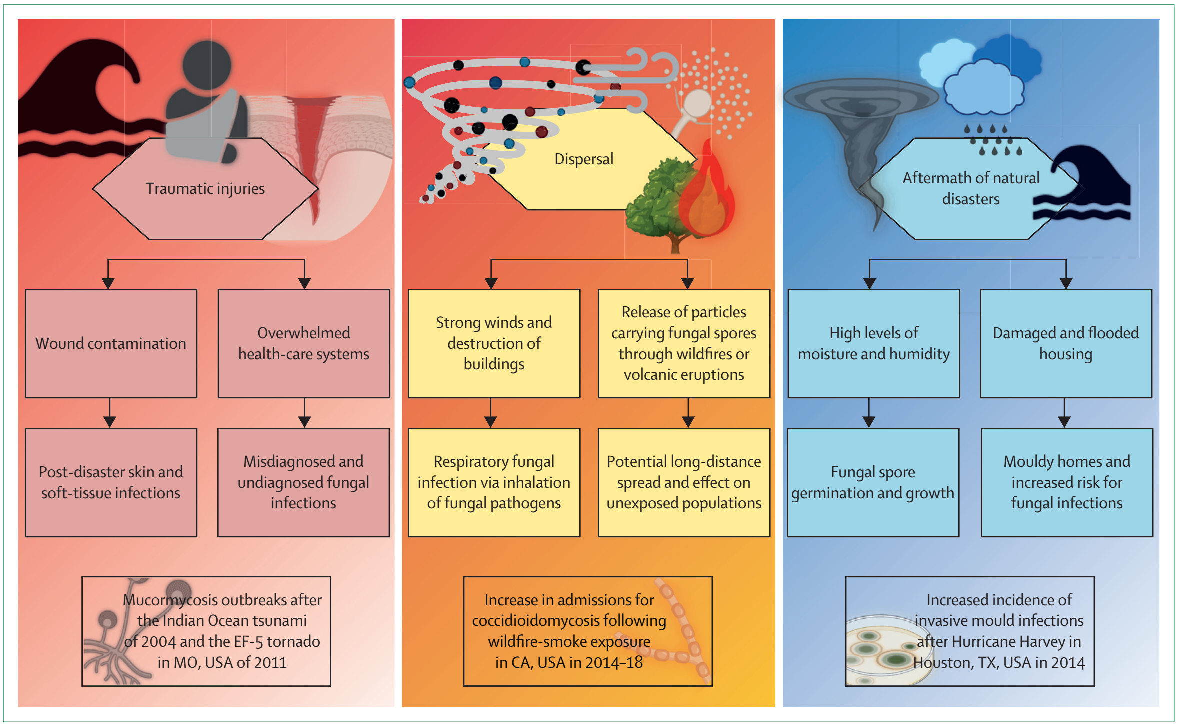

Figure 3: **Mechanisms through which natural disasters trigger fungal outbreaks**
The mechanisms outlined include traumatic injuries, dispersal of fungal spores, and housing damage in the aftermath of floodings.

Mechanisms through which natural disasters trigger fungal outbreaks

Natural disasters can cause extensive damage to natural habitats and urban areas, which can create conditions conducive to fungal growth or opportunities for fungal exposure that would not exist otherwise (figure 3). Natural disasters present a high risk for traumatic injuries, such as lacerations, abrasions, or contusions with disruption of the skin barrier, which provide entry routes for fungal pathogens. Wound contamination with organic matter can lead to post-disaster mould infections in the skin and soft tissues. Traumatic injuries and psychological stress also have complex and profound adverse effects on the immune system, making natural disaster victims highly susceptible to opportunistic fungal infections.

Wildfires and volcanic eruptions leave ash that affects ecosystems through altered soil pH values and increased nutrient contents, potentially altering microbial communities and creating new niches for the colonisation of fungi with functional traits that enable them to survive such extremes and cause harm to humans.[64,65] Wildfires leave deeper soil horizons at temperatures that are higher than normal and are suitable for breaking the dormancy of fungal spores, which poses a substantial threat for high fungal burden exposures in the context of skin injuries and inhalation. Notably, fungal spores can travel in a fire plume over long distances, impacting distant unexposed populations.[8,66] Widespread contamination with mycotoxin-producing moulds has also been reported after flooding events,[67] as high levels of humidity and moisture can cause fungal spores and biological fragments (such as mycotoxins) to become airborne and disperse across large areas, further facilitated by strong winds.[68,69] Fungal spores can be carried over long distances through extreme storms or volcanic eruptions where particles carrying fungal spores are released into the atmosphere.[70]

Eventually, the dispersal of fungal pathogens following building damage, storms, or wildfires can cause chronic inflammation, asthma, or respiratory fungal infections through inhalation of fungal spores small enough to reach the alveolar surface of the lungs.[10] Tsunamis, floods, and heavy rainfalls can also lead to marked increases in humidity and housing damage in affected areas, providing conditions favourable for fungal germination and growth.[71]

Finally, tsunamis might present a condition in which the land is seeded with pathogenic waterborne microbes. This fascinating hypothesis might link the tsunami that followed the Great Alaskan Earthquake in 1964 to the subsequent introduction of the subtropical fungus *Cryptococcus gattii* in the Pacific Northwest 35 years later.[72]

Epidemiology of fungal outbreaks following natural disasters

The first fungal outbreaks associated with natural disasters were described nearly 40 years ago,[73] when in 1985 eight victims injured during a volcano eruption in Colombia developed soft-tissue infections due to *Rhizopus arrhizus,*

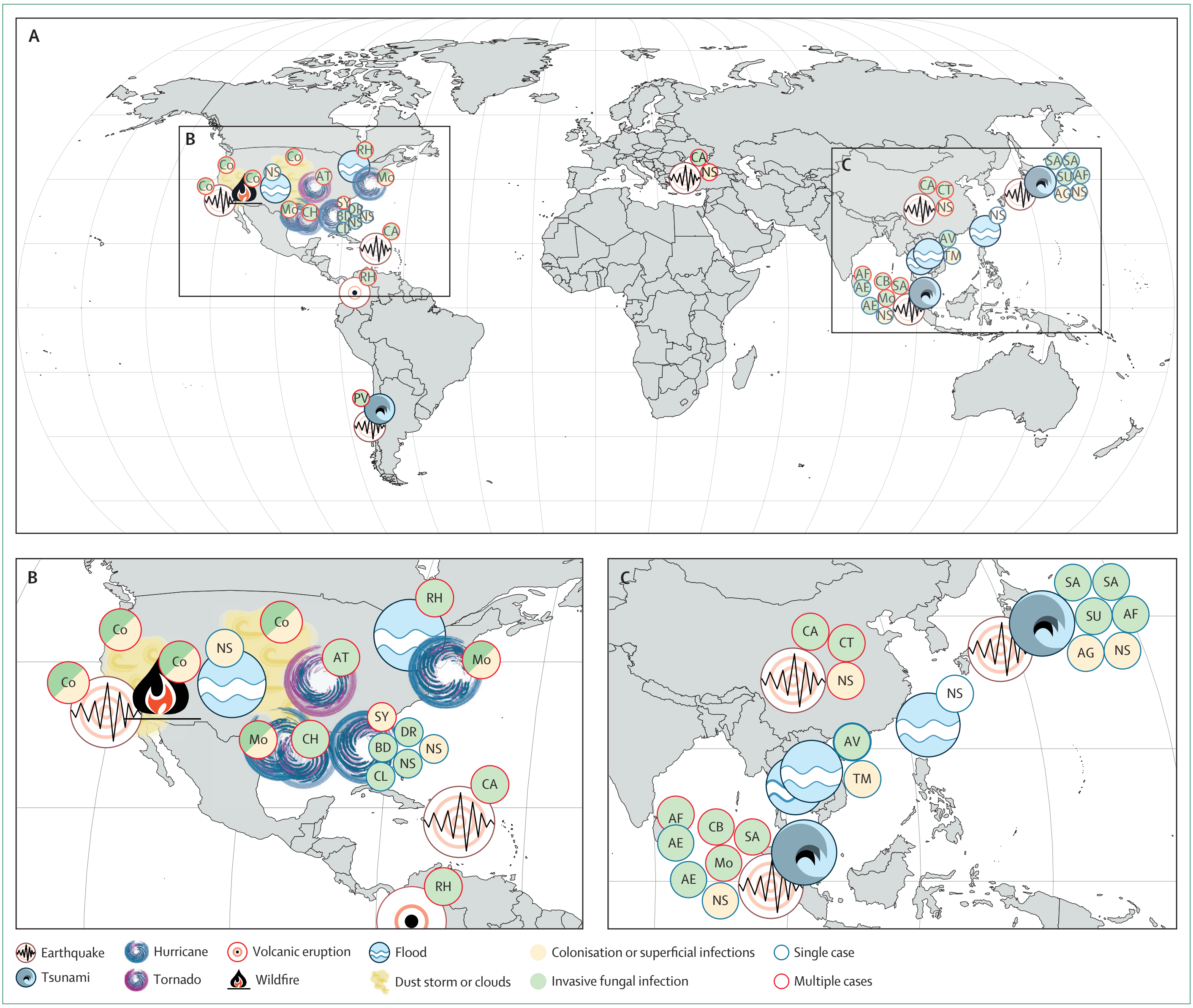

Figure 4: **Global distribution of fungal outbreaks related to natural disasters**

(A) World map. (B) North and middle America. (C) Southeast Asia. AE=*Apophysomyces elegans*. AF=*Aspergillus fumigatus*. AG=*Aspergillus glaucus*. AT=*Apophysomyces trapeziformis*. AV=*Aspergillus flavus*. BD=*Blastomyces dermatitidis*. CA=*Candida albicans*. CB=*Cladophialophora bantiana*. CH=unknown (chromoblastomycosis). CL=*Cladosporium* spp. CT=*Candida tropicalis*. CO=*Coccidioides immitis*. DR=*Drechslera* spp. Mo=moulds. NS=not specified. RH=*Rhizopus arrhizus*. PV=*Paecilomyces variotii*. SA=*Scedosporium apiospermum*. SU=*Scedosporium aurantiacum*. SY=*Syncephalastrum* spp. TM=*Trichosporon mucoides*.

and since then, their frequency has been increasing globally[74] (figure 4 and appendix pp 39–45).

Specifically, multiple fungal outbreaks were associated with traumatic injuries and wound contamination. After the catastrophic Indian Ocean tsunami in 2004, several cases of trauma-related invasive mould infections due to *Apophysomyces elegans*, other species within Mucorales, *Cladophialophora bantiana*, *Scedosporium* spp, and *Aspergillus* spp were reported for victims in the affected regions.[75,76] Following the Great Sichuan Earthquake of 2008 in China, affected individuals showed *Candida tropicalis* and other fungal pathogens in blood and wound cultures.[77] During the few days that followed the EF-5 tornado that struck Joplin, MO, USA, 13 patients were diagnosed with necrotising soft-tissue infections attributed to *Apophysomyces trapeziformis*, all of which were consequences of the traumatic injuries sustained during the tornado.[9] Of interest, tornadic shear–stress challenge transiently induced a hypervirulent phenotype with various pathogenic species within Mucorales.[78]

Near-drowning victims swept away by tsunamis and floods show a high risk for developing severe fungal infections due to aspiration of contaminated mud and water. After the tsunami following the Great Japan Earthquake in

2011, cases of severe infections with *Scedosporium* and *Aspergillus* spp were reported.[79] Traumatic injuries and wound contamination with muddy water in flooded areas can further add to the risk of developing fungal infections in these individuals.

Overwhelmed health-care systems following natural disasters contribute to fungal outbreaks, as evident from an outbreak of five *A fumigatus*-related meningitis cases that was linked to contaminated medical equipment in a tsunami-affected hospital in Sri Lanka.[80] Cumulative breaches of host defence due to traumatic injuries, weakened immunity following traumatic injuries, displacement and homelessness, insufficient hygiene, malnutrition, contaminated food and water supplies, and suboptimal health care in post-disaster settings can increase the susceptibility of populations to fungal infections and promote outbreaks.

Even months after natural disasters that include flooding, individuals might be at risk for developing a life-threatening fungal disease, as observed after the Chagrin River flooding in Cleveland, OH, USA (related to the tropical storm Alberto) in 1994, as high spore counts and inhalation of *Stachybotrys chartarum* in water-damaged houses led to pulmonary haemorrhages in infants.[81] In addition, outbreaks of severe fungal infections were linked to hurricane flooding, as seen following Hurricane Harvey in Texas, USA, in 2017. The category 4 hurricane affected Houston, TX, and the incidence of invasive mould infections was significantly higher within 1 year after the hurricane (3·69 cases per 10 000 health-care encounters) than within 1 year before the hurricane (2·50 cases per 10 000 health-care encounters).[82] Of note, the results of another study involving more stringent culture-based definitions showed no significant increase in proven or probable invasive mould infections after Hurricane Harvey, but trends were observed in terms of worse post-hurricane invasive mould infection outcomes and higher instances of mould-positive respiratory specimens and systemic antifungal use.[83]

Although acute and life-threatening fungal outbreaks have rarely been reported following flooding disasters, the risk of developing respiratory symptoms, eye irritation, allergies, asthma, and other chronic diseases from exposure to mould in water-damaged environments is high, as observed after Hurricane Katrina in 2005 and Hurricane Sandy in 2012 (appendix pp 14–16).

Immediate and long-term remediation efforts under consideration as protective measures and professional assistance are therefore crucial for avoiding persistent exposure to mould spores and mycotoxins (appendix pp 16–20). These efforts should include intensive cleaning, ensuring adequate ventilation, and educating individuals to obtain and maintain healthy indoor environments.

Reports on fungal outbreaks following wildfires are increasing. In 2017, a coccidioidomycosis outbreak occurred among state-prison inmates who were employed to fight wildfires in California, USA, leaving some with severe complications, including meningitis.[84] Between 2014 and 2018, hospital admissions for coccidioidomycosis within a 200-mile radius of wildfires in California, USA increased by 20% in the month following wildfire-smoke exposure. An association between smoke exposure and the rate of invasive aspergillosis was not seen.[66] Dispersion of moulds and allergens can also occur following tornadoes, hurricanes, and dust storms due to turbulent winds, and during the clean-up and renovation activities following a disaster due to movement of building materials and the disturbance of soil, thereby increasing the risk for developing respiratory symptoms, asthma, eye irritation, and invasive fungal diseases.[85,86] Within 2 months after the Northridge Earthquake in 1994 that caused huge landslides leading to immense dust clouds in central California, USA, an outbreak of 203 cases of acute coccidioidomycosis due to *C immitis* was reported.[87]

A substantial mismatch in the geographical distribution of reports of natural disaster-associated fungal outbreaks (figure 4) versus the geographical distribution of natural disasters indicates that fungal outbreaks following natural disasters are likely to be under-reported. Such under-reporting could be attributed to several factors, including challenges in conducting research in disaster-affected regions (where health-care facilities operate at reduced capacity), when the primary focus is on immediate health concerns. In this chaotic environment, a diagnosis of invasive fungal disease might be compromised by a scarcity of appropriate diagnostic tools[88] and professionals trained in mycology as well as little awareness and knowledge regarding the epidemiology of invasive fungal diseases following natural disasters. Misdiagnosis, the absence of diagnosis, and the absence of validated invasive mould infection case definitions could result in their exclusion from systematic reporting. Low research funding can further hamper systemic documentation of fungal diseases in the setting of natural disasters. In the future, efforts to identify cohorts or populations at high risk of fungal infections following natural disasters might benefit from artificial intelligence, which has already been used to predict the risk of mucormycosis in individuals with COVID-19 in India.[89]

To reduce the devastating impact of natural disaster-associated fungal outbreaks, monitoring fungal exposure following natural disasters, including risk assessment as well as short-term and long-term remediation efforts, is of utmost importance (appendix pp 16–20, 46).

Conclusions and recommendations

The changing climate has substantially affected the spread and acquisition of fungal diseases, promoted the emergence of new fungal pathogens, and resulted in increased dispersion of fungi. Fungi are becoming more thermotolerant, resulting in the emergence of new species that are pathogenic to humans, such as *C auris* and *C deuterogattii*. As the planet continues to warm, pathogens, including fungi, also adapt and expand their virulence and reach. Several fungi, once confined to specific regions, have emerged as major health threats in areas unaccustomed to such infections.

Panel: Open questions—future research topics

- Does global warming increase the propensity of dimorphic fungi to acquire their most invasive form?
- How do interactions between environmental fungi and plants, bacteria, or amoebae shape virulence and fungal resistance in the warming planet?
- What constitutes the eco-evolutionary framework and which factors (eg, precipitation, alterations of cool and warm episodes, dark–light cycles, urbanisation, and pollution) influence fungal thermal adaptation?
- Does the emergence of thermally adapted human fungal pathogens require an intermediate host first (eg, a species jump), and is such a species jump fungus-specific?
- Is fungal adaptation to thermal and other environmental stress caused by genetic adaptation, by evolution and genetic selection, or by epigenetic priming changes?
- Are some fungi climate-insensitive? If so, which are the mechanisms underlying their adaptability to different climates?
- What is the effect of pyroaerobiology on fungal spore dispersal and how does this effect influence the ecologic dynamics of fungal populations?
- What is the effect of global warming on the migratory patterns of birds acting as potential carriers of fungal spores and the transmission of fungal diseases?
- What are the late effects of the environmental ecosystems following natural disasters?

Search strategy and selection criteria

We searched PubMed, Google Scholar, and Web of Science with the keywords “natural disaster”, “cyclone”, “earthquake”, “flooding”, “hurricane”, “tornado”, “tsunami”, “wildfire”, “resistance”, “outbreak”, “climate change“, “fungal”, “mold“, “Aspergillus“, “Mucorales“, “Candida”, “epidemiology”, “thermotolerance”, “adaption”, “emergence”, “global warming”, “virulence”, “Histoplasma”, “Coccidioides”, “ecosystem(s)”, and “remediation“ to select relevant clinical and animal studies published between Jan 1, 1970, and Sept 1, 2023. We also searched the reference lists of all relevant publications for additional references.

Climate change has also been associated with water supply and food scarcities owing to its adverse effects on crop yields and agricultural productivity. In response to these scarcities, the reliance on fungicides to protect crops continues to increase and might contribute to the development of antifungal resistance in fungal pathogens, representing a crucial One Health problem and posing a major threat to both agriculture and human public health.

Following natural disasters, fungal pathogens can spread through the air or in contaminated water or residential buildings, affecting the ecosystems in these environments and resulting in fungal exposure. Factors such as traumatic injuries during disasters, along with the weakened immunity and health of displaced people owing to homelessness and malnutrition, provide favourable conditions for fungal pathogens and substantially increase the susceptibility of populations to fungal infections. Fungal outbreaks can then occur, which could be detrimental in a setting with a compromised medical infrastructure and low capabilities in terms of diagnosing or treating fungal infections. The implications, therefore, are profound, and the response should be robust and multifaceted.

Mitigating the impact of climate change on fungal ecosystems and the prevalence of invasive fungal diseases is an increasingly complex global challenge. Therefore, the actions needed to address this challenge transcend borders and require attention and coordination from decision makers and policy makers, including those in the public health and health-care sectors and those in the various One Health domains. Climate change mitigation strategies should be a shared responsibility, with global efforts to reduce greenhouse gas emissions and limit temperature increases. High-income countries, with greater resources and advanced health-care infrastructures, have an obligation to substantially invest in collective efforts to address global challenges. What was once seen as an issue confined to low-resource settings is now becoming increasingly relevant globally. After natural disasters, prompt and proper cleaning of affected areas can prevent large outbreaks of fungal infections. Finally, social vulnerability, climate change, and risks posed by natural disasters are strongly inter-related, and populations vulnerable to both climate change and natural disasters will continue to be at an increased risk from fungal diseases in the future.

Funding is an essential component of the response. Allocating resources for research, innovation, surveillance, and public awareness campaigns is essential. In low-income and middle-income countries, where health-care systems are under strain, investing in health-care infrastructure, access to antifungal medications and health care, and training for health-care professionals is essential. Investment in disaster resilience is also paramount across the board. Finally, collaboration is key. Countries and regions can learn from the experiences of one another and share best practices in managing fungal diseases in a changing climate. Working together, resources to address this global health challenge effectively can be shared, as could be the resultant pool of knowledge. Furthermore, collective efforts can address several questions regarding the pathogenesis, associated risk factors, and epidemiology of fungal infections in the context of climate change. Opportunities for future research on this important topic are covered in the panel. Potential limitations of this Review include the underdiagnosis or under-reporting of natural disaster-related fungal outbreaks from some regions of the world as well as the lack of standardized reporting practices of such events.

In conclusion, the impact of climate change on fungal pathogens and diseases, exacerbated by natural disasters and population displacement due to climate change, is an urgent global issue that affects both high-income countries as well as low-income and middle-income countries. Systematic and collaborative global efforts to mitigate the deleterious effects of climate change and increased understanding of the inter-relatedness between climate change, natural disasters, and fungal infections could help to improve efforts in terms of prevention, detection, and treatment.

Contributors

MH, DPK, DS, SW, and JDJ conceived and designed the study. DS, SW, JDJ, J-PG, MH, and DPK wrote the initial draft. SW, DPK, DS, ME, RS, J-PG, and MH created the figures. HS, NPF, AA-I, AC, OC, and GRT provided crucial comments. DS, SW, and JDJ extracted the relevant information from the literature. MH and DPK verified the extracted information in a second review of the articles. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Declaration of interests

DS received speaker fees from Pfizer and Hikma Pharmaceuticals, all outside of the submitted work. AC is a Fellow of the CIFAR Program Fungal Kingdom: Threats & Opportunities. JDJ received research funding from Astellas, F2G, and Pfizer, all outside of the submitted work. J-PG received speaker fees and travel support from Gilead, Mundipharma, Pfizer, and Shionogi, all outside of the submitted work. RS received speaker fees from Akademie für Infektionsmedizin, Hikma, and Pfizer, and travel support from Pfizer, all outside of the submitted work. OC reports grants or contracts from Amplyx, Basilea, BMBF, Cidara, DZIF, EU-DG RTD (101037867), F2G, Gilead, Matinas, MedPace, MSD, Mundipharma, Octapharma, Pfizer, and Scynexis; consulting fees from AbbVie, Amplyx, Biocon, Biosys, Cidara, Da Volterra, Gilead, IQVIA, Janssen, Matinas, MedPace, Menarini, Molecular Partners, MSG-ERC, Noxxon, Octapharma, Pfizer, PSI, Scynexis, and Seres; honoraria for lectures from Abbott, AbbVie, Al-Jazeera Pharmaceuticals, Astellas, Gilead, Grupo Biotoscana/United Medical/Knight, Hikma, MedScape, MedUpdate, Merck/MSD, Mylan, Noscendo, Pfizer, and Shionogi; payment for expert testimony from Cidara; participation on a Data Safety Monitoring Board (DSMB) or Advisory Board from Actelion, Allecra, Cidara, Entasis, IQVIA, Janssen, MedPace, Paratek, PSI, Pulmocide, Shionogi, and The Prime Meridian Group; a patent at the German Patent and Trade Mark Office (DE 10 2021 113 007.7); stocks from CoRe Consulting, and EasyRadiology; and other interests from DGHO, DGI, ECMM, EHA, ISHAM, MSG-ERC, and Wiley. GRT received research and consulting fees from Astellas, Amplyx, Cidara, F2G, Mayne, Melinta, Mundipharma, and Scynexis and served on the DSMB for Pfizer, all outside of the submitted work. MH received research funding from Gilead, Astellas, MSD, IMMY, Mundipharma, Scynexis, F2G, and Pfizer, all outside of the submitted work. DPK received honoraria and research support from Gilead Sciences and Astellas Pharma; received consultant fees from Astellas Pharma, MSD, and Gilead Sciences; and is a member of the Data Review Committee of Cidara Therapeutics, AbbVie, Scynexis, and the Mycoses Study Group, all unrelated to the submitted work. All other authors declare no competing interests.

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