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Climate Change and Natural Disasters: Impact on Fungal Infections

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1.0 Climate change: Impact on fungal diseases

1.1 Adaptation of pathogenic fungi to heat stress and climate change

Temperature sensing mechanisms are part of the fungi's ability to sense and respond to environmental cues, to ensure their survival, leading ultimately, in the case of pathogenic fungi, to their ability to invade cells and cause infections. These mechanisms remain scarcely studied in pathogenic fungi. Hypotheses, mostly derived from research on bacteria, plants, and the saprophytic yeast *Saccharomyces cerevisiae*, suggest adaptive physiological and biochemical changes in membrane fluidity, sensing of stress-induced acidification, responses to misfolded proteins, and possibly “RNA thermometers”[1].

In contrast to the understudied temperature sensing mechanisms, the highly conserved downstream pathways driving the heat stress response of pathogenic fungi are better defined. Given that environmental stress is often multidimensional – such as heat stress in turn promotes oxidative damage – molecular downstream mechanisms involved in fungal heat adaptation are orchestrated with other stress responses. Specifically, the fungal heat shock factor/heat shock protein system and calcineurin signaling cascades are widely regarded as multifunctional regulators that are broadly involved in stress adaptation, growth, morphogenesis, biofilm formation, and virulence of many human-pathogenic fungi[1-3]. For instance, in *Cryptococcus neoformans*, calcineurin signaling has well-described roles in heat and pH adaptation, cell wall integrity, antifungal drug tolerance, hyphal filamentation and septation, and virulence[3,4]. Similarly, in pathogenic molds, the calcineurin pathway is pivotal for adaptation to heat stress and other environmental stress conditions, hyphal growth and septation, dimorphic transitions, cell wall integrity, responses to mechanical shear stress, virulence, interference with host immunity, and possibly toxin production[4-7]. Therefore, successful fungal thermo-adaptation may coincide with other fitness gains and virulence attributes[1,8].

Given the pleiotropic effects of these pathways and their interplay with cellular shape and growth, fungal heat adaptation has been broadly associated with structural and morphological changes. For example, dimorphic transitions of *Candida albicans* as well as the endemic fungi *Histoplasma capsulatum* and *Paracoccidioides brasiliensis* are controlled by a temperature-sensitive regulatory network and are tightly interrelated with virulence[9-11]. Furthermore, thickening and remodeling of the cell wall are common structural changes implicated in environmental stress responses of various fungi. For instance, adaptation of *C. neoformans* to a variety of stress conditions, including heat stress, has been associated with capsule enlargement and increased melanin production[12]. Heat-induced cell wall thickening and melanin production have also been described in pathogenic *Aspergillus* spp.[13-15]. Besides thermoregulation, melanin confers protection against other determinants of climate change, including increased UV exposures, dryness, and oxidative stress[16]. Importantly to human health, melanin also raises inhibitory antifungal concentrations, thus leading to increased antifungal resistance[17].

Additionally, several metabolic changes have been described to counteract heat-induced cell damage, protein degradation, and intracellular oxidation. This includes heat-induced secretion of detoxifying enzymes, such as catalase and superoxide dismutase to eliminate free radicals[18]. Furthermore, heat-inducible production of protein-stabilizing metabolites, including glutathione, pyruvate, and trehalose, was shown in several fungal taxa[19-23]. Glutathione protects fungal nucleic acids from oxidative damage and facilitates its repair in response to environmental stressors, including UV damage. Glutathione secretion can also contribute to the detoxification of extracellular harmful chemicals[19] and xenobiotics. Pyruvate accumulation counteracts protein degradation while also stabilizing the mitochondrial membrane potential that is vital for cellular survival and energy production[20]. Similarly, heat-induced activation of trehalose synthesis contributes to the stabilization of protein conformations and membrane protection[21-23], and increases *in-vivo* virulence of several pathogenic fungi[24,25]. For example, heat exposure of *Penicillium expansum* spores resulted in an up to 20-fold increase in germination compared to untreated conidia due to stress-induced increase in the synthesis of trehalose in the heat-treated spores[26]. As another mechanism to increase the thermostability of proteins, thermophilic fungi produce peptidases with higher proportions of charged and hydrophobic residues and a decrease in polar residues[27].

Sourcing of a sufficient nutrient supply in stressful environments is another critical determinant of fungal fitness and survival. Consequently, fungal heat adaptation could be associated with increased metabolic flexibility, such as broadened amino acid utilization by Mucorales[8]. Another well-described mechanism to secure enough amino acid supply is the expression of amino acid permeases in the cell wall of *C. neoformans* in response to heat and other environmental stress conditions[28].

On the genome level, changes in ploidy and allelic balance, such as loss of heterozygosity in *C. albicans*, have been identified as a fungal adaptation mechanism to achieve genetic plasticity and rapid fitness gains under stressful conditions[29,30]. Increased movement of transposons (mobile DNA elements) within the fungal genome, as shown in *C. neoformans*, is another rapid genomic adaptation mechanism to environmental stress[31].

In summary, mediated by evolutionary conserved cascades, heat stress induces a complex and dynamic adaptive response of fungal cells, including morphological changes, cell wall remodeling, production of antioxidants and detoxifying agents and enzymes, broadened nutrient utilization, and genomic adaptation. Together, these adaptive responses not only drive fungal thermotolerance but also enhance fungal fitness and virulence in hostile environments, including the human body (**Figure 1**).

On a cautionary note, experimental approaches used to identify these molecular and metabolic determinants of fungal thermo-adaptation commonly relied on short-term exposures to acute heat stress. Therefore, simple *in-vitro* models of thermotolerance cannot provide an accurate representation of long-term adaptation to climate change. Although more pathophysiological representative experimental protocols have been developed to study fungal microevolution during gradual exposures to slight temperature increments over multiple generations[32], these protocols are slow and laborious, and are therefore not practical for most mycology laboratories. Furthermore, these protocols continue to rely on static heat exposures that cannot reproduce the circadian fluctuations in environmental temperatures, along with many other interrelated variables, such as humidity or substrate utilization. Although omics-driven comparisons of mesophilic and thermophilic isolates can yield critical insights into fungal heat adaptation from an evolutionary perspective, the development of more nuanced and multidimensional *in-vitro* micro-ecosystems, and their cross-validation with field studies, will be critical to gain more insights into fungal microevolution in the context of climate change[33].

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

Modelling studies predict that fungal communities will change at a broad spatial scale[34]. Saprotrophic fungi may tolerate higher temperatures than ectomycorrhizal fungi, as suggested by an *in-situ* warming study in an experimental forest site[35]. Notably, saprotrophic fungi decompose carbon present in dead and decaying organic matter, including recalcitrant substrates in boreal peatlands and forests[36]. The rates of decomposition in areas with fast warming rates is likely to increase, further increasing atmospheric CO_2 concentration[37].

Additionally, urban population density and the connectivity of urban centers can increase the transmissibility of infectious diseases[38]. However, for fungal infections, this association is subject to many confounding and interrelated socio-economic and healthcare-related variables, as discussed elsewhere[39]. As with urbanization, the ever-expanding demand for global connectivity and intercontinental travel as well as medical tourism is not only a significant contributor to carbon emissions and climate change, but can also lead to fungal dissemination and outbreaks. For instance, several travel-related case clusters of *C. auris* candidiasis have been identified in the U.S., including due to healthcare exposures abroad[40], as have cases of CNS fusariosis associated with medical tourism in Mexico[41].

Increased susceptibility to fungal infection and climate change-related shifts in fungal epidemiology are not limited to human-pathogenic fungi, as fungal plant pathogens are evolving with climate change and threaten food security. For example, stripe rust caused by *Puccinia striiformis* is a global pathogen to wheat crops. Previously showing a preference for colder temperatures, it has adapted to warming temperatures over the past two decades, with newer strains being more thermotolerant and spreading to warmer regions[42]. Similarly, a pathogen to wheat and other cereal crops, *Fusarium graminearum* spp. complex, can result in yield losses of up to 75%, particularly during warmer and humid years[43]. This species produces high amounts of mycotoxins, especially at higher temperature and humid conditions. Thus, the severity of crop losses from these pathogens is likely to increase as global temperatures rise.

Lastly, climate change is a major driver of human migration, with estimates of up to 200 million environmental refugees in the coming decades due to sea-level rise, coastal flooding, loss of arable land, famines, and increased severity and frequency of natural disasters, combined with underlying social and economic factors[44,45]. Migration, especially forced displacement, can lead to a temporary lack of basic necessities such as access to nutritious foods and clean water, loss of social networks, limited healthcare access, and unsanitary or crowded living conditions, such as in refugee camp[45,46]. These factors contribute to an increased risk of refugees for fungal diseases, especially superficial fungal infections[47,48]. In addition, compared to non-migrants, HIV acquisition is more common among both male and female migrants[49] due to vulnerability to sexual violence, exploitation, limited access to preventative care, and limited legal protection. Ultimately, endangered food security, increasing economic inequality, social inequity, and enforced relocation can spark structural conflict, resulting in the destruction of social networks and physical infrastructure, further displacement, and exposures to various fungal and other microbial agents, including due to combat-related injuries, as extensively reviewed elsewhere.

1.3 Impact of climate change on the epidemiology of fungal infections

C. auris has emerged as a significant global health threat, particularly within healthcare settings, causing large and enduring outbreaks in intensive care units, and assisted living facilities worldwide. Since its first report in 2009, the pathogen has been documented in over 56 countries across all six WHO regions[50-54]. Notably, a concerning aspect is the presence of strains displaying resistance to multiple antifungal treatments. Accordingly, *C. auris* has been recognized as one of the four fungal pathogens of critical importance in the recently published World Health Organization (WHO) fungal pathogens priority list[55].

Similarly, *Cryptococcus deuterogattii*, a species within the *C. gattii* complex, has higher levels of thermotolerance than other *C. gattii* complex members[56] and is an emerging pathogen to both humans and animals, with recent reports of spread to British Columbia, Canada, as well as Washington and Oregon states in the U.S.[57]. Climate change has been implicated as a cause of its spread[58].

Likewise, several *Bipolaris* and *Cladosporium* species that were historically plant pathogens have been linked to human infections such as surgical site infections. However, the detailed mechanisms of cross-kingdom pathogenesis of these plant-pathogenic fungi and their association with climate change are largely unknown[59].

Our understanding of the epidemiology of *Emergomyces* spp. (formerly *Emmonsia*) is evolving, although cases of emergomycosis have been reported in South Africa, Uganda, Europe, India, China, the U.S., and Canada[60]. While there have been indications that the increasing environmental burden of *Emergomyces africanus* might be associated with cooler temperatures and high precipitation, the climate sensitivity of these emerging dimorphic fungi, consisting of at least 5 pathogenic species with disparate geographical spread[61,62], remains scarcely studied.

Several fungal infections with predominantly cutaneous or ocular manifestations are also considered to be affected by climate change[63]. For instance, dermatophyte infections due to *Epidermophyton*, *Microsporum*, or *Trichophyton* species have a predilection for moist and warm conditions, show considerable seasonality, proliferate in climate zones with higher humidity, and have been associated with climatic changes[64]. Similarly, chromoblastomycosis has been reported predominantly in tropical and subtropical regions and climate change could result in an expansion of its endemic area and/or shifts in etiological agents[65]. Furthermore, its causative agents, including *Fonsecaea pedrosoi* and *Phialophora verrucosa*, can proliferate in adverse environments and withstand heavily polluted habitats, possibly conferring an ecological advantage in the context of anthropogenic pollution[66]. Other cutaneous fungal infections that are associated with tropical or subtropical climates and/or seasonal patterns during rainy season include sporotrichosis[67], talaromycosis[68], and mycetoma[69]. The incidence of fungal eumycetoma, a chronic infection of the skin and underlying structures which is endemic in the tropical/sub-tropical areas, has increased in some parts of the world, and was linked to climatic changes and their sequalae[63,70,71]. Lastly, a sharp increase in fungal keratitis cases has been observed over the past two decades and might be linked to climate change and increasing environmental spore burden[72].

2.0 Natural disasters and fungal outbreaks

Natural disasters that predispose populations in affected areas to fungal outbreaks include tornados, hurricanes, floods, tsunamis, earthquakes, wildfires, and even volcanic eruptions[73-79]. Natural disasters primarily result from extreme weather events, geological activities, or a combination of both, with a mostly unpredictable potential to immediately disrupt ecosystems and human communities. Natural disasters, increasingly related to global warming, can also lead to outbreaks of fungal diseases irrespective of a country's economic status. Factors such as traumatic injuries during disasters, along with the weakened immunity and health of displaced people due to homelessness and malnutrition, provide favorable conditions for fungal pathogens, and significantly increase populations susceptibility to fungal infections.

In the weeks and months after trauma, individuals may develop persistent inflammation–immunosuppressive catabolism syndrome, with decreased production of cytokines, impaired function of monocyte-macrophages and effector T cells and increased number of myeloid-derived suppressor cells (MDSCs), which have potent immunosuppressive activity[80].

Pyroaerobiology is the integration of microbiology and aerobiology, smoke and atmospheric sciences, fire behavior, and fire ecology in a coherent effort to understand the ecological and societal impacts of smoke-vectored microbes, including fungi. Pyroaerobiology is in its infancy as it relates to fungal spread. To understand the spread of fungal organisms by wildfires, a foundational understanding of the capacity of wildland fire to aerosolize viable fungal spores is needed[81].

The potential to acquire or select for hypervirulent traits under extreme conditions has been demonstrated for laboratory-induced, shear-stress challenged *Rhizopus arrhizus*. This phenomenon was linked to the calcineurin/heat shock protein 90 stress response pathway[6].

In 2005, Hurricane Katrina affected wide areas around New Orleans, U.S., and during the months following the return of the population to the affected areas, the admissions from respiratory symptoms, eye, throat and nose irritation were higher post-hurricane compared to the period prior to Hurricane Katrina[82]. In addition, an abundance of airborne allergens and an inability to access medication in the chaotic aftermath of natural disasters, such as in Hurricane Katrina, can contribute to asthma exacerbation[82,83]. Hurricane Sandy, which affected wide stretches at the Atlantic coast of the U.S. in 2012, left states like New York and New Jersey severely flooded. Those involved in post-hurricane clean-up and reconstruction of damaged infrastructure in affected areas where the dispersion of fungal spores occurred were twice as likely to develop lower respiratory symptoms due to mold exposure, compared to those without such exposure[84]. Taken together, these findings may indicate an increase of chronic forms of mold disease including hypersensitivity and asthma following hurricanes and other natural disasters leaving environment and housing water damaged.

Determinants of vulnerability to natural disasters overlap significantly with social determinants of health (SDOH)[85]. Prevention efforts could target circumstances driving vulnerability to natural disasters, including low income and poverty, which may predispose people to live in areas with lower cost of living that is at higher risk of natural disasters[86,87], poor housing which may not withstand natural disasters and thereby further increasing vulnerability[88], and occupational work that results in higher exposure to fungal pathogens that can cause infection, such as rescue work following natural disasters. Of note, there are also highly relevant disparities in post- disaster access to quality healthcare that could impact the diagnosis and successful treatment of IFDs[89].

3.0 Monitoring and remediation of fungal exposure in times of natural disasters

Natural disasters are not only responsible for increased outdoor fungal exposure, but they also significantly contribute to indoor fungal exposure in buildings that are afflicted by moisture damage and dampness. In the aftermath of hurricanes and tsunamis, disruption of essential infrastructure can leave houses damaged for prolonged periods of time. Such increased and prolonged exposure to fungal spores can induce both acute and chronic infections but also allergic diseases, depending on individual underlying risk factors.

In post-disaster scenarios, monitoring outdoor fungal spore counts can be indicative for the presence of mold growth in damaged buildings. Monitoring and preventing the outdoor exposure to virulent yeasts and filamentous micromycetes such as *Aspergillus* spp. and mucormycetes, especially in persons with immunocompromising conditions, can prevent life-threatening invasive fungal infections as well as chronic fungal infections. Similarly, even outside of the natural disaster setting, limiting the recurrent exposure to fungi in residential or occupational environments can result in a decrease of care consumption, particularly among individuals with chronic respiratory diseases such as asthma, COPD, and cystic fibrosis[90].

3.1 Risk assessment

A first step in flood-damage assessment is the observation of visible fungi, particularly on building surfaces following water damage after a hurricane or tsunami. Visible fungal colonies can serve as a clear indicator of moisture infiltration and potentially high spore counts in indoor air. For long-term follow-up after remediation has removed any visible fungal colonies, or in case of lighter contamination, indirect markers such as dampness spots, moldy odor, peeling-off wallpaper and blistering paint, must be considered. Modeling of the prediction of microbial contamination in large-scale floods could enable local authorities to better plan the provision of temporary accommodation and remediation prioritization[91,92].

Mycological tools may also be pertinent to monitor indoor fungal contamination using different matrix such as air, dust, surface and water samples[93]. Then, multiple techniques for detection of fungi in the environment can be used (**Supplemental Figure 1**):

(i) If the objective is to assess the risk for infection for immunosuppressed individuals, fungal culture will provide quantitative and qualitative data on the viable flora, including species identification and potentially in vitro sensitivity testing to antifungals. The recommended culture media are Malt extract agar and Dichloran Glycerol (DG18) that allow more fungal diversity, and the addition of chloramphenicol reduces bacterial contamination. Incubation temperature of plates varies between 25°C and 30°C according to the literature in order to recover a wide panel of environmental species[93]. In vitro susceptibility testing is feasible in case of positive culture with virulent fungal species that can acquire resistance to antifungal agents in the environment.

(ii) On a larger scale, if the challenge is to prevent allergic risks in the dwellings of a district or a city, then the whole flora (viable and non-viable) is the target and culture-independent techniques are usually preferred. Detection of abnormal fungal development in buildings can rely on molecular tools from qPCR (with specific or pan-fungal primers) to NGS (ITS or 18s targeted metagenomics)[93-95], but also on detection of fungal cell wall components such as ergosterol or glucans or secondary metabolites such as volatile organic compounds and mycotoxins[96-98].

Regarding endemic mycosis such as coccidioidomycosis, improved training regarding the potential risks for developing coccidioidomycosis and preventive measures such as limiting dust exposure through implementing a respiratory protection program may decrease the chance of outbreaks in the general population but also among rescue workers[99].

3.2. Remediation

Immediate remediation in times of natural disasters aims at controlling indoor mold growth and moisture by drying all items and cleaning up the moldy areas. According to the U.S. Environmental Protection Agency[100], inhabitants of contaminated buildings can clean up moldy areas that are less than about 10 square feet. In cases of larger moldy areas, seeking professional remediation services is advised. Individuals remediating these areas must wear gloves, glasses, and masks (N95 or FFP2) and use strong biocides such as chlorine bleach. Even among those at high-risk for fungal disease, such as immunocompromised individuals, compliance with recommended use of personal protective equipment (PPE), such as respiratory protection, can be low, as seen during the cleanup efforts following Hurricane Harvey in Houston, Texas, in 2017[101].

Long-term remediation efforts for obtaining and maintaining healthy indoor environments should be based on a combination of several strategies. Physical methods, such as intensive cleaning, repairing water leaks, ensure proper ventilation, such as through the use of air purifiers or mechanical ventilation systems, and through heating are required to eliminate fungal spores from surfaces, lower indoor humidity level and thus, prevent future fungal growth. Equally important is the behavioral education on how to sustain low humidity levels within homes while maintaining a clean living environment. A lower humidity level also contributes to decreasing the burden of house dust mites in carpeting[96,102,103]. Trained healthcare workers can play a crucial role in implementing such preventive measures in homes of individuals with chronic respiratory conditions, such as asthma[104].

The prevention of fungal exposure through effective remediation efforts can have significant clinical implications, especially for individuals living in affected areas[90]. A notable case is the aftermath of Hurricane Katrina in New Orleans, Louisiana. Following the devastation caused by the hurricane in 2004, individuals were exposed to elevated levels of mold spores that resulted in an increase of respiratory symptoms in individuals in the affected area. Six months after the hurricane , an overall decrease in mold levels were associated with an improvement in children's health with a decrease in respiratory symptoms[105].

Lastly, the combination of exposure to other microorganisms beside fungi such as bacteria, protozoa and viruses, as well as to carbon dioxide that may indicate poor ventilation in indoor spaces, and other chemical compounds that can lead to respiratory irritation, allergies or more severe health effects upon prolonged exposure, should not be underestimated since they can adversely affect health.

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Supplemental Table 1 Occurrence of Fungal Infections in Geophysical and Volcanic Natural Disasters and Climatological Events

Disaster Name	Disaster Time / period	Affected Regions	N cases	Invasive vs superficial/ colonization	Anatomic site	Fungal agent	Comment	Study Period	Ref.
Dust Storm									
Great Bakersfield Dust Storm/Southern San Joaquin Valley Dust Storm	1977, Dec 20-21	USA, California, San Joaquin Valley	18	Colonization, invasive	Lung, disseminated	*Coccidioides immitis*	N=18 symptomatic coccidioidomycosis cases; N=4 with disseminated disease	1977, Dec 20-21	[106]
			555	Colonization, invasive	Lung, disseminated	*Coccidioides immitis*	N=555 dust-storm-related coccidioidomycosis (115 dust-storm-related IFI + 435 colonizations) in 16 weeks post-storm *vs N*=175 within same time frame prior to storm; N=115 dust-storm-related coccidioidomycosis (*Coccidioides immitis*), 16/115 disseminated disease; 15/100.000 white and 44/100.000 black population	1977, Dec 20-21	[107]
Dust Storms Western US	1998 – 2011	Western United States	NS	NS	NS	*Coccidioides*	8-fold increase in coccidioidomycosis incidence from 1998 to 2011 in endemic areas (Arizona, California, Nevada, New Mexico, and Utah) (5.3/100,000 population in 1998 to 42.6/ 100,000 in 2011; higher incidence of persons aged 60–79 years: 69.1/100,000 in 2011	1998 - 2011	[108,109]
Volcanic eruption									
Stratovolcano Tolima eruption-associated Lahar	1985, Nov 13	Colombia, Nevado del Ruiz	8	Invasive	Soft tissue	*Rhizopus arrhizus*	N=8 mucormycosis necrotizing fasciitis: N=5 *Rhizopus* spp., N=3 NOS	1985, Nov 13	[110]
Earthquake									
Northridge Earthquake Mw 6.7-associated landslides/dust clouds	1994, Jan 17	USA, California, Simi Valley	170	Colonization, invasive	Lung or erythema nodosum	*Coccidioides immitis*	N=170 laboratory evidence of acute coccidioidomycosis between January 24 - March 15, 1994 (active surveillance) vs N=52 in January - December 1993 (passive surveillance)	1994, Jan - Mar 15	[111]
			203	Colonization, invasive	Lung, disseminated	*Coccidioides immitis*	N=203 coccidioidomycosis cases (*Coccidioides immitis*) between January 24 - March 15, 1994; N=3 fatalities	1994, Jan 24 - Mar 15	[112]
Izmit earthquake Mw 7.5	1999, Aug 17	North-western Turkey (Marmara)	2	NS	Wound/Soft tissue	*Candida albicans*	N=960 injured patients were admitted, N=321 were hospitalized, N=60 had crush syndrome, N=40 with microbiological data, 2 samples grew *C. albicans*	1999, Aug	[113]

			205	Superficial	Cutaneous superficial	NS	Higher incidence of infectious diseases during months 1-3 post-earthquake compared to same period pre-earthquake; cutaneous superficial fungal infections in 17.4% of all admitted patients within 3 months post-earthquake vs. 13.2% within same months pre-earthquake	1998, Aug 23 - 1999, Feb 23 (pre-earthquake) vs 1999, Aug 23 - 2000, Feb 23 (post-earthquake)	[114]
			2	NS	Lung	*Candida albicans*	35 hospitals: 639 crush syndrome renal victims, 223 with infectious complications: Candida albicans grew from sputum or trachea cultures in 2 cases	1999, Aug	[115]
Sumatra–Andaman earthquake Mw 9.1-9.3 Boxing Day Tsunami	2004, Dec 26	Sri Lanka	6	Invasive	Meningitis	*Aspergillus fumigatus*	6 cases in Sri Lanka, Colombo Hospital, associated to contaminated equipment donated post-Boxing Day Tsunami in December 2004	2005, Jun 21 - July 17	[116,117]
		Coastal areas of South Asia	12	Superficial	Cutaneous superficial fungal infections	NS	N=86 infection-infestations in 235 patients, N=12 (14.3%) were superficial fungal infections (mostly tinea corporis)	2005, Jan 5 - 25	[118]
		Indonesia	1	Invasive	Osteomyelitis knee	*Scedosporium apiospermum*	Immunocompetent patient 2 years after tsunami-related traumatic rupture of patellar tendon and surgery	2004, Dec 26	[119]
		Sri Lanka	1	Invasive	Wound/Soft tissue	*Apophysomyces elegans*	Immunocompetent patient with tsunami-related traumatic injuries	2004, Dec 26	[120]
		Thailand	1	Invasive	Wound/Soft tissue	*Apophysomyces elegans*	Immunocompetent patient with soft-tissue injuries and bilateral fractures of pubic rami	2004, Dec 26	[121]
		Thailand	2	Invasive	Wound/Soft tissue	*Cladophialophora bantiana*	2/42 injured tsunami victims treated in Sweden developed skin infection post-skin-transplantation 4-6 weeks after the tsunami	2004, Dec - 2005, Feb	[122]
		Thailand	2	Invasive	Spondylodiscitis, brain abscess	*Scedosporium apiospermum*	Immunocompetent patients developed *Scedosporium*-associated infections several weeks post-tsunami after near drowning (1) and deep cutaneous traumatic injuries and fractures (1)	2004, Dec - 2005, Feb	[123]
		Thailand	4	Colonization, invasive	Wound, blood, sinus	*Candida albicans, Aspergillus fumigatus, Fusarium solani, Mucor* spp.	17 severely injured tsunami survivors transferred to Cologne, Germany: *Mucor* spp., *Fusarium solani, Aspergillus fumigatus, Candida albicans, C. tropicalis, C. glabrata isolated from wounds; C. albicans and C. tropicalis positive blood cultures; A. fumigatus* from nasal swab	2004, Dec - 2005, Feb	[124,125]
Great Sichuan earthquake Mw 7.9	2008, May 12	China, Sichuan province	NS	NS	NS	NS	Fungi identified in 7.6% of 210 non-replicate clinical isolates from 42 patients with earthquake-related crush syndrome	2008, May 12	[126]

			NS	Colonization, invasive	Wound/Soft tissue	NS	19 fungal strains from 340 secretion specimens isolated from 148 cases of orthopedic open wounds of earthquake victims	2008, May 12 – Jun 22	127
			NS	Colonization, invasive	Wound/Soft tissue, blood	*Candida tropicalis*	2,135 smears of secretion from 1,823 hospitalized wounded victims of Wenchuan Earthquake, Pathogens found in 725 cases within 1 month post-earthquake = *C. tropicalis* (8.3%)	2008, May 12 – Jun 22	128
			NS	NS	Wound/Soft tissue	*Candida* spp.	M=464 non-duplicate clinical isolates from wound and/or pus from 330 patients out of 1,823 earthquake victims admitted mostly with open wounds and tissue damage = fungal pathogens identified in 2.4% of isolates, not further specified	2008, May 12 – Jun 22	129
			NS	NS	Wound/Soft tissue	*Candida albicans*	N=725 non-duplicate isolates of 1,823 severely injured hospitalized victims - 9.7% positive for *Candida* spp.	2008, May 12 - Jun 11	130
			NS	NS	NS (crush syndrome)	NS	N=571 wound secretions, sputum, or blood from 123 patients with earthquake-related injuries: 78/123 with positive culture (19 fungal strains not further specified)	2008, May 12 - Jun 31	131
			NS	NS	NS (crush syndrome)	NS	N=210 non-replicate clinical isolates from wound, blood, sputum, urine and catheter samples from 42 of 66 earthquake victims with crush syndrome - fungal pathogens in 7.6% of the isolates, not further specified	2008, May 12 - June 22	126
West Indian Island of Hispaniola earthquake Mw 7.0	2010, Jan 14	Haiti	NS	Invasive	Wound, CRBSI	Wound: *Candida albicans* (1), *C. tropicalis* (1), *C. glabrata* (1), catheter: *C. albicans* (1)	N=30 children injured children - in 3 wound samples *Candida albicans* (1), *Candida tropicalis* (1) and *Candida glabrata* (1) was identified, in 1 blood sample *C. albicans* was identified	2010, Jan 14 - Feb 6	132
2010 Chile Earthquake Mw 8.8 and tsunami	2010, Feb 27	Chile, Central region	3	Invasive	Peritoneum	*Paecilomyces variotii*	Within 4 months post-earthquake: *Paecilomyces variotii*-associated peritonitis in peritoneal dialysis patients (PD fluid bags storage with exposure to excessive dust and destruction of the architecture of the storage holds post-earthquake)	2010, Mar 26 - Jun 12	76
Great East Japan Earthquake, 東日本大震災 Mw 9.1 and	2011, Mar 11	Japan	1	Invasive	Sinus, CNS	Unknown	1 month after near drowning, without pathogen identification	2011	133
			1	Invasive	Lung, brain	*Scedosporium apiospermum*	1 month after near drowning	2011	134
			1	Invasive	vertebral osteomyelitis	*Scedosporium apiospermum*	1 month after near drowning	2011	77

associated tsunami			1	Invasive	Lung, brain	*Scedosporium aurantiacum*	1 month after near drowning first symptoms (diagnosis 2 months post-near drowning)	2011	[135]
			1	Invasive	Pulmonary, disseminated	*Aspergillus fumigatus*	2 weeks after near drowning	2011	[136]
Tornado									
Joplin EF-5 Tornado	2011, May 22	USA, Missouri, Joplin	13	Invasive	Wound/Soft tissue	*Apophysomyces trapeziformis*	Cutaneous mucormycosis following lacerations and penetrating injury from airborne material diagnosed within 3 weeks post-tornado	2011	[137]
			2	Invasive	Wound/Soft tissue	*Apophysomyces trapeziformis*		2011	[138]
			13	Invasive	Wound/Soft tissue	*Apophysomyces trapeziformis*	Cutaneous mucormycosis following lacerations and penetrating injury from airborne material diagnosed within 3 weeks post-tornado	2011	[139]
Wildfire									
California wildfires	2017, Aug	USA, California	10	NS	NS	*Coccidioides* NOS	N=10/198 inmate firefighters (112 answered the survey) with coccidioidomycosis (four clinical and six laboratory-confirmed), two were hospitalized; none reported wearing respiratory protection on this wildfire	2017	[140]
Wildfires >5,000 acres	2014 - 2018	USA, California	UNK	Colonization, invasive	Any	*Coccidioides* NOS	Increased risk of coccidioidomycosis in months after a large fire (> 5000 acres) within a 200-mile radius of that hospital. Invasive *Candida* infections served as controls. No change in IA incidences.	2014, Oct - 2018, May	[141]

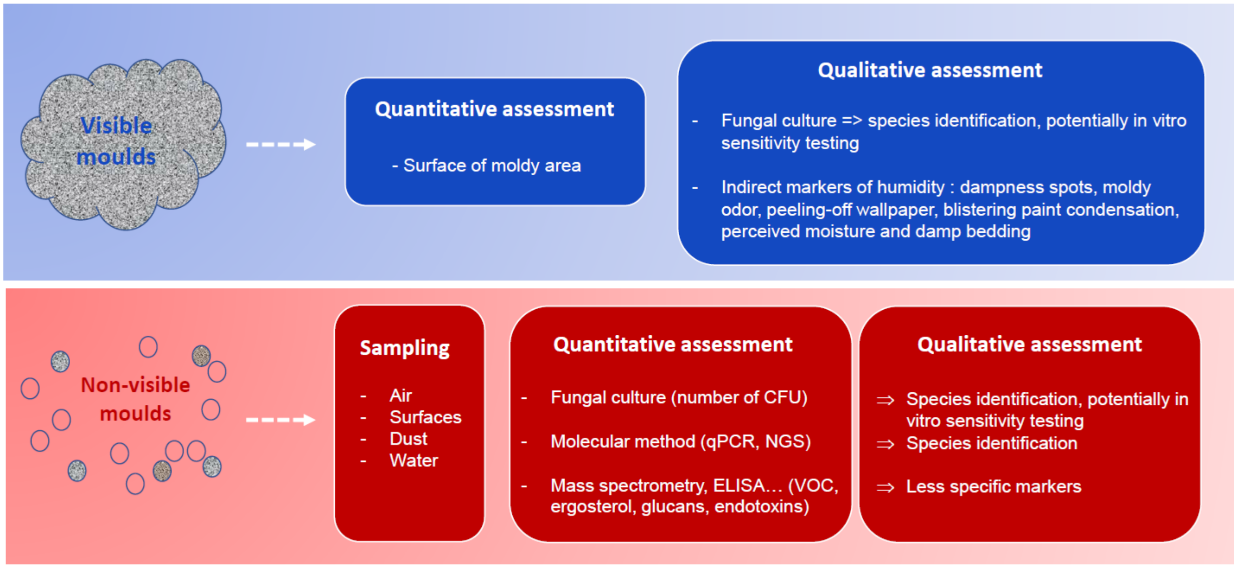
Supplemental Table 2 Occurrence of Fungal Infections in Hydrometeorological Natural Disasters (e.g., Rain and Floods)

Disaster Name	Disaster Time / period	Affected Regions	N cases	Invasive vs superficial/ colonization	Anatomic site	Fungal agent	Comment	Study Period	Ref.
Hurricane									
Katrina Category 5 Hurricane	2005, Aug 29	USA, Florida, Louisiana, Cayman Islands, New Orleans	3	Invasive	Neuro-ophtalmic	*Drechslera* spp., NS	N=3 Neuro-ophthalmic fungal infections; *Drechslera* spp. (1), septate hyphae (2)	2005, Aug	[142]
			1	Invasive	Lung	*Cladosporium* spp.		2005, Aug	[73]
			NS	NS	Sinus, neurological signs NOS ("symptoms associated with mold exposure")	NS	595/2,834 US Coast Guard responders to hurricanes Katrina and Rita reported mold exposure (10% of 595 had severe respiratory problems of which 42% sought medical treatment for it; 90% of 595 had mild respiratory symptoms of which 10% sought medical treatment for it)	2005, Aug - 2006, Nov	[143]
			1	Invasive	CNS, lung	*Blastomyces dermatitidis*	Immunocompetent person working at a farm hit by Hurricane Katrina	2006	[144]
			8	contamination without evidence of IFI	Lung, ear, sinus	*Syncephalastrum* spp.	N=8 *Syncephalastrum* identified in clinical specimen vs N=0 pre-hurricane since January 1, 2004	2005, Sep 12 - 2006, Feb 12	[145]
Ike Category 2 Hurricane	2008, Sep 13	Cuba, USA, Texas	3	Invasive	Soft tissue	Unspecified agent of chromoblastomycosis		2008	[146]
Sandy Category 3 Hurricane	2012, Oct 29	USA, New York City	7	Invasive	Skin/Soft tissue	*Aspergillus fumigatus*, *A. niger*, *A. flavus*, *A. terreus*, *Fusarium*, *Mucor*, *Curvularia*, *Bipolaris*, *Trichosporon*, *Aureobasidium*	Burn intensive care unit pre- and post-Sandy (2009-2016): high rate of polymicrobial infections post-hurricane vs pre-hurricane: 1/10 IMI patients with multiple species between Jan 2009 - Oct 2012 *vs* 4/7 patients with 2-4 molds Dec 2012 - Apr 2014; rate of mold infections: 0.6/1,000 patient days pre-Sandy Jan 2009 - Oct 2012, 2.0/1,000 during outbreak Dec 2012 - Apr 2014, 0.8/1,000 post-Sandy May 2014 - Dec 2016	2009 - 2016	[147]
			NS	mold exposure-associated LRS	(Lower respiratory tract symptoms)	Molds, NOS	Respiratory symptoms: exposure to mold/damp environments (AOR 2.0; 95% CI: 1.7–2.4), reconstruction work (AOR: 1.8; 95% CI: 1.5–2.1), and other potential respiratory irritants (AOR: 2.2; 95% CI: 1.8–2.6) post-Sandy were associated with lower respiratory symptoms with significant dose-response relationship compared to those	2012, Oct 29 - 2023, Nov	[84]

Disaster Name	Disaster Time / period	Affected Regions	N cases	Invasive vs superficial/ colonization	Anatomic site	Fungal agent	Comment	Study Period	Ref.
							not exposed to respective environments or irritants		
Harvey Category 2 Hurricane	2017, Aug 25	USA, Texas, Houston	2	Invasive	Skin	Mucorales NOS	N=2 cutaneous mucormycosis after liver transplantation post-Harvey	2017	148
			NS	NS	NS	*Aspergillus* spp., Mucorales, NS	1 year pre- vs. 1 year post-hurricane Harvey incidence (based on diagnosis codes) in Houston: aspergillosis (5.4 vs. 5.2 per 100,000 enrollees), mucormycosis (0.9 vs. 0.7), and unspecified mycoses (26.3 vs. 28.9) not significantly different; diagnosis codes for mold exposure (6.3 vs. 11.0 (RR 1.7, 95% CI 1.0-3.1)	2016, Sep - 2018, Aug	149
			103	Invasive	Lung, sinus, CNS, other	Aspergillus, Mucorales, NS	1 year pre- vs. 1 year post-hurricane Harvey, 4 hospitals in Houston: IMI (N=73 vs N=109; incidence 2.5 vs 3.7, RR 1.48, 95% CI 1.10-2.00); Aspergillosis 64/73 (87%) vs 96/109 (88%); Mucormycosis 7/73 (10%) vs 6/109 (6%)	2016, Sep - 2018, Aug	150
			36	Colonization, invasive	NS	*Aspergillus*, *Fusarium*, *Rhizopus*, *Syncephalastrum*, *Conidiobolus*	1 year pre- vs. 1 year post-hurricane Harvey, single center, Houston: 36 patients with culture positive mold infections pre-Harvey *vs* 67 post-Harvey, rate of invasive fungal infection was not significantly increased post-Harvey; Slightly higher in-hospital mortality in IMI patients post-Harvey	2016, Sep - 2018, Aug	151
			34	Colonization, invasive	NS	*Aspergillus*, *Fusarium*, *Rhizopus*, Conidiobolus, *Syncephalastrum*	1 year pre- vs. 1 year post-hurricane Harvey, single center, Houston: 34 patients with mold-positive cultures/202,365 patient-days pre-Harvey *vs* 43/207,373 post-Harvey; Rate of culture positive mold infections 0.17 pre-Harvey vs 0.21 post-Harvey/1,000 patient-days (no difference)	2016, Sep - 2018, Aug	152
Flooding									
Tropical storm Alberto-associated Chagrin River flooding	1994, Jul	USA, Ohio, Cleveland	10	NS	Lung/hemorrhage - no confirmation if fungus related	NS, high burden of *Stachybotrys chartarum* in air samples	N=10 infants (≤1 year) with pulmonary hemorrhage after living in moldy homes post-flooding (*Stachybotrys chartarum* isolated from those homes)	1994, Nov	153
Northern and central Thailand	2006, Sep	Thailand	2	Superficial	Skin	*Trichosporon mucoides*, non-spore	N=2 fungal dermatoses in 96 patients admitted with skin conditions during floods	2006, Oct	154

Disaster Name	Disaster Time / period	Affected Regions	N cases	Invasive vs superficial/ colonization	Anatomic site	Fungal agent	Comment	Study Period	Ref.
flooding						forming hyaline fungi			
Morakot Typhoon (Category 2)-associated flooding	2009, Aug 7	Taiwan	NS	NS	Wound, pus	NS	1 month pre- vs post-typhoon: higher rate of polymicrobial growths in post-typhoon patients, fungal growth not further specified	2009, Jul 9 - Sep 6	[155]
Thailand Floods	2011, Sep	Thailand, Bangkok area	1	Invasive	Skin	*Aspergillus flavus*	Within 6 weeks post-flood: skin lesions reported in 96 out of 707 army personnel and flood victims exposed to flood water - *Aspergillus flavus* (1)	2011, Oct - Dec	[156]
Denver-Boulder metropolitan area flooding	2013, Sep	USA, Colorado	4	Invasive	Rhino-orbital-cerebral	*Rhizopus arrhizus*	Within 1 month post-flood: 4 rhino-orbital cerebral mucormycosis	2013, Sep - Oct	[157]

Supplemental Figure 1. Remediation after Natural Disasters: quantitative and qualitative assessment of fungal contamination in buildings.



CFU: colony forming units; ELISA: enzyme linked immunosorbent assay; NGS: next-generation sequencing; qPCR: quantitative polymerase chain reaction; VOC: volatile organic compound.