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Review

The effects of climate change on fungal diseases with cutaneous manifestations: A report from the International Society of Dermatology Climate Change Committee

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ABSTRACT

Climate change affects all aspects of ecosystems, including humans and numerous microorganisms. Fungi are especially sensitive to climate extremes. Persistently warmer temperatures at increasingly higher latitudes are contributing to ongoing expansion of the geographic ranges of known fungal pathogens. Alongside fungal species' advancement into new territories, many have the capacity to develop thermotolerance. Consequently, a greater number of previously unharmful or underappreciated fungal species may emerge due to climate change.

More frequent extreme weather events - including heat waves, drought, and flooding - also foster circumstances that favor the survival and infectivity of pathogens. We reviewed the English language literature in order to describe what is known about the effects of climate change on fungal infections with cutaneous manifestations. We included candidal infections, dermatophytoses, other mold infections, and deep fungal infections, and focused on organisms with evidence of climate sensitivity, particularly those that are emerging, spreading to new geographic regions, or both. This may provide an important tool for understanding the epidemiological patterns underlying fungal transmission, predicting future outbreaks, and adopting effective control strategies.

1. Introduction

Climate variables – such as temperature, humidity, and rainfall – affect all aspects of ecosystems, including humans, other animals, plant life, and a host of microorganisms. Fungal organisms are especially sensitive to climate extremes; for example, most fungal species cannot tolerate mammalian body temperatures, and as such, even those with pathogenic potential have historically been limited in their pathogenicity by the mammalian "endothermy thermal barrier [1]." However, many fungal species have the capacity to develop thermotolerance [2,3]. As ambient temperatures rise worldwide from anthropogenic climate change, a greater number of previously unharmful or underappreciated pathogenic fungal species may become infectious. Likewise, persistently warmer temperatures at higher latitudes are expanding the geographic ranges of known fungal pathogens into new territories [4]. The relationship between climate change and precipitation is also complex. Some regions will incur greater rainfall and humidity, leading to increased soil moisture that favors the growth of specific fungal species [5,6]. Other geographic locations may experience drought or become more arid, supporting other mycoses' growth [7]. Additionally, more frequent extreme weather events related to climate change, like heat waves, drought, flooding, and severe storms, foster circumstances in which existing infectious fungi flourish *and* novel mycoses emerge. Importantly, while therapeutic advances have improved the care of patients with malignancy, prior organ transplant, and autoimmune conditions, the iatrogenic immune suppression from these treatments increases patients' risk of developing severe fungal infections, broadening the pool of susceptible individuals [8].

Climate change presents substantial challenges to healthcare systems worldwide. Physicians who completed their training or practice in regions where certain climate-sensitive fungal diseases were historically rare or absent may find it difficult to recognize, diagnose, and treat them. With the objective of improving the knowledge gap, we reviewed the literature in order to describe what is known about the effects of climate change on fungal infections with cutaneous manifestations. Furthermore, we identified and discussed topics for

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further investigation that would increase our understanding of climate change's diverse impact. Although research on climate variables and fungal diseases continues to grow, we acknowledge some disparities in the level of available evidence for specific infections. To address these differences, the following mycoses are grouped together by the strength of support in the literature for their respective climate-sensitivities.

2. Methods

We performed a literature review of English-language articles available on PubMed using each of the search terms climate, climate change, environment, temperature, and humidity, in combination with each of the following individual terms: fungus, fungal, candida, candidiasis, *Candida parapsilosis, Candida albicans, Candida auris, Candida glabrata*, tinea, dermatophyte, dermatophytosis, aspergillus, mucormycosis, mucor, talaromycosis, *Talaromyces, Penicillium, Cryptococcus*, cryptococcal, *Coccidioides*, coccidioidomycosis, *Blastomyces*, blastomycosis, histoplasmosis, *Sporothrix*, sporotrichosis, phaeohyphomycosis, and chromoblastomycosis. Relevant references within the initial articles identified were also reviewed. We focused on fungal infections with skin manifestations and evidence of climate sensitivity, particularly those that are emerging, spreading to new geographic regions, or both.

3. Fungi with strong evidence for climate-sensitivity

Fungal infections with strong evidence for climate-sensitivity – including their cutaneous and systemic manifestations – are summarized in Table 1.

3.1. Dermatophytosis

Dermatophytoses are infections of the hair, skin, or nails caused by the mold species *Microsporum, Epidermophyton,* or *Trichophyton.* Causative organisms are classified as anthrophilic (human reservoir), zoophilic (animal reservoir), or geophilic (soil reservoir); zoophilic and geophilic organisms usually cause more inflammatory lesions. Skin findings typically include red, scaly, often annular plaques (Fig. 1). Dermatophytes are sensitive to temperature and moisture changes, evidenced by their typical distribution at warm, moist sites of the body [9]. Indeed, dermatophyte keratinase activity, a proxy of skin infectability, is highest at 30-40 °C [10]. Diagnoses of tinea corporis have been reported in multiple countries to be higher in the wet season [5]. In France, a marked increase in dermatophytosis caused by *T. verrucosum* was observed between 2012-2016, and the number of annual cases correlated positively with mean annual rainfall [6]. In a study of three eco-climatic zones in Mali, tinea capitis prevalence was highest in zones with greater humidity [11]. Superficial fungal infections, particularly tinea corporis, have been reported to occur more frequently following flooding, for example, after the Indian Ocean Tsunami (2004) [12] and Hurricane Katrina in the US (2005) [13]. In addition, tinea pedis occurs more frequently after prolonged exposure and immersion of feet in floodwater [14].

In the last two decades, the epidemiology of dermatophytoses in India has shifted substantially. First, its overall prevalence is rising, and it has been hypothesized that increasing average ambient temperature and humidity in India, where there have been especially severe heat waves recently, may be a causative factor [15]. Second, there has been a change in the clinical characteristics at presentation toward a greater burden of extensive, highly inflammatory lesions as well as a shift in the most common causative organisms [16]. One study confirmed *T. mentagrophytes* as the cause of over 90% of dermatophytoses in India, whereas *T. rubrum* was previously the most prevalent pathogen [17]. This is well above this species' 20-25% worldwide prevalence [15]. This finding is concerning as resistance of *T. mentagrophytes* to terbinafine, a prominent treatment option, is emerging [16]. Of note, terbinafine-resistant *T. rubrum* dermatophytoses have also recently been reported from Europe [18].

3.2. Chromoblastomycosis

Chromoblastomycosis is a neglected tropical disease (NTD) caused by a group of dematiaceous (darkly pigmented) fungi [19]. *Fonsecaea pedrosoi* is the most common causative pathogen, followed by *Phialophora verrucosa* and *Cladophialophora carrionii* [7,19]. Cutaneous lesions arise after traumatic inoculation from an environmental source [20]. Skin findings may include skin-colored papules that evolve into verrucous, crusted, or ulcerated nodules. An acral predilection is common (Fig. 2), and sequelae can be debilitating [19]. Secondary lymphedema and pruritus may occur [19].

Chromoblastomycosis is caused by climate-sensitive pathogens. While this condition is seen globally, it's most prevalent in tropical and subtropical regions. Climate also influences which infectious organism predominates. In subtropical rain forests, chromoblastomycosis is most often caused by members of the *Fonsecaea* species, while in arid conditions, *Cladohialophora carrionii* is the primary pathogen [7]. Further, chromoblastomycosis has been reported after hurricane-related flooding [21] and in flooded areas of Thailand following the Indian Ocean Tsunami (2004) [22]. Of concern, agents of chromoblastomycosis are enriched in polluted environments where toxic monoaromates are prevalent, particularly in human-made niches such as wood treated by phenolic preservatives, toxic mine waste, or oil-polluted soils [23].

Table 1
Fungi with strong evidence for climate-sensitivity.

Clinical condition	Morphology	Organism(s)	Skin manifestations	Systemic manifestations	Climate sensitivity
Dermatophytosis	Mold	*Microsporum, Epidermophyton,* or *Trichophyton* species	Tinea capitis, corporis, cruris, and pedis; onychomycosis [9]	Rare [15]	Predilection for warm, moist body sites [9]; activity highest at 30-40 °C [10]. Associated with high humidity, high rainfall, and heavy flooding [5,6,11–13].
Chromoblastomycosis	Mold	*Fonsecaea pedrosoi, Cladophialophora carrionii,* & *Phialophora verrucose*	Verrucous, crusted, and/or ulcerated nodules resembling cauliflowers. Typically affects distal extremities [19]	Rare [19]	Highest prevalence in tropical/subtropical regions [7]. Cases reported after hurricane- and tsunami-induced flooding [21,22].
Coccidioidomycosis	Dimorphic fungus	*Coccidioides immitis* & *C. posadasii*	Primaryinoculation: nodules and ulcers Disseminateddisease: multifocal (umbilicated) papules/nodules [28]	Pulmonary: Fever, cough, fatigue, dyspnea Disseminated: headache, myalgias, meningeal signs, inflammatory arthritis [27]	Thrives in areas with low rainfall, few winter freezes [24,25]. Geographic range expanding to include Northwest US [29,30].

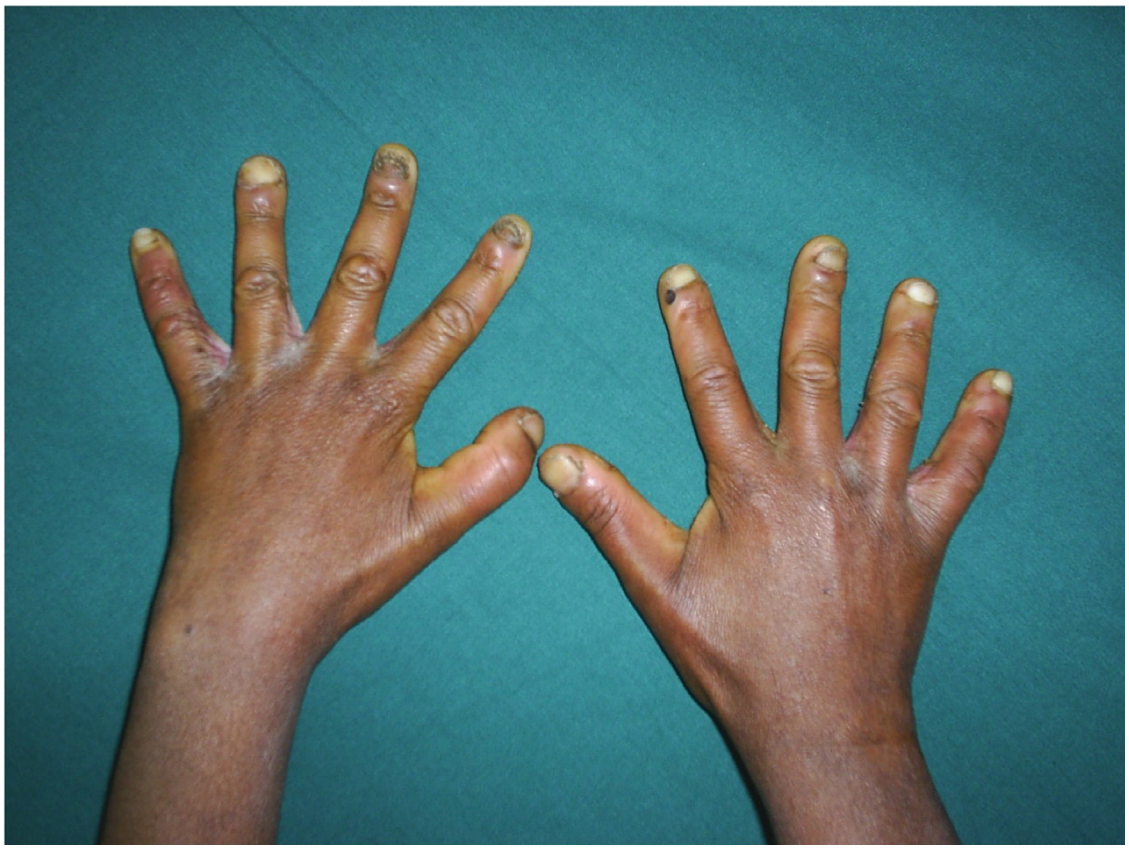

Fig. 1. Tinea corporis. Widespread, scaly, annular, erythematous plaques in a patient with widespread tinea corporis.

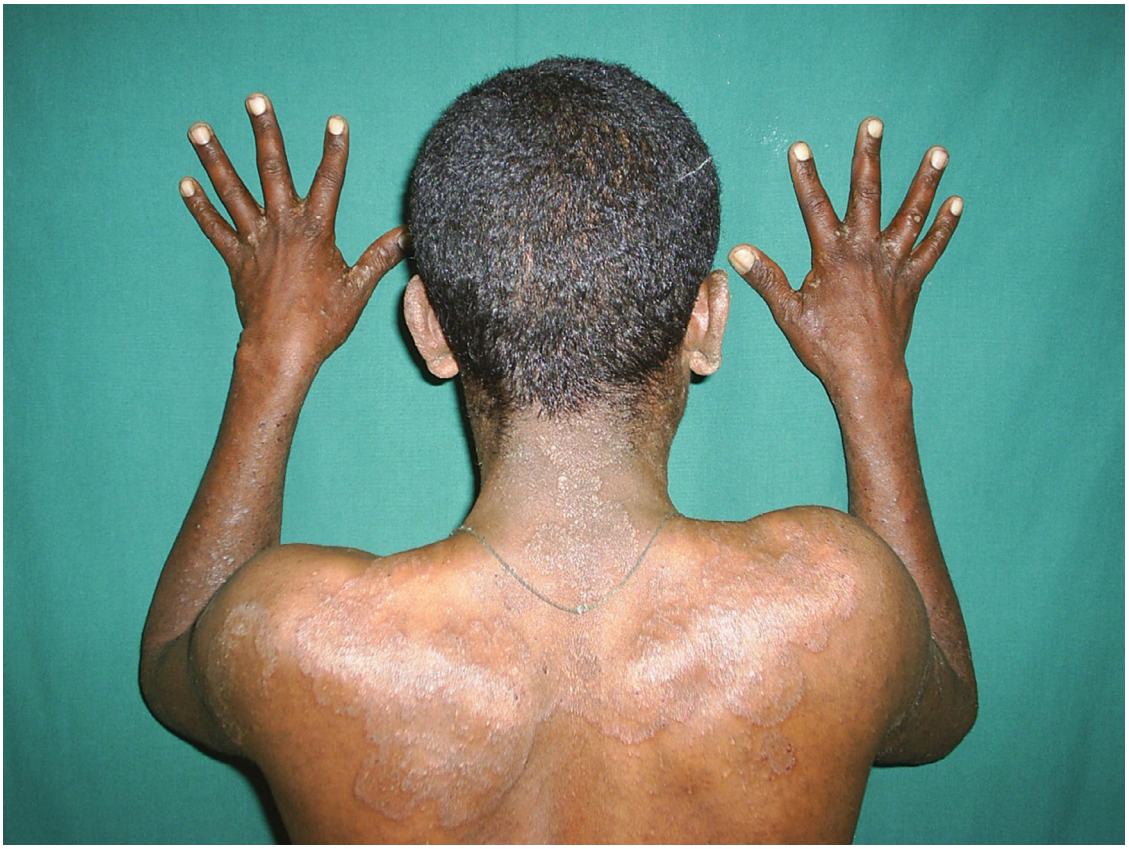

Fig. 2. Chromoblastomycosis. Chronic, progressively expanding leg lesion with verrucous and nodular features that developed over the course of 8 years following a traumatic injury to the extremity.

3.3. Coccidioidomycosis

Coccidioidomycosis, or Valley Fever, is a systemic mycosis caused by the dimorphic organisms *C. immitis* and *C. posadasii.* These are endemic to arid regions of North and South America, and their confined geographic range reflects their climate sensitivity. *Coccidioides* spores, which are found in soil approximately 10 cm below the surface, require increased soil moisture to germinate, followed by a dry spell to aerosolize. Wind and precipitation are the main drivers of spore aerosolization [24,25]. During prolonged heat waves, high temperatures sterilize topsoil, reducing competition against *Coccidioides* by other microbes [26]. In endemic areas, up to 50% of the population have been exposed, mainly via spore inhalation or, less commonly, via cutaneous inoculation [24]. Only 40% of infected persons develop symptoms, and 1% develop disseminated disease [27]. Skin lesions can be seen in three scenarios: 1) they can be the result of acute pulmonary infection, presenting as an "acute pulmonary exanthema"; 2) they can reflect the presence of a disseminated infection involving the skin; or, rarely, 3) they can represent a primary infection from direct inoculation. Therefore, skin findings vary widely, and include erythema nodosum-type lesions, acute exanthema, erythema multiforme, multifocal umbilicated papules or nodules, and indurated nodules with ulceration [28]. Groups that experience higher mortality include transplant recipients, certain ethnic groups (i.e., Filipino-Americans), and patients with malignancy, diabetes, and HIV infection [27].

The incidence of coccidioidomycosis has risen steadily in the last two decades [29]. Reasons for this include better diagnostic tests, regional population growth that increased the size of vulnerable populations, and progressively favorable environmental conditions for organism growth. The geographic range of *Coccidioides* species is also expanding – spores have now been identified far north of the warm, arid Southwestern US, including in the soil of Oregon [30] and Washington states; since 2010, at least 16 cases have occurred in south-central Washington, all in patients with negative travel histories [31]. Heavy rainfall in 2016 following a prolonged drought in California was linked to an increased incidence of cutaneous coccidiomycosis [32,33]. Incidence is also known to increase following environmental disasters such as wildfires [34]. By 2100, the area of endemicity for coccidioidomycosis is projected to double in size if climate change continues its current trajectory. The number of affected US states could rise from 12 to 17, with a caseload increase of 50% [35].

4. Fungi with moderate evidence for climate-sensitivity

Fungal infections with moderate evidence for climate-sensitivity – including their cutaneous and systemic manifestations – are summarized in Table 2.

4.1. Mycetoma

Mycetoma is an NTD that causes a chronic, granulomatous, progressive inflammatory disease of the skin and subcutaneous tissues following traumatic inoculation of causative organisms. Persons with inadequate footwear and those who work outdoors are at especially high risk. Mycetoma may be caused by either fungi (eumycetes) or bacteria (actinomycetes), resulting in eumycetoma or actinomycetoma, respectively [36]. Lesions usually begin as painless, slowly progressive nodules at sites of trauma, most often the lower extremities. A fully developed mycetoma is characterized by the classic triad of tumefaction, draining sinuses, and presence of grains (Fig. 3) [37]. Infection may spread to subcutaneous structures including bone,

Table 2
Fungi with moderate evidence for climate-sensitivity.

Clinical condition	Morphology	Organism(s)	Skin manifestations	Systemic manifestations	Climate sensitivity
Mycetoma	Mold	Numerous cauasative bacterial and fungal species	Painless subcutaneous nodules, sinuses; discharge with grains [37]	Rare, but may cause fatal bone infection [39]	Highest prevalence in tropical/subtropical regions [36,38,39]. Eumycetoma reported in areas with higher rainfall [36].
Talaromycosis	Mold	*Talaromyces marneffei* (formerly *Penicillium marneffei*)	Small, painless (umbilicated) papules on face, extremities, palate; chronic genital sores [44]	Fever, malaise, weight loss, cough, dyspnea, lymphadenopathy, hepatosplenomegaly [19]	Infections higher in rainy seasons (Thailand, Vietnam) [45,46] and associated with high relative humidity [47].
Sporotrichosis		*Sporothrix schenckii* & *brasiliensis*	Localized (most common form): erythematous ulcers and/or nodules, in a lymphocutaneous pattern Disseminateddisease: multifocal nodules or ulcers [49]	Pulmonary: fevers, cough, dyspnea, chest pain Osteoarticular: inflammatory oligoarthritis Disseminated: fever, oligoarthritis, headache, confusion [19]	Optimal growth at 22 °C-27 °C and 90% humidity [49]; endemic to tropical/subtropical regions. Cases peak in rainy season [50]. Increasing reports of local cases in areas not previously known to be endemic [51–54].
Blastomycosis	Dimorphic fungus	*Blastomyces dermatitidis & B gilchristii*	Primaryinoculation: solitary or multifocal verrucous nodules/ulcers with irregular borders Disseminateddisease: verrucous nodules/ulcers [56]	Pulmonary: fatigue, fever, cough, night sweats, chest pain Osteoarticular–Pain in involved areas (most commonly long bones or vertebrae) Disseminated: osteoarticular signs as above; prostatitis (men), adnexal pain (women); meningeal signs, focal neurologic deficits [56]	Temperature sensitive (cases peak in autumn) [63]. Soil moisture influences growth/dispersal [58]. Incidence rising in known endemic areas. Geographic range expanding from central US [59,60].
Histoplasmosis	Dimorphic fungus	*Histoplasma capsulatum var. capsulatum* & *var. duboisii*	Primaryinoculation: nodules, cellulitis, necrotizing fasciitis [69–71] Disseminateddisease: umbilicated papules [65]	Pulmonary: fevers, dyspnea, cough, chest pain Disseminated: fatigue, weight loss, meningeal signs, hepatosplenomegaly, shock [65]	Grows best in areas with high soil humidity [67]. Geographic range expanding from central US [76].

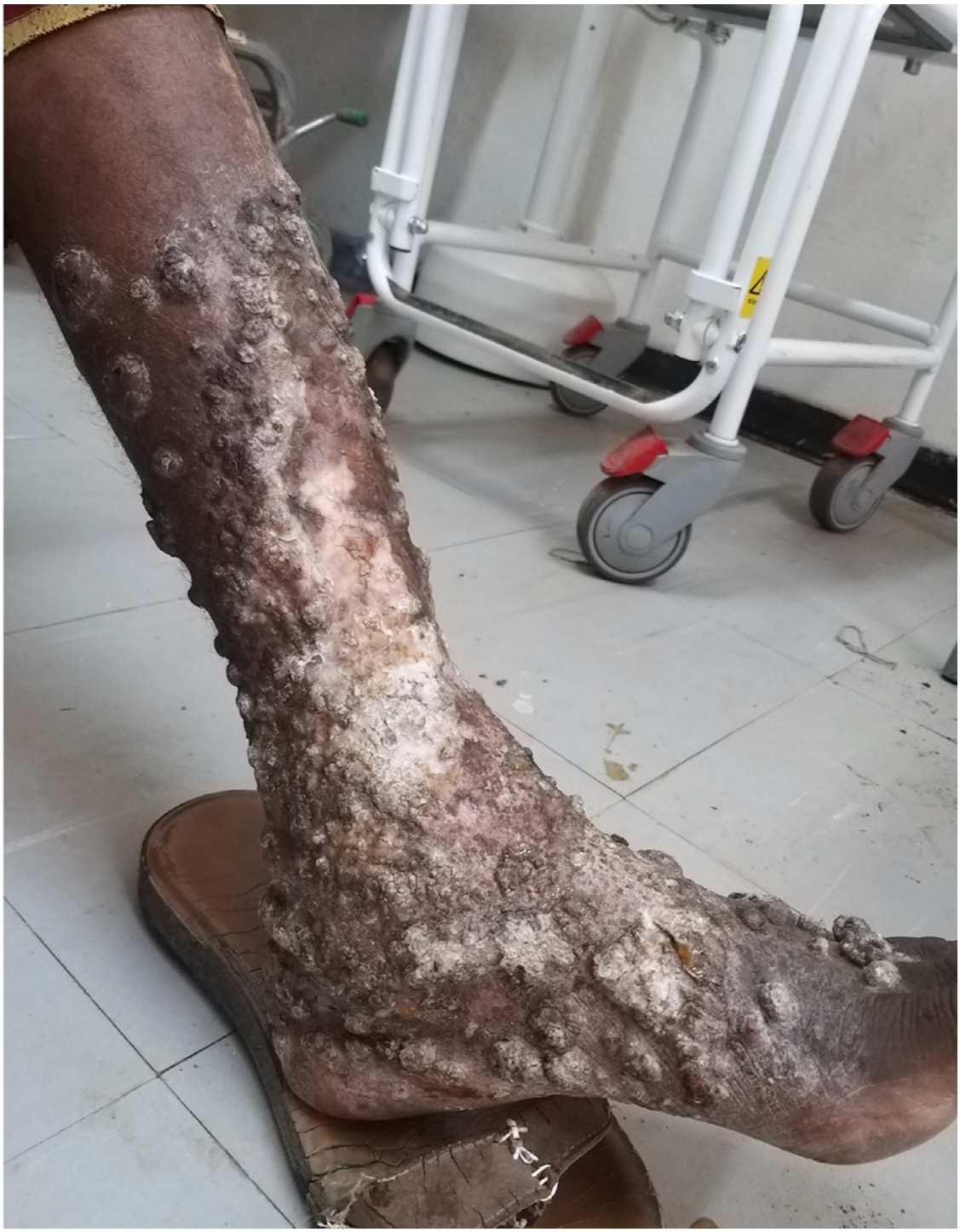

Fig. 3. Mycetoma. Chronic, edematous, progressively destructive mycetoma of the foot that developed over 6 years.

resulting in deformity and loss of function with serious social and economic implications [36].

Most reported cases of mycetoma are from tropical and subtropical areas, in the so-called "mycetoma belt", which includes Mexico, Venezuela, Mauritania, Senegal, Chad, Ethiopia, Sudan, Somalia, Yemen, and India [36,38,39]. Eumycetoma occurs more often in areas with high rainfall [36]. A study from India suggested a rising incidence of mycetoma was due to changes in climatic conditions, such as heavy rainfall, increased irrigation by the Rajasthan Canal, urbanization of villages, and modification in agriculture [40]. The geographic range of the organisms that cause mycetoma may be expanding, or is at least broader than previously known. There have recently been reports from new areas, including the Comoro Islands, Laos, Venezuela, and Brazil [38]. Additionally, autochthonous cases have recently been reported from Europe [41] and the US [42].

4.2. Talaromycosis

Talaromycosis is an AIDS-defining opportunistic infection caused by *Talaromyces marneffei* (formerly known as *Penicillium marneffei*), mainly reported in persons living in South and Southeast Asia [43]. Manifestations may be mucocutaneous, including papules on the face and extremities, palatal papules, and chronic genital sores [44].

P. marneffei infections have been reported to occur more frequently in the rainy seasons in Northern Thailand [45] and Vietnam [46]. A later study from Vietnam noted that *P. marneffei* admissions were strongly associated with humidity ($P < .001$), although rain, temperature, and wind were not statistically significant associations [47]. Talaromycosis has also been reported in immunosuppressed travelers returning from endemic areas, presenting a diagnostic challenge for unaware clinicians [48].

4.3. Sporotrichosis

Sporotrichosis is a subacute to chronic infection caused by thermally dimorphic *Sporothrix* species, especially *S. schenckii* [19]. It is found throughout the world in soil and on decaying plant matter. Localized infection occurs after skin injury, manifesting with erythematous nodules, ulcers, or both, classically distributed in a lymphocutaneous pattern (Fig. 4) [49].

Sporotrichosis is climate sensitive. It is endemic to tropical and subtropical regions, growing best at temperatures ranging from 22 °C-27 °C and at 90% humidity [49]. Notably, it is hyperendemic to Mexico, Peru, Colombia, and Brazil [50]. In Australia, case rates peak during the rainy season [50]. Recently, cases presumed to be autochthonous have been reported in areas not previously thought to be endemic, including northern Japan [51], southern Italy [52], France [53], and Portugal [54]. In addition, zoonotic (domestic cat-related) sporotrichosis is being reported more frequently in Brazil, where widespread outbreaks have occurred in multiple regions of the country; however, the relationship of this to climate variables is unknown [55].

There have also been documented cases of sporotrichosis following extreme flooding in China. From 1991 to 1993, over 400 villagers contracted sporotrichosis, reporting a history of contact with reeds

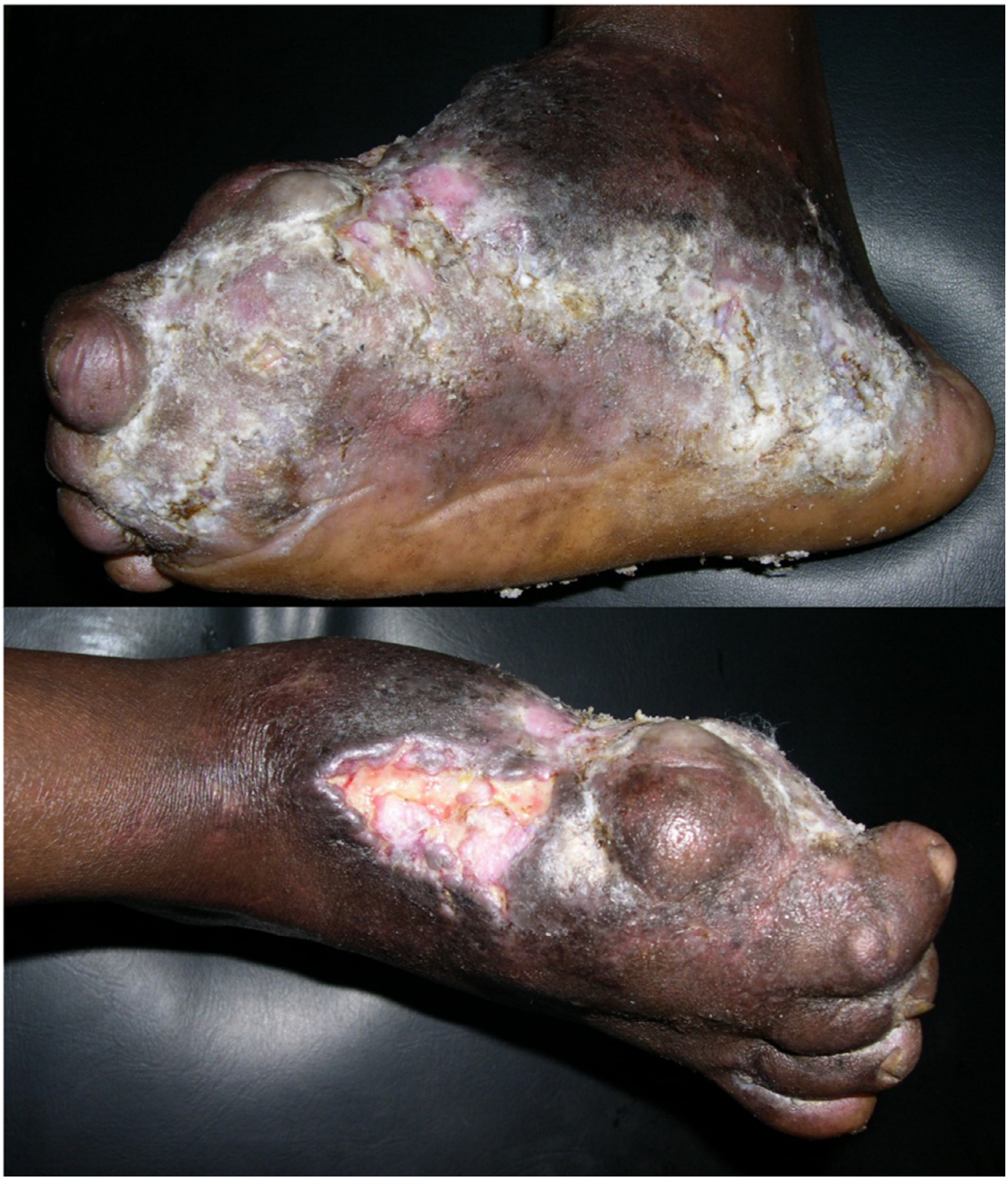

Fig. 4. Localized sporotrichosis. Erythematous nodules with overlying scale in a lymphocutaneous distribution in a patient with localized sporotrichosis.

[49]. Researchers later isolated *Sporothrix* from these plants. Furthermore, 224 villagers who collected reeds that washed ashore from the Nen River flood developed sporotrichosis after three to six months [49].

4.4. Blastomycosis

Blastomycosis is caused by the dimorphic fungal organisms *B. dermatitidis* and *B. gilchristii*. As with *Coccidioides*, infection is mainly acquired via inhalation of spores that are aerosolized during activities that disrupt decaying wood or soil [56]. Once inhaled, spores convert to their yeast form [56]. Most infections lack symptoms; if symptomatic, patients may present with acute or chronic pulmonary infection, and more rarely with hematogenous dissemination to other organs such as the skin, genitourinary tract, and osteoarticular structures [56]. In patients with extrapulmonary blastomycosis, cutaneous lesions occur in up to 60% of cases, typically appearing as ulcers with heaped-up borders and an exudative base, or verrucous nodules with irregular borders [56]. Cutaneous blastomycosis can also be acquired primarily, via direct inoculation, typically presenting as verrucous nodules, ulcerations, or both; lesions may be solitary or multifocal [56].

Historically, the geographic range of *Blastomyces* included Central and Southern North America, especially the waterways in the Great Lakes region (US) [57]. Hyperendemic foci exist in western Ontario and north-central Wisconsin [58]. Evidence suggests the organism's range may be growing [58]; since 2000, cases have been identified as far west as Saskatchewan [59] and as far east as New York [60]. The incidence also appears to be rising in certain states – Illinois, Indiana, and Wisconsin [58]. However, the reasons behind this trend as well as the lateral geographic expansion of blastomycosis are unknown, requiring further evaluation. While the factors contributing to *B. dermatitidis*' environmental persistence are not fully understood, soil moisture plays an important role [58]. Most cases of sporadic and outbreak-related cases of blastomycosis occur by waterways [61]. Climate and weather also likely influence conditions fostering growth and dispersal. Blastomycosis clusters have been observed after heavy rainfall followed by low precipitation or drought [61]. Additionally, three months following Hurricane Katrina, a case of blastomycosis with CNS, pulmonary, and cutaneous manifestations was reported in a previously healthy 26-year-old man [62].

Blastomycosis is known to be climate-sensitive; its presentation has seasonal variation, with case rates peaking in autumn and nadiring in spring, which suggests relatively lower infectivity during colder months [63]. The relationship with temperature is less clear; in one analysis of endemic regions, maximum temperature was *inversely* related with the odds of being a high cluster area [64]. To our knowledge, there is no data published on shifts in seasonal patterns as weather patterns change, which could elucidate greater information about the association between temperature and blastomycosis incidence. Further research conducted on climate variables impacting the virulence of blastomycosis-causing organisms would also be beneficial.

4.5. Histoplasmosis

Histoplasmosis is caused by several dimorphic fungal organisms of the *Histoplasma* species. Compared to *Coccidioides* and *Blastomyces, Histoplasma* appears to be less geographically restricted, accounting for the fact that it is the most common of the three endemic mycoses [65]. These organisms grow particularly well in organic matter enriched by bird or bat droppings [66]. Histoplasmosis growth is greatest in areas with high soil humidity, and soil moisture influences the temperature at which *Histoplasma* can survive [67]. Infection is acquired when contaminated soil is inhaled [65]. Specific risk factors for exposure include bird and bat watching, cave exploration, outdoor construction occupations, and bamboo removal and burning [68]. Primary cutaneous histoplasmosis occurs after direct inoculation and may appear as cutaneous nodules [69], cellulitis [70], or necrotizing fasciitis [71]. Patients with disseminated (extrapulmonary) disease may present with numerous umbilicated papules [65].

The geographic range of *Histoplasma* was historically established, via nationwide skin histoplasmin sensitivity testing, to include the Ohio and Mississippi River Valleys in North America [58]. The organism is also endemic to much of Latin America [65] and is even more widely distributed than previously appreciated [58]. Specifically, the HIV/AIDS pandemic and widespread use of immunosuppressive agents have increased knowledge of its global distribution [43]. Cases have been acquired in regions not previously considered to be endemic, including Florida [72], Montana [73], Idaho [73], Nebraska [74], Minnesota [74], Wisconsin [74], Michigan [74], and Alberta, Canada [75], with calls for increased state surveillance [74]. A known factor associated with outbreaks is environmental disruption of the organism's habitat [66]. However, it is unknown whether there is an increased caseload seen after extreme weather events. While the overall frequency of histoplasmosis outbreaks appears to have decreased over time, cases are hypothesized to be underreported since histoplasmosis – as of 2016 – was not a nationally notifiable disease in 80% of US states [66]. Moreover, there were >5000 national hospitalizations for histoplasmosis in 2012. By subtracting estimated hospitalizations from states with surveillance data from the national total, researchers calculated a percentage of hospital admissions from other states. The unusually high predicted percentage likely indicated a greater number of undiagnosed infections in surveilled states, some of which are regions not traditionally recognized for endemicity [74].

Despite the northward expansion of *Histoplasma*, thermotolerance of this species is poorly understood. However, modeling of environmental conditions involving distance to open water, soil pH, and land cover type demonstrated expanded geographical reach of *H. capsulatum* from climate change [76]. Certain environmental stressors may also drive pathogenicity. Strains of *H. capsulatum* that grow at elevated temperature or those exposed to more light show increased virulence, which may be of concern if temperatures and exposure to UV increases as predicted [76].

5. Fungi with weaker evidence for climate-sensitivity

Fungi with weaker evidence for climate-sensitivity – including their cutaneous and systemic manifestations – are summarized in Table 3.

5.1. Candidiasis

Candidiasis may be caused by numerous *Candida* species and is classified as either superficial (cutaneous, mucocutaneous, hair, and nail involvement) or invasive (e.g., meningitis, visceral infection, and candidemia). The favored areas of involvement for superficial cutaneous candidiasis are the skin folds, genital and oral mucosa, fingertips, and nails. Superficial cutaneous candidiasis typically presents as beefy-red patches and plaques with fine scale and satellite papules or pustules peripherally [77]. Alternative manifestations may include erosion interdigitalis blastomycetica (Fig. 5), balanitis, or diaper dermatitis. *Candida* is also the most common non-dermatophyte cause of onychomycosis [78]. Cutaneous manifestations of invasive candidiasis may include ecthyma gangrenosum-like lesions, a folliculitis-like eruption, subcutaneous nodules, and tense hemorrhagic bullae [77]. The most common risk factors for infection include extremes of age, malnutrition, obesity, diabetes, and immune deficiency [79]. Although a predilection for moist body sites suggests temperature and humidity sensitivity [80], more research is needed into the specific effects of climate change and extreme weather events on the prevalence of candidal skin infections.

Table 3
Fungi with weaker evidence for climate-sensitivity.

Clinical condition	Morphology	CaUnited Statestive organism	Skin manifestations	Systemic manifestations	Climate sensitivity and geographic range
Candidiasis	Yeast	Numerous species, including *C. albicans, C. parapsilosis, C. glabrata*, & *C. auris*	Superficialcutaneous: bright pink-red papules/ vesicles, diaper dermatitis, erosio interdigitalis blastomycetica, balanitis [77] Onychomycosis: nail dystrophy [78] Mucocutaneous: stuck-on white papules/plaques Candidemia: folliculitis-like eruption, subcutaneous nodules, hemorrhagic bullae [77]	Bacteremia: fever, chills, fatigue, constellations of endocarditis [77]	Predilection for warm, moist body sites in superficial cutaneous candidiasis [80].
Cryptococcosis	Yeast	*C. gattii* or *C. neoformans*	Nodules, papules, pustules, abscesses, cellulitis, ulcers [81,84–86]	Pulmonary: Fever, cough, fatigue, dyspnea CNS: meningeal signs, focal neurologic deficits [82,83]	Previously seen in tropical/ subtropical climates; wider geographic range observed than before [87–89].
Mucormycosis	Mold	*Rhizopus, Lichtheimia*, & *Mucor* species	Echthyma-gangrenosum-like lesions, mucutaneous ulceration and eschar, necrotic papulonodules, cellulitic plaques, necrotizing fasciitis [77]	Rhinocerebral: unilateral facial swelling, headache, congestion, fever Pulmonary: fever, cough, chest pain, dyspnea Gastrointestinal: vomiting abdominal pain, bleeding Disseminated: meningeal signs, confusion, coma [77]	Inoculated by wind- and water-borne debris during natural disasters [92,93]

5.2. Cryptococcosis

Cryptococcosis is a potentially fatal mycosis caused by *C. gattii* or *C. neoformans*. The condition frequently affects the lungs, central nervous system (CNS), and skin. Cryptococcosis normally occurs after inhalation and deposition of fungal spores into the pulmonary alveoli [81]. *C. gattii* primarily affects apparently healthy individuals, typically causing granulomatous lesions (cryptococcomas) in the lung and brain, and occasionally in the skin [82,83]. *C. neoformans* most often affects immunocompromised persons, e.g. AIDS patients and organ transplant recipients [84,85]. Cutaneous presentations may include nodules, papules, pustules, abscesses, subcutaneous swelling, cellulitis-like erythema, and pyoderma gangrenosum-like ulcerations [81,84–86].

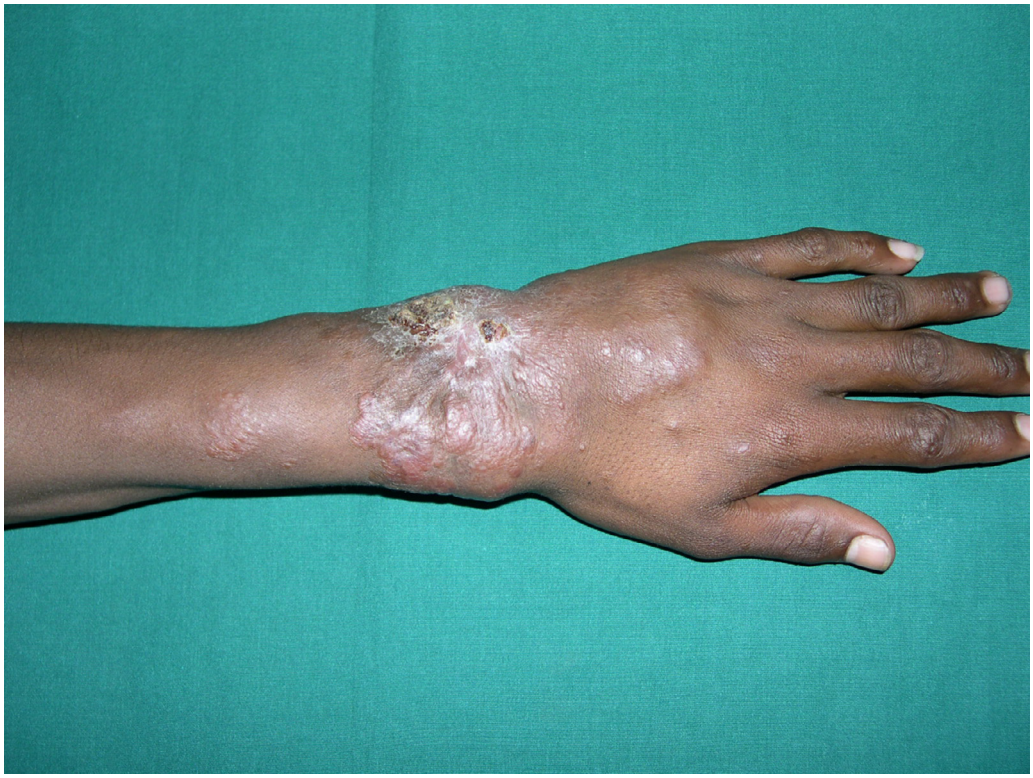

Fig. 5. Candidiasis. Macerated, scaly erythematous papules and plaques in two patients with localized superficial candidiasis of the webspaces and distal fingertips. A common risk factor is prolonged exposure to moisture.

C. gattii infections prior to 1999 were most often found in tropical and subtropical climates. Since that time, there has been an expansion of the geographic range of *C. gattii* to climates such as the Pacific Northwest (Washington and Oregon, US, and British Columbia, Canada). This emergence may be due to climate change, altered land use, and host susceptibility [87–89].

5.3. Mucormycosis

Mucormycosis, also known as zygomycosis, is caused by molds from the genera *Rhizopus, Lichtheimia*, and *Mucor* [90]. Cutaneous disease may present after traumatic skin injury [91]. Alternatively, spores may be inhaled or ingested [91] with skin findings occurring as a secondary consequence of underlying invasive mucormycosis; this form is most commonly seen in patients with diabetes mellitus and severely immunocompromised patients, such as those who have undergone hematopoietic stem cell transplantation [90]. Skin manifestations may include ecthyma gangrenosum-like lesions, mucocutaneous ulceration and eschars, necrotic papulonodules, cellulitic plaques, or any combination of these [77].

Mucormycosis has been reported several times following natural disasters, during which the organisms are inoculated via penetrating injuries from wind- or water-borne debris. Necrotizing fasciitis due to mucormycosis, which has a high mortality, was notably reported in victims of the 2004 Indian Ocean Tsunami [92] and the 2011 Missouri tornado [93]. These cases were hypothesized to be the result of soil dispersal and displacement. Currently, there is insufficient evidence in the literature to evaluate relationships between climate variables and the virulence or geographic range of genera causing mucormycosis. Further research is recommended to understand any associated environmental factors.

6. Conclusion

Several superficial, deep, and systemic mycoses are caused by organisms known to be sensitive to climate variables such as temperature, humidity, and rainfall. Anthropogenic climate change, through its widespread and varied effects on ecosystems, is having and will continue to have effects on the pathogenicity of fungal organisms. More specifically, sustained higher temperatures at increasingly higher latitudes account partially for the expanding geographic ranges of the dimorphic fungi *Coccidioides, Blastomyces, Histoplasma*, and *Sporothrix*. Natural disasters such as floods, hurricanes, and drought are associated with increased incidence of several specific fungal infections; the burden posed by these infections may grow as a result of increasingly frequent climate change-related extreme weather events. In addition, novel climate-sensitive pathogenic fungal organisms, such as *Tinea mentagrophytes* (in India), have emerged in recent years in the setting of global warming. In the future, sustained higher ambient temperatures globally may lead to the emergence of previously unknown or underappreciated pathogenic fungal species. This presents a potential challenge for physicians who trained or practice in regions with novel or previously rare fungi. Through increased awareness of new patterns related to climate change, physicians can collaborate more effectively with multi-disciplinary teams to offer sustained coordination and follow-up for affected patients. Importantly, while there is mounting evidence of the impact of climate variables on fungal pathogens, there is also a continued need for global research to evaluate changes in the incidence and prevalence of mycotic species with cutaneous manifestations. Long-term monitoring of shifting geographic spreads may improve both detection and care.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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