

Assessment of the emerging risk of human fungal infections after natural disasters and extreme weather events

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EXECUTIVE SUMMARY

Human fungal infections are relatively rare compared to viral and bacterial infections, due at least in part to their preferential growth at temperatures lower than that of the human body. However, global temperature rises coupled with increases in natural disasters and extreme weather events are triggering an increase in these infections.

The aim of this report is to provide a summary of what is known about fungal infections associated with natural disasters and extreme weather events to support health professionals in identifying these infections. Due to overlap in routes for infection, events were grouped into flooding events, which includes tsunamis, cyclones and extreme rainfall; events associated with dispersal of fungal propagules, such as tornadoes, earthquakes, landslides, dust storms, wildfires; and volcanic eruptions.

The main routes for infection during flooding events are aspiration of floodwaters, infection of traumatic wounds and exposure to mould proliferating on water-damaged surfaces and materials. Most reported fungal infections related to aspiration of floodwaters involved *Scedosporium* species which often disseminated from the lungs to other sites in the body. These infections were frequently misdiagnosed due to the rarity of these infections in immunocompetent individuals, and the propensity for symptoms to present some time after the near drowning. Several different fungal species have been associated with infection of traumatic wounds in flood victims, including *Scedosporium* and *Apophysomyces elegans* – a member of the order Mucorales notable for causing rare life-threatening necrotic infections. Several studies reported potential links between mould exposure after flooding events and invasive fungal infections, particularly in immunocompromised individuals.

The main routes for fungal infection associated with dispersal events are inhalation of fungal propagules and infection of traumatic wounds. Most reports associated with inhalation involved *Coccidioides* species which are not known to be present in New Zealand. However, this route for exposure is still relevant, particularly as aerosolised dust and wildfire smoke can transmit other fungal species. Indeed, inhalation of dust aerosolised by a 2011 tornado in Joplin, USA and the 2011 Japanese earthquake has been associated with *Aspergillus* lung infections. The majority of reported fungal wound infections associated with dispersal events related to the Joplin tornado and involved *Apophysomyces trapeziformis* – another Mucorales associated with necrotising infections.

Few reports of fungal infections associated with volcanic eruptions exist, although cases of necrotising fasciitis caused by *Rhizopus arrhizus* were documented in victims of an eruption of Nevado del Ruiz in Colombia in 1985. More recently, two burn victims of the 2019 Whakaari White Island eruption developed fatal *Aspergillus fumigatus* infections.

This report also highlights how fungal infections are likely to disproportionately affect vulnerable populations in New Zealand in the aftermath of natural disasters and extreme weather events, particularly Māori who are at increased risk due to higher prevalence of comorbidities and higher likelihood of living in mould-affected housing and geographically remote areas more vulnerable to extreme weather events.

Finally, this report highlights the importance of early detection of fungal infections in victims of natural disasters and extreme weather events and emphasises that a potential fungal cause should be considered where wound infections don't respond to antibiotic treatment.

1. INTRODUCTION

Most fungi are adapted to grow at temperatures below that of the human body, and their low thermal tolerance is considered a key factor limiting human fungal infections (Seidel et al., 2024). As such, the potential for outbreaks of human fungal diseases is often overlooked in favour of viral and bacterial diseases. However, as global temperatures rise due to climate change, fungi are adapting to higher temperatures and becoming more thermotolerant, leading to increased virulence of known fungal pathogens, emergence of new pathogens, and geographical spread of pathogens into previously non-endemic areas (Seidel et al., 2024). Compounding this are increases in the frequency and severity of natural disasters and extreme weather events which can act to further spread pathogenic fungi and trigger outbreaks of disease (Seidel et al., 2024) (Figure 1).

Natural disasters and extreme weather events can trigger fungal disease outbreaks via several routes including increasing susceptibility of individuals to infection (eg, via traumatic wounds or burns), dispersal of potentially pathogenic fungi present in the environment (eg, via dust storms, wildfire smoke, strong winds) and increased exposure in the aftermath of disasters due to fungal proliferation (eg, mould in water-damaged buildings) (Figure 2).

1.1. AIM AND SCOPE

The aim of this report is to assess what is known about fungal infections following natural disasters and extreme weather events in order to support health professionals in identifying potentially life-threatening fungal infections in the aftermath of these events. Man-made disasters (eg, radiation accidents, acts of terrorism) are outside the scope of this report.

Due to overlap in potential routes for infection between different natural disasters and extreme weather events, they have been grouped as follows: events associated with flooding (eg, cyclones, tsunami, extreme rainfall); events associated with dispersal of fungal propagules (eg, tornadoes, dust storms, wildfires, landslides, earthquakes); and volcanic eruptions. There will undoubtedly be some overlap between groups as many of these events also have the potential to cause traumatic wounds which are ideal routes for fungal infection.

To evaluate the relevance of the fungal infections identified in this report within the New Zealand context, the presence of the various pathogens in New Zealand was investigated using the New Zealand Organisms Register (<https://www.nzor.org.nz/search>). The findings from this assessment are presented in the Appendix.

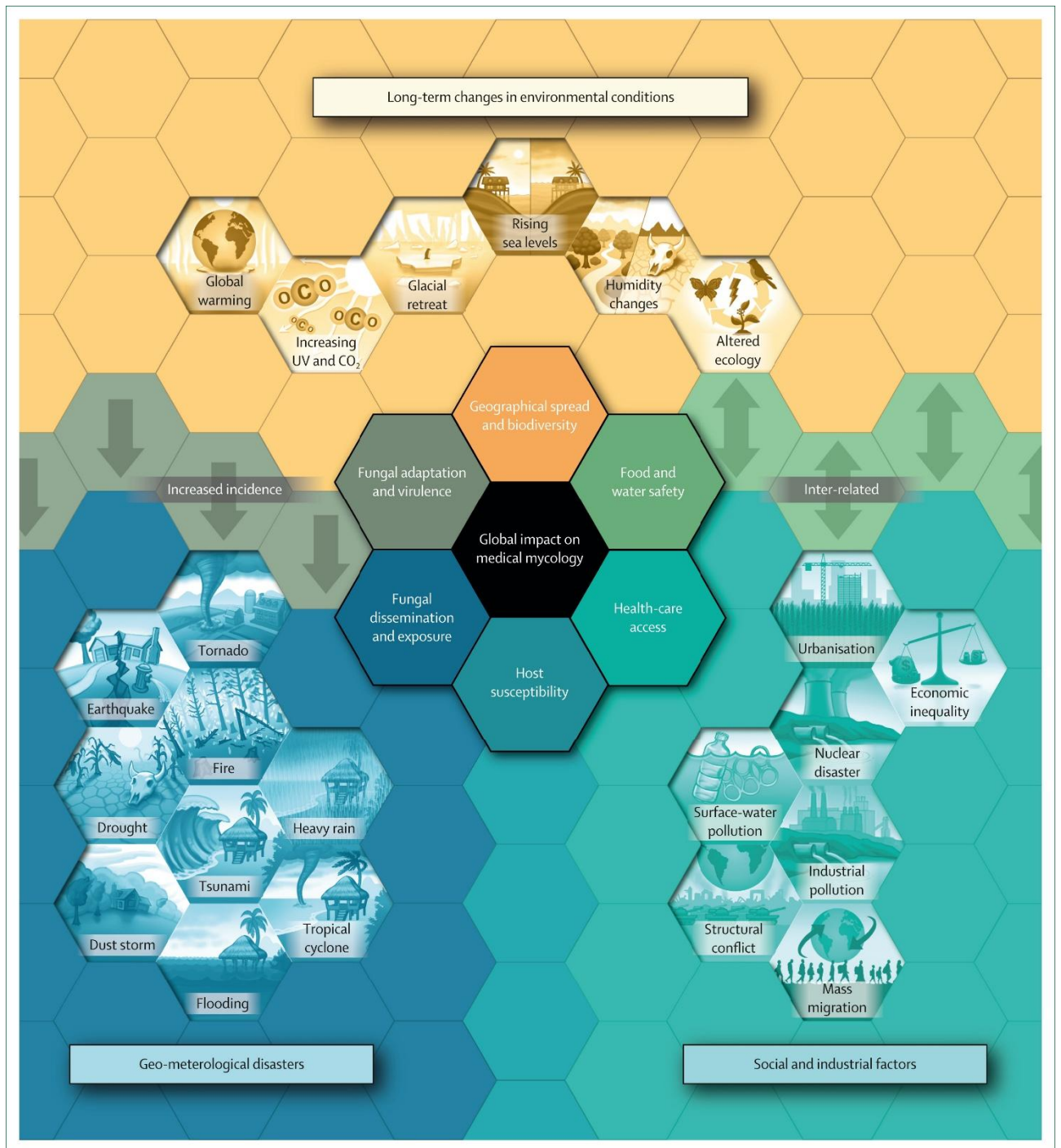


Figure 1: Impacts of climate change and natural disasters on fungi

Reproduced from Seidel et al. (2024).

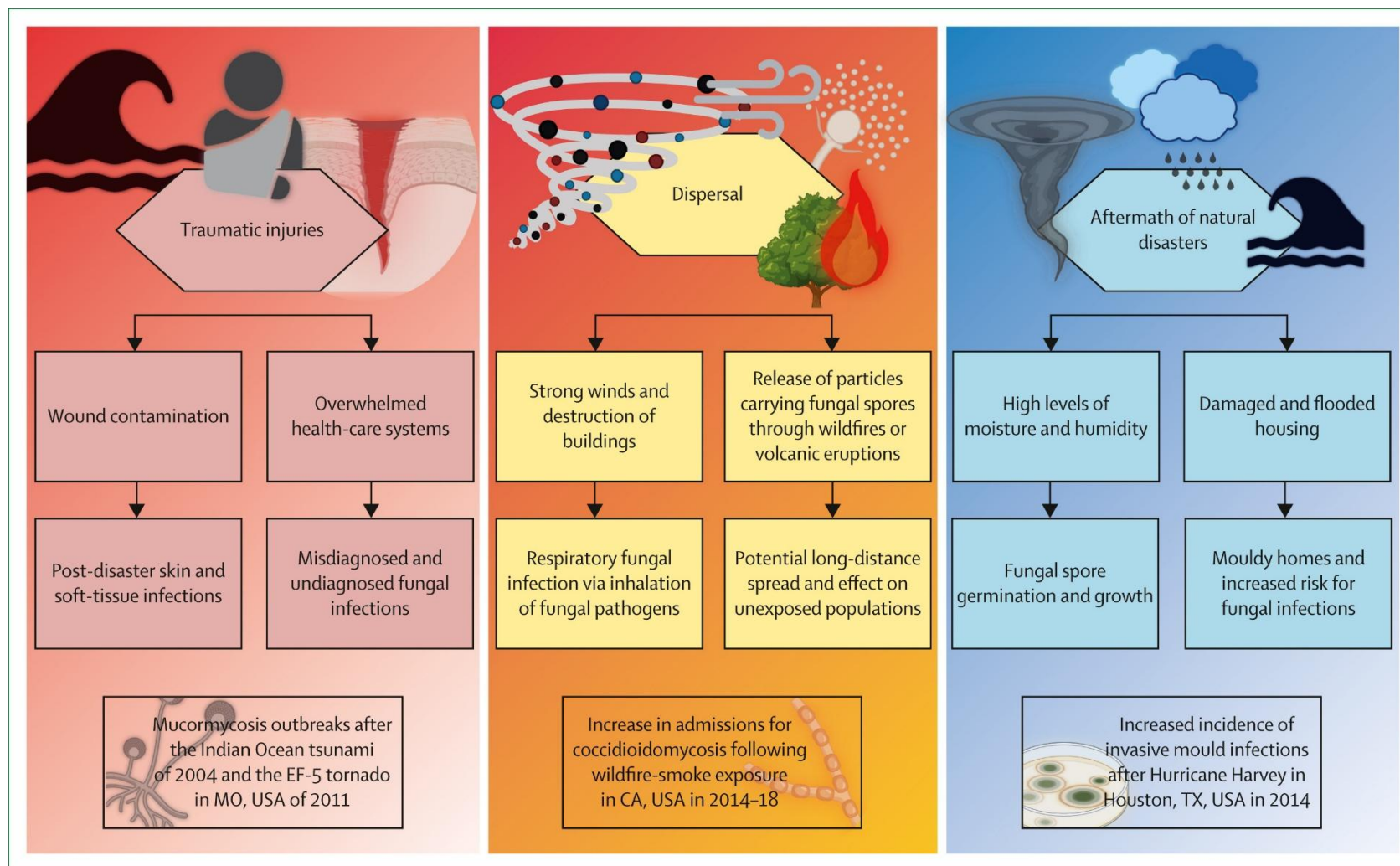


Figure 2: Schematic summarising mechanisms by which natural disasters trigger fungal outbreaks

Reproduced from Seidel et al. (2024).

2. FLOODING EVENTS

The main natural disasters or extreme weather events associated with flooding are tsunamis, cyclones (also referred to as hurricanes or typhoons depending on where the storm system develops), and extreme rainfall. During flooding events, and in their immediate aftermath, there are three major routes of exposure to fungal pathogens: aspiration of flood waters, contamination of traumatic wounds, and exposure to mould growing on water-damaged building materials. These three routes will be discussed below, drawing on details from published examples (summarised in Table 1).

2.1. ASPIRATION

During flooding events, one of the main routes for infection by pathogenic fungi is aspiration of floodwaters by near drowning victims. Floodwaters are often contaminated with a variety of materials including soil, sewage and other anthropogenic substances such as oil or petrol. As such, these waters contain a wide variety of microbial species capable of causing disease, and most cases of aspiration pneumonia are polymicrobial, meaning multiple different microbial species are present (Kawakami et al., 2012). Infections involving members of the fungal genus *Scedosporium* are becoming increasingly recognised as important, and often life-threatening, complications in near drowning victims due to their ability to cause invasive disseminated infections in otherwise healthy individuals (Garzoni et al., 2005; Nakadate et al., 2012; Nakamura et al., 2013; Nakamura et al., 2011; Shimizu et al., 2014).

Scedosporium spp. are ubiquitous in the environment and are frequently resistant to multiple antifungal agents (Cortez et al., 2008). Under normal circumstances, most *Scedosporium* infections occur in immunocompromised individuals (Cortez et al., 2008). However, in the case of near drowning victims, they are often immunocompetent. As such, infections in near drowning victims are frequently initially misdiagnosed. For example, a 33-year-old victim of the 2011 Japanese tsunami with aspiration pneumonia was initially treated with antibiotics but died 17 days after admission from necrotising pneumonia and lung haemorrhage (Nakadate et al., 2012). It was not until after her death that *Scedosporium* was identified in bronchoalveolar lavage (BAL) fluid (fluid obtained using a bronchoscope to deliver a small amount of liquid into the lungs, which is then recollected and used for analysis).

Where infections spread to other locations in the body, symptoms may not develop until after the symptoms of the near drowning have apparently resolved. For example, another victim of the Japanese tsunami who was initially treated with broad-spectrum antibiotics for tsunami-related aspiration pneumonia (also referred to as ‘tsunami lung’) and discharged after three weeks hospitalisation, represented at hospital with a 1-month history of severe lower back pain which had started immediately after his initial discharge (Shimizu et al., 2014). The treating physicians suspected a bacterial spine infection and started antibiotics. The pain worsened over the following month and eventually endoscopic debridement (removal of dead or infected tissue), and irrigation of the intervertebral disc was performed. It was only when intraoperative cultures grew *S. apiospermum* that the fungal cause of infection was discovered.

Table 1: Flood-related fungal infections

	Event	Date	Location	Cases (Age/Sex)	Description	Reference
TSUNAMI	Boxing Day Indian Ocean tsunami	26 Dec 2004	Indonesia	1 (27/F)	Osteomyelitis due to <i>S. apiospermum</i> identified > 4 years after traumatic knee injury obtained during the tsunami	Angelini et al. (2013)
			Sri Lanka	1 (56/M)	Necrotising soft tissue wound infection caused by <i>Apophysomyces elegans</i>	Andresen et al. (2005)
			Thailand	1 (59/M)	Spondylodiscitis ~ 4 months after near drowning due to <i>S. apiospermum</i>	Garzoni et al. (2005)
				1 (51/F)	Brain abscesses due to <i>S. apiospermum</i> , developed ~ 8 weeks after hospitalisation for traumatic wounds and pneumonia	
				not specified	<i>Aspergillus fumigatus</i> and <i>Candida</i> infections of traumatic wounds	Maegele et al. (2005)
				not specified	Infection of wounds with <i>A. fumigatus</i> , <i>Fusarium solani</i> and <i>Mucor</i> species	Maegele et al. (2006)
				2 (61/M; 59/F)	Infection of intact skin close to the site of severe soft tissue wounds with <i>Cladophialophora bantiana</i>	Petrini et al. (2006)
				1 (F)	Necrotising wound infection caused by <i>A. elegans</i>	Snell and Tavakoli (2007)
	Great East Japan tsunami	11 Mar 2011	Japan	1 (59/F)	Lung and brain abscesses caused by <i>S. apiospermum</i> after near drowning	Nakamura et al. (2011)

	Event	Date	Location	Cases (Age/Sex)	Description	Reference
TSUNAMI	Great East Japan tsunami	11 Mar 2011	Japan	1 (73/F)	Fungal sinusitis and meningitis subsequent to aspiration pneumonia	Igusa et al. (2012)
				1 (68/F)	<i>A. fumigatus</i> lung infection subsequent to near drowning which disseminated throughout the body resulting in death	Kawakami et al. (2012)
				1 (33/F)	<i>S. apiospermum</i> and <i>Scedosporium prolificans</i> detected in BAL fluid from a patient who died 17 days post tsunami from massive lung haemorrhage	Nakadate et al. (2012)
				1 (68/F)	Lung and brain infection caused by <i>Scedosporium aurantiacum</i> in victim of near drowning	Nakamura et al. (2013)
				1 (45/M)	Vertebral osteomyelitis due to <i>S. apiospermum</i> discovered 1 month after near drowning	Shimizu et al. (2014)
CYCLONE	Hurricane Katrina	Aug 2005	Florida, Louisiana, and New Orleans, USA	1 (26/M)	Exposed while working in hurricane-impacted fields. Systemic fungal infection with multiple brain lesions, bilateral lung infiltrates and spreading facial rash positive for <i>Blastomyces dermatitidis</i>	Szedler et al. (2007)
				8 (Sep 2005 – Jan 2006)	<i>Syncephalastrum</i> identified in sputum, bronchoalveolar lavage, endotracheal aspirate, ear and nasal swabs of patients with variable mould exposure post Katrina. Transient colonisation with no evidence of infection	Rao et al. (2007)

	Event	Date	Location	Cases (Age/Sex)	Description	Reference
CYCLONE	Hurricane Katrina	Aug 2005	Florida, Louisiana, New Orleans, Cayman Islands	1 (25/F)	Exposed while working in a hurricane-damaged classroom. Painless vision loss in left eye post Katrina. Necrotic fungal mass removed from the sinus cavity; contained <i>Drechslera</i>	Sridhar et al. (2012)
				1 (58/F)	Exposed during cleaning of hurricane-damaged hotel rooms. History of left-sided headache and double-vision. Necrotic mass containing fungal hyphae removed from the sinus cavity	
				1 (64/M)	Exposed while working as an inspector in mould-infested trailers. History of vision loss in the right eye and right-sided headache. Fungal hyphae found surrounding the right optic nerve and in the sinus cavity	
	Hurricane Ike	Sep 2008	Texas, USA; Cuba	3 (60/M; 64/M; 74/M)	Three cases of chromoblastomycosis – cutaneous or subcutaneous fungal skin infections generally caused by implantation of spores beneath the skin following trauma involving plant material (eg, thorns, splinters)	Riddel et al. (2011)

	Event	Date	Location	Cases (Age/Sex)	Description	Reference
CYCLONE	Hurricane Sandy	Oct 2012	New York, USA	5	Nosocomial outbreak of polymicrobial fungal infections in patients in a burn intensive care unit potentially related to water and air infiltration during Hurricane Sandy	Sood et al. (2018)
	Hurricane Harvey	Aug 2017	Houston, Texas, USA	5	Acute invasive mucormycoses: two diabetic patients with rhino-cerebral infections (both fatal); cystic fibrosis patient with visceral mucormycosis; two liver transplant patients with severe cutaneous infections	Kueht et al. (2019)
				not specified	Increase in fungal infections in immunocompromised cancer patients in Hurricane Harvey-affected counties	Wurster et al. (2022)
				not specified	Increase in patients presenting with invasive fungal infections post Hurricane Harvey (2.50 cases/10,000 healthcare encounters in 12 months pre-Harvey vs 3.69 cases/10,000 post-Harvey)	Toda et al. (2023)
EXTREME RAINFALL		Sep 2006	Thailand	25	Superficial skin infections - <i>Trichosporon mucoides</i> and non-spore forming hyaline fungi identified by culturing	Vachiramon et al. (2008)
		July 2011 – Jan 2012	Thailand	1	Skin legions caused by <i>Aspergillus flavus</i>	Thongtaeparak et al. (2016)
		Sep 2013	Colorado, USA	4	Four cases of rhino-orbital-cerebral mucormycosis (<i>Rhizopus oryzae</i> / <i>R. arrhizus</i>) within 1 month of flooding, two cases fatal	Davies et al. (2017)

A similar pattern was seen for another Japanese tsunami victim who initially presented with aspiration pneumonia and was treated with antibiotics (Nakamura et al., 2013). The patients' condition initially improved but by day 26 worsened with computed tomography (CT) showing multiple lesions in both lungs. High levels of the fungal marker β -D-glucan were detected in clinical samples, suggestive of a fungal infection so antifungal treatment was started. After 42 days the patient was transferred to another hospital and remained symptomatic despite a decrease in β -D-glucan levels, so antibiotics were restarted. Bronchoalveolar lavage fluid taken on day 2 at the new hospital was found to contain fungal hyphae, so the antifungal treatment was continued. However, by day 16 the patient had developed right-sided paralysis and β -D-glucan levels had increased. By day 29 the patient lost consciousness and brain magnetic resonance imaging (MRI) revealed multiple abscesses. By day 45, high levels of β -D-glucan levels were detected in cerebrospinal fluid. On day 62, 60 days after BAL samples were collected, PCR analysis identified *S. aurantiacum*. Fortunately, the patient was successfully treated with antifungal agents.

This spread of *Scedosporium* to the brain was also reported for another victim of the Japanese tsunami who initially presented with respiratory failure due to aspiration pneumonia (Nakamura et al., 2011). Whilst her initial head CT on day 5 was normal, a brain MRI on day 63 identified multiple abscesses eventually attributed to *S. apiospermum*. Importantly, although filamentous fungi were observed in BAL fluid on day 28, PCR diagnosis of *S. apiospermum* was not obtained until day 69. As *Scedosporium* species are often resistant to multiple antifungal agents, early identification of the cause of infection will play an important role in selecting the best antifungal treatment.

In a third reported case of disseminated brain infection by *Scedosporium* in a near drowning victim of the Japanese tsunami, the patient was less fortunate, succumbing to the infection (Igusa et al., 2012). In this case, the patient was initially diagnosed with aspiration pneumonia and antibiotics started. However, symptoms persisted and 21 days after the tsunami she was transferred to another hospital where a brain CT identified lesions in the sinus cavities. Testing of BAL fluid identified antibiotic-resistant *E. coli*, so the antibiotic treatment was changed, leading to improvement of the respiratory symptoms but the fever persisted, and the patient had a low level of consciousness. A repeat brain MRI showed the lesions had spread, and antibiotic-resistant *E. coli* was detected in nasal samples. However, the cerebrospinal fluid contained β -D-glucan, suggestive of fungal rather than bacterial meningitis. A combination of antifungal agents was started but after 12 days the patient died.

It is important to note that these disseminated *Scedosporium* infections are not unique to the Japanese tsunami. For example, a victim of the 2004 Boxing Day tsunami successfully treated for aspiration pneumonia re-presented one month after discharge with back pain, revealed to be due to spondylodiscitis (infection of the intervertebral disc and adjacent vertebrae) caused by *S. apiospermum* (Garzoni et al., 2005). In this case, the patient was successfully treated with antifungal agents.

It is also important to note that *Scedosporium* spp. are not the only fungal species capable of causing invasive infections in near drowning victims, with another Japanese tsunami near drowning victim succumbing to an *Aspergillus fumigatus* infection (Kawakami et al., 2012). In this case, as for many discussed above, fungal infection was not initially considered as the patient was immunocompetent, so no initial testing for β -D-glucan was performed. *Aspergillus* pneumonia was not diagnosed until day 13, based on the result of a sputum

culture from day 6 and slightly elevated β -D-glucan levels on day 10. Antifungal treatment was started but the patient eventually died on day 18.

2.2. INFECTION OF TRAUMATIC WOUNDS

Another key route for infection by fungal pathogens during flooding events is via traumatic wounds. Invasive infections of wound sites with fungal species of the order Mucorales (mucormycoses), which includes *Apophysomyces* and *Rhizopus* species (Skiada et al., 2020), have been identified in victims of the 2004 Boxing Day tsunami. For example, a victim with a soft-tissue wound on her right buttock rapidly developed necrosis, requiring surgical debridement (Snell & Tavakoli, 2007). Despite antibiotic treatment, the patient worsened, becoming septic and requiring intensive care. A week later, *Apophysomyces elegans* was identified in tissue cultures. The patient was then successfully treated with amphotericin for 42 days, coupled with further surgical debridement and topical negative pressure dressings. The authors emphasised that early identification of the causative agent of necrosis is important, so that an appropriate course of treatment can be chosen (Snell & Tavakoli, 2007). A second tsunami victim developed a necrotising *A. elegans* infection at the site of a large thigh wound and in two other non-contiguous superficial abrasion sites (Andresen et al., 2005). The thigh wound was initially debrided, and antibiotics were started. By day five, the two abrasion sites had developed necrosis. The wounds were debrided and fungal hyphae consistent with cutaneous mucormycosis were identified in the removed tissue. Antifungal treatment was started as was hyperbaric oxygen therapy. Eventually *A. elegans* was identified in cultures from the debrided tissue. The authors noted that diagnosis of cutaneous mucormycosis based on just clinical observations is difficult and normally requires tissue biopsy so that histology and culturing can be performed. They also noted that “the best outcomes from cutaneous mucormycosis have been associated with early detection, aggressive surgical debridement, early use of amphotericin B, and correction of predisposing factors” (Andresen et al., 2005). Importantly, they highlight that “mucormycosis and other invasive fungal infections should be considered in survivors of natural disasters whose wound infections are progressive despite antibacterial treatment and debridement” (Andresen et al., 2005).

Scedosporium species have also been found to infect traumatic wounds of tsunami survivors, and similar to what has been reported for some near drowning victims above, these infections may not become apparent until some time after admission. For example, another Boxing Day tsunami victim with deep cutaneous wounds colonised by multi-drug-resistant bacteria, developed a brain infection caused by *S. apiospermum* which was not detected until eight weeks after hospitalisation (Garzoni et al., 2005). Although the patient eventually recovered, they required more than six months of antifungal treatment. In a second example, Angelini et al. (2013) detail the case of a patient suffering from a bone infection (osteomyelitis) caused by *S. apiospermum*, in which the causative agent was not identified until more than four years after a traumatic knee injury obtained during the 2004 Boxing Day tsunami. The initial injury required surgical intervention to repair ruptured tendons, which was completed around one month post injury. Over the following two-year period the patient complained of chronic mild pain, but no major issues were seen by x-ray or MRI. A follow-up MRI a year later found a cyst had developed but after its removal, no microbial growth was detected in surgical cultures. Two-and a half years later, an open

biopsy of the knee recovered fungal tissue identified as being from a *Scedosporium* species. Antifungal treatment was started, and symptoms initially improved but then over the following six-months deteriorated. *Scedosporium apiospermum* was finally identified from cultures grown from fine-needle aspirate. Surgical debridement of infected tissue was performed, and the antifungal treatment was changed to a more aggressive approach, with the patient on antifungals for more than a year. This again highlights the importance of early diagnosis of fungal infection and correct species identification so that an effective antifungal treatment approach can be chosen.

Cladophialophora bantiana, a rare but virulent human pathogen most well-known for causing brain abscesses (Lortholary et al., 2024) has also been identified in invasive wound infections from two survivors of the Boxing Day tsunami (Petrini et al., 2006). Both patients had severe soft tissues wounds which required multiple surgeries and skin grafts. Four to six weeks after the tsunami the patients developed purulent infections in the non-traumatised intact skin outside of the skin graft margins. Culture analysis of biopsies taken from the infected regions identified *C. bantiana* and *Mycobacterium abscessus*. The patients were treated for one month with antifungal agents chosen based on known sensitivity of *C. bantiana*, after which skin biopsies no longer contained fungus. This highlights how careful selection of an antifungal agent, taking into consideration known sensitivities results in a good clinical outcome.

Infections of traumatic wounds of tsunami victims with a range of other fungal species have also been reported, including *A. fumigatus*, *F. solani* and *Candida* species (Maegele et al., 2005; Maegele et al., 2006). Fungal wound infections have also been reported for three individuals who suffered minor skin trauma whilst cleaning up brush and downed trees in the aftermath of Hurricane Ike (Riddel et al., 2011). All three developed chromoblastomycosis, a cutaneous/subcutaneous fungal skin infection generally caused by implantation of spores beneath the skin following trauma involving plant material.

In addition to infections related to traumatic wounds, superficial fungal skin infections have also been reported following exposure to floodwaters during extreme rainfall events. For example, 25 people reportedly suffered from superficial fungal skin infections after flooding in Thailand in 2006 (Vachiramon et al., 2008), and another person developed skin lesions caused by *Aspergillus flavus* after exposure to floodwaters during flooding in Thailand in 2011 (Thongtaeparak et al., 2016).

2.3. MOULD PROLIFERATION IN INDOOR ENVIRONMENTS

The third potential route for fungal infections following flooding events relates to increased exposure in the aftermath of these events due to fungal proliferation on water damaged surfaces. Several reports have detailed an increase in invasive fungal infections after flooding events. For example, Toda et al. (2023) reported that the incidence of invasive fungal infections in Houston, Texas post Hurricane Harvey increased significantly from 2.50 to 3.69 cases per 10,000 healthcare encounters. The most common fungal species identified were *Aspergilli*, followed by *Fusarium* and *Penicillium* species. Multiple cases of fungal sinus infection were reported in individuals working in Hurricane-damaged buildings after Hurricanes Katrina and Wilma (Sridhar et al., 2012). The first case involved a schoolteacher

who had been working in a hurricane damaged classroom and developed vision loss in her left eye over a 10-month period. A large mass in the sinus cavity was identified by MRI, and on examination after surgical removal it was found to contain necrotic material and fungal hyphae, with *Drechslera* identified by culturing. The patient was treated with antifungal medications, but allergic fungal sinusitis (AFS) persisted. Unfortunately, the patient did not seek further treatment, so the outcome of this case is unclear. The second case involved a woman who had worked cleaning Hurricane-damaged hotel rooms. Around eighteen months later she developed left-sided headache and six months later sought treatment as it progressed to double vision. An MRI revealed a large mass in the sinus cavity, and a large necrotic mass containing fungal hyphae was removed. The patient was treated with a one-year course of antifungal medication and at a two-year checkup her symptoms had resolved, although it was noted that there was residual sinus disease. In the third case, a Federal Emergency Management Agency (FEMA) employee who worked inspecting mould-infested trailers after Hurricane Katrina developed vision loss in his right eye. An MRI identified a mass in the sinus cavity which on removal was found to contain fungal hyphae. Although he was given antifungal treatment, two months later the infection was more extensive. The patient declined surgery to remove the mass so instead received a combination of antifungal agents intravenously for 10 months, and his symptoms gradually improved.

A separate report by Davies et al. (2017), documented four cases of rhino-orbital-cerebral mucormycosis in Colorado in 2013 following a widespread flooding event. All four cases involved immunocompromised individuals. The first, a man suffering from acute myelogenous leukaemia, presented with sinus disease, presumed to be invasive fungal sinusitis and antifungal treatment targeted at mucormycosis was started. *Rhizopus oryzae* (*R. arrhizus*) was cultured from tissue debrided from the sinus. Although the patient was treated with aggressive surgical debridement and antifungal therapy, he unfortunately died 11 days after admission. The remaining three cases who all had poorly controlled diabetes, developed fungal sinusitis due to *R. oryzae*. One patient recovered after multiple stages of radical debridement and resection of necrotic soft tissue, combined with antifungal medications, but required major facial reconstruction. In another of the patients, the infection spread to the cerebrum, and he succumbed to the infection. The final patient, recovered after radical surgical removal of infected tissue and antifungal treatment, but was to remain on the antifungal medication for life.

An increase in fungal infections in immunocompromised individuals has also been reported after hurricane Harvey, with one study showing that the number of immunocompromised cancer patients from counties affected by the hurricane with mould-positive cultures doubled from 36 to 67 post hurricane (Wurster et al., 2022). Of the 67 patients with positive mould cultures post Harvey, 34 had probable/confirmed invasive fungal infections versus 23/36 pre-Harvey. Another study reported that at one hospital, five immunocompromised patients presented with acute invasive fungal infections in the month after hurricane Harvey (Kueht et al., 2019). Two patients who both suffered from diabetes were diagnosed with rhino-cerebral infections, which in both cases proved fatal. A third patient who suffered from cystic fibrosis had visceral mucormycosis of the stomach which required a gastrectomy (removal of the stomach). The remaining two patients who were both liver transplant recipients were diagnosed with cutaneous mucormycosis. The first developed this infection at the site of a leg IV around 16 days post-surgery. The wound required serial debridement, antifungal treatment and an eventual skin graft, with antifungal treatment scheduled for a 1-year

duration post discharge. The second case, who in addition to the liver transplant, had diabetes and hepatitis, presented with a cutaneous abscess on his chest wall two days prior to the liver transplant. The abscess was drained and the tissue debrided but the infection progressed to a wound 430 cm². The patient was successfully treated with antifungal therapy including use of a biologic skin substitute impregnated with amphotericin and discharged after three months with ongoing oral antifungal treatment.

A report by Sood et al. (2018) also highlighted the potential for fungal disease outbreaks in healthcare facilities that have experienced water damage during flood-related events, with a polymicrobial fungal outbreak occurring in a burn intensive care unit temporally associated with air and water permeation into the facility during Hurricane Sandy.

Whilst the studies discussed above have highlighted the potential for fungal infections as a result of increased exposure after flooding events, caution must be exercised to distinguish colonisation from true infection (Eid et al., 2024). Indeed, a study by Rao et al. (2007) highlighted the presence of *Syncephalastrum* in BAL fluid, sputum, ear and nose swabs and endotracheal aspirate of eight patients with variable mould exposure post hurricane Katrina, all of whom displayed no evidence of infection, suggesting the patients were transiently colonised by, rather than infected with *Syncephalastrum*.

Additionally, it should be noted that infections related to increased fungal proliferation in the aftermath of flooding events are not restricted to indoor exposure, with a farmer working in fields after Hurricane Katrina developing a systemic *Blastomyces dermatitidis* infection (Szeder et al., 2007). The patient initially presented with a three-month history of headache, cough (including coughing up blood), fever, fatigue, reduced cognitive function and a facial rash. A chest x-ray revealed infiltration in both lungs, and on suspicion of fungal infection, antifungal treatment was started. Fungal infection was confirmed by skin biopsy. A brain MRI showed multiple lesions prompting a change in antifungal treatment to one more optimal for central nervous system infection. The patient was discharged on an antifungal treatment regime and at a four-month follow-up symptoms had resolved, and the brain lesions had reduced in size. The patient remained on antifungal treatment for one year.

3. DISPERSAL-RELATED EVENTS

The main natural disasters or extreme weather events associated with dispersal of fungal propagules are tornadoes, earthquakes, landslides, dust storms and wildfires. During, and in the immediate aftermath of these events the main routes for infection are inhalation of aerosolised fungal propagules (eg, spores, hyphal fragments) and contamination of traumatic wounds. These two routes will be discussed below, drawing on details from published reports, summarised in Table 2.

3.1. INHALATION OF FUNGAL PROPAGULES

An increase in fungal respiratory infections has been reported after several natural disasters during which environmental dust was aerosolised. For example, a report by Forshee-Hakala (2015) documented an increase in cases of pneumonia in the town of Joplin Missouri, USA in the month after a devastating EF-5 tornado in May 2011. A comparison of the microbial species isolated from sputum of pneumonia patients prior to and after the tornado revealed the presence of several atypical pathogens most commonly found in decaying vegetation, soil and water – including *Aspergillus* species.

An increase in respiratory illness was also documented after the 2011 Japanese earthquake, with numerous people suffering from chronic cough (Ogawa et al., 2012). Culturing of sputum from six of these patients identified *A. fumigatus*, *A. flavus* and basidiomycetous fungi. These same species were also cultured from the floor of a refuge where many people were staying after the disaster. The authors hypothesised that environmental fungi dispersed into the air due to the collapse of houses were responsible for the respiratory symptoms displayed by these residents.

Pappagianis et al. (1994) also documented an increase in fungal respiratory infections after an earthquake, with 170 people diagnosed with coccidioidomycosis (also known as Valley fever) in Ventura County, California, USA in the two months after the 1994 Northridge earthquake. This greatly exceeded the 52 cases diagnosed during the whole of 1993. *Coccidioides* species live in soil and produce resistant spores which can become dispersed in the air by events which result in aerosolisation of soil particles. As such, it was hypothesised that the Northridge earthquake caused an increase in airborne dust, which was laden with *Coccidioides* spores, thereby increasing exposure of residents to this pathogen. Another report noted that the majority of cases occurred in Simi Valley, which is located at the base of a mountain and had experienced several landslides during the earthquake, with the amount of time residents spent in a dust cloud significantly affecting their risk of acute coccidioidomycosis (Schneider et al., 1997). An increase in cases of coccidioidomycosis has also been reported after dust storms in California (Flynn et al., 1979; Williams et al., 1979). Although a more recent study of data from 2006 – 2020 found no consistent link between dust storms and coccidioidomycosis (Comrie, 2021). It is important to note, however, that *Coccidioides* species responsible for Valley fever, *C. immitis* and *C. posadasii*, are not present in New Zealand (Figure 3). Although, like many other fungi, the geographic spread of this pathogen is likely to increase due to climate change (Wadman, 2025).

Table 2: Reported fungal infections associated events which lead to dispersal of fungal propagules

	Event	Date	Location	Cases (Age/Sex)	Description	Reference
TORNADOES	Joplin EF-5 tornado	22 May 2011	Joplin, Missouri, USA	2 (16/M; 13/F)	Infection of traumatic wounds with <i>Apophysomyces trapeziformis</i> (other fungi also present)	Weddle et al. (2012)
				4 (50/F; 48/M; 17/M; 75/F)	Necrotising infections of traumatic wounds with <i>A. trapeziformis</i> . One case fatal	Austin et al. (2014)
				NS	<i>Aspergillus</i> present in sputum of pneumonia patients only post-tornado potentially due to aerosolisation of soil-associated fungal propagules	Forshee-Hakala (2015)
DUST STORMS	Great Bakersfield dust storm	Dec 1977	San Joaquin Valley, California, USA	18	Increase in cases of symptomatic coccidioidomycosis diagnosed at the Naval hospital, Lemoore, California in the 2 – 4 weeks following the dust storm	Williams et al. (1979)
				550	550 cases of coccidioidomycosis reported to the California Department of Health Services in the first 16 weeks of 1978 cf. maximum of 175 cases for the same period over the previous 10 years	Flynn et al. (1979)
WILDFIRES		2014 – 2018	California, USA	not specified	Retrospective study of 22 Californian hospitals showing 20% increase in admission for coccidioidomycosis in the month following wildfire smoke exposure	Mulliken et al. (2023)
		2017	California, USA	10	Cluster of cases of coccidioidomycosis in California state prison inmate wildland firefighters	Laws et al. (2021)
		2021	California, USA	7	Cluster of cases of coccidioidomycosis in wildland firefighters	Donnelly et al. (2022)

	Event	Date	Location	Cases (Age/Sex)	Description	Reference
EARTHQUAKE	Landslides associated with the Northridge earthquake	17 Jan 1994	Ventura County, California, USA	203	Large outbreak of coccidioidomycosis likely related to dust clouds generated during landslides associated with the Northridge earthquake	Schneider et al. (1997)
	Northridge magnitude 6.7 earthquake			170	Increase in acute coccidioidomycosis following the earthquake, likely due to inhalation of airborne dust	Pappagianis et al. (1994)
	Marmara magnitude 7.4 earthquake	17 Aug 1999	Northwest Turkey	not specified	<i>Candida albicans</i> isolated from wounds of crush injury patients	Kazancioglu et al. (2002)
				not specified	<i>C. albicans</i> isolated from blood cultures of two crush victims and from sputum of a crush victim suffering with pneumonia	Keven et al. (2003)
	Great Sichuan magnitude 8.0 earthquake	12 May 2008	Sichuan province, China	56	<i>C. albicans</i> isolated from various clinical samples from earthquake victims	Kang et al. (2009)
				not specified	<i>C. albicans</i> isolated from wounds	Tao et al. (2009)
				not specified	<i>C. albicans</i> isolated from sputum and blood of earthquake injury victims	Wang et al. (2010)
	Hispaniola magnitude 7.0 earthquake	14 Jan 2010	Haiti	not specified	Various <i>Candida</i> species isolated from clinical samples of child victims	Arquès et al. (2013)
	Chilean magnitude 8.8 earthquake	27 Feb 2010	Central Chile	6	<i>Paecilomyces variotii</i> peritonitis in dialysis patients potentially linked to dust contamination of dialysis bags	Torres et al. (2014)
	Japanese magnitude 9.0 earthquake	11 Mar 2011	Eastern Japan	6	<i>A. fumigatus</i> , <i>A. flavus</i> and basidiomycetous fungi cultured from sputum of survivors with chronic, non-productive cough	Ogawa et al. (2012)

NS, not specified.

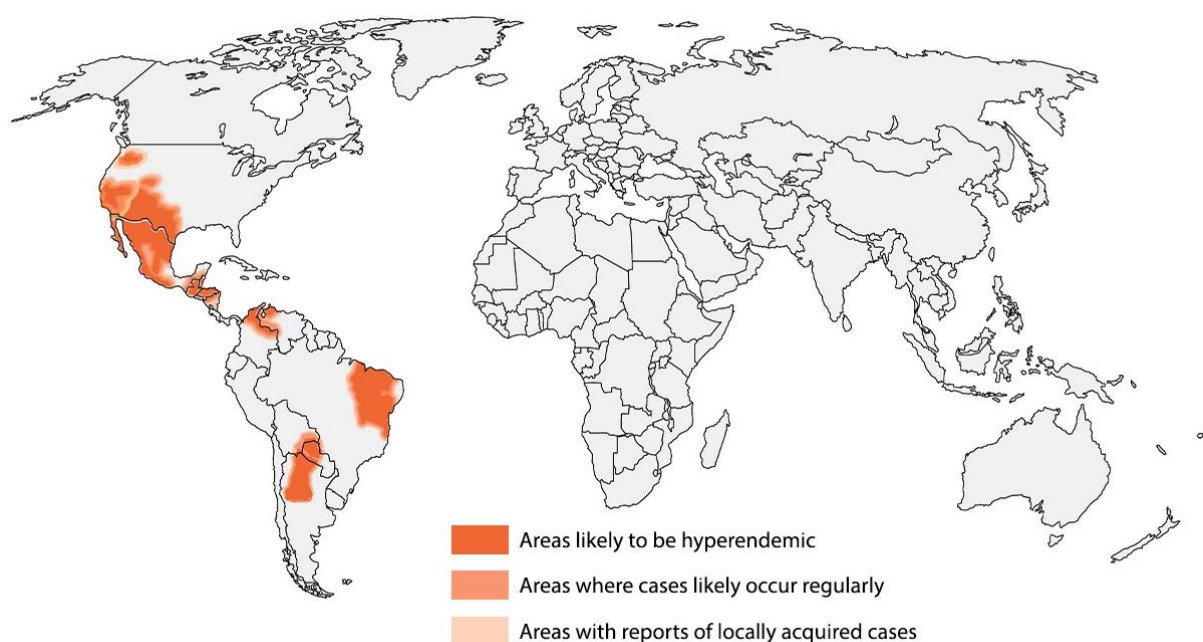


Figure 3: Map showing areas where *Coccidioides* species responsible for Valley fever are found

Reproduced from <https://www.cdc.gov/valley-fever/areas/index.html>

An increase in cases of coccidioidomycosis has also been reported in wildland firefighters, potentially due to increased dust exposure whilst digging and moving soil (Donnelly et al., 2022; Laws et al., 2021). However, this may not be the sole cause, as hospital admissions for coccidioidomycosis at 22 hospitals in California were found to increase by 20% in the month following wildfire smoke exposure, with a 2% increase for each day of smoke exposure in the prior month (Mulliken et al., 2023). This suggests that *Coccidioides* spores may be transported in wildfire smoke. Interestingly, there was no similar pattern observed for hospital admissions for aspergillosis. This study is not the first to suggest microbial cells may be transported in wildfire smoke. A 2004 study showed that viable spores of several fungal genera could be transported in smoke from biomass fires (Mims & Mims, 2004). Another study demonstrated that concentrations of fungal spores in the air peak around 10 days after the fire event (Camacho et al., 2018). This study also noted that “it is likely that fire-induced convections promote release of fungal spores from their natural habitat and that the soil heating conditions could trigger the germination of fungi colonising post-fire plant residues and soil” (Camacho et al., 2018). A more recent study also highlighted the potential for wildfire smoke to be infectious, consisting of a bioaerosol of fungal and bacterial cells (Kobziar & Thompson, 2020). This study also simulated the transport of microbial cells < 2.5 µm in size in wildfire smoke from a fire in Utah, as can be seen in Figure 4. Indeed, wildfire smoke has been found to contain four times as many microbial cells compared to background air, with around 78% predicted to be viable (Kobziar et al., 2022). A wide range of fungal species were identified in the smoke, including *Cladosporium*, *Alternaria*, *Aureobasidium* and *Elasticomyces* species. It has been estimated that under typical conditions, a wildfire in the western United States emits around 3.71×10^{14} microbial cells per hectare (Kobziar et al., 2022).

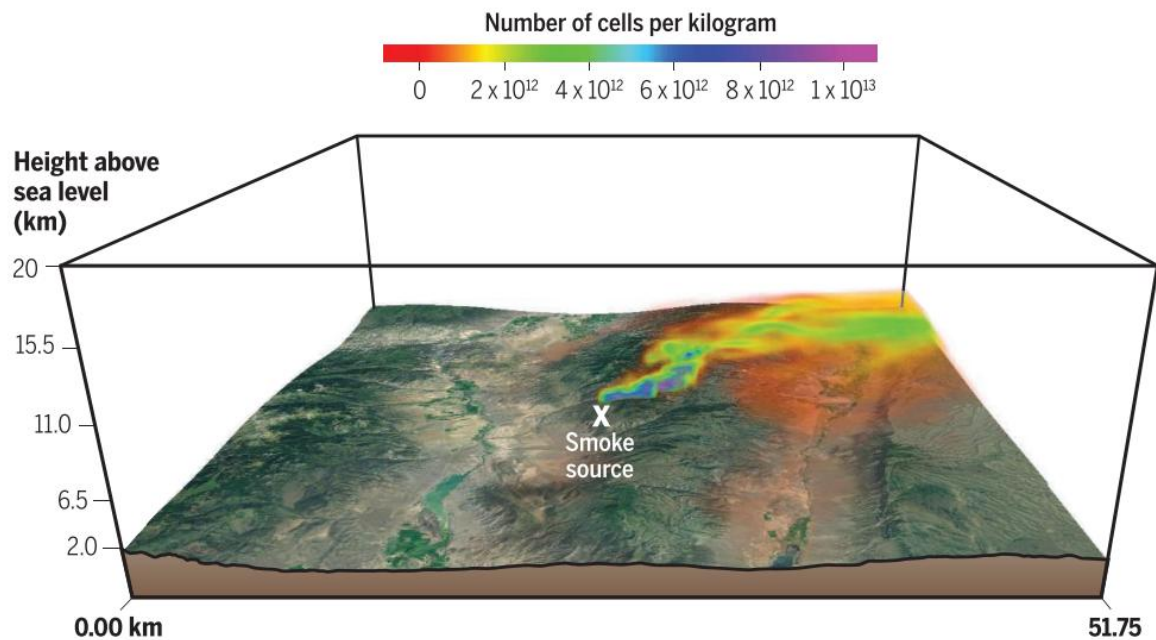


Figure 4: Simulated dispersal of microbial cells in a smoke plume
Reproduced from Kobziar and Thompson (2020).

3.2. INFECTION OF TRAUMATIC WOUNDS

The second key route for fungal infection during dispersal-related events is infection of traumatic wounds. Several cases of cutaneous mucormycosis were reported after the Joplin EF-5 tornado, prompting an onsite Center for Disease Control (CDC) investigation (Kaye, 2011). Overall, thirteen *A. trapeziformis* infections were identified, of which five proved to be fatal (Benedict et al., 2011). Specific details of six cases were published in two separate reports. Austin et al. (2014) documented four cases, noting that “onset of this fungal infection can occur without clinical signs, presenting several days to several weeks after injury, delaying diagnosis”. The first patient had a scalp wound which showed signs of infection on day 9 when necrosis at the edge of the wound became evident. The necrotic tissue was debrided, resulting in exposure of the cranium. Although *Zygomycete* fungi were identified in histology samples, the exact species was not initially identified. The wound continued to progress, requiring extensive debridement of the scalp, forehead and eyelid. Within 24 hours the infection progressed further with patches of fungal growth visible, and the patient died 16 days after their injury. Samples were sent to the University of Texas Health Science Center (UTHSC) and the CDC, from which *A. trapeziformis* was identified. The second patient had a flank laceration which displayed signs of infection and necrosis two days post admission. The wound was debrided and appeared to improve, so the patient was discharged on antibiotics. Three days later the wound became more painful, swollen and necrosis and discharge were visible. Multiple bacterial pathogens were identified from wound cultures, and 11 days post injury a *Zygomycete* infection was confirmed. The wound underwent several rounds of surgical debridement. *Apophysomyces trapeziformis* was identified in samples sent to UTHSC and the CDC. The patient was treated with antifungal therapy and eventually recovered but required skin grafts to cover the extensive wound. The third case

involved a teenage boy with multiple lacerations. Six days post admission he developed cellulitis around a leg laceration and underwent extensive debridement of the necrotic tissue, which was repeated twice. Sixteen days post injury *Zygomycete* fungi and *Pseudomonas aeruginosa* were identified in wound cultures. The patient was treated with antibiotics and antifungal medications and eventually recovered. *Apophysomyces trapeziformis* was later identified in samples sent to UTHSC and the CDC. In the fourth case, the patient developed *A. trapeziformis* infection at the site of a knee wound which had already been reopened and debrided several days after it was initially closed. The knee infection worsened and progressed to a large necrotic wound. The wound was extensively debrided, including removal of 'curd-like fungus-appearing material'. Nineteen days post injury, a *Zygomycete* infection was identified by pathology. The patient was treated with antifungal therapy and eventually recovered. *Apophysomyces trapeziformis* was again identified in tissue samples sent to UTHSC and the CDC.

A separate report by Weddle et al. (2012) documented two paediatric *A. trapeziformis* infections. The first involved infection at the site of a thoracotomy (surgical opening of the chest wall) used to insert hardware to stabilise rib fractures. The patient developed fever 10 days post-surgery and necrosis was visible at the thoracotomy site. The wound underwent several extensive rounds of debridement, but the infection and necrosis progressed. Overall, the patient underwent 12 separate rounds of debridement and was treated with a range of antifungal medications. Culturing of initial surgical samples identified *Candida tropicalis* and *A. fumigatus*, with *A. trapeziformis* not identified until day 14. After identification of *A. trapeziformis*, an additional antifungal medication was added. The patient eventually recovered and was discharged after three months. The second case involved infection of a scalp wound, which was initially irrigated and closed, with the patient on antibiotics. Ten days post injury the wound became red with purulent discharge. Treatment with the antifungal fluconazole, and two additional antibiotics was commenced, but the wound became necrotic and required debridement. Several bacteria and *Candida* species were cultured from tissue samples, with *A. trapeziformis* not identified until repeat cultures on day 14 post admission. This prompted addition of three more antifungal medications into the treatment regime. The patient eventually recovered after a total of 10 debridement surgeries and skin grafts.

Several reports have also documented fungal infections of traumatic wounds in earthquake victims, particularly involving *Candida* species. For example, Kang et al. (2009) and Tao et al. (2009) both isolated *C. albicans* from wounds of survivors of the 2008 Great Sichuan earthquake. A separate study also reported isolation of *C. albicans* from sputum and blood samples collected from survivors of the same earthquake (Wang et al., 2010). *Candida albicans* has also been isolated from wounds (Kazancioglu et al., 2002) and blood cultures (Keven et al., 2003) taken from crush syndrome victims of the 1999 Marmara earthquake in Northwest Turkey. In addition to *C. albicans*, *Candida tropicalis*, and *Candida glabrata* have also been isolated from samples collected from the wounds of child victims of the 2010 Haiti earthquake (Arquès et al., 2013).

4. VOLCANIC ERUPTIONS

Two reports of fungal infections in victims of volcanic eruptions were identified. The first of these pertains to the eruption of Nevado del Ruiz in Colombia in November 1985, which resulted in lahars (mudflows containing debris and volcanic material) that destroyed the town of Armero, killing over 23,000 people and injuring more than 4,500 (Patiño et al., 1991). Eight victims of the eruption with traumatic wounds subsequently developed necrotising fasciitis due to mucormycosis caused by *Rhizopus arrhizus* (Patiño et al., 1991). Of these eight infections, six proved fatal despite radical debridement of infected tissue.

The second report pertains to the 2019 Whakaari White Island eruption in which 47 people visiting the volcano, which was at the time a tourist attraction, were caught up in an eruptive event, initially leaving eight people dead and 31 with serious burns (Baker et al., 2022). Of these burn victims, two developed invasive *Aspergillus fumigatus* infections which ultimately resulted in their deaths on days 13 and 50 despite radical surgical debridement of infected tissue and treatment with systemic antifungal agents (Baker et al., 2022). Burn patients are at high risk for fungal infections due to the long period they spend without a skin barrier, combined with antibiotic treatment that removes the normal bacterial flora that would otherwise protect against fungal infection (Eid et al., 2024).

Both reports demonstrate the potential for life-threatening invasive fungal infections in victims of volcanic eruptions. Early diagnosis of these infections is crucial to the outcome of these infections (Patiño et al., 1991), as it will play an important role in deciding the course of treatment, especially given the high degree of antifungal resistance in many of these species.

Table 3: Reported fungal infections following volcanic eruptions and associated activity

Event name	Date	Region affected	People affected	Description	Reference
Mount Tolima lahar	13 Nov 1985	Nevado del Ruiz, Colombia	8	Severe necrotising fasciitis with mucormycosis (<i>Rhizopus arrhizus</i>); 75% of cases fatal	Patiño et al. (1991)
Whakaari White Island eruption	9 Dec 2019	Whakaari White Island, New Zealand	2	Invasive fungal infections of burn patients with <i>A. fumigatus</i> leading to death 13- and 50-days post eruption	Baker et al. (2022)

5. EQUITY CONSIDERATIONS

Vulnerable populations are disproportionately affected by natural disasters due to a range of factors including less access to healthcare, inadequate housing quality (including living in overcrowded housing) and increased likelihood of living in geographically vulnerable areas (George et al., 2025; Jenks et al., 2023) (Figure 5). They are also more likely to suffer from comorbidities, which increase their susceptibility to fungal infections (George et al., 2025). Climate change will further act to exacerbate these inequities (Seidel et al., 2024). This chapter will discuss these different equity factors as they pertain to New Zealand.

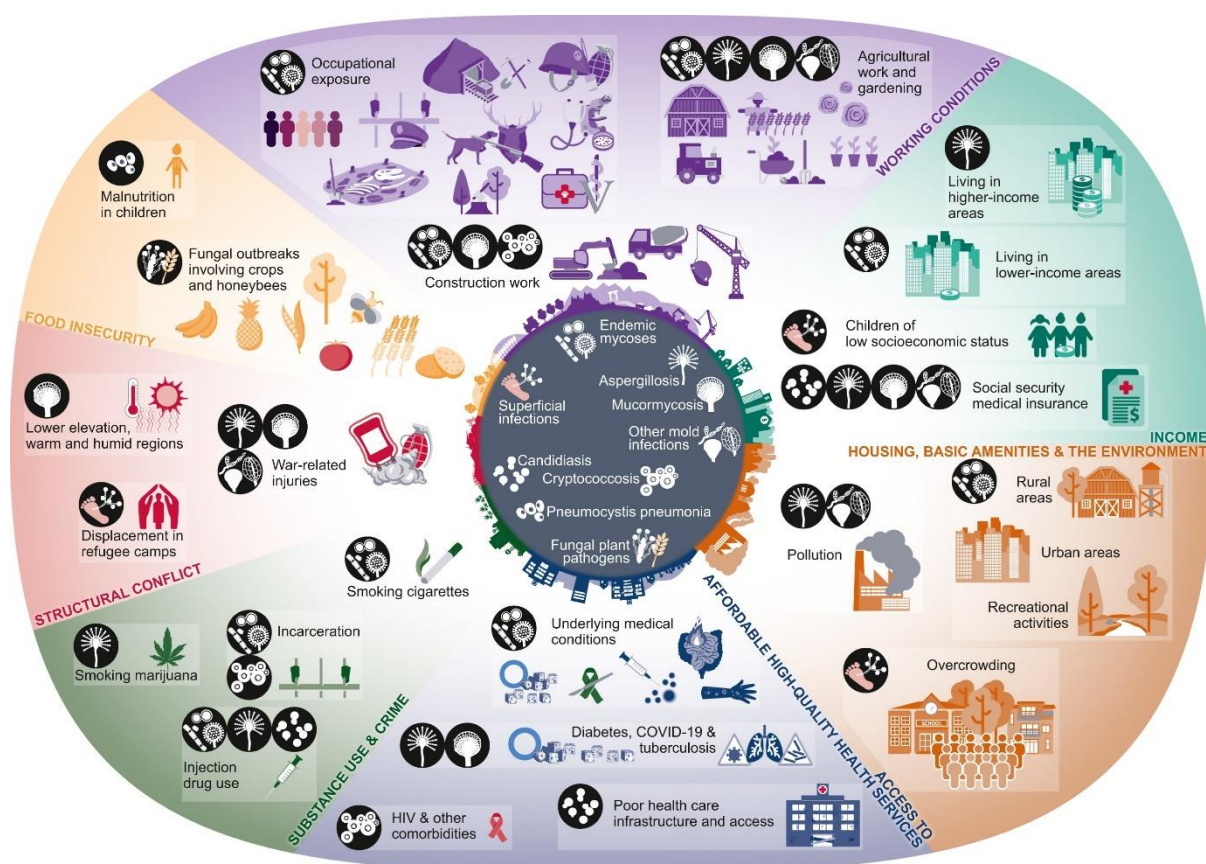


Figure 5: Socioeconomic factors influencing fungal infections

Reproduced from Jenks et al. (2023).

5.1. COMORBIDITIES

Several pre-existing medical conditions are known to increase the risk of fungal infections, including diabetes (Khanna et al., 2021), HIV, chronic respiratory disease, cancer, organ transplants and post-primary tuberculosis infection¹. Indeed, several of the clinical reports

¹ <https://www.who.int/news/item/25-10-2022-who-releases-first-ever-list-of-health-threatening-fungi>

Accessed 10 September 2025

documented herein highlighted poor outcomes of fungal infections in immunocompromised patients (including cancer and organ transplant patients) and those suffering from diabetes.

Inequities exist for many of these conditions in New Zealand, leading to increased risk of fungal infection for certain population subgroups after natural disasters or extreme weather events. For example, the prevalence of diabetes is roughly three times higher in Māori and Pacific peoples compared to Europeans (Teng et al., 2025). Māori are also more likely to suffer from diabetes-related complications (Harwood & Tipene-Leach, 2007). Rates of diabetes are also higher in the elderly population of New Zealand, with 16% of people 65 and older estimated to have diabetes, compared to only 8% of people aged 45 – 64, and 1.3% of people under 45². With regards to HIV, although its overall prevalence in New Zealand is low, a report by Saxton et al. (2022) noted that there are ‘profound inequities’ for gay and bisexual males. Inequities also exist for Māori, who are more likely to present with advanced HIV³.

There are also inequities with respect to chronic respiratory disease in New Zealand, with Māori and Pacific peoples known to have the highest respiratory health burden (Telfar-Barnard & Zhang, 2024). The prevalence of asthma is significantly higher for Māori compared to non-Māori, and rates of COPD (chronic obstructive pulmonary disease) are also higher for Māori and Pacific peoples compared to other ethnic groups (Telfar-Barnard & Zhang, 2024). Overall hospitalisation rates for respiratory disease are also known to be 2.2 times higher for the most deprived socioeconomic quintile in New Zealand compared to the least deprived (Telfar-Barnard & Zhang, 2024).

5.2. POOR QUALITY HOUSING

Several studies documented in this report have highlighted a link between exposure to mould growing in flood-damaged buildings and fungal infections. Dampness and mould are common in New Zealand homes, and there are stark inequities for different socioeconomic classes and ethnic groups (Stats NZ, 2020). For example, more than 40% of Pacific peoples and 30% of Māori are likely to live in homes with visible mould greater than A4 in size (at least some of the time), compared to only 17% of Europeans (Stats NZ, 2020). Lower income households are also more likely to live in mould affected housing compared to higher income groups (Stats NZ, 2020).

Thorough removal or remediation of water-damaged building materials and furnishings after flooding events is key to reducing exposure to mould, and in turn fungal infections. However, the extent to which this occurs is likely to be impacted by a households’ insurance coverage. It has previously been estimated that only 32% of Māori have house insurance compared to the New Zealand average of 48%, and only 40% have contents insurance compared to the average of 55%⁴. Lower income groups are also less likely to have house or contents insurance.

² <https://bpac.org.nz/2019/diabetes-elderly.aspx> Accessed 10 September 2025

³ <https://www.rnz.co.nz/news/national/571362/researchers-target-rising-hiv-diagnoses-among-maori> Accessed 10 September 2025

⁴ <https://retirement.govt.nz/news/latest-news/low-insurance-among-maori> Accessed 10 September 2025

5.3. GEOGRAPHICALLY VULNERABLE AREAS

People living in remote areas of New Zealand are some of the most vulnerable to natural disasters and extreme weather events, both due to their greater exposure to the elements (eg, low-lying coastal areas), and limited access to healthcare. It has been estimated that almost a third of the people living in the most remote areas are Māori (Gurney, 2022; Whitehead et al., 2022). Additionally, an assessment by University of Auckland PhD student Haukapuanui Vercoe found that one third of New Zealand's 869 marae are in flood prone areas⁵. Indeed, a report on the flooding in Hawke's Bay due to Cyclone Gabrielle found that "Mana whenua were disproportionately affected by this flood event, with a number of marae, papakāinga and urupā and other taonga significantly affected due to their being located on marginal, flood-prone land adjacent to river channels" (HBIFR, 2024). Combined with the inequities detailed above, this positions Māori as some of the most vulnerable in New Zealand to potential fungal infections after natural disasters or extreme weather events and highlights the need for these inequities to be addressed.

⁵ <https://www.rnz.co.nz/news/te-manu-korihi/512771/survey-finds-third-of-marae-in-flood-prone-areas-up-to-30-percent-prone-to-landslides>

6. RISK MINIMISATION APPROACHES

There are several steps which can be taken prior to, and in the immediate aftermath of natural disasters and extreme weather events to minimise the risk of fungal infections. This chapter will focus on highlighting these steps for individuals and healthcare professionals.

6.1. ADVICE FOR INDIVIDUALS

Advice for individuals to reduce their risk of fungal infections is focused on minimisation of exposure to mould in water-damaged environments. Several publications provide practical advice for minimising mould exposure after flooding events, including a previous report prepared for the Ministry of Health on decontamination of mould-affected environments (Cressey, 2019). Key points include:

- Children, immunocompromised individuals and allergy sufferers should avoid exposure to mould-contaminated environments.
- Wear safety goggles, rubber gloves and a mask when cleaning mouldy surfaces and materials. The CDC recommends using an N95 respirator mask⁶.
- Shower and wash your clothing after any mould remediation work.
- Ensure areas undergoing mould remediation are well-ventilated to avoid buildup of fungal spores (Metts, 2008).
- Ensure appropriate safety precautions are taken when using antifungal agents or biocides as they can be toxic (Metts, 2008).
- Seek health care if you experience any symptoms which may be related to mould exposure (Metts, 2008).

Care should also be taken to minimise exposure to silt accumulated outdoors after flooding events as this will also likely contain fungal propagules which can be easily aerosolised, including whilst driving through flood-affected areas.

6.2. ADVICE FOR HEALTHCARE PROFESSIONALS

Perhaps the most important action which can be taken to reduce the risk of fungal infections associated with natural disasters and extreme weather events is to raise the awareness of this risk within the healthcare sector. Many of the clinical studies summarised in this report highlight the importance of early detection of fungal infections, and correct species identification so that an appropriate treatment path can be taken. Particularly in the case of species where antifungal resistance limits therapeutic options. However, due to the relative rarity of many of these infections, and their propensity to present after an initial period of apparent recovery, they are often initially overlooked, leading to delayed detection and poorer outcomes for patients. Educating healthcare professionals on the potential for

⁶ <https://www.cdc.gov/mold-health/about/clean-up.html> Accessed 14 September 2025

invasive fungal infections in victims of natural disasters will lead to earlier detection of these infections, leading to better clinical outcomes (Andresen et al., 2005).

Another important action which can be taken by healthcare professionals to reduce the risk of fungal infections in the aftermath of these events is to ensure that medical supplies do not become contaminated with fungal spores. Fungal infections potentially arising from contamination of medical supplies has previously been reported in Sri Lanka after the Boxing Day tsunami (Gunaratne et al., 2007) and Chile after a 2010 earthquake (Torres et al., 2014). An outbreak of fungal infections in patients in a burn intensive care unit in Baltimore, USA after Hurricane Sandy (Sood et al., 2018) also highlights the importance of ensuring any damage to healthcare facilities during natural disasters or extreme weather events is appropriately remediated before these facilities return to an operational state.

Lastly, support from healthcare professionals in promoting the messaging for individuals highlighted in Section 6.1 will further help to minimise the risk of fungal infections associated with natural disasters and extreme weather events.

7. SUMMARY

Human fungal infections, once considered relatively rare due to a lack of thermotolerance limiting their growth at body temperature, are increasing, driven by rising global temperatures. Adding to this is an increase in the frequency of natural disasters and extreme weather events, which further increase our susceptibility to fungal pathogens.

The aim of this report was to summarise what is known about fungal infections following natural disasters and extreme weather events, based on published clinical reports, to aid health professionals in identification of these potentially life-threatening infections. Due to overlap in the routes by which these events increase susceptibility to fungal infection, they were grouped into flooding events, events associated with dispersal of fungal propagules, and volcanic eruptions.

Flooding events considered in this report include tsunamis, cyclones and extreme rainfall. There are three main routes by which these events increase the risk of fungal infections: aspiration of floodwaters, contamination of traumatic wounds, and exposure to mould growing on water-damaged materials and surfaces. Several reports have documented invasive fungal infections following aspiration of floodwaters by near drowning victims, particularly in survivors of the 2011 Japanese tsunami. Most of these infections involved *Scedosporium*, a species ubiquitous in the environment and renowned for its resistance to antifungal agents. *Scedosporium* infections seldom occur in otherwise healthy individuals, so these infections were often initially misdiagnosed. These infections also tended to disseminate from the lungs to other sites in the body, with symptoms not apparent until the symptoms of the near drowning were largely resolved.

Scedosporium infection of traumatic wounds has also been reported in flooding victims. As for infections initially arising from aspiration, these infections tended to disseminate to other bodily locations, with symptoms not apparent until some time after the initial injury. Infections of traumatic wounds with *A. elegans*, a species of the order Mucorales notorious for causing life-threatening necrotic infections known as mucormycoses have also been reported. In both cases the fungal cause of infection was not initially considered until antibiotic treatment failed. These reports highlighted the importance of early identification of the causative agent of necrotic infections for the best clinical outcomes and emphasised that a fungal cause of infection should be considered in survivors of natural disasters whose wound infections do not improve with debridement and antibiotic treatment.

Several studies have demonstrated a potential link between fungal infections and mould exposure in water damaged buildings in the aftermath of flooding events, including a study showing a significant increase in invasive fungal infections in Houston, Texas after Hurricane Harvey. A number of cases of severe rhino-orbital-cerebral fungal infections were documented in individuals exposed to mould after flooding events, including several immunocompromised patients. An outbreak of fungal infections in a burn intensive care unit was also potentially linked to Hurricane-associated water damage. A separate study in which *Syncephalastrum* was cultured from various clinical samples taken from asymptomatic individuals with variable mould exposure after Hurricane Katrina highlights, however, the need to distinguish true infection from colonisation.

Dispersal events considered in this report include tornadoes, earthquakes, landslides, dust storms and wildfires. There are two main routes by which these events increase susceptibility to fungal infections: inhalation of aerosolised fungal propagules and contamination of traumatic wounds.

The majority of reports of fungal infections associated with inhalation of fungal propagules involved *Coccidioides* species naturally present in the soil. As *Coccidioides* species are not present in New Zealand, these studies are not directly relevant. However, as the range of *Coccidioides* is increasing due to climate change it may pose a threat in the future. Additionally, several studies highlighted that in addition to *Coccidioides*, spores from a range of other fungal species can also be transported in wildfire smoke. Infections caused by *Aspergillus* species likely associated with inhalation of aerosolised environmental dust have also been reported following a 2011 tornado in Joplin, USA and the 2011 Japanese earthquake.

Several cases of cutaneous mucormycosis associated with infection of traumatic wounds were reported after the 2011 Joplin tornado, many of which proved to be fatal. Symptoms of many of these necrotic infections did not present until some time after the initial injury thereby delaying diagnosis and appropriate treatment, resulting in many patients requiring multiple rounds of radical surgical debridement. Additionally, several reports have also documented *Candida* infection of traumatic wounds in earthquake victims.

Two reports documented fungal infections associated with volcanic eruptions. The first involved multiple cases of necrotising fasciitis caused by *R. arrhizus* in victims of an eruption in Colombia in 1985, with 75% of cases fatal. The second relates to the recent 2019 Whakaari White Island eruption, with two burn patients developing invasive *A. fumigatus* infections which in both cases proved fatal.

The studies discussed in this report also highlighted how vulnerable populations are disproportionately affected by natural disasters. On consideration of this in the New Zealand context, several inequities became apparent. Firstly, Māori, Pacific peoples and the elderly are at higher risk of fungal infections due to their higher prevalence of diabetes. As HIV also increases the risk of fungal infections, Māori and gay and bisexual males are also at higher risk due to inequities related to HIV in both population groups. Risk for Māori and Pacific peoples is also elevated due to higher rates of respiratory disease in these populations.

Certain population subgroups are also at higher risk of fungal infections due to their increased exposure to mould. These include Māori and Pacific peoples, and low-income households who are more likely to live in mould affected housing. People living in remote areas of New Zealand are also at higher risk due to being in areas at greater risk from natural disasters and extreme weather events. Māori, in particular, are at the highest risk as almost a third of people living in the most remote areas are Māori and one third of New Zealand's marae are in flood prone areas.

The main step individuals can take to reduce their risk of fungal infections following natural disasters and extreme weather events is to limit exposure to mould developing on water-damaged materials and surfaces. For healthcare professionals, the most important step which can be taken to reduce fungal infections associated with these events is to highlight the potential for these otherwise rare infections in victims of natural disasters and extreme weather events.

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APPENDIX

To assess whether the various fungal pathogens discussed in this report are present in New Zealand, the New Zealand Organisms Register (<https://www.nzor.org.nz/search>) was interrogated. Species identified only to the genus level were excluded from the analysis. Results are summarised in Table 4 below.

Table 4 Occurrence of reported fungal species in New Zealand

Species	Presence in New Zealand
<i>Apophysomyces elegans</i>	Absent
<i>Apophysomyces trapeziformis</i>	Absent
<i>Aspergillus flavus</i>	Present
<i>Aspergillus fumigatus</i>	Present
<i>Blastomyces dermatitidis</i>	Absent
<i>Candida albicans</i>	Present
<i>Candida glabrata</i>	Present
<i>Candida tropicalis</i>	Present
<i>Cladophialophora bantiana</i>	Present
<i>Coccidioides immitis</i>	Absent
<i>Coccidioides posadasii</i>	Absent
<i>Fusarium solani</i>	Present
<i>Paecilomyces variotii</i>	Present
<i>Rhizopus arrhizus/Rhizopus oryzae</i>	Present
<i>Scedosporium apiospermum</i>	Present
<i>Scedosporium aurantiacum</i>	Present
<i>Scedosporium prolificans</i>	Absent
<i>Trichosporon mucoides</i>	Absent



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