Infectious Diseases After Hydrologic Disasters

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KEYWORDS

• Flood • Hurricane • Tsunami • Infectious diseases • Soft tissue infections
• Respiratory infections • Gastrointestinal infections • Vector-borne diseases

KEY POINTS

- Skin and soft tissue infections following a hydrologic disaster can arise in the setting of traumatic injury and exposure to contaminated water.
- Gastrointestinal and respiratory infections are common among displaced populations, and are shaped by living conditions, access to clean water, and pre-existing endemic diseases.
- Leptospirosis is a zoonotic infection that has been associated with severe floods and population displacement.
- Vector-borne diseases can be influenced by hydrologic disasters; outbreaks are often multifactorial in nature.
- Disaster responders can reduce their risk of illness due to infectious diseases through careful planning, preparation, and preventive measures.

INTRODUCTION

Natural disasters are defined as disturbances in the ecosystem that impede a native community's ability to adapt, often requiring external interventions for survival.[1] They can arise from hydrologic, atmospheric, or geologic events.[2] Hydrologic events include hurricanes, tsunamis, and storm surges, as well as excessive rainfall, floods, and even drought. Recent hydrologic disasters such hurricanes Harvey, Irma, and Maria in the latter half of 2017 are poignant reminders of the power and destruction these events can unleash. When hydrologic disasters occur, concerns about the threat of

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infectious diseases associated with human remains often arise.[2,3] However, endemic infectious diseases affecting vulnerable and displaced populations pose the greatest risk to human health, particularly in resource-poor settings.[2–5] This article discusses general principles of infectious diseases following a hydrologic disaster. Next, it focuses on skin and soft tissue infections, gastrointestinal infections, respiratory infections, and zoonotic and vector-borne infectious diseases commonly encountered among survivors. Finally, it provides personal safety guidance for emergency physicians and other disaster responders providing care after a hydrologic disaster.

PRINCIPLES OF INFECTIOUS DISEASE AFTER HYDROLOGIC DISASTERS

The risk for infectious diseases following a hydrologic disaster can be contextualized using the classic epidemiologic triad or triangle, comprised of an external agent (microorganism), a susceptible host, and an environment that brings the host and agent together. In most instances, agents responsible for infections are ones that existed naturally in the affected region prior to the disaster, albeit with varying levels of contribution to human disease.[3] For this reason, it is possible to generate a rational differential diagnosis of microorganisms responsible for specific infectious disease syndromes based on geography and individual exposure history. Hosts, including survivors and responders alike, are susceptible to infection through traumatic injury and exposure to contaminated environments following a hydrologic event. Poor hygiene, poor sanitation, and lack of access to clean water and uncontaminated food further increase host vulnerability to various common communicable infectious diseases.[2,3] A hydrologic event disrupts the environment on multiple levels and can eliminate pre-existing barriers separating hosts and agents. Water sources can become contaminated with microbe-laden sewage, wastewater, and agricultural runoff.[5] Standing water can serve as a breeding site for arthropod vectors (eg, mosquitos). Displaced human populations lacking shelter are likely to encounter contaminated water, animals, and arthropod vectors, while those in temporary shelter may be subject to infections associated with crowded living conditions.

The timeline following a natural disaster is often broken down into an impact (0–4 days), postimpact (4 days to 4 weeks), and recovery phase (after 4 weeks).[3] Infections during the impact phase are likely to be associated with traumatic injuries (eg, lacerations, punctures) sustained while escaping imminent danger or performing initial clean-up and repairs after a hydrologic event. However, most acute infections including those involving wounds or related to population displacement are likely to emerge during the postimpact phase.[6] Vector-borne diseases (eg, dengue, malaria), uncommon infections related to environmental contamination (eg, leptospirosis), and infections with longer incubation or latent periods are more apt to emerge during postimpact and into the recovery phase.[3,6]

SKIN AND SOFT TISSUE INFECTIONS

Skin and soft tissue infections (SSTIs) are common after hydrologic disasters.[7–10] Compromised skin integrity in the setting of environmental water exposure, traumatic wounds, and water-related dermatologic conditions (eg, contact dermatitis, immersion foot) provide skin and waterborne pathogens an avenue for infecting underlying soft tissue. Gram-positive organisms including *Staphylococcus aureus* and *Streptococcus* species are typical bacterial pathogens associated with these infections, which may be exacerbated in crowded living conditions. During Hurricane Katrina in 2005, a cluster of methicillin-resistant *S aureus* (MRSA) SSTIs involving adults and children occurred at an evacuee facility in Dallas, Texas.[11] Empiric antibiotic therapy directed

against these common pathogens, paired with incision and drainage of abscesses when appropriate, is no different from standard clinical practice in many cases.[12]

Gram-negative organisms specifically associated with water exposure also play a role in SSTIs after hydrologic events.[13,14] *Vibrio vulnificus* is naturally found in saltwater or brackish water and has been associated with wound infections in southern US states bordering the Gulf of Mexico. Infections begin with cellulitis around the wound and can progress to the formation of hemorrhagic bullae, altered mental status, and septic shock, with a mortality rate of up to 30%.[15] Patients with underlying liver disease (eg, cirrhosis) and immunosuppression are particularly susceptible to infection and poor outcomes. Eighteen cases of wound-associated *V vulnificus* and *V parahaemolyticus* infection were reported after Hurricane Katrina, with 5 cases resulting in death.[16] Wound and blood cultures are necessary to establish the diagnosis. Severe infections should be treated using a combination of a third-generation cephalosporin (eg, ceftazidime, ceftriaxone) and doxycycline; fluoroquinolones may also be considered.[12,14,17] Timely surgical debridement of the wound reduces mortality in severe wound and necrotizing soft tissue infections involving this organism.[18]

Aeromonas species are gram-negative organisms that inhabit fresh and brackish water and have also been implicated in wound infections following hydrologic disasters. *Aeromonas* species was the most common wound isolate recovered from survivors with SSTI transferred to four hospitals in Bangkok, Thailand, following the 2004 Indian Ocean tsunami.[19] Most survivors likely had contaminated freshwater exposure from surrounding reservoirs after flooding from the tsunami wave.[9] High concentrations of *Aeromonas* species were detected in floodwater from Lake Pontchartrain throughout New Orleans weeks after Hurricane Katrina.[20] Onset of infection is typically within 48 hours and may present as a simple cellulitis, with erythema, warmth, and pain to the affected region. Infection can spread deeper, resulting in myonecrosis and necrotizing soft tissue infection. Antibiotic coverage for *Aeromonas* consists of a combination of doxycycline and either ciprofloxacin or ceftriaxone.[12,14] Wound culture with antibiotic susceptibility testing and surgical debridement when indicated are important guides to appropriate care.

Polymicrobial SSTIs including other gram-negative organisms such as *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and *Escherichia coli*, some multidrug-resistant, were common among survivors of the 2004 Indian Ocean tsunami.[19,21,22] Similar microbiological trends were observed in SSTIs following floods in Taiwan after Typhoon Morakot in 2009.[23] In general, a history of immersion in contaminated waters with subsequent SSTI should raise a concern for gram-negative or polymicrobial infection. Broad empiric antibiotic coverage of gram-positive and gram-negative organisms is recommended pending wound culture results. Indolent, late-onset skin infections and those that fail to improve with conventional antibiotic regimens increase suspicion for an uncommon organism. Several cases of cutaneous infection due to *Burkholderia pseudomallei*, the causative agent for melioidosis, and nontuberculous mycobacteria have been reported among tsunami survivors.[24–27]

Necrotizing soft tissue infection (NSTI) complicating contaminated traumatic soft tissue injuries is one of the gravest concerns after a hydrologic disaster. Infections can be polymicrobial (type I), involving aerobic and anaerobic bacteria, or monomicrobial (type II), classically due to group A *Streptococcus* (GAS; also known as *S pyogenes*), other β-hemolytic streptococci, or *S aureus*. GAS was identified using rapid whole-genome sequencing in a case of wound-associated NSTI after floodwater exposure during Hurricane Harvey in Houston, Texas, in 2017.[28] Although less common, *V vulnificus* and *Aeromonas* species can also cause NSTI, with *V vulnificus* NSTI reported in a patient with hepatitis C who was

evacuated from New Orleans following a boat rescue during Hurricane Katrina.[16] Cutaneous mucormycosis leading to NSTI has also been described in tsunami survivors with contaminated soft tissue injuries.[29,30] Subtle erythema and edema accompanied by pain out of proportion to physical findings can give way to skin bullae, ecchymosis, necrosis, and systemic toxicity. Progression of disease is often rapid, and death can ensue within hours of presentation. Prompt surgical debridement is combined with empiric broad-spectrum antibacterial or antifungal therapy (eg, liposomal amphotericin B), depending on the organism of concern, and fluid resuscitation.[12]

Clostridium tetani is a toxin-producing anaerobe naturally found in soil. An outbreak involving 106 cases of tetanus was reported a month after the 2014 Indian Ocean tsunami in Indonesia, where population tetanus immunization status was suboptimal at baseline.[31] Traumatic puncture wounds from debris inoculate *C tetani* spores into soft tissue. Subsequent germination and production of tetanus toxin lead to clinical symptoms after an incubation period of 3 to 21 days. With generalized tetanus, the most common form of the disease, painful, involuntary muscle spasm and rigidity frequently involve the jaw (trismus), neck, trunk, and extremities. Diagnosis of tetanus is clinical. Definitive treatment requires wound debridement for source control of spores, administration of human tetanus immune globulin (HTIG) to neutralize unbound toxin (passive immunization), active immunization with tetanus toxoid at a site different from that of HTIG, and initiation of antibiotic therapy (eg, metronidazole, penicillin G). As tetanus is a vaccine-preventable disease, appropriate wound care after a traumatic injury with active and passive immunization against tetanus based on US Centers for Disease Control and Prevention (CDC) guidelines is recommended when resources permit.[32]

GASTROINTESTINAL INFECTIONS

Diarrheal illnesses contribute up to 40% of deaths after a natural disaster, particularly in the setting of population displacement.[3] Contaminated food and water, disrupted sewage systems, compromised sanitation, poor hygiene, and crowded living situations can facilitate fecal-oral transmission of several gastrointestinal pathogens leading to outbreaks and even widespread epidemics of infectious diarrhea, particularly in resource-poor settings and developing countries. In the United States, flooding after several hydrologic disasters has also been associated with diarrheal illness.[33–37]

V cholerae remains a widespread cause of bacterial diarrheal illness globally, with significant morbidity and mortality. An estimated 2.9 million cases of cholera occur annually, resulting in 95,000 deaths across 69 endemic countries, with the greatest global burden of disease centered in sub-Saharan Africa and southeast Asia.[38] *V cholerae* was the most common cause of diarrhea during flood-associated diarrheal epidemics in 1988, 1998, 2004, and 2007 in Bangladesh, where the disease is endemic, significantly affecting older patients and those of lower socioeconomic status.[39–41] In contrast, cholera is rare and sporadic in nonendemic countries such as the United States, with no direct flood-associated cases or epidemics identified after Hurricane Katrina in 2004 or Hurricane Rita in 2005 along the Louisiana Gulf Coast.[16,42] Transmitted through contaminated water, *V cholerae* causes a profuse, secretory diarrhea leading to dehydration, muscle cramps, electrolyte derangements, acute renal failure, altered mentation, and hypotension. Abdominal pain and vomiting are common early on. Definitive diagnosis of cholera is established through stool culture; basic laboratory testing can help identify patients with significant hypoglycemia and

electrolyte losses. Treatment centers on aggressive fluid resuscitation and supportive care. Although often reserved for severe cases with significant volume depletion, antibiotic therapy reduces duration of illness, total stool volume, stool shedding of *V cholerae*, and fluid requirements.[43] Doxycycline, azithromycin, ciprofloxacin, and ceftriaxone are appropriate choices to treat *V cholerae*, depending on local antibiotic resistance patterns.[44]

Enterotoxigenic *E coli* has been a significant cause of epidemic diarrhea, particularly among children after flooding in Bangladesh.[39,41] Other enteric pathogens associated with diarrhea after floodwater exposure include *Salmonella* and *Shigella* species throughout parts of Asia.[40,45,46] In Massachusetts, the risk of *Clostridium difficile* infection increased in the 2 weeks following a flood.[47] Diarrhea accompanied by fever, bloody or mucoid stool, severe abdominal pain, or signs of sepsis should prompt stool testing for *Salmonella*, *Shigella*, *Campylobacter*, *Yersinia*, shiga toxin-producing *E coli* (0157), and *C difficile* with guideline-directed antibiotic therapy and fluid resuscitation when indicated.[44]

Norovirus is the most common cause of acute viral gastroenteritis worldwide. Low infectious dose, presence of virus in vomitus, and continued viral shedding in stool weeks after patient recovery make norovirus highly contagious.[48] This is further compounded by its stability on environmental surfaces. Following Hurricane Katrina, an outbreak of acute gastroenteritis caused by norovirus affected more than 1000 evacuees and relief workers in temporary shelter at Reliant Park in Houston, Texas, over a period of 11 days.[49,50] A smaller outbreak of what was likely norovirus occurred in an evacuation shelter in New York City after Hurricane Sandy in 2012.[51] Symptoms including nausea, vomiting, abdominal pain, and nonbloody diarrhea, with or without fever, usually develop within 2 to 3 days of infection. Diagnosis of norovirus is usually clinical, but can be confirmed using molecular or immunologic assays of stool specimens. Treatment, as with other acute viral gastroenteritis, is supportive, and infection prevention measures emphasizing hand hygiene, contact isolation, and rigorous environmental disinfection are critical to halting an outbreak.

Rotavirus was responsible for an outbreak of diarrheal illness in a temporary shelter in India following the 2004 Indian Ocean tsunami, and has been a frequent agent of flood-associated diarrhea in Bangladesh.[40,52] In 2014, rotavirus played a prevalent role in a diarrheal outbreak in Honiara following significant flooding and population displacement that subsequently evolved into a nationwide epidemic throughout the Solomon Islands.[53]

Hepatitis A virus (HAV) and hepatitis E virus (HEV) are endemic in many developing countries, with the greatest mortality reported in sub-Saharan Africa and Asia.[54] Both viruses are transmitted through contaminated food and water and thrive in poor sanitary conditions. Flood-associated outbreaks of HAV and HEV have been reported previously in Sudan and India.[55,56] Severe flooding in 2007 in Anhui Province, China, was significantly associated with an increased incidence of HAV infection in affected areas.[57] In Bangladesh, increased incidence of HEV infection after flooding has been attributed to sewage contamination of piped water, with high mortality rates reported among women in the third trimester of pregnancy.[58] Symptoms can include fever, fatigue, nausea, vomiting, abdominal pain, and jaundice. Most infections are self-limited; a small fraction can progress to acute liver failure. Although HAV infection is readily diagnosed by serology, testing options for HEV may vary and are more limited in availability. Treatment is supportive and should focus on fluid resuscitation and avoiding further hepatic insult. HAV infection is preventable through widely available vaccines; in contrast, HEV vaccines may be more challenging to access.

RESPIRATORY INFECTIONS

Acute respiratory infections are common following natural disasters, particularly among displaced populations and young children.[2] Although data are limited, excess morbidity and mortality caused by acute respiratory infections is significant in crisis-affected populations, with case fatality rates as high as 35%.[59] Overcrowding, poor nutrition, and lack of health care are significant risk factors for infection. The incidence of acute respiratory tract infections increased from 295 to 1205 per 100,000 residents in a Nicaraguan municipality immediately after Hurricane Mitch in 1998.[60] Following the 2014 Indian Ocean tsunami, syndromic surveillance identified more than 50,000 cases of acute respiratory infection among survivors in Aceh Province, Indonesia.[61] Acute respiratory infections have likewise been common following several large hydrologic disasters in the United States.[11,33,35,36,62,63] Pertussis was diagnosed in an infant who was rescued from a rooftop in New Orleans after Hurricane Katrina.[11] In most instances, upper respiratory infections are viral in origin, while lower respiratory infections (pneumonia) are likely attributable to common bacterial (eg, *Streptococcus pneumoniae*) and viral pathogens. Management should follow standard clinical practices.

Immersion and near-drowning after a hydrologic event can lead to aspiration of contaminated floodwater, with subsequent inoculation of bacteria into the respiratory tract. Following the 2004 Indian Ocean tsunami, multiple cases of gram-negative and polymicrobial aspiration pneumonitis and pneumonia were reported among survivors.[21,26,64–66] Common gram-negative organisms isolated in sputum culture included *P aeruginosa*, *Klebsiella* species, *E coli*, and *Aeromonas* species. Among severely injured European tourists repatriated to their native countries following the tsunami, several multidrug-resistant gram-negative organisms were recovered from upper respiratory cultures, likely acquired from environmental and healthcare exposure.[21] Aspiration-related melioidosis, manifesting with multilobar pneumonia, pulmonary abscess, and sepsis, was also reported in Thailand and Indonesia among tsunami survivors.[64,67] Although *B pseudomallei* is endemic to southeast Asia and northern Australia, sporadic cases of melioidosis have been described in the Americas, with 1 case occurring after floodwater exposure in Puerto Rico.[68] Inhalation of aerosolized particles (eg, high-pressure washing of contaminated environments) may also predispose individuals to gram-negative and polymicrobial pneumonia during the postimpact phase after a hydrologic event.[6] In general, patients with pneumonia not responding to conventional antibiotic regimens for community-acquired pneumonia should undergo microbiological investigation for unusual or polymicrobial infections.

Pulmonary tuberculosis can present unique challenges to public health after a hydrologic disaster when populations are displaced. Following Hurricane Katrina, a homeless man who was evacuated from New Orleans to Philadelphia was identified on entry screening with suspicious symptoms, isolated, and diagnosed with tuberculosis.[11] Additional new tuberculosis cases were identified among evacuees to 3 other states.[69] All the while, intense public health efforts were underway to track 195 individuals with known tuberculosis throughout Alabama, Louisiana, and Mississippi to confirm continuation of their treatment.[11] Transmitted through airborne droplet nuclei, *Mycobacterium tuberculosis* can linger for hours in enclosed and poorly ventilated spaces, posing significant infection risks in crowded living conditions. Chronic cough (>3 weeks), fever, chills, night sweats, and recent weight loss in high-risk patients (eg, immunosuppression, drug use, incarceration, close household contact with another person with pulmonary tuberculosis, or birth in a tuberculosis-endemic region) should all raise concern for tuberculosis.

ZOONOTIC INFECTIONS

Zoonotic infections stem from pathogens that are transmitted from animals to people. Chief among these, leptospirosis is a significant concern after hydrologic events associated with flooding and population displacement. More than a million estimated cases of leptospirosis contribute upwards of 59,000 deaths annually, with the highest morbidity and mortality seen in resource-poor countries.[70] Leptospirosis is caused by spirochetes from the genus *Leptospira*, which are free-living in freshwater and moist soil, and widely distributed in temperate and tropical regions around the world. Animals, particularly rodents, can become infected and serve as reservoirs, shedding high concentrations of *Leptospira* in urine back into the environment. Subsequent human infection occurs when nonintact skin (eg, abrasions or lacerations) and mucous membranes (conjunctiva, nasopharynx) come in direct contact with contaminated water and other environmental sources of *Leptospira*. Leptospirosis is common in endemic regions in the setting of heavy rainfall, freshwater flooding, increases in rodent population due to poor sanitation, and situations that place rodents and people in close proximity with one another.[2] Several outbreaks have been reported after flooding related to typhoons and unusually heavy rainfall throughout southeast Asia, Australia, and South America.[71–77] A fourfold increase in leptospirosis cases was identified in Puerto Rico shortly after Hurricane Hortense in 1996.[78] Another rise in cases in Puerto Rico following Hurricane Maria in late 2017 remains under investigation.

After a 5 to 14 day incubation period, patients with leptospirosis develop a flu-like illness with fever, chills, malaise, myalgias, headache, cough, nausea, vomiting, and diarrhea. Conjunctival suffusion is often present. Most cases of leptospirosis are self-limited; however, a subset can be complicated by aseptic meningitis, jaundice, renal failure, pulmonary hemorrhage, or acute respiratory distress syndrome. Diagnosis of leptospirosis is often clinical as confirmatory laboratory testing may take considerable time, depending on availability. Serology (eg, microscopic agglutination test, enzyme-linked immunosorbent assay) and increasingly molecular diagnostics (eg, polymerase chain reaction assay) support definitive diagnosis; blood culture has low sensitivity and can take several weeks. Antibiotic therapy for mild cases should consist of oral doxycycline or amoxicillin; intravenous penicillin or ceftriaxone is used to treat severe disease. Those with severe disease may require hospital admission, renal replacement therapy, ventilatory support, or even extracorporeal membrane oxygenation (ECMO).[79] Chemoprophylaxis with doxycycline following severe floods in endemic countries after high-risk exposure and during outbreaks may be effective in reducing leptospirosis cases.[73,76,80]

Animal bites are a common traumatic injury following the displacement of human and animal populations. After Hurricane Ike in 2008, many bites resulted from contact with domesticated pets (primarily dogs and cats) known to victims, some complicated by soft tissue infection.[81] Delayed wound care and lack of antibiotic prophylaxis can increase the risk for infections related to oral flora of the biting animal or human skin flora, particularly with deep wounds. Canine and feline oral flora include *Pasteurella multocida*, *Capnocytophaga canimorsus*, *Staphylococcus*, *Streptococcus*, and anaerobes; *Bartonella henselae* is found primarily in cat saliva.[82] Antibiotic prophylaxis after a dog or cat bite with amoxicillin-clavulanate provides adequate coverage in many instances.[12] Mild infections can likewise be treated with amoxicillin-clavulanate if MRSA is not a concern; severe infections may be treated with ampicillin-sulbactam or other broad-spectrum antibiotic regimens with coverage of anaerobes and antibiotic-resistant organisms when appropriate. Management of infections related to unusual

and uncommon types of animal bites should be based on anticipated oral flora, wound culture, and infectious disease consultation.[82]

Although rare in the United States and other countries with established animal vaccination programs, rabies remains endemic in many parts of the world, with infections largely attributed to animal bites, primarily involving dogs.[83] Prophylaxis following an animal bite or other high-risk contact (eg, bat exposure), including the administration of human rabies immune globulin (HRIG) and rabies vaccine, should be guided by local epidemiology, risk assessment, public health infrastructure for animal testing and monitoring, and availability of HRIG and vaccine in resource-limited settings.[84,85]

VECTOR-BORNE DISEASES

Infectious diseases transmitted by arthropod vectors, particularly mosquitos, are variably influenced by hydrologic disasters and their aftermath in endemic regions. Initial high winds and heavy flooding can reduce vector populations and disrupt existing breeding sites, decreasing the risk of infection during the impact phase. Standing water left behind may establish new vector breeding sites in the postimpact and recovery phase. Differing levels of exposure to disease-carrying vectors and active public health interventions to control vectors further shape the landscape of vector-borne disease after a hydrologic disaster.

Dengue virus (DENV) is an arthropod-borne virus (arbovirus) that is transmitted by the *Aedes aegypti* and *Aedes albopictus* mosquito. Common throughout the tropics, DENV is responsible for an estimated 390 million infections annually, of which a quarter manifest with clinically significant disease.[86] Heavy precipitation and other climatic changes have predicted increases in DENV infection in several endemic regions weeks to months later.[87–90] In Cuba, heavy rainfall from Hurricane Michelle may have further potentiated an ongoing DENV outbreak in 2001.[91] Severe flooding may have a similar additive effect in endemic resource-poor settings, as seen in 2010 during a severe DENV epidemic in Pakistan.[92] Dengue cases briefly exceeded epidemic threshold 2 months after Typhoon Haiyan struck the Philippines in late 2013; however, no large outbreak occurred due in large part to aggressive nationwide vector control activities in affected areas.[93] A similar response following flash flooding in Honiara, Solomon Islands, likely also prevented any large DENV outbreaks despite significant population displacement.[94]

Symptoms of DENV infection, including fever, headache, retro-orbital pain, arthralgias, myalgias, rash, and hemorrhagic manifestations (eg, epistaxis, gingival bleeding, petechia, ecchymosis, gastrointestinal bleeding, or vaginal bleeding), develop within 4 to 7 days after inoculation. Leukopenia, thrombocytopenia, and transaminitis are common. A fraction of patients may develop severe disease with plasma leakage, severe bleeding, hypotension, and multiorgan failure. Diagnosis of DENV infection is clinical. Laboratory confirmation is made by serologic or molecular testing (reverse transcriptase polymerase chain reaction, RT-PCR). Treatment is supportive, with aggressive fluid hydration and administration of acetaminophen for fever and pain control.[95] Aspirin, ibuprofen, and other nonsteroidal anti-inflammatory drugs are to be avoided given the risk of exacerbating bleeding. Management of severe DENV infection may require critical care interventions including blood transfusion, renal replacement therapy, and hemodynamic support with vasopressors.

Malaria is a protozoal infection that is transmitted by the *Anopheles* mosquito throughout most tropical regions. In 2013, an estimated 95 to 284 million cases occurred worldwide, resulting in anywhere from 703,000 to 1,032,000 deaths.[96] Of

the 5 species known to cause human disease, *Plasmodium falciparum* accounts for most worldwide disease and associated mortality. Malaria outbreaks have been reported after heavy rainfall and severe flooding in southeast Asia and sub-Saharan Africa where disease is endemic and populations are highly vulnerable to infection.[97–100] In 1963, Haiti was the site of a malaria epidemic totaling some 75,000 cases following heavy rainfall and flooding from Hurricane Flora 2 months prior.[101]

The incubation period for malaria ranges anywhere from 7 to 30 days, depending on the infecting *Plasmodium* species. Initial symptoms are protean including fever, chills, rigors, diaphoresis, malaise, myalgias, arthralgias, headache, cough, nausea, vomiting, abdominal pain, and diarrhea. As the infection progresses, fever can become cyclic. With severe malaria, patients may develop significant anemia, altered mental status (including encephalopathy with cerebral malaria), renal failure, liver failure, coagulopathy, metabolic acidosis, acute respiratory distress syndrome, and septic shock. Diagnosis is classically made by identification and quantification of parasitemia in thick and thin blood smears with light microscopy. In resource-limited settings and endemic countries, rapid detection tests (RDTs) targeting specific antigens have emerged as a reliable means for diagnosing uncomplicated *P falciparum* malaria.[102] A positive RDT is highly specific for *P falciparum*, but a negative result cannot rule infection. It is recommended that all RDTs be confirmed with traditional blood smears. Antimalarial therapy is imperative and should be guided by prevailing regional malaria epidemiology and drug resistance patterns.[103,104] Young children, immunocompromised patients, those with severe malaria and/or high parasitemia, and other patients at high-risk for complications should be hospitalized for treatment and may require critical care.

HUMAN REMAINS AND INFECTIOUS DISEASES

Human remains and their potential to spread infectious diseases after a natural disaster have long been a concern among many. However, most initial deaths associated with a hydrologic disaster are caused by traumatic injuries or drowning and not infection. Human remains pose little risk to the general public in regions not endemic for certain infectious diseases (eg, *V cholerae*, *M tuberculosis*).[105–108] No known epidemics of infectious disease after recent natural disasters have been attributed to the presence of human remains.[108]

Without a living human host, most medically significant pathogens do not survive for a considerable time, particularly in the setting of desiccation. Although bloodborne pathogens (eg, human immunodeficiency virus, hepatitis C virus) can persist in human remains, infection generally requires body fluid exposure with nonintact skin or mucous membranes or a percutaneous injury (eg, needle, bone fragment).[105] In regions with endemic infectious diseases, human remains resulting from a hydrologic disaster are unlikely to transmit infection except in unique circumstances. For example, *V cholerae* is environmentally resilient and naturally exists in aquatic environments. Corpses harboring *V cholerae* could contaminate drinking water sources during an ongoing epidemic, contributing to spread of disease alongside more significant factors (eg, overcrowding, poor sanitation), but are not likely to be the primary trigger for an epidemic.[108] Remains of a person with pulmonary tuberculosis in which respirations have ceased are unlikely to disseminate *M tuberculosis,* although certain precautions should be taken to further reduce exposure (eg, covering the mouth of the body to prevent escape of air and ensuring adequate ventilation of the surrounding environment during handling of remains).[105] Avoiding death rituals that involve close unprotected family contact with corpses associated with pathogens known to be transmitted in

this manner (eg, *V cholerae*, Ebola virus) is highly prudent, irrespective of a coinciding hydrologic disaster.[109,110] In general, the risk of infectious diseases from human remains is negligible compared with that of survivors of a disaster.

GUIDANCE FOR EMERGENCY RESPONDERS

Prevention of infectious diseases following a hydrologic disaster requires systems-based public health and emergency management strategies that address population displacement. Evacuation of survivors from contaminated environments and access to safe shelter, clean water and food, and basic health care services are integral components of a coordinated disaster response. Avoidance of overcrowding, promotion of personal hygiene, restoration of a functional sanitation infrastructure, and aggressive vector control in vulnerable regions help further mitigate the risk of communicable infectious diseases. Awareness of the spectrum of infectious disease possible after a hydrologic disaster informs early recognition, definitive diagnosis, and management, and helps prevent transmission to others, depending on the causative pathogen.

Emergency physicians, nurses, fire and emergency medical services professionals, and other first responders providing aid in a hydrologic disaster can reduce their risk of infectious disease through preparation, attention to preventive health, and use of appropriate personal protective equipment. Prior to a disaster, providers should ensure they are current with routinely recommended immunizations, including tetanus and influenza, in accordance with annual CDC guidance (www.cdc.gov/vaccines/schedules/hcp/adult.html). In addition, health care professionals should receive immunization against hepatitis B virus; those anticipating responses in countries with high or intermediate hepatitis A endemicity should also receive HAV immunization. Other immunizations related to responses outside the United States may be considered in consultation with an infectious disease or travel medicine specialist, or based on routinely updated recommendations for travelers, such as the CDC Yellow Book (www.cdc.gov/travel).

At the time of disaster, medical planning should include information gathering about ongoing infectious disease outbreaks as well as diseases endemic to the anticipated area of operations, particularly if deploying internationally. The CDC travelers' health Web site (including the Yellow Book) and International Society for Infectious Diseases' internet-based Program for Monitoring Emerging Diseases (ProMed; www.promedmail.org) are regularly updated resources that provide timely information for assessing infectious disease risks by geographic region. If mosquitos and other arthropods are likely to be a concern, appropriate clothing minimizing exposed skin and use of insect repellent can help reduce bites, particularly important in areas with known endemic vector-borne diseases. Likewise, chemoprophylaxis for certain endemic diseases (eg, malaria and leptospirosis) may be considered, particularly if high-risk exposures are anticipated (eg, sleeping outdoors and immersion in contaminated water).

Use of appropriate personal protective equipment while providing health care after a hydrologic disaster should follow standard precautions; transmission-based precautions (eg, airborne, droplet, and contact precautions) should be guided by clinical suspicion for certain pathogens, particularly when caring for a patient with respiratory complaints, uncontrolled diarrhea, or draining wounds. Hand hygiene underpins all infection prevention practices, perhaps even more so in disaster settings where resources are limited. For rescuers and those likely to be significantly exposed or immersed in floodwater, protective clothing and equipment minimizing skin and mucous membrane contact with contaminated water are advised whenever

possible. Following such exposures, clothing, equipment, and responders should undergo decontamination (eg, soap and clean water) to remove residual microbial and chemical burden from floodwater. For those likely to navigate through or handle debris, appropriate head protection, eyewear, work gloves, and boots protect against traumatic injury in contaminated environments. Masks or respirators may be considered if there is potential for aerosolization of floodwater or other environmental particulates. Growing literature focusing on emergency responders and deployment health during past hydrologic disasters can help inform future preventive strategies.[111–113]

Access to safe, uncontaminated food and water is critical to preventing infectious diseases among disaster responders as much as it is to the affected populations they serve. Responders should remain vigilant for signs or symptoms of infection and seek medical care when indicated during and after participation in disaster aid, as some illnesses may not clinically manifest until after returning home.

SUMMARY

Infectious diseases have long been associated with hydrologic events and their aftermath, although outbreaks are uncommon in developed countries with intact emergency management and public health infrastructures. Most communicable infections surface among displaced populations in the setting of inadequate shelter, overcrowding, poor sanitation, and lack of access to clean food and water. Increased awareness and knowledge of the potential infectious disease risks after a hydrologic disaster optimize emergency care to survivors and safeguards the health of disaster responders.

REFERENCES

1. Lechat MF. The epidemiology of health effects of disasters. Epidemiol Rev 1990; 12:192–8.
2. Watson JT, Gayer M, Connolly MA. Epidemics after natural disasters. Emerg Infect Dis 2007;13:1–5.
3. Kouadio IK, Aljunid S, Kamigaki T, et al. Infectious diseases following natural disasters: prevention and control measures. Expert Rev Anti Infect Ther 2012;10: 95–104.
4. Ivers LC, Ryan ET. Infectious diseases of severe weather-related and flood-related natural disasters. Curr Opin Infect Dis 2006;19:408–14.
5. Cann KF, Thomas DR, Salmon RL, et al. Extreme water-related weather events and waterborne disease. Epidemiol Infect 2013;141:671–86.
6. Allworth A. Infectious disease considerations related to sudden flooding disasters for the emergency physician. Emerg Med Australas 2011;23:120–2.
7. Lee SH, Choi CP, Eun HC, et al. Skin problems after a tsunami. J Eur Acad Dermatol Venereol 2006;20:860–3.
8. Tempark T, Lueangarun S, Chatproedprai S, et al. Flood-related skin diseases: a literature review. Int J Dermatol 2013;52:1168–76.
9. Bandino JP, Hang A, Norton SA. The infectious and noninfectious dermatological consequences of flooding: a field manual for the responding provider. Am J Clin Dermatol 2015;16:399–424.
10. Dayrit JF, Bintanjoyo L, Andersen LK, et al. Impact of climate change on dermatological conditions related to flooding: update from the International Society of Dermatology Climate Change Committee. Int J Dermatol 2018; 57(8):901–10.

11. Centers for Disease Control and Prevention. Infectious disease and dermatologic conditions in evacuees and rescue workers after Hurricane Katrina–multiple states, August-September, 2005. MMWR Morb Mortal Wkly Rep 2005;54:961–4.
12. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis 2014;59:147–59.
13. Lim PL. Wound infections in tsunami survivors: a commentary. Ann Acad Med Singapore 2005;34:582–5.
14. Diaz JH, Lopez FA. Skin, soft tissue and systemic bacterial infections following aquatic injuries and exposures. Am J Med Sci 2015;349:269–75.
15. Strom MS, Paranjpye RN. Epidemiology and pathogenesis of Vibrio vulnificus. Microbes Infect 2000;2:177–88.
16. Centers for Disease Control and Prevention. *Vibrio* illnesses after Hurricane Katrina–multiple states, August-September 2005. MMWR Morb Mortal Wkly Rep 2005;54:928–31.
17. Liu JW, Lee IK, Tang HJ, et al. Prognostic factors and antibiotics in *Vibrio vulnificus* septicemia. Arch Intern Med 2006;166:2117–23.
18. Chao WN, Tsai CF, Chang HR, et al. Impact of timing of surgery on outcome of *Vibrio vulnificus*-related necrotizing fasciitis. Am J Surg 2013;206:32–9.
19. Hiransuthikul N, Tantisiriwat W, Lertutsahakul K, et al. Skin and soft-tissue infections among tsunami survivors in southern Thailand. Clin Infect Dis 2005;41:e93–6.
20. Presley SM, Rainwater TR, Austin GP, et al. Assessment of pathogens and toxicants in New Orleans, LA, following Hurricane Katrina. Environ Sci Technol 2006;40:468–74.
21. Maegele M, Gregor S, Steinhausen E, et al. The long-distance tertiary air transfer and care of tsunami victims: injury pattern and microbiological and psychological aspects. Crit Care Med 2005;33:1136–40.
22. Doung-ngern P, Vatanaprasan T, Chungpaibulpatana J, et al. Infections and treatment of wounds in survivors of the 2004 Tsunami in Thailand. Int Wound J 2009;6:347–54.
23. Lin PC, Lin HJ, Guo HR, et al. Epidemiological characteristics of lower extremity cellulitis after a typhoon flood. PLoS One 2013;8:e65655.
24. Nieminen T, Vaara M. *Burkholderia pseudomallei* infections in Finnish tourists injured by the December 2004 tsunami in Thailand. Euro Surveill 2005;10:E050303.4.
25. Svensson E, Welinder-Olsson C, Claesson BA, et al. Cutaneous melioidosis in a Swedish tourist after the tsunami in 2004. Scand J Infect Dis 2006;38:71–4.
26. Garzoni C, Emonet S, Legout L, et al. Atypical infections in tsunami survivors. Emerg Infect Dis 2005;11:1591–3.
27. Appelgren P, Farnebo F, Dotevall L, et al. Late-onset posttraumatic skin and soft-tissue infections caused by rapid-growing mycobacteria in tsunami survivors. Clin Infect Dis 2008;47:e11–6.
28. Long SW, Kachroo P, Musser JM, et al. Whole-genome sequencing of a human clinical isolate of emm28 *Streptococcus pyogenes* causing necrotizing fasciitis acquired contemporaneously with Hurricane Harvey. Genome Announc 2017;5[pii:e01269-17].
29. Andresen D, Donaldson A, Choo L, et al. Multifocal cutaneous mucormycosis complicating polymicrobial wound infections in a tsunami survivor from Sri Lanka. Lancet 2005;365:876–8.

30. Snell BJ, Tavakoli K. Necrotizing fasciitis caused by *Apophysomyces elegans* complicating soft-tissue and pelvic injuries in a tsunami survivor from Thailand. Plast Reconstr Surg 2007;119:448–9.
31. Aceh Epidemiology Group. Outbreak of tetanus cases following the tsunami in Aceh Province, Indonesia. Glob Public Health 2006;1:173–7.
32. Liang JL, Tiwari T, Moro P, et al. Prevention of pertussis, tetanus, and diphtheria with vaccines in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 2018;67:1–44.
33. Centers for Disease Control and Prevention. Tropical Storm Allison rapid needs assessment–Houston, Texas, June 2001. MMWR Morb Mortal Wkly Rep 2002; 51:365–9.
34. Wade TJ, Sandhu SK, Levy D, et al. Did a severe flood in the Midwest cause an increase in the incidence of gastrointestinal symptoms? Am J Epidemiol 2004; 159:398–405.
35. Centers for Disease Control and Prevention. Hurricane Ike rapid needs assessment - Houston, Texas, September 2008. MMWR Morb Mortal Wkly Rep 2009; 58:1066–71.
36. Noe RS, Schnall AH, Wolkin AF, et al. Disaster-related injuries and illnesses treated by American Red Cross disaster health services during Hurricanes Gustav and Ike. South Med J 2013;106:102–8.
37. Wade TJ, Lin CJ, Jagai JS, et al. Flooding and emergency room visits for gastrointestinal illness in Massachusetts: a case-crossover study. PLoS One 2014;9: e110474.
38. Ali M, Nelson AR, Lopez AL, et al. Updated global burden of cholera in endemic countries. PLoS Negl Trop Dis 2015;9:e0003832.
39. Qadri F, Khan AI, Faruque AS, et al. Enterotoxigenic *Escherichia coli* and *Vibrio cholerae* diarrhea, Bangladesh, 2004. Emerg Infect Dis 2005;11:1104–7.
40. Schwartz BS, Harris JB, Khan AI, et al. Diarrheal epidemics in Dhaka, Bangladesh, during three consecutive floods: 1988, 1998, and 2004. Am J Trop Med Hyg 2006;74:1067–73.
41. Harris AM, Chowdhury F, Begum YA, et al. Shifting prevalence of major diarrheal pathogens in patients seeking hospital care during floods in 1998, 2004, and 2007 in Dhaka, Bangladesh. Am J Trop Med Hyg 2008;79:708–14.
42. Centers for Disease Control and Prevention. Two cases of toxigenic *Vibrio cholerae* O1 infection after Hurricanes Katrina and Rita–Louisiana, October 2005. MMWR Morb Mortal Wkly Rep 2006;55:31–2.
43. Leibovici-Weissman Y, Neuberger A, Bitterman R, et al. Antimicrobial drugs for treating cholera. Cochrane Database Syst Rev 2014;(6):CD008625.
44. Shane AL, Mody RK, Crump JA, et al. 2017 Infectious Diseases Society of America clinical practice guidelines for the diagnosis and management of infectious diarrhea. Clin Infect Dis 2017;65:1963–73.
45. Vollaard AM, Ali S, van Asten HA, et al. Risk factors for typhoid and paratyphoid fever in Jakarta, Indonesia. JAMA 2004;291:2607–15.
46. Ni W, Ding G, Li Y, et al. Effects of the floods on dysentery in north central region of Henan Province, China from 2004 to 2009. J Infect 2014;69:430–9.
47. Lin CJ, Wade TJ, Hilborn ED. Flooding and *Clostridium difficile* infection: a case-crossover analysis. Int J Environ Res Public Health 2015;12:6948–64.
48. Atmar RL, Opekun AR, Gilger MA, et al. Determination of the 50% human infectious dose for Norwalk virus. J Infect Dis 2014;209:1016–22.

49. Centers for Disease Control and Prevention. Norovirus outbreak among evacuees from Hurricane Katrina–Houston, Texas, September 2005. MMWR Morb Mortal Wkly Rep 2005;54:1016–8.
50. Yee EL, Palacio H, Atmar RL, et al. Widespread outbreak of norovirus gastroenteritis among evacuees of Hurricane Katrina residing in a large "megashelter" in Houston, Texas: lessons learned for prevention. Clin Infect Dis 2007;44:1032–9.
51. Ridpath AD, Bregman B, Jones L, et al. Challenges to implementing communicable disease surveillance in New York City evacuation shelters after Hurricane Sandy, November 2012. Public Health Rep 2015;130:48–53.
52. Sugunan AP, Roy S, Murhekar MV, et al. Outbreak of rotaviral diarrhoea in a relief camp for tsunami victims at Car Nicobar Island, India. J Public Health (Oxf) 2007;29:449–50.
53. Jones FK, Ko AI, Becha C, et al. Increased rotavirus prevalence in diarrheal outbreak precipitated by localized flooding, Solomon Islands, 2014. Emerg Infect Dis 2016;22:875–9.
54. Stanaway JD, Flaxman AD, Naghavi M, et al. The global burden of viral hepatitis from 1990 to 2013: findings from the Global Burden of Disease Study 2013. Lancet 2016;388:1081–8.
55. McCarthy MC, He J, Hyams KC, et al. Acute hepatitis E infection during the 1988 floods in Khartoum, Sudan. Trans R Soc Trop Med Hyg 1994;88:177.
56. Pal S, Juyal D, Sharma M, et al. An outbreak of hepatitis A virus among children in a flood rescue camp: a post-disaster catastrophe. Indian J Med Microbiol 2016;34:233–6.
57. Gao L, Zhang Y, Ding G, et al. Identifying flood-related infectious diseases in Anhui Province, China: a spatial and temporal analysis. Am J Trop Med Hyg 2016;94:741–9.
58. Mamun Al M, Rahman S, Khan M, et al. HEV infection as an aetiologic factor for acute hepatitis: experience from a tertiary hospital in Bangladesh. J Health Popul Nutr 2009;27:14–9.
59. Bellos A, Mulholland K, O'Brien KL, et al. The burden of acute respiratory infections in crisis-affected populations: a systematic review. Confl Health 2010;4:3.
60. Campanella N. Infectious diseases and natural disasters: the effects of Hurricane Mitch over Villanueva municipal area, Nicaragua. Public Health Rev 1999;27:311–9.
61. World Health Organization. Epidemic-prone disease surveillance and response after the tsunami in Aceh Province, Indonesia. Wkly Epidemiol Rec 2005;80:160–4.
62. Centers for Disease Control and Prevention. Surveillance in hurricane evacuation centers–Louisiana, September-October 2005. MMWR Morb Mortal Wkly Rep 2006;55:32–5.
63. Centers for Disease Control and Prevention. Injury and illness surveillance in hospitals and acute-care facilities after Hurricanes Katrina And Rita–New Orleans area, Louisiana, September 25-October 15, 2005. MMWR Morb Mortal Wkly Rep 2006;55:35–8.
64. Athan E, Allworth AM, Engler C, et al. Melioidosis in tsunami survivors. Emerg Infect Dis 2005;11:1638–9.
65. Kateruttanakul P, Paovilai W, Kongsaengdao S, et al. Respiratory complication of tsunami victims in Phuket and Phang-Nga. J Med Assoc Thai 2005;88:754–8.
66. Yorsaengrat W, Chungpaibulpatana J, Tunki B, et al. Respiratory complication of tsunami diaster victims in Vachira Phuket Hospital. J Med Assoc Thai 2006;89:518–21.

67. Chierakul W, Winothai W, Wattanawaitunechai C, et al. Melioidosis in 6 tsunami survivors in southern Thailand. Clin Infect Dis 2005;41:982–90.
68. Christenson B, Fuxench Z, Morales JA, et al. Severe community-acquired pneumonia and sepsis caused by *Burkholderia pseudomallei* associated with flooding in Puerto Rico. Bol Asoc Med P R 2003;95:17–20.
69. Centers for Disease Control and Prevention. Tuberculosis control activities after Hurricane Katrina–New Orleans, Louisiana, 2005. MMWR Morb Mortal Wkly Rep 2006;55:332–5.
70. Costa F, Hagan JE, Calcagno J, et al. Global morbidity and mortality of leptospirosis: a systematic review. PLoS Negl Trop Dis 2015;9:e0003898.
71. Kawaguchi L, Sengkeopraseuth B, Tsuyuoka R, et al. Seroprevalence of leptospirosis and risk factor analysis in flood-prone rural areas in Lao PDR. Am J Trop Med Hyg 2008;78:957–61.
72. Su HP, Chan TC, Chang CC. Typhoon-related leptospirosis and melioidosis, Taiwan, 2009. Emerg Infect Dis 2011;17:1322–4.
73. Dechet AM, Parsons M, Rambaran M, et al. Leptospirosis outbreak following severe flooding: a rapid assessment and mass prophylaxis campaign; Guyana, January-February 2005. PLoS One 2012;7:e39672.
74. Amilasan AS, Ujiie M, Suzuki M, et al. Outbreak of leptospirosis after flood, the Philippines, 2009. Emerg Infect Dis 2012;18:91–4.
75. Smith JK, Young MM, Wilson KL, et al. Leptospirosis following a major flood in Central Queensland, Australia. Epidemiol Infect 2013;141:585–90.
76. Chusri S, McNeil EB, Hortiwakul T, et al. Single dosage of doxycycline for prophylaxis against leptospiral infection and leptospirosis during urban flooding in southern Thailand: a non-randomized controlled trial. J Infect Chemother 2014;20:709–15.
77. Mohd Radi MF, Hashim JH, Jaafar MH, et al. Leptospirosis outbreak after the 2014 major flooding event in Kelantan, Malaysia: a spatial-temporal analysis. Am J Trop Med Hyg 2018;98:1281–95.
78. Sanders EJ, Rigau-Perez JG, Smits HL, et al. Increase of leptospirosis in dengue-negative patients after a hurricane in Puerto Rico in 1996 [correction of 1966]. Am J Trop Med Hyg 1999;61:399–404.
79. Delmas B, Jabot J, Chanareille P, et al. Leptospirosis in ICU: a retrospective study of 134 consecutive admissions. Crit Care Med 2018;46:93–9.
80. Schneider MC, Velasco-Hernandez J, Min KD, et al. The use of chemoprophylaxis after floods to reduce the occurrence and impact of leptospirosis outbreaks. Int J Environ Res Public Health 2017;14.
81. Warner GS. Increased incidence of domestic animal bites following a disaster due to natural hazards. Prehosp Disaster Med 2010;25:188–90.
82. Abrahamian FM, Goldstein EJ. Microbiology of animal bite wound infections. Clin Microbiol Rev 2011;24:231–46.
83. Hampson K, Coudeville L, Lembo T, et al. Estimating the global burden of endemic canine rabies. PLoS Negl Trop Dis 2015;9:e0003709.
84. Manning SE, Rupprecht CE, Fishbein D, et al. Human rabies prevention–United States, 2008: recommendations of the Advisory Committee on Immunization Practices. MMWR Recomm Rep 2008;57:1–28.
85. Rupprecht CE, Briggs D, Brown CM, et al. Use of a reduced (4-dose) vaccine schedule for postexposure prophylaxis to prevent human rabies: recommendations of the Advisory Committee on Immunization Practices. MMWR Recomm Rep 2010;59:1–9.

86. Bhatt S, Gething PW, Brady OJ, et al. The global distribution and burden of dengue. Nature 2013;496:504–7.
87. Hashizume M, Dewan AM, Sunahara T, et al. Hydroclimatological variability and dengue transmission in Dhaka, Bangladesh: a time-series study. BMC Infect Dis 2012;12:98.
88. Sang S, Gu S, Bi P, et al. Predicting unprecedented dengue outbreak using imported cases and climatic factors in Guangzhou, 2014. PLoS Negl Trop Dis 2015;9:e0003808.
89. Chuang TW, Chaves LF, Chen PJ. Effects of local and regional climatic fluctuations on dengue outbreaks in southern Taiwan. PLoS One 2017;12:e0178698.
90. Sirisena P, Noordeen F, Kurukulasuriya H, et al. Effect of climatic factors and population density on the distribution of dengue in Sri Lanka: a GIS based evaluation for prediction of outbreaks. PLoS One 2017;12:e0166806.
91. Hsieh YH, de Arazoza H, Lounes R. Temporal trends and regional variability of 2001-2002 multiwave DENV-3 epidemic in Havana City: did Hurricane Michelle contribute to its severity? Trop Med Int Health 2013;18:830–8.
92. Hassan U, Loya A, Mehmood MT, et al. Dengue fever outbreak in Lahore. J Coll Physicians Surg Pak 2013;23:231–3.
93. Aumentado C, Cerro BR, Olobia L, et al. The prevention and control of dengue after Typhoon Haiyan. Western Pac Surveill Response J 2015;6(Suppl 1):60–5.
94. Shortus M, Musto J, Bugoro H, et al. Vector-control response in a post-flood disaster setting, Honiara, Solomon Islands, 2014. Western Pac Surveill Response J 2016;7:38–43.
95. World Health Organization. Dengue: guidelines for diagnosis, treatment, prevention and control. New edition. Geneva (Switzerland): World Health Organization; 2009.
96. Murray CJ, Ortblad KF, Guinovart C, et al. Global, regional, and national incidence and mortality for HIV, tuberculosis, and malaria during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet 2014;384:1005–70.
97. Brown V, Abdir Issak M, Rossi M, et al. Epidemic of malaria in north-eastern Kenya. Lancet 1998;352:1356–7.
98. Kondo H, Seo N, Yasuda T, et al. Post-flood–infectious diseases in Mozambique. Prehosp Disaster Med 2002;17:126–33.
99. Memon MS, Solangi S, Lakho S, et al. Morbidity and mortality of malaria during monsoon flood of 2011: South East Asia experience. Iran J Public Health 2014;43:28–34.
100. Boyce R, Reyes R, Matte M, et al. Severe flooding and malaria transmission in the western Ugandan highlands: implications for disease control in an era of global climate change. J Infect Dis 2016;214:1403–10.
101. Mason J, Cavalie P. Malaria epidemic in Haiti following a hurricane. Am J Trop Med Hyg 1965;14:533–9.
102. Abba K, Deeks JJ, Olliaro P, et al. Rapid diagnostic tests for diagnosing uncomplicated P. falciparum malaria in endemic countries. Cochrane Database Syst Rev 2011;(7):CD008122.
103. Centers for Disease Control and Prevention. Guidelines for treatment of malaria in the United States. Atlanta (GA): Centers for Disease Control and Prevention; 2013.
104. World Health Organization. Guidelines for the treatment of malaria. 3rd edition. Geneva (Switzerland): World Health Organization; 2015.

105. Morgan O. Infectious disease risks from dead bodies following natural disasters. Rev Panam Salud Publica 2004;15:307–12.
106. de Ville de Goyet C. Epidemics caused by dead bodies: a disaster myth that does not want to die. Rev Panam Salud Publica 2004;15:297–9.
107. Kirkis EJ. A myth too tough to die: the dead of disasters cause epidemics of disease. Am J Infect Control 2006;34:331–4.
108. Pan American Health Organization. Management of dead bodies after disasters: a field manual for first responders. 2nd edition. Washington (DC): Pan American Health Organization; 2016.
109. Sack RB, Siddique AK. Corpses and the spread of cholera. Lancet 1998;352:1570.
110. Dietz PM, Jambai A, Paweska JT, et al. Epidemiology and risk factors for Ebola virus disease in Sierra Leone-23 May 2014 to 31 January 2015. Clin Infect Dis 2015;61:1648–54.
111. O'Leary DR, Rigau-Perez JG, Hayes EB, et al. Assessment of dengue risk in relief workers in Puerto Rico after Hurricane Georges, 1998. Am J Trop Med Hyg 2002;66:35–9.
112. Tak S, Bernard BP, Driscoll RJ, et al. Floodwater exposure and the related health symptoms among firefighters in New Orleans, Louisiana 2005. Am J Ind Med 2007;50:377–82.
113. Rusiecki JA, Thomas DL, Chen L, et al. Disaster-related exposures and health effects among US Coast Guard responders to hurricanes Katrina and Rita: a cross-sectional study. J Occup Environ Med 2014;56:820–33.