Epidemics After Natural Disasters

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Abstract

Epidemics of infectious disease are rare following natural disasters, especially in developed countries. Observations from previous natural disasters suggest that skin, diarrheal, and respiratory infections are the most common infectious diseases in survivors. The etiologies of disease outbreaks are usually predictable, reflecting infectious diseases endemic in the affected area before the disaster. Injury and soft tissue infections are expected during the first few days after a disaster. In contrast, airborne, water-borne, and food-borne diseases are anticipated for up to one month after a disaster. A feared consequence of natural disasters is the potential exposure to dead bodies, both human and animal. No evidence exists that exposure to bodies after a disaster leads to infectious disease epidemics. To be discussed are specific epidemics that have followed earthquakes, floods, tornadoes, tsunamis, volcanic eruptions, landslides, and drought. Preventative public health and safety measures aimed at attenuation of such epidemics will be reviewed.

Introduction

A disaster has been defined as "a result of a vast ecological breakdown in the relation between humans and their environment, a serious or sudden event on such a scale that the stricken community needs extraordinary efforts to cope with it, often with outside help or international aid."[1] Such disasters may be natural disasters, transportation disasters, the result of terrorism or technological events, and pandemics. This article will focus on epidemics following natural disasters. Such disasters include earthquakes, floods, tornadoes, tsunamis, volcanic eruptions, landslides, and drought.

Much misunderstanding surrounds the potential for communicable diseases after disasters, with a widely held belief that epidemics are inevitable. This is partially due to an overestimation of the capacity for disease spread from dead bodies.[2] In fact, the primary driver of disease spread after natural disasters is population displacement and crowding and its interplay with endemic disease and with a breakdown in infrastructure. If a disease is not endemic to a disaster area and is not introduced after a disaster, then it won't cause an epidemic after the event.[2] Factors contributing to disaster severity include human vulnerability due to poverty and social inequality, environmental degradation, and rapid population growth, especially among the poor.

Natural disasters have killed millions of people over the last twenty years, impacting the lives of at least one billion more people, and resulting in enormous economic damages.[3] In the decade 1994-2004, there were approximately one million thunderstorms, 100,000 floods, tens of thousands of landslides, earthquakes, wildfires and tornadoes, and several thousand hurricanes, tropical cyclones, tsunamis, and volcanoes.[3] Table 1 shows the relative risk of communicable diseases following specific disasters.

Because developing countries may lack resources, infrastructure, and disaster-preparedness systems, they may be disproportionately affected by natural disasters.[4]

Phases of natural disasters include an impact, post-impact, and recovery.[5] The impact phase occurs from 0-4 days, during which extrication of victims and treatment of immediate soft tissue infections takes place. Hypothermia, heat, illness, and dehydration are characteristic of this phase. In the post- impact

phase, which takes place from four days to four weeks post-disaster, airborne, foodborne, waterborne, and vector diseases are seen. Examples of diseases in this phase are cholera, bacterial dysentery, cryptosporidiosis, rotavirus, norovirus, salmonella, typhoid and paratyphoid, giardiasis, hepatitis A and E, and leptospirosis. Communicable respiratory infections in post-disaster settings include viral (e.g., influenza, RSV, adenoviruses), bacterial (e.g., *Strep pneumoniae*, pertussis, tuberculosis, Legionella, *Mycoplasma pneumoniae*), and diseases transmitted via the respiratory route (e.g., measles, varicella, *nisseria meningitides)*. Tetanus is also seen in this phase.

Table 1: Risk of communicable diseases after disasters, by mode of transmission[1]

DISASTER TYPE	PERSON-TO-PERSON*	WATER-BORNE#	FOOD-BORNE+
Volcano	Medium	Medium	Medium
Earthquake	Medium	Medium	Medium
Hurricane	Medium	High	Medium
Tornado	Low	Low	Low
Heat wave	Low	Low	Low
Cold wave	Low	Low	Low
Flood	Medium	High	Medium
Famine	High	High	Medium
Air pollution	Low	Low	Low
Industrial accident	Low	Low	Low
Fire	Low	Low	Low
Radiation	Low	Low	Low
Civil war/ refugees	High	High	High

*Shigellosis, streptococcal skin infections, scabies, infectious hepatitis, pertussis, measles, diphtheria, other respiratory infections, giardiasis, HIV/AIDS, other sexually-transmitted diseases, meningococcal disease, plague.
#Typhoid and paratyphoid fevers, cholera, leptospirosis, infectious hepatitis, shigellosis, campylobacter, salmonella, E. coli, cryptosporidiosis.
+ Typhoid and paratyphoid fevers, cholera, infectious hepatitis, shigellosis, campylobacter, salmonella, E. coli, amebiasis, giardiasis, cryptosporidium.

The recovery phase begins after four weeks, and diseases with long incubation periods, vectorborne, and chronic diseases manifest in this phase. Examples of organisms with long incubation periods are leishmaniasis and leptospirosis. Vectorborne illnesses include malaria, western/Saint Louis encephalitis, dengue, yellow fever, and West Nile virus. Chronic diseases seen in the recovery phase after hurricane Katrina, for example, included cardiac disease, hypertension, diabetes, and asthma. Special needs for shelters, wheelchairs, oxygen, large cots for the obese, glucometers, access to dialysis, and lacking prescription medications were also seen.[5]

Wound infections among survivors of the 2004 tsunami in Southeast Asia were polymicrobial, with over 600 organisms ultimately identified. Most prominent among these were *Aeromonas* species, *E.coli*, *Klebsiella pneumonia*, and *Pseudomonas aeruginosa*. Some of these organisms were resistant to all licensed antibiotics.[6] Among hurricane evacuees from the New Orleans area, a cluster of infections with methicillin-resistant *Staphylococcus aureus* (MRSA) was reported in approximately 30 pediatric patients at one evacuee facility in Dallas, Texas.[7] Additionally, 24 cases of hurricane-associated *Vibrio vulnificus* and *V. parahaemolyticus* wound infections were reported, with six deaths.[8]

Among the factors that contribute to disease transmission after disasters are environmental considerations, endemic organisms, population characteristics and crowding, the pre-event structure and type of public health systems and facilities and levels of immunization, and the magnitude of the disaster itself.[9] Environmental considerations include climate, with cold conditions favoring airborne pathogens and warm conditions favoring waterborne pathogens. In temperate climates, the winter is associated with influenza, while summer is a time for enterovirus infections. Similarly, rainfall during El Nino contributes to malaria, while drought leads to malnutrition-related disease. Geography may play a role in epidemics by isolating victims from needed resources. Organisms endemic to a region will be present after the disaster, while those that are not endemic before the event are unlikely to be present afterwards. On the other hand, as was demonstrated with the appearance of *Vibrio* infections after hurricane Katrina, the lack of reports of an organism prior to a disaster does not guarantee that the organism is not endemic.[8] This phenomenon was also demonstrated by the appearance of enterotoxicogenic *Shigella dysenteria* type 1, *Neisseria meningitidis*, and hepatitis E, following disasters in Africa.[9] As happened in Haiti with cholera, introduction of non-endemic organisms is possible when relief workers carry it to a disaster area. In fact, cholera has emerged as a serious disease in Latin America only in recent years.[2] Examples of endemic disease outbreaks to a region will be present after the disaster, including a nine-fold increase in coccidiomycosis (Valley fever) from January- March 1994 after the Northridge, California, earthquake and the giardiasis outbreak in 1980 after the eruption of Mount St. Helens, Montana.[9]

Population characteristics include the density and age of victims and the preponderance of chronic diseases. Displaced populations may be crowded in refugee camps. Disease incidence is usually increased in the elderly and in the very young. Baseline immunity to specific diseases is another critical feature of populations. Floods in Nepal in 1973 and in Sudan in 1988 displaced hundreds of thousands of victims. Similarly, the eruption of Mount Pinatubo in the Philippines in 1991 and hurricane Katrina resulted in mass displacement and temporary crowded shelter conditions for victims. Refugees living in crowded, temporary settings, of whom there may currently be over 50 million worldwide, are subject to explosive outbreaks of communicable disease of low endemicity, such as malaria, schistosomiasis, and leishmaniasis. Most recently reported, the human immunodeficiency virus has affected almost 10% of refugee Sudanese men since their forced migration to Ethiopia.[10]

Table 2: Waterborne, Vectorborne, and Direct Contact Diseases[26]

Disease	Clinical Features	Incubation Period	Diagnosis	Treatment
WATERBORNE DISEASES: SUMMARY				
Cholera	profuse watery diarrhea, vomiting	2 hrs - 5 days	direct microscopic observation of V. cholerae in stool	rehydration therapy; antimicrobials
Leptospirosis	sudden onset fever, headache, chills, vomiting, severe myalgia	2 - 28 days	leptospira- specific IgM serological assay	penicillin, amoxi, doxyxycline, erythromycin, cephalosporins
Hepatitis	jaundice, abdominal pain, nausea, diarrhea, fever, fatigue and loss of appetite	15 - 50 days	serological assay detecting anti- HAV of anti-HEV IgM antibodies	supportive care; hospitalize/ barrier nursing for severe cases; monitoring of pregnant women
Bacillary dysentery	malaise, fever, vomiting, blood and mucous in stool	12 - 96 hrs	suspect if bloody diarrhea, confirm by isolation of organism	nalidixic acid, ampicillin; hospitalize seriously ill or malnourished; rehydration
Typhoid fever	sustained fever, headache, constipation	3 - 14 days	culture from blood, bone marrow, bowel fluids; rapid antibody tests	ampicillin, trimethoprim-sulfamethoxazole, ciprofloxacin
VECTORBORNE DISEASES: SUMMARY				
Malaria	fever, chills, sweats, head and body aches, nausea and vomiting	7 - 30 days	parasites on blood smear observed using a microscope; rapid diagnostic assays if available	chloroquine, sulfadoxine-pyrimethamine
Dengue	sudden onset severe flu- like illness, high fever, severe headache, pain behind the eyes, and rash	4 - 7 days	Serum antibody testing with ELISA or rapid dot-blot technique	intensive supportive therapy
Japanese encephalitis	quick onset, headache, high fever, neck stiffness, stupor, disorientation, tremors	5 - 15 days	serological assay for JE virus IgM specific antibodies in CSF or blood (acute phase)	intensive supportive therapy
Yellow fever	fever, backache, headache, nausea, vomiting; toxic phase- jaundice, abdominal pain, kidney failure	3 - 6 days	serological assay for yellow fever virus antibodies	intensive supportive therapy
DIRECT CONTACT DISEASES: SUMMARY				
Pneumonia	cough, difficulty breathing, fast breathing, chest indrawing	1 - 3 days	Clinical presentation; culture respiratory secretions	co-trimoxazole, chloramphenicol, ampicillin,
Measles	rash, high fever, cough, runny nose, red and watery eyes; serious post-measles complications (5-10% of cases) - diarrhea, pneumonia, croup	10 - 12 days	generally made by clinical observation	supportive care; nutrition/ hydration; vitamin A; control fever; antibiotics in complicated cases
Bacterial meningitis	Sudden onset fever, rash, neck stiffness; altered consciousness; bulging fontanelle in <1 yrs of age	5 - 15 days	Examination of CSF – elevated WCC, protein; gram negative diplococci	Penicillin, ampicillin, chloramphenicol, ceftriaxone, cefotaxime, co-trimoxazole; diazepam (seizures)
Tetanus	difficulty swallowing, lockjaw, muscle rigidity, spasms	3 - 21 days	entirely clinical	immune globulin

Malnutrition, diabetes, and heart disease among victims also make them more vulnerable to infections. Another important population characteristic includes level of education, with less educated people tending to be less responsive to disaster teams. Religious beliefs may thwart public health efforts as was seen in the polio epidemic in Nigeria in 2004. The underlying health education and hygiene of the public and the types of trauma present (e.g., penetrating, blunt, burns) are also important population characteristics.

Critical pre-disaster variables include sanitation, primary health care and nutrition, medical infrastructure, equipment and medications, disaster preparedness, disease surveillance, and roads and transportation. The spread of microorganisms during a natural disaster is facilitated by disruption of public water and sewage systems, crowded living conditions, air borne transmission, lack of immunization, and injury-related infection due to exposure to debris.

The type of disaster can determine the variety of trauma and other post-disaster noninfectious illnesses. Earthquake, hurricane, and tsunami can lead to crush injury and other penetrating injuries. Tsunami and floods lead to near-drowning and electrocution. Floods cause about 50% of all deaths from natural disasters in the United States.[9] The majority of these are due to vehicle-related drowning during flash floods. Flooding also leads to carbon monoxide exposure and death.

According to the World Health Organization (WHO), the most common causes of death in a disaster are diarrhea, acute respiratory infections, measles, malaria, and malnutrition.[11]

Diseases Associated with Natural Disasters

Diseases associated with natural disasters can be divided generally into those associated with overcrowding, waterborne diseases, vectorborne diseases, and other diseases (Table 2). Crowding after disasters has contributed to epidemics of acute respiratory illness and pneumonia, measles, and meningitis. Evacuation to camps following natural disasters is prone to lead to infectious diseases. Camps combine high population density and poor sanitation, synergistic preconditions for fecal-oral and airborne droplet transmission of disease.[12]

These illnesses are spread person-to-person by airborne droplet transmission. A measles outbreak followed the eruption of Mount Pinatubo, Philippines, in 1991. Most of the more than 100,000 displaced persons were members of the Aeta tribe that lived on the slopes of the volcano. More than 18,000 cases of measles were reported within two months of the eruption, with a 25% mortality rate. Since that time, fewer cases of measles have followed natural disasters, as immunization has been more widespread.

Acute respiratory infections (ARI) are a significant contributor to death and disability after disasters, and children less than five are affected disproportionately.[4] Respiratory pathogens in post-disaster settings include viral (influenza, RSV, adenoviruses), bacterial (*Strep pneumoniae*, pertussis, tuberculosis, Legionella, *Mycoplasma pneumoniae*), and diseases transmitted via the respiratory route *(*measles, varicella*, nisseria meningitides)*. Among reported illnesses after Hurricane Katrina, the proportion of ARI was 12% four days after the levee overflowed and 20% during the next four weeks.[9]

Exposure to open-flame cooking, malnutrition, and lack of access to health care and antibiotics contribute to morbidity and mortality from ARI. ARI was responsible for most of the deaths among survivors of the tsunami in Aceh in 2004.[11] The incidence of ARI quadrupled in Nicaragua in the month following Hurricane Mitch in 1998.[13] Aspiration pneumonia is seen after flooding and tsunamis and is due to inhalation of soil-contaminated salt water. "Tsunami Lung" is a syndrome of cavitary lung disease and brain abscess seen among Southeast Asia tsunami survivors. It is initiated by the aspiration of soil, sand, and other particulate matter as a consequence of near-drowning. The syndrome is a polymicrobial pneumonia process that occurs up to six weeks later. Identified pathogens include water-borne organisms *Aeromonas, Pseudomonas, Streptococcal* species*, Nocardia, Pseudallescheria boydi,* and *Burkholderia pseudomallei.*[9,14]

Flooding constitutes approximately 40% of natural disasters, and it promotes both waterborne and vectorborne diseases.[11] Upper respiratory infections and pneumonias were reported among multiple victims of hurricane Katrina, including a case of pertussis in a two-month-old infant who was rescued from a rooftop in New Orleans and evacuated to Tennessee. Appropriate antimicrobial prophylaxis was provided, and no additional cases were reported.[7,15]

Waterborne diseases include diarrheal pathogens, hepatitis, and leptospirosis. Several pathogens have been associated with diarrhea after disasters. *Vibro cholerae* and enterotoxigenic *Escherichia coli* have been isolated after flooding in West Africa. Diarrheal diseases have followed hurricanes and flooding in Bangladesh, Sudan, and Nepal and are the most lethal public health threat to refugees overall.[2] Over 70% of deaths among Kurdish refugees in 1991 were due to diarrheal disease. The cholera epidemic that followed the 2010 Haiti earthquake sickened more than 170,000 people and killed more than 3,600.[14] The US Centers for Disease Control and Prevention (CDC) confirmed that the form of cholera detected in Haiti is one that is typically found in South Asia and Africa.[15] The Haitian outbreak originated from contaminated water near a facility that housed Nepalese troops, who are thought to have introduced the strain to an immunologically naïve local population. From Haiti, several new cases were identified in previously unaffected regions.

The Dominican Republic detected its first case of cholera in a migrant worker who had returned home from Haiti after the outbreak there. Additional cases of cholera have since been

reported in Bolivia, Brazil, Chile, Colombia, Nicaragua, Panama, Peru, and Venezuela. Subsequently, confirmed imported cases have been reported in Florida. The CDC has reported 13 suspected imported cases, with five confirmed as of December 2010. Researchers estimate that an additional 200,000 cases of cholera could arise in the Caribbean within the next 18 months.[15]

Paratyphoid fever, caused by *E Salmonella enterica* serotype Paratyphi A, resulted in diarrhea outbreaks in Indonesia in 1992–1993. *Cryptosporidium parvum*-related diarrhea followed flooding in Indonesia in 2001–2003.[16] An outbreak of diarrheal diseases after flooding in Bangladesh in 2004 involved more than 17,000 cases of *V.Cholerae* and *E.Coli* infections.[16] All survivors of the December 2004 tsunami in Aceh province in 2004 were forced to drink from unprotected wells, and 85% of these residents who were studied reported diarrhea.[11] The risk of diarrheal disease outbreaks following natural disaster is higher in developing countries than in industrialized countries. The association of communicable diseases with malnutrition is well known. In many of these malnourished victims, diseases such as measles, malaria, ARI, and diarrheal diseases are the most common causes of death.

After Hurricanes Allison and Katrina, initial reports were that *Escherichia coli* counts were 100 times higher than those normally found in river runoff. Among the 24,000 Katrina evacuees who were housed in the Reliant Park building in the Houston Astrodome complex, 18% developed acute gastroenteritis from September 2–12, 2005. Approximately 25% of adult and 40% of pediatric medical visits during this time were due to GI illness. Secondary spread to shelter and medical staff also occurred. While 50% of specimens were positive for norovirus, no other pathogen was identified.[9,17] Clusters of diarrheal disease were reported among persons in evacuation centers in Louisiana, Mississippi, Tennessee, and Texas. In Louisiana, approximately 20 clusters of diarrheal illness in evacuation centers were reported to and investigated by the CDC. In Memphis, gastrointestinal illness was the most common acute disease complaint among evacuees. Approximately 1,000 cases of diarrhea and vomiting were reported among evacuees in Mississippi and Texas. Norovirus was detected in stool specimens from patients in Texas. Nontyphoidal *Salmonella*, nontoxigenic *V. cholerae* O1, and other infections were also identified. No confirmed cases of *Shigella* dysentery or typhoid fever were reported in evacuees, and by three weeks after the initial relocations following Katrina few additional cases of diarrheal disease were being reported.[7,8] Following the 1976 Friuli, Italy, earthquake, there was a five- to sixfold increase in Salmonella secondary to food contamination, poor hygiene, and overcrowding.[9]

Following Hurricane Katrina, an increase in mortality was reported by CDC from *Vibrio* (*Vibrio vulnificus* and *parahaemolyticus*) soft tissue infections. Sixty percent of cases were wound infections and 40% resulted from eating raw shellfish. Necrotizing complicated some cases, and overall *Vibrio* mortality was 40%, and it was 20% in the subset with *Vibrio* from wounds. *Vibrio parahemolyticus* cases after Katrina were mostly diarrhea, while *V. vulnificus* cases were mostly septicemia and wound infection, and very few were diarrhea. For those two species, secondary transmission is not seen. During the flooding following Katrina, *Vibrio* cases were also reported in Arkansas, Texas, and Louisiana. These were mostly in immuno-compromised, chronically ill people who were exposed seawater. Once contact with brackish seawater ceased, case reports ended and so did any apparent epidemic potential.[8,16,17]

In most refugee settings, the case fatality rates (CFR) for cholera are between 2-5%. In the 1994 Rwandan refugees in the Zairian town of Goma (now Republic of the Congo), the rate rose to almost 25%, with almost 90% of adult deaths due to diarrheal illness. This outbreak, which has come to be known as "The Great Lakes Disaster," caused at least 48,000 cases and 23,000 deaths within one month in the refugee camps in Goma, Zaire. Shigella also erupted. Shigella dysenteria type 1, seen in displaced African populations, has a CFR of 10% in young children and in the elderly. Following Hurricane Mitch in 1998, cholera outbreaks occurred in Guatemala, Nicaragua, and Belize.[4,5,6]

The fecal-oral route (Tables 2 and 3) spreads Hepatitis A, B, and E. Usually infection results from drinking contaminated water, and there is current debate about the role of person-to-person spread. Hepatitis A and B are endemic in most developing countries, so children in these areas develop immunity to it early in life. In hepatitis E-endemic areas, generally mild disease outbreaks have followed flooding. Hepatitis E has only recently been introduced in most parts of Africa, so adults are unlikely to have immunity to it. Thus, any hepatitis-like illness in this region is assumed to be hepatitis E. Pregnant women are especially vulnerable, however, with mortality as high as 25%. Both hepatitis A and hepatitis E were noted in Aceh after the December 2004 tsunami and after the Pakistan earthquake in 2005. An increase in hepatitis A followed the 1983 Popaya, Columbia earthquake.[9]

Leptospirosis is a bacterial disease that is spread by contact with contaminated water. Rodents shed the organisms in their urine, and contact of the skin and mucous membranes with contaminated water, soil, or vegetation. Several floods in the last decade were followed by leptospirosis outbreaks, notably in Taiwan after Typhoon Nali in 2001, in Mumbai after flooding in 2000, in Portugal in 1967, and Brazil in 1975.[4]

Measles incidence and spread transmission after a disaster depends upon the baseline immunization rate of the affected population, especially those under15 years of age. More than 18,000 measles cases occurred after the eruption of Mount Pinatubo in the Philippines in 1991.[18] Increased death rates from measles were reported in camps in Bangladesh (1978), Somalia (1980), Sudan (1985), Ethiopia (1987), Malawi (1988–1990), Mozambique (1988–89, 1991), Philippines (1991), Darfur, Somalia (1994), Haripur, Afghanistan, and Kakuma refugee

camps, Kenya (2005). CFR of 2.3% up to 32% were reported.[9] Crowded living conditions, as is common among people displaced by natural disasters, contributes to rapid transmission of the organism.[12] Also contributing to measles outbreaks is a low baseline vaccination coverage rate among the affected population and, in particular, among children aged <15 years. These conditions that facilitate transmission are also ones that necessitate higher immunization coverage levels to prevent outbreaks. *Neisseria meningitidis* outbreaks have followed flooding and displacement in Pakistan and Aceh. *Meningococcal meningitis* outbreaks occurred also in refugees in Thailand in 1979, and among displaced Sudanese in1988, in Khartoum.[9,16] Rapid response with antimicrobial prophylaxis interrupts transmission in disaster-affected populations.

Important vectorborne diseases following disasters include malaria, dengue, Japanese encephalitis, and yellow fever, all of which are transmitted by mosquitoes. Disruption of water supplies promoting standing water, especially when associated with large numbers of displaced people sleeping outside, contributes to outbreaks. Months of severe flooding in Bolivia in 2007 triggered a dengue outbreak that killed 35 people. A dramatic rise in malaria cases followed the 1991 earthquake in Costa Rica, associated with changes in habitat that promoted mosquito breeding. Over 75,000 cases of *Plasmodium falciparum* malaria were associated with the 1966 Haitian hurricane Flora.[17] Malaria-specific mortality is especially severe in situations in which refugees fleeing an area of low endemicity travel through or migrate to an area of high endemicity.[2] Outbreaks of louse-borne relapsing fever were seen after refugee concentration in Somalia and Ethiopia.

Other diseases that are seen after disasters are tetanus and coccidiomycosis. Spores of *C. tetani* reside in soil, entering the tissue in contaminated wounds, especially in populations where vaccination levels are low. Once in the wounds, the spores produce a metalloprotease, tetanospasmin, which travels by retrograde movement into the central nervous system. There, the toxin blocks neurotransmission and disinhibits the motor cortex, leading to extensive spasm. Contributing to the occurrence of tetanus after disasters are penetrating injury with spore delivery, co-infection with other bacteria, localized tissue ischemia, and devitalized tissue. An outbreak of tetanus peaked two and one-half weeks after the tsunami in Aceh. The 106 cases that were reported from December 30 – January 26, 2004, occurred in Banda Aceh 4–30 days post-tsunami. CFR for this outbreak was 18.9%.[9] The infection is not transmitted person to person. *Coccidioides immitis* is a fungus that is found in soil in certain semiarid areas. Coccidiomycosis (valley fever) can be associated with exposure to increased levels of airborne dust after landslides and earthquakes. An outbreak occurred after the 1994 earthquake in Southern California. Large dust clouds generated by the earthquake dispersed the spores of Coccidioides. The disease incidence peaked two weeks after the earthquake, and there was a ninefold increase in coccidiodomycosis from January–March 1994. Two hundred and three cases were reported in Ventura County, California, including three deaths. There were 30 cases per 100,000 population. Fifty-six percent of the cases and the highest attack rate (114 / 100,000) was in the town of Simi Valley, at the base of a mountain range that had numerous landslides associated with the earthquake. Risk was associated with being within, and amount of time spent in, the dust cloud.

While tuberculosis (TB) is not a disease that is associated with natural disasters, it has emerged as a disease among displaced populations. Inadequate access to healthcare among refugees has resulted in an increased spread of TB among them.[19] During the war in Bosnia and Herzegovina in 1991 and during the civil war and famine in Somalia in 1991-92, the incidence of TB increased four-fold.[2] In 1985, 26% of deaths among refugees in Somalia were attributable to TB. Co-infection with HIV and malnutrition also contribute to the transmission, morbidity, and mortality of TB in displaced peoples. Control of TB among evacuees has consisted of both detecting new cases and providing treatment continuity for previously known cases.

Immediately after hurricane Katrina, TB program staff sought out known TB patients to check their status and assure that therapy continued. As of September 23, 2004, all 27 currently known TB patients who resided in Alabama, all 21 in Mississippi, and 105 (71%) of 147 in Louisiana had been located. Of the 42 TB patients from Louisiana not yet located, 41 were considered noncontagious at the time the hurricane made landfall.[19] A homeless person without a previous diagnosis of TB who was evacuated from New Orleans to Philadelphia was identified with symptoms consistent with pulmonary TB. The patient was isolated and begun on treatment for TB disease; a subsequent culture confirmed TB. At least eight other evacuees initially identified as potentially having TB were subsequently determined to have other conditions (e.g., lung cancer and infection with nontuberculous mycobacteria).[7,17,19]

Diagnosis of Communicable Diseases

The diagnosis of waterborne, vectorborne, and direct contact diseases is summarized in Table 2. Diagnosis of cholera is made by the direct microscopic observation of the *V. cholerae* organisms in the stool of victims. Leptospira and hepatitis are detected by specific IgM serologic assays and by detecting anti-HAV or anti-HEV IgM antibodies, respectively. Typhoid is identified by culturing *Salmonella typhi* from blood, bone marrow, and bowel fluids, and by rapid antibody tests.

The etiology of ARI is often suggested by the clinical presentation, and culture of respiratory secretions may identify a specific pathogen, such as *Streptococcus pneumoniae* or *Haemophilus influenzae*.

Measles is generally diagnosed by its clinical features, while the etiology of meningitis is made by examination of the cerebrospinal fluid. In the case of vectorborne diseases, parasites are identified on blood smear and serum antibody testing with

Table 3: Hepatitis A and E[17]

VIRUS	FEATURES	DURATION/SYMPTOMS	DIAGNOSIS/TREATMENT
Hepatitis A	Most reported vaccine-preventable disease. 33K acute cases. 61K new infections.70% children < 6yrs area symptomatic. Fecal-oral transmission via contaminated food, water,shellfish.	Incubation 28 days average, (range 15-50 days).Viral shedding 2 weeks prior to onset jaundice. Jaundice in 70% of patients, abdominal pain, fatigue, loss of appetite, nausea, diarrhea, fever.15% with relapsing symptoms 6-9 months.	Serology positive for IgM antibody (anti-IgM HAV) 5-10 days after onset jaundice up to 6 months. IgG antibody (anti-IgG HAV) positive early in course, confers lifetime immunity.Treatment supportive. Vaccine available for prevention.
Hepatitis E	Most common cause of non-A, non-B hepatitis worldwide. 2004 outbreak in Darfur, Sudan: 7791 cases and 99 deaths. Most common in young to middle-aged adults. Fecal-oral transmission via contaminated food, water,shellfish. Increased risk with flooding.	Incubation 40 days average, (range 15-60 days). Jaundice, abdominal pain, fatigue, dark urine, loss of appetite, nausea, vomiting. Secondary symptoms: arthralgias, diarrhea, pruritus, urticaria. Viral shedding for 2 weeks after infection.	Serology positive for IgM antibody (anti-IgM HEV) 5-10 days after onset jaundice up to 6 months. However, not commercially available in US; research lab only. Also PCR, IFA research lab only. IgG treatment supportive. No vaccine available for prevention.

ELISA. A rapid dot-blot technique may be used in suspected dengue. Serologic assays are available for detecting IgM specific antibodies in Japanese encephalitis in CSF or blood. Similarly, serological assay is used for detection of yellow fever virus antibodies.

The Significance of Dead Bodies

There is a widely held fear that the presence of dead bodies after a disaster contributes to disease transmission. This potential is vastly overstated.[4] In reality, when death is directly due to the natural disaster, human remains pose no risk for outbreaks. There are a few specific circumstances in which dead bodies pose any risks, such as deaths from cholera[21-24] or hemorrhagic fevers.[22] It is recommended that workers who handle bodies employ universal precautions for blood and body fluids and that they use and correctly dispose of gloves. Hand washing with soap after handling bodies and before eating and use of body bags are also important. Except in cases of cholera, shigellosis, or hemorrhagic fever, bodies do not need disinfection before disposal, but disinfection of vehicles and equipment is suggested. In addition, burial, rather than cremation, is recommended, with the bottom of graves above the water table (Table 3).[4,20-23]

Disease Control after Disasters

An accurate risk assessment for communicable diseases after a disaster is required in order to identify existing epidemic and endemic diseases that are common in the affected area. Similarly, the living conditions of the affected population are evaluated, with special attention to the availability of safe water and adequate sanitation facilities. Also assessed are the age distribution, nutritional status, and immunization coverage of those affected and their degree of access to healthcare.

Prevention and control of disease begins with hand washing, with proper handling of water and food, and with sewage disposal. Oral rehydration therapy (ORT) is initiated for diarrheal illness. Where possible, victims and aid workers must avoid entering contaminated water. Preventive Health Measures against the transmission of infectious agents related to natural disasters have been addressed by the WHO.[11] WHO recommends: keep hands and vessels clean, avoid preparing food directly in areas surrounded by flood water, separate raw and cooked food, cook food thoroughly, keep food at safe temperatures, use safe water, wear appropriate protective clothing during rescue and clean-up operations, and immunization.

Hepatitis and polyvalent *Strep pneumonia* vaccination may be appropriate. In the event of measles, rapid mass vaccination should be instituted within 72 hours of an initial case report, and vitamin A is administered in children six months to five years of age to prevent complications and to reduce mortality. Rapid mass vaccination is also instituted for meningitis.

Malaria response includes mosquito control, provision of insecticide-treated nets, bedding, and clothing, and the emptying of standing water containers. In the case of dengue, Japanese encephalitis, and yellow fever, mosquito control, isolation of cases, and mass vaccination are implemented.

Table 4: Recommended Handling of Dead Bodies[11]

SUGGESTED MEASURES FOR HANDLING DEAD BODIES
Universal precautions for blood and body fluids.
Disposal or disinfection of used gloves.
Avoiding cross-contamination of personal items.
Washing hands after handling bodies and before eating.
Disinfection of vehicles and equipment.
Use of body bags, especially for badly damaged bodies.
Hepatitis B vaccination.
No special arrangements, such as disinfection, with disposal of bodies.
New burial areas sited at least 250 meters away from drinking water sources,and with at least 0.7 meters of distance above the saturated zone.

Table 5: Disaster Myths and Realities[13]

MYTH	REALITY
Foreign medical volunteers with any kind of medical background are needed.	1. The local population almost always covers immediate lifesaving needs. 2. Only skills that are not available in the affected country may be needed. 3. Few survivors owe their lives to outside teams.
Any kind of assistance is needed, and it's needed now!	1. A hasty response not based on impartial evaluation only contributes to chaos. 2. Unrequested goods are inappropriate, burdensome, divert scarce resources, and more often burned than separated and inventoried. 3. Not wanted, seldom needed – used clothing, OTC, prescription drugs, or blood products; medical teams or field hospitals.
Epidemics and plagues are inevitable after every disaster.	1. Epidemics rarely ever occur after a disaster. 2. Dead bodies will not lead to catastrophic outbreaks of exotic diseases. 3. Proper resumption of public health services will ensure the public's safety (e.g., immunizations, sanitation, waste disposal, water quality, and food safety). Caveat: Criminal or terror-intent disasters require special considerations.
Disasters bring out the worst in human behavior.	1. While isolated cases of antisocial behavior exist, the majority of people respond spontaneously and generously.
The community is too shocked and helpless to respond.	1. Many find new strengths. 2. Cross-cultural dedication to common good is most common response to natural disasters. 3. Thousands volunteer to rescue strangers and sift through rubble after earthquakes from Mexico City, California, and Turkey. 4. Most rescue, first aid, and transport are from other casualties and bystanders.

Source: Noji E. Cutler lecture reference.

Conclusions

Natural disasters have killed millions of people during the past two decades and have adversely affected the lives of over one billion others. These events include earthquakes, volcanic eruptions, landslides, tsunamis, floods, and drought. Because of their lack of resources, infrastructure, and emergency medical and disaster systems, developing countries are disproportionately affected by natural disasters.

The potential for such disasters to be followed by epidemics of communicable diseases is often presumed to be very high, but this has been overstated (Table 5). In particular, the contribution of dead bodies in epidemic spread after disasters has been vastly exaggerated. In fact, dead bodies are not a significant contributor to disease. Rather, increases in the risk of communicable disease transmission has been demonstrated to be dependent upon: 1) the size, health status, and living conditions of the population displaced by the disaster; 2) crowding; 3) inadequate water and sanitation; and 4) poor access to health services. These factors are also characteristic of sudden population displacement in general.[2]

An understanding of epidemics after natural disasters has led to improved detection of, and response to, communicable diseases in these settings. By monitoring the incidence of such diseases and by documenting their impact, it is possible to better quantify the risk of outbreaks following natural disasters. Thus, by preparation for the inevitable, medical, epidemiological, and disaster response personnel will be more able to ameliorate the suffering that will occur after future natural disasters.

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