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CHAPTER

7

SYNDEMICS AND THE WORLDS THEY MADE

After studying this chapter, you should be able to

- Understand why the occurrence and importance of syndemics in human health was not often recognized in the past.
- Identify the role of specific syndemics in shaping past events.
- Recognize that wars and syndemics have been commonly entwined events in human history.

BEFORE NOW

In his book *The Healthy Body and Victorian Culture*, Bruce Haley (1978) offers the following account of syndemic disease patterns in the United Kingdom during the year 1847:

As had happened a decade earlier, typhus occurred simultaneously with a severe influenza epidemic, one which carried off almost thirteen thousand. There was also a widespread dysentery epidemic, and as if all this were not enough, cholera returned in the autumn of 1848, assailing especially those parts of the island hardest hit by typhus and leaving about as many dead as it had in 1831. . . . Diseases like cholera, typhus, typhoid, and influenza were more or less endemic at the time, erupting into epidemics when the right climatic conditions coincided with periods of economic distress. The frequency of concurrent epidemics gave rise to the belief that one sort of disease brought on another; indeed, it was widely believed that influenza was an early stage of cholera [p. 34].

As a result of such experiences, Haley argues, Victorians were more concerned with health than with almost all, if not all, other issues in their lives, including politics, religion, and their emergent ambivalence about change and modernity. The imprint of entwined syndemics on Victorian culture may be seen in that era's romantic poetry, utopian and other literature (novels like *The Mill on the Floss*, *The Egoist*, and *Tom Brown's School Days*, for example), views on spiritual perfection and virtue, commitment to hiking and other athletic recreation (such as calisthenics), ideas about the benefits of dieting and bathing, use of architectural tiling depicting plants and flowers, and resurgence of interest in the ancient Greek and Roman cultures, especially the embrace and celebration of Hygeia, the Greek goddess of health. Exemplary is John Ruskin's *Unto This Last* (1877), a set of essays in which a healthy Britain is likened to a human body after exercise: "Thus the circulation of wealth in a nation resembles that of the blood in the natural body. There is one quickness of the current which comes of cheerful emotion or wholesome exercise; and another which comes of shame or of fever. There is a flush of body which is full of warmth and life; and another which passes into putrefaction" (p. 140).

Similarly, John Kay, a leading figure during this era's emergent public health movement, wrote that "the moral leprosy of vice [is] capable of corrupting the body of society, like an insidious disease" (quoted in Adams, 1999, p. 175). This frequent cultural linking of the health of the social body to the health of physical bodies (Schepers-Hughes & Lock, 1987) draws attention to an important component of the social nature of sickness.

Although the rate at which new syndemics develop has accelerated over time as human populations have become larger and more concentrated (consider, for example, the growing number of megacities around the world), as the speed of transportation (carrying people, animals, commodities, and pathogens) has increased through new technologies, and as the human impact on the earth's climatic and other environmental systems has grown, syndemics are not a new phenomenon. The title of this chapter is borrowed from medieval historian Norman Cantor's book *In the Wake of the Plague: The Black Death and the World It Made* (2002). In this book (which is enlivened, if somewhat scientifically diminished, by its serious consideration at one point of the notion that the Black Death might have had an extraterrestrial origin), Cantor questions the received wisdom about the cause of the epidemic. Its metaphorical designation, the Black Death, is often attributed to the dark skin blotches that characterized victims, but that name more likely was intended to convey the devastating social impact of this epidemic, which swept through Europe in the mid-fourteenth century and killed at least a third (if not far more) of the total population. Epidemiological and history texts usually assert that the outbreak was caused solely by the rat-borne pathogenic bacterium *Yersinia pestis*. Cantor, however, picking up an idea introduced by British zoologist Graham Twigg, cogently argues that the engine driving the Black Death was a syndemic uniting the effects of *Y. pestis* with anthrax, an acute animal and human disease caused by a different microorganism, the spore-forming bacterium

Bacillus anthracis. Similarly, in his book *A History of Bubonic Plague in the British Isles*, J.F.D. Shrewsbury (1970), a bacteriologist with a keen interest in disease history, argued that the Black Death was the consequence of a co-epidemic involving yersinial plague and louse-borne typhus.

More recently, DeWitte and Wood (2008) used skeletal remains of 490 Black Death victims (from the East Smithfield Black Death cemetery in London) to test the assumption that the Black Death was so virulent its victims were killed indiscriminately. Using the methods of paleodemographic age estimation and a multistate model of selective mortality, these researchers counted bone lesions suggestive of prior infections and other health problems on the skeletons of the individuals in their sample. They also assessed dental development to determine approximate age. These findings were then compared with data for 291 individuals buried in cemeteries in the medieval Danish towns of Viborg and Odense shortly before the Black Death epidemic began. As would be expected, findings from the Danish sample suggested that bone lesions were associated with earlier death (that is, those with more health problems had died at a younger age). Significantly, this was also true of the Black Death victims from London, indicating that the epidemic tended to take the lives of those who were already in poor health. In particular, the lesions found on the London skeletons DeWitte and Wood examined appeared to have been caused by malnutrition. This suggests that the Black Death was a syndemic caused by diet-related immune system damage and infection with *Y. pestis*.

Whichever of these various explanations of the virulence of the Black Death prevails (and they are not mutually exclusive, multiple infections and malnutrition may have been involved), it is evident that syndemics have had an impact on the course of history, have helped to shape the world as we know it, and will continue to do so in the future. As the multiple disease entwinements of HIV/AIDS, the largest syndemic in human history, make clear, the impacts of disease interactions on human life can be profound. If, as Marx suggested, the goal is not merely to understand the course of history but to change it, then the lessons learned in the study of syndemics may be equally profound.

Although a worthy endeavor, researching syndemics of the past is hampered on the one hand by the limited availability and quality of health records and on the other hand by a less than complete level of disease knowledge. The further back in time one goes, the more problematic the quality of the health records, from the standpoint of both systematic data collection and precision of terminology. When one considers the symptom descriptions provided by ancient Latin authors such as Celsus (25 B.C.–37 A.D.) and Artaeus of Cappadocia (circa 200 A.D.), for example, it is evident that they were applying the disease term *elephantiasis* to cases of leprosy and not to the helminth infection that now bears that label. Moreover, although epidemics have been recorded since the beginning of writing, the tendency has been to focus on periodic outbreaks that were seen as being caused by a single disease. The result is year-by-year listings of a small, rotating set of diseases, implying that only one disease was present in any

given year. However, as Herring and Sattenspiel (2007) observe, "The historical record documents epidemic after epidemic of newly introduced diseases, but these diseases did not occur in a vacuum; rather, they interacted with those already present" (p. 200). Thus Sallares (2002) has suggested that the cases of typhoid fever described in the *Epidemios* volumes of the Corpus Hippocraticum (see Chapter Two) "could very easily have been infected with malaria as well, but this would not be apparent from the description given in the ancient text" (p. 127). Similarly, in commenting on the national mortality statistics for Italy toward the end of the nineteenth century, a malaria researcher noted that "medical science shows that for each death attributed to malaria there are several other deaths, which are attributed to other causes, but nevertheless are directly linked to malaria or indirectly caused by the debilitating effects of malaria infection" (Bonelli quoted in Sallares, 2002, p. 119).

Evidence of unrecognized disease interactions of the past does in fact appear in malaria data from rural Italy. Prior to the 1930s, the Italian hill town of Sermoneta, on the edge of the Pontine Marshes, for example, was recorded as having a crude death rate of 41 per 1,000 population, and 8 percent of these deaths were attributed to malaria. Eradication of mosquito breeding sites in the marshes, however, halved the annual death rate (Hackett, 1937), suggesting a synergistic interaction between malaria and the diseases that were recorded as the cause of death. The same pattern is seen in pre-independence British Guyana, where malaria is assumed by modern researchers to have been the cause of death at a level almost four times the number of deaths actually attributed to it (Jones, 2000). Overall, the use of death records to assess disease interaction is hampered, as Noymer and Garene (2000) observe, "because even if contributory causes are listed on the death certificate [which they often are not], a unique cause of death is recorded" (p. 573).

Syndemics in the past were not often recognized as such by physicians or public health officials of the day. An example of this is what was once called *typho-malarial fever*, a disease label dating to the period before the pathogenic etiology of malaria was determined in the late 1800s. Patients exhibited typhoid's gastrointestinal symptoms and malaria's debilitating effects, but the medical experts of the time saw these as symptoms of single disease. The existence of two separate but interacting diseases was not realized. Such confusion is understandable given the tendency for diseases of diverse origin to generate similar symptoms. Adding to the difficulty of identifying syndemics is the atypical disease expression that sometimes occurs when diseases interact.

Despite these challenges, it is possible to discern from the existing archival records the critical or at least influential role disease interaction has had on history. It is the argument of this chapter that attention to the issue of disease interface—which includes an analytical focus on the array of both local and wider social conditions that foster disease spread, clustering, and exchange—contributes to an improved understanding of the history of diseases and the diseases of history. The syndemics concept, in other words, is of value to both history and historical epidemiology.

The remainder of this chapter examines a number of past syndemics in their historical and social contexts:

- The Irish famine syndemic of 1741
- The Gibraltar cholera syndemic of 1865
- The Massachusetts scarlet fever syndemic of the 1800s
- The global influenza syndemic of 1918
- The syndemics among Native Americans on the American frontier
- The syndemics of the Mormon migration
- The syndemics of war

IRISH FAMINE SYNDEMIC OF 1741

On December 27, 1739, temperatures across Ireland fell below freezing; they felt even colder because of bitter easterly gales. Thus began the Great Frost, the most severe period of extreme freezing cold on record for Ireland and beyond. Although much of Europe was hit, the resulting crisis was particularly severe in Ireland. The blast of Arctic weather, a parting nod from the last ice age, hung on for almost two months, followed by an erratic spring that saw no rainfall. As a result, both cereal and root crops failed, and stored potatoes froze and became inedible; meanwhile, domestic animals died in great numbers, ill-preparing people for a snowy winter and continued drought the following year.

What followed was the first great Irish famine, characterized not only by the loss of the staple food sources but also by a painful economic recession tied to the breakout of war between Britain and Spain and a resulting loss of overseas trade. Tens of thousands of Irish were reduced to begging and to the consumption of famine foods like nettles, seaweed, and dock leaves. Rising malnutrition and unhygienic living conditions stemming from the faltering economy and a breakdown of the social infrastructure led in the later months of 1740 to a series of overlapping epidemics: typhus (caused by the louse-borne bacteria *Rickettsia prowazekii*), dysentery (caused by unsanitary drinking water containing various microorganisms), and relapsing fever (a tick- and body lice-borne infection caused by *Borrelia recurrentis*).

Soon mortality rates began to rise, peaking the following year. Although the records are seriously inadequate for establishing the actual death rate thrust upon the Irish by the famine, Lee (1989) draws on various sources to suggest a rate of mortality similar to that of the better known Irish potato famine of the 1840s, during which 10 percent of the population succumbed. As Clarkson and Crawford (2001) comment: "We can only guess at how many died, but on the basis of contemporary accounts and burials recorded in parish registers, there may have been between 300,000 and 480,000 famine-related fatalities in the two years 1740–1741, the majority of them in the south and east of the country" (p. 126).

The Irish people's experience of the catastrophe is expressed in the folk name given to the deadly famine: *bláin an áit*, or "year of the slaughter" (Post, 1977). According to one eyewitness: "Multitudes are daily perishing. . . . I have seen the labourer endeavouring to work at his spade, but fainting for want of food and forced to quit it. I have seen the aged father eating grass like a beast . . . the helpless orphan exposed on the dunghill, and none to take him in for fear of infection . . . the hungry infant sucking at the breast of the already expired parent" (BBC, 2008). Another observer commented: "We have a great mortality among the poor people, who die in great numbers from fevers and fluxes. One poor man buried eight of his family in a few days" (BBC, 2008). In even starker terms a third witness, the Reverend Philip Skelton, curate of Monaghan parish, recorded the demise of "whole parishes in some places . . . the dead have been eaten in the fields by dogs for want of people to bury them. Whole thousands in a barony have perished, some of hunger and others of disorders occasioned by unnatural, unwholesome, and putrid diet" (BBC, 2008).

In sum, the Irish famine syndemic of 1741 began with a mix of both environmental factors and political and economic factors that led to a radical drop in available food sources and a breakdown of the social and hygienic infrastructure. Malnutrition ushered in and united with several diseases of poverty to possibly kill one of every ten people in Ireland, with the poor disproportionately shouldering the brunt of the disaster. Although unique in its particular array of events and conditions, this syndemic shares many features of others that were to follow on the local or global stage of subsequent history. Unlike the well-known famine that was to strike the Emerald Isle 100 years later, this earlier crisis caused no migration to distant shores, a factor that has obscured popular awareness of it even within Ireland. On the summit of Killiney Hill, in Dublin, overlooking the wealthy neighborhood that sports the expansive mansions owned by people like Van Morrison and Bono, however, there is a an obelisk that commemorates the famine and the relief program that carried many families through the harshest days of the crisis.

out and ultimately became a world affair, with a significant number of deaths recorded in Europe and North America. John Snow (1855) described the arrival of cholera in London during this second epidemic wave:

The first case of decided Asiatic cholera in London, in the autumn of 1848, was that of a seaman named John Harold, who had newly arrived by the Elbe steamer from Hamburg, where the disease was prevailing. He was seized with cholera on the 22nd of September, and died in a few hours. Now the next case of cholera, in London, occurred in the very room in which the above patient died. A man named Blenkinsopp [suffered] rice water evacuations [profuse diarrhoea], and, amongst other decided symptoms of cholera, complete suppression of urine from Saturday till Tuesday morning, and after this the patient had consecutive fever [p. 1].

A third pandemic commenced in the 1850s and took its major toll primarily in Russia. The fourth pandemic began in 1863 and raged across Europe and Africa, hitting Gibraltar in 1865. It is its impact on Gibraltar, or the Rock as it is known colloquially, that is of concern here.

From the researcher's point of view, there is an excellent reason for focusing on this tiny European country of 3.6 square miles, with a population that even today does not surpass 30,000. Larry Sawchuk, a professor of anthropology at the University of Toronto, and his colleagues and students have pored over historical archives to assess the country's social history of health and disease, including syndemically related diseases. These records (Sawchuk 2001), which are linked to Gibraltar's status since 1713 as a British military garrison and colonial outpost (though physically connected by a small thread of land to Spain and 1,000 miles from Britain), include the Gibraltar Police Death Registry, the Gibraltar Government Census of 1868, and the Colonial Blue Books (a record of death counts). An important finding of careful examination of this archival material is that cholera had the single greatest impact of any disease on the life expectancy of people on the Rock during the mid-1800s and was responsible for reducing the average duration of life by twelve years.

A particular configuration of social conditions and social relations formed the context for health on Gibraltar in the mid-nineteenth century. The local "medical topography" was summarized by Hennen (1839), a British physician who subsequently died in an epidemic on the Rock, as being strongly influenced by a population that was "overcrowded, perhaps, beyond any community in the world" at the time (p. 71). The dwellings of the poor were described as being small, dirty, and poorly ventilated, places at high risk, Hennen believed, for febrile miasmas, a view in keeping with the prevailing ideology of disease causation at that time. The cause of these conditions, as Sawchuk and Burke (2003) observe, "was the fact that landowners were more concerned with making large and quick profits than with providing proper accommodation for the poor working classes" (p. 178). Further, as a garrison town Gibraltar had come to be a temporary home to several thousand military families. The regular relocation of troops

GIBRALTAR CHOLERA SYNDEMIC OF 1865

Cholera possesses the dubious distinction "of being one of the most fatal diseases in history" (Sawchuk & Burke, 2003, p. 199). The earliest known reference to this deadly ailment dates from fourth-century B.C. India, a land where cholera has been endemic since ancient times. The world, of course, came to know cholera not as a distinctly Indian or even Asian disease, although it still sometimes is referred to as Asian cholera, but rather as a Grim Reaper making periodic sweeps of the planet. People outside of India began witnessing pandemic undulations of cholera in the 1800s, a century in which there were six great waves, each of which, "[l]ike a forest fire . . . pushed along a rough direction and sparked blazes sporadically in its way" (Auyang, 2003, p. 12). The first of these waves began in 1820 and spread across India and up into China to the east and to the Caspian Sea to the west, leaving in its wake a trail of death and suffering. Military movements during the Russian, Persian, and Turkish wars (1826–1829) and the crushing of the 1831 Polish revolt helped to spread the disease during this outbreak. Twenty years later a second cholera pandemic broke

from Gibraltar to sites across Britain's dispersed empire offered a ready mechanism for population mixing and the continual arrival of infectious diseases. Additionally, access to clean water was minimal, with the purity of the well water continually sabotaged by the poor disposal of sewage and refuse. At the same time, adequate nourishment was also a problem, especially during the hot summer months. Finally, maintenance of domestic animals in close proximity to dwellings resulted in additional pollution. For the poorer families of Gibraltar, susceptibility to disease "was not simply the result of a lack of wealth or lax hygiene, but, rather, a constellation of factors that were experienced together in a poverty complex" (Sawchuk & Burke, 2003, p. 203).

Within this social environment, the people of Gibraltar were regularly beset by what Sawchuk and Burke refer to as the *pathogens of everyday life*. By this they mean a cluster of persistent endemic infections that routinely ensured significant rates of morbidity and mortality. During the first years of the colony, years in which the residents' lives were fragile at best, diarrheal diseases—"the product of synergy between enteric infections, malnutrition, repeated insults by infectious disease, and a poor weaning diet that [set] up a vicious cycle of gastroenteritis/malnutrition lowering overall disease resistance"—resulted in considerable and consistent sickness and death among children (Sawchuk, Herring, & Waks, 1995, p. 619). Moreover, "even infants who survived the infection were prone to other opportunistic infectious diseases" as well as to "the periodic outbreak of synergistic infectious diseases such as measles" (Sawchuk & Burke, 2003, p. 188). In adulthood, respiratory tuberculosis, another endemic condition, was the primary cause of death of Gibraltarians during this era. Records suggest a high annual rate of TB infection, with virtually everyone being infected by age twenty. In addition, an array of respiratory conditions formed a "disease cluster" that included pneumonia, bronchitis, and other inflammations of the lungs (Sawchuk & Burke, 2003, p. 189).

This overall description suggests important components of the local medical topography (to borrow Hennan's term again) on Gibraltarians at the time of the arrival of cholera in the summer of 1865, thrusting Gibraltarians from high but normal levels of mortality from various diseases into an episode of what is referred to in demography as *crisis mortality*. During this brief period, almost 600 people died. Mortality was not equally distributed in the population, however. Simply put, "poverty put some Gibraltarians at greater risk than others" (Sawchuk & Burke, 2003, p. 203). It was among the poor that cholera—as a result of its syndemic interaction with debilitating social conditions, malnutrition, and various other infectious diseases—wreaked the greatest havoc and took the steepest toll.

MASSACHUSETTS SCARLET FEVER SYNDEMIC OF THE 1800S

In her classic novel *Little Women*, Louisa May Alcott uses the expression "castle in the air" metaphorically to refer to the gap that commonly emerges between one's dreams and lived reality. First published in 1868, the book describes the lives and relationships of four sisters growing up in Massachusetts during the Civil War. One of the sisters, Beth, the second youngest, a shy but charitable girl, experiences the death of a neighbor's

baby in her arms. Soon she too develops symptoms of the disease that took the baby's life. She explains to her sister Jo what happened when the baby died: "I just sat and held it softly till Mrs. Hummel came with the doctor. He said it was dead, and looked at Heinrich and Minna, who have sore throats. 'Scarlet fever, ma' am. Ought to have called me before,' he said crossly. Mrs. Hummel told him she was poor, and had tried to cure the baby herself, but now it was too late, and she could only ask him to help the others and trust to charity for his pay" (Alcott, 1872, p. 166).

Realizing that she too has been infected, Beth tries to convince her sister that she will be all right: "'Don't be frightened, I guess I shan't have it badly. I looked in Mother's book, and saw that it begins with headache, sore throat, and queer feelings like mine, so I did take some belladonna, and I feel better,' said Beth, laying her cold hands on her hot forehead and trying to look well" (p. 166). At first, it appears that Beth will survive her bout with scarlet fever, but she develops rheumatic fever and ultimately dies of congestive heart failure, affirming the metaphor around which the book is constructed. During the time that Alcott was writing *Little Women*, the world was in the midst of a pandemic of scarlet fever that ran from 1820 to 1880. Notes Krause (2001):

[S]carlet fever most likely occurred for centuries either as an endemic disease or as localized epidemics. And then, in the early part of the 19th century, a pandemic of often fatal scarlet fever appeared suddenly and swept through Asia, Europe, and the United States.... Physicians in 1830, reflecting on their past experience, noted a striking increase in mortality not seen previously, and fatality rates of up to 30% were often reported. Scarlet fever became the most common fatal infectious childhood disease, more fatal than measles, diphtheria, or pertussis, a fact that is difficult to comprehend today [p. 15].

One of the places hit by the pandemic, in 1858–1859 and again in 1867–1868, was the Connecticut River Valley of western Massachusetts. Anthropologists Alan Swedlund and Alison Donta (2003) of the Connecticut Valley Historical Demography Project have studied archival records of this segment of disease history, focusing their research on the population of a fertile agricultural valley at the onset of the industrial revolution and especially on four towns spread across the valley. These towns, Deerfield, Greenfield, Montague, and Shelburne, varying in their level of dependence on agriculture, commercial, and industrial economies, form a natural laboratory. Although all these towns had doctors when scarlet fever arrived, there were no effective medical treatments at the time for the disease. (Indeed, even after *Streptococcus pyogenes* was identified in the third quarter of the nineteenth century as the immediate pathogenic cause, no effective treatment became available until the emergence of antibiotic pharmacology.)

The 1858–1959 epidemic in the Connecticut River Valley was severe. Throughout the state of Massachusetts this first scarlet fever epidemic caused over 2,000 deaths, 95 percent of which were among children. In the four towns studied by Swedlund and

Donta, there were over 89 deaths among children that were tied to scarlet fever in 1858–1859 and 1867–1868. In Deerfield, for example, twelve households lost children to scarlet fever in 1859. The average age of those who died was three years and eleven months. In examining cases of scarlet fever in the target towns, it was not evident to research team members that nutritional or socioeconomic characteristics were significant risk factors for scarlet fever infection. They note, however, the importance of “the close and perhaps synergistic interrelationship between nutrition and infection that . . . can be influential not so much in who becomes infected, but in who survives” (Swedlund & Donta, 2003, p. 17). As noted in Chapter Six, nutrition plays an important role in immune health, including the ability of mothers to pass temporary immune protection to newborns.

GLOBAL INFLUENZA SYNDEMIC OF 1918

A world-shaping syndemic was sparked by the global influenza pandemic of 1918, which epidemiologists estimate was responsible for the deaths of between 40 and 100 million people worldwide, making it one of the most deadly events in human history.

More people died during the 1918 pandemic, which lasted two years, than during all four years of the Black Death outbreak (although of course the world population was by 1918 far larger than it had been during the Middle Ages). Arriving during the closing phase of World War I, the pandemic had a significant impact on mobilized national armies. Half of the U.S. soldiers who died during the Great War, for example, were victims of influenza, not of enemy bombs and bullets. It is estimated that almost three-quarters of a million Americans died during the pandemic. Its impact was so harsh that the average life span in the United States fell by ten years in the second decade of the twentieth century. Among those struck was President Woodrow Wilson, who became ill early in 1919 while in Versailles negotiating the treaty to end the world war (Tice, 1997).

The pandemic was truly global in its impact, showing up in almost all heavily populated areas but in sparsely populated Arctic settlements and in remote Pacific Islands as well. In this respect the pandemic exposed the early stages of the most recent wave of globalism and its reconstruction of the modern world as a socially smaller, more integrated place.

The lingering if muted presence of what has been called the **forgotten pandemic**

(Crosby, 2003) is captured by Restivo (2000):

There is a cemetery in a small railroad town in northern Ohio where I grew up that tells a sliver of the story of the great ‘Spanish’ influenza pandemic of 1918. One section of the cemetery is full of simple, rough, limestone markers that sit willy-nilly in the perpetually damp sod of the graveyard. These unembellished memorials mark the graves of unfortunate souls who died one week during the fall of 1918 from the flu. In other towns all over the country there are similar hastily prepared grave sites. At least 10,000 people died in the city of Philadelphia during one three-week period during the month of October, overwhelming doctors, hospitals and undertakers [p. 12].

Moreover, as the panicked reaction to the recent SARS outbreak suggests, perhaps the 1918 epidemic is not such a forgotten pandemic after all.

An important part of the death toll in 1918 and 1919 was caused by viral pneumonia characterized by extensive bleeding in the lungs and resulting in suffocation. Many victims died within forty-eight hours of the appearance of the first symptoms. In fact it was not uncommon for people who appeared quite healthy in the morning to have perished by sunset. Historian Adolph Hoehling (1961), for example, relays the remembrances of Henrietta Burt, a secretary, who visited a friend’s home to play bridge during the epidemic: “We played until long after midnight,” she recalled. “When we left we were all apparently well. By eight o’clock in the morning I was too ill to get out of bed, and the friend at whose house we played was dead” (p. 163). Eino Hautala, a child of eleven living on his family’s farm in Minnesota, remembered a local barn that was pressed into use as a morgue. Because it was a particularly brutal winter and the ground was frozen, inside the barn the bodies of flu victims were “stacked like cordwood until the spring came” (“Personal Histories,” 2006).



The devastating impact of the 1918 pandemic on the U.S. military during World War I led the nation’s surgeon general to establish the Pneumonia Commission in July of 1918 to investigate the nature and cause of the disease outbreak. Among the five members of the commission was Captain Francis G. Blake. In letters that he wrote while on the commission, Blake poignantly described the misery caused by the 1918 pandemic on U.S. army bases. On September 25, for example, he wrote from Camp Pike: “You ought to see this hospital tonight. Every corridor and there are miles of them with a double row of cots and barracks about the Camp turned into emergency infirmaries and the Camp closed” (quoted in Pettit & Baille, 2008, p. 92). A few days later, after another round of new cases, Blake wrote, “I am getting too tired to write about anything that is going on here. There is only death and destruction anyway” (p. 95).

Even before the 1918 pandemic, researchers were confused about what caused the *grippe*, as the disease was then known. Some thought that the source might be a bacterial infection. At the time, virology had not emerged as a discipline, but bacteriology was already a flourishing field. As Crosby (2003) notes, at autopsy, “By far the commonest microorganism in the lungs of influenza victims was found to be Pfeiffer’s bacillus [*Bacillus influenzae*]” (p. 40), and hence this microbe was at one time proposed as the source of the pandemic. Subsequent research, however, showed that it was not present in every case, provoking caution about its role. Writing during the second year

of the pandemic, John Ruhrhah (1920), a professor of diseases of children at the University of Maryland, summarized what the scientists knew:

In 1892, Pfeiffer announced his discovery of the influenza bacillus and since that time to the present this has generally been credited with being the cause. These statements have not, however, passed entirely unchallenged and Rosenthal, in 1900, and others, have questioned the role of the Pfeiffer bacillus in influenza. In 1910, Vincent suggested that epidemics of grippe might be due to a filtrable virus. In the epidemic of last year the bacillus of influenza was reported in large numbers by certain observers and in smaller numbers by others, and sometimes it was not found at all. The Conference of Bacteriologists at the British War Office under Leshman believed that the influenza bacillus played a part of great importance in the epidemic, although they were not sure of its being the primal cause [p. 160].

Ultimately, it was confirmed that the pandemic was caused by a virus, specifically an influenza A virus of the H1N1 subtype (Taubenberger & Morens, 2006). By way of a virological or epidemiological pathway that is not yet fully understood, the virus had successfully “jumped” the species barrier (presumably through a genetic mutation that allowed infection of human cells). The origin species was likely avian, as ducks and geese appear to be the primary animal reservoirs for all known influenza viruses (although horses, pigs, and even marine mammals can be infected). Although in humans (and bird species like chickens) influenza is a respiratory infection of varying degrees of intensity, in waterfowl the infection targets the gastrointestinal tract and tends to produce subclinical infection.

Among people who survived the first several days of infection during the pandemic, however, many subsequently died of secondary conditions. Most notable among these was bacterial pneumonia, and it was this tendency to promote secondary complications that made the 1918 influenza so deadly. As summarized by Morens and Fauci (2007), individuals suffering from viral infection during the 1918 pandemic were at risk for the development of “aggressive bronchopneumonia featuring epithelial necrosis, microvasculitis/vascular necrosis, hemorrhage, edema, and widely variant pathology in different parts of the lung, from which pathogenic bacteria could usually be cultured at autopsy. . . . In a few autopsies, severe bronchopneumonia was seen without evidence of bacteria, but studies generally showed a close correlation between the distributions of pulmonary lesions and cultured bacteria . . . identifying the major bacteria as the organisms now known as *S. pneumoniae*, *S. pyogenes*, and, less commonly, *Haemophilus influenzae* and *Staphylococcus aureus*” (p. 1020).

In other words, the bacteria found in the lungs of many victims that at first were thought to be the source of the influenza were actually evidence that a syndemic interaction involving a virus and one or more strains of bacteria was an important factor in the lethality of the 1918 pandemic. This interpretation is supported by research with

animal models that identified a lethal synergism between influenza virus and *Streptococcus pneumoniae* (McCullers & Rehg, 2002). This research showed that if influenza infection precedes bacterial infection, there is a 100 percent rate of mortality. If the reverse occurs and bacterial infection precedes viral infection, a countersyndemic reaction occurs that confers protection from influenza and leads to improved rates of survival.

Moreover, it has been argued that countless numbers of those who expired quickly from the disease were coinfected with tuberculosis, which would explain the plummet in TB cases after 1918; many carriers of the disease may have perished during the pandemic. Noymer and Garenne (2000), who believe that “[t]uberculosis and influenza very likely interacted in 1918” (p. 573), analyzed a data set of 1918 influenza cases originally developed by Raymond Pearl, a well-known biologist and statistician long affiliated with Johns Hopkins University, who exhibited an early interest in disease interactions (for example, Pearl, 1919, 1928). Although Noymer and Garenne (2000) noted the limitations of the data set (for example, a lack of controls for age, sex, and socioeconomic status, a focus only on white households, and the inclusion only of households with at least one case of tuberculosis), their regression analysis led them to conclude that “many influenza deaths in 1918 took place among the tuberculous—persons with clinical disease or latent infection with *Mycobacterium tuberculosis*. That the 1918 influenza virus, known to be atypical, should interact pathologically with *M. tuberculosis* seems likely” (p. 573). It is this syndemic interaction, they assert, which accounts for various features of the 1918 epidemic, including both its extreme mortality rate and the age group (twenty-five- to thirty-four-year-olds) that was hardest hit (tuberculosis being a disease of adulthood, not old age). The significant drop in tuberculosis cases after 1918 (likely due to the high fatality rate during the epidemic) can in this light be interpreted as a countersyndemic outcome, although one with a high initial cost in human lives.

SYNDEMICS AMONG NATIVE AMERICANS ON THE AMERICAN FRONTIER

An example of a consequential syndemic from the nineteenth century can be found on the reservations on which indigenous peoples were confined with the closing of the U.S. frontier. For example, the Sioux (a term derived from the French spelling of an Ojibwa word for “little snakes” that was officially applied to the Lakota, Dakota, and Nakota peoples in 1825) are known historically for their abilities as mobile foragers and fierce warriors, but were militarily forced by the U.S. government onto a series of small reservations, including Rosebud, Pine Ridge, Lower Brule, Crow Creek, Cheyenne River, and Standing Rock, where they could not practice their previous subsistence patterns (for example, the last bison hunt occurred in 1882, by which point not a single wild bison remained in Lakota territory) and were treated as a conquered population. The Rosebud Sioux, for example, descendants of the Sicangu Oyate of the Tetonwan Division of the Lakota people, had their broader tribal homeland recognized

by treaties signed in 1851 and 1868 but then watched it being reduced by the U.S. Congress to its current boundaries in south central South Dakota as part of the Great Sioux Agreement of March 2, 1889. Moreover, the U.S. government reneged on its promises to adequately provision Sioux reservations; such failures to respect treaty terms with Native Americans were typical. Food that was provided was often of low quality. Living under extremely stressful conditions and having inadequate diets, as well as being the victims of overt racism on the part of the registration agents appointed to oversee their reserves, the Sioux suffered exposure to various infectious diseases transmitted by contact with whites.

One source for the Sioux experience with European disease is indigenous records. The Sioux subgroups or bands traditionally documented their histories using a calendrical system known as the *waniyetu wowapi*, or “winter count.” These pictorial calendars, produced by designated tribal historians, or “keepers” (each affiliated with a subgroup or band maintaining its own records), consisted of drawings on muslin cloth or hide that recorded defining events during each year. The images used and name of the year were selected by the tribal historian in consultation with band elders. Some of these calendars are held by the Smithsonian Institution. A record in the Smithsonian collection for the year 1849–1850, for example, depicts a man suffering from stomach pains and diarrhea and is named “Many died of the cramps.” For the following year, 1850–1851, another winter count drawing portrays a rough human shape covered with spots and is named “Many died of the smallpox.” For the year 1873–1874, a drawing of a man covered with blotches is named “Measles and other sickness used up the people in winter” (Greene & Thornton, 2007). As these examples suggest, winter count records indicate a series of epidemics that took many lives and caused much illness among reservation residents.

During the closing years of the nineteenth century, the Sioux endured a number of particularly lethal epidemics. Charles Alexander Eastman (1936/1977), a Sioux physician and writer who worked on the Pine Ridge Reservation during this period, recorded his observations and reactions to the events he witnessed: “Rations had been cut from time to time; the people were insufficiently fed, and their protests and appeals were disregarded. Never was more ruthless fraud and graft practiced upon a defenseless people than upon these poor natives by the politicians! Never were there more worthless ‘scraps of paper’ anywhere in the world than many of the Indian treaties and Government documents! Sickness was prevalent and the death rate alarming, especially among the children” (p. 99).

Black Elk, a noted Sioux folk healer, told his biographer: “There was hunger among my people before I went across the big water to Europe in 1886, because the Wasichus [whites] did not give us all the food they promised in the Black Hills treaty. . . . But it was worse when I came back [1889]. My people looked pitiful. . . . We could not eat lies and there was nothing we could do” (Neihardt, 2004, p. 177). James Mooney (2006), an anthropologist and representative of the Bureau of Indian Affairs sent to investigate a possible Sioux rebellion, described the health situation on the reservations similarly in 1896: “In 1888 their cattle had been diminished by disease. In

1889, their crops were a failure. . . . Sullenness and gloom, amounting almost to despair, settled down on the Sioux. . . . The people said their children were all dying from the face of the earth, and they might as well be killed at once. Then came another entire failure of crops in 1890, and an unexpected reduction of rations, and the Indians were brought face to face with starvation. Thus followed epidemics . . . with terrible fatal results” (pp. 826–827).

During this period, the Sioux endured epidemics of measles, influenza, and pertussis in rapid succession. Tuberculosis and skin-based TB diseases like scrofula were also frequent. Other common diseases among those living on the reservations were bronchitis, pneumonia, and pleurisy. As this list of clustered health conditions suggests, it is likely that during this period the Sioux were victims of a syndemic that combined a number of interacting infectious diseases, malnutrition, self-medication alcohol abuse, and stressful and extremely disheartening life conditions. As a result, while the official annual mortality rate on the Sioux reservations was between 1 and 2 percent, the actually death rate is believed by a number of historians to have been probably closer to 10 percent, a devastatingly high toll that helped to shape the final phases of frontier history.

SYNDEMIC OF THE MORMON MIGRATION

The Western frontier and the mid-nineteenth-century westward migration have long been seen as compelling influences in U.S. cultural development, giving rise to the so-called frontier mentality that has often been proposed as the source of everything from a core cultural emphasis on individualism and self-reliance to high rates of interpersonal violence, an enduring social restlessness and geographical mobility, a profound automobile obsession, and a distinctive exuberance and action orientation. Tempering the optimistic aspects of the frontier experience, however, were persistent epidemic cycles and overlapping lethal diseases that cut life short and left lonely grave sites all along the migration pathways (McNeil, 1980).

Among the thousands of migrants from European and East Coast points of origin were those who headed west to Zion as members of the Church of Jesus Christ of Latter-day Saints (LDS). The Mormons, as church members came to be known, moved westward in droves, some 70,000 between 1840 and 1869 (the year the transcontinental railroad was completed), in one of the largest organized migrations in American history. As Black et al. (1998) report, the migration presented both emotional and physical challenges: “The long journey was filled with the natural fears of fatigue, possible illness, concern for food and water, questions about the durability of their wagons or handcarts or their domestic animals, the potential dangers of Indian attacks—indeed, all the fears that go with the unknown. Adding to these fears was the sorrow many felt as they left extended family members behind, most never to be seen again” (p. 40).

Although it would be impossible to calculate actual morbidity rates during the migration, LDS historians, using the vast genealogical and other archival data collected and maintained by their church, estimate that between 4,600 and 6,000 Saints (6.6 to 8.6 percent of their total migrant flow) died en route, either while crossing the ocean

from Europe or on one of the land trails leading to their final destination in Utah or contiguous areas (Black et al., 1998). The immediate causes of death included violence, accidents, and acute diseases. Among the latter cholera was particularly significant and was responsible for many deaths among the European Mormon immigrants who landed in New Orleans between 1849 and 1854. A major epidemic of yellow fever also hit New Orleans in 1853, resulting in the deaths of one of every fifteen people and leading city folk to say that since so many people were dying "soon people will have to dig their own graves" (Louisiana State Museum, 2007). To limit exposure to epidemics along the way, Brigham Young, head of the Mormon church after the death of founder Joseph Smith, was forced to send messages to European migrants instructing them to avoid disembarking in New Orleans.

In addition to acute diseases, other health factors played important roles in contributing to Mormon migrant mortality; as Baker (2002) observes: "Malnutrition, intestinal parasites, or other chronic disease conditions made a person more susceptible to disease. Insufficient food, combined with inadequate sanitation, represented the greatest factor that led to serious outbreaks among Mormon immigrants at sea" (p. 86). Kimball (quoted in Baker, 2002) adds: "Injury, sickness and death were commonplace. Emigrants suffered cuts; broken bones; gun wounds; burns; scaldings; animal, insect and snake bites; stampedes; overturned wagons; shifting freight; drownings; quicksand; black scury [acute fever with hemorrhagic skin lesions]; black cancer (probably diphtheria); cholera; typhoid fever; ague [malaria]; quick consumption (tuberculosis); headaches; piles [hemorrhoids]; rumps; asthma; inflammation of the bowels; scrofula; erysipelas [streptococcal skin infection]; diarrhea; small pox; itch; and infections of all kinds, including puerperal fever" (p. 86). Similarly, Mormon church records (Church of Jesus Christ of Latter-day Saints, 1996) note that conditions at longer term encampments set up by the Mormons before moving on to Utah were also characterized by multiple threats to health: "Life in these settlements was almost as challenging as it had been on the trail. In the summer they suffered from malarial fever. When winter came and fresh food was no longer available, they suffered from cholera epidemics, scurvy, toothaches, night blindness, and severe diarrhea" (p. 60).

As these accounts suggest, the significant toll taken by syndemically related diseases, disorders, and injuries was a critical aspect of the Mormon migration to Zion, and likely not one of minor historical significance for the survival of the Mormon church. Enduring sickness, death, and other hardships and sacrifices provided the survivors with important "faith promoting experiences" (Kimball, 1995) that no doubt helped to increase Mormon resolve and contributed to group solidarity and organization in the face of subsequent challenges, including, ultimately, open conflict with the U.S. government over the practice of polygamy and, more importantly, control of the Western territories, conflict that led to federal arrest of group leaders, military occupation of Salt Lake City, and the temporary confiscation of church property. In response to the health and other challenges faced during the migration, the Mormon church developed an internal system of support and welfare that continues to serve its members.

SYNDEMIC^S OF WAR

War and other forms of physical conflict rival infectious disease as global causes of morbidity and mortality. Moreover, they are breeding grounds of syndemics. Since the bloody end of World War II, there have been at least 160 wars around the world with as many as 25 million (and probably many more) people killed, most of them civilians. Directly or indirectly, war touches the lives of most people on the planet, often with enduring and costly impact. Preparation for war takes a further toll by diverting a people's resources from the health sector and other social needs. Further, because war destroys housing, disrupts social institutions, and displaces persons, it creates impoverished and highly vulnerable refugee populations (currently totaling 12 million people worldwide, as well as 20 to 25 million internally displaced persons) who often face severe problems of adequate nutrition, exposure to contagious diseases, and substandard health care.

In a famous passage about the intimate relationship of war and disease, the renowned bacteriologist Hans Zinsser (1925/2000) observed, "Soldiers have rarely won wars. They more often mop up after the barrage of epidemics" (p. 153). In the defeat of New World empires, for example, European diseases such as smallpox and measles were far more deadly than European weapons. Similarly, during the American Civil War, even though the average soldier might have feared the dreadful minié ball as his greatest threat to life and limb, "disease was the biggest killer of the war" (Civil War Society, 2002). In the federal army roughly three out of five died of disease, and among Confederate soldiers two out of three deaths were caused by disease (Bollet, 2004). Typhoid, dysentery, and malaria, often in some combination, were the primary pathogen-generated enemies on the Civil War battlefield. Together they have been called the third army that participated in the war. Similarly, despite the enormous firepower of modern warfare, in World War II's Pacific theater casualties were greater from diseases like malaria than they were from actual combat.

Usually an unintended consequence, disease also has been used intentionally as a weapon of war. This is not a new strategy; for example, during the fourth century B.C. Scythian archers covered the tips of their arrows with animal excrement to cause wound infections; during the Middle Ages the corpses and feces of individuals who had died of bubonic plague were catapulted over castle walls onto besieging armies; and more recently, during World War II, Winston Churchill at one point contemplated an anthrax attack on German cities. Indeed, in modern times biological warfare has come to be a grave threat.

Twin Scourges: War and Disease

The nature of the connection between war and disease, two of the greatest sources of human suffering, is complex; disease can trigger human conflict but it can also so decimate armies (as HIV/AIDS has done in the case of several African armies) that it forces a suspension or even an end to fighting. In either case, soldiers and civilians on

both sides of the battlefield have endured often terrible health consequences (Smallman-Raynor & Cliff, 2004). Further, war has played a role in creating new diseases, reactivating old ones, and significantly elevating the impact of others, and, as a result the popular names of a long list of diseases reflect their close association with the battlefield environment: for example, trench fever, an infection caused by the louse-borne pathogen *Bartonella quintana*; trench foot, a condition characterized by blisters, open sores, and swelling and caused by prolonged exposure of the feet to a damp, unsanitary, and cold environment; trench mouth, a severe form of infected and bleeding gums; soldier's disease, an old term for opium addiction stemming from the use of opium to treat the pain of war wounds; and combat stress reaction (also called battle fatigue, shell shock, war neurosis, and soldier's heart, a lexical proliferation suggesting its frequency), an intense and usually short-term stress reaction to war experiences that impairs mental functioning (and which in prolonged form is now called posttraumatic stress disorder).

Additionally, down through the ages, as seen in the global transmission of cholera during the nineteenth century and the influenza pandemic of 1918 (which was first observed in the United States at Fort Riley in Kansas, a site so severely hit by the outbreak that the army ran out of coffins to bury the many victims), war has been a handmaiden of syndemics in many if not all regions of the world. By directly causing massive death and injury on the one hand, and by disrupting trade and destroying social infrastructure, including subsistence, hygienic, and medical systems, on the other, war has often set the stage for famine and epidemic diseases, or as often as not both at once as syndemically interacting sources of illness and death. Further, as soldiers are moved about the landscape of shifting battlefields, war has been a major spreader of disease, and consequently it has also brought diseases into contact that were previously isolated by geographical distance or topological barriers. War, in short, has been a powerful syndemogenic force in world history. As a result, the nexus of war and syndemics has played a major role in shaping the worlds as we know it.

Darfur: Physical Violence, Social Suffering, and Syndemics

A particularly grim and disturbing expression of the impact of war on health is the toll conflict takes on the lives of children. Countries that have been ravaged by internal wars, such as Sierra Leone, Angola, Afghanistan, Liberia, and Somalia, tend to have the highest child mortality rates in the world. In Sierra Leone, for example, over 250 out of every 1,000 children die before reaching five years of age, compared to 8 per 1,000 in the United States, according to UNICEF (Machel, 1996). Since 1990, 90 percent of conflict-related deaths have been among civilians, and of these 80 percent have been among women and children. UNICEF reports that in a typical war the death rate among children under five years of age goes up by 13 percent. The causes are multiple and usually entwined and include accidental or intentional violence (for example, military bombardment of civilian residences, gang rape by soldiers of women and girls, and ethnic cleansing); the spread of diseases facilitated by the destruction or chaos-driven breakdown of societal infrastructure; and the creation of vulnerable populations like refugees, who often wind up in overcrowded camps with inadequate

or polluted water, lack of adequate food supplies, unhygienic living conditions, and limited health care. Moreover, many children are left orphaned, while others are abducted to be used as compliant soldiers.

The emotional scars children bear from exposure to violence, loss of family members, dislocation from their homes, their resulting impoverishment, and other social suffering can be lifelong. Typical is Sumaya, a fifteen-year-old girl from the Sudan, whose village was attacked in 2005 by an Arab tribal militia called the Janjaweed. Sumaya, one of over 2.5 million Sudanese refugees, was interviewed by UNICEF workers in the Kalma refugee camp in South Darfur:

"I was at school when they attacked us," says Sumaya. My sisters ran back to the village, and I ran with some friends. My cousin Mona was running ahead of me when she was shot. I stopped and held her hand. When she died, her hand slipped out of mine. Some boys came and told me that I had to run, so I did." Along the way Sumaya found her grandmother and her 4-year-old brother, Mozael (whom everyone calls Baba). She took the little boy in her arms and started running. "We ran and ran until I felt that I couldn't go on any longer," remembers Sumaya. "I thought about throwing my brother in the grass because he was so heavy, but my grandmother took my hand and told me that we should all stay together." Two agonizing weeks went by before Sumaya and Baba were reunited with the rest of the family. Together, they walked 147 kilometres to Kalma Camp [along with 70,000 other refugees] [UNICEF, 2006].

By the time the refugees of Darfur reached refugee camps, many children had been pushed to the brink by a combination of trauma, lack of food and water, and disease. Exemplary is Mukhtar, a one-month-old child treated by physician Sayid Obeid Bakhet at his small clinic in the camp at Zam Zam in the Sudan.

The boy's legs were limp. Folds of skin hung loosely from his bones, easily holding the shape of the doctor's pinch—a telltale sign of dehydration. His face glowed with fever, and his narrow chest heaved and fluttered. His milky eyes darted desperately around the dim tent. He was a month old but weighed less than five pounds. Dr. Bakhet knew immediately that Mukhtar needed attention at once. His mother, Mariam Ahmed, a fire of panic burning in her eyes, urgently pressed the tiny child into the doctor's arms. "He vomits everything," she said. "It looks like he cannot breathe." Dr. Bakhet listened to the boy's labored chest and shook his head. "Pneumonia," he said. He felt the soft spot on top of Mukhtar's still-forming skull. It was sunken. "Dehydration," he added. . . Dr. Bakhet grew nervous. He had lost one little boy 10 days earlier to a deadly combination of disease just like Mukhtar's and could not bear to see it happen again [Polgreen, 2006].

Labeled by many observers as an act of genocide by the central Sudanese government against the impoverished and restive people of the Darfur region, and deemed the “world’s worst humanitarian crisis” by the United Nations in 2004 (BBC, 2004), the tragedy in Darfur largely escaped world attention until tens of thousands of people (ultimately 450,000 in some estimates) had already fallen victim to the deadly syndrome that united war, gastrointestinal diseases, malaria, other infectious diseases, dehydration due to lack of water and to intestinal infection, and malnutrition. The government’s announcement in 2005 that the ABCO corporation had begun drilling for oil in Darfur prompted some humanitarian workers to conclude that the government’s brutal scorched-earth policies were intended to drive people off the land to open it to drilling by transnational oil companies.

SUMMARY

This chapter has examined a set of syndemics that played major roles in shaping local, national, or even international social histories. The purpose was to show that syndemics can dramatically alter individual lives, the course of social events, and the character of human experience in suffering over time.

KEY TERMS

- forgotten pandemic
- pathogens of everyday life
- social suffering
- syndemics of history
- syndemics of war

QUESTIONS FOR DISCUSSION

1. What are the challenges in assessing the role of syndemics in shaping the course of human history? Why were syndemics in the past often not recognized as disease interactions?
2. Discuss the role of syndemics in the Irish famine of 1741, the deterioration of health among Native Americans following internment on reservations, the health problems faced by Mormon migrants to Utah, the cholera epidemics in Gibraltar in 1865, and the scarlet fever epidemics of the nineteenth century.
3. The global influenza pandemic of 1918 has reemerged as an issue of keen public health interest because of the growing recognition that we are at significant risk for future influenza pandemics. How did syndemic disease interaction play a key role in the health impact of the 1918 pandemic? Discuss the countersyndemic aspects of the 1918 global influenza with regard to tuberculosis.
4. How does war contribute to the development of syndemics?

CHAPTER

8

A WORLD OUT OF BALANCE

Emergent and Reemergent
Ecosyndemics

After studying this chapter, you should be able to

- Recognize the reasons for the rapid rise in the number of emergent and reemergent diseases and the resulting rapid rise in the potential for novel syndemics to form.
- Understand the role of the environment and environmental change in disease interactions and syndemic development.
- Be familiar with studies that explicate the role of animal populations as reservoirs for future syndemics.
- Define and work with the concepts of superinfection and the iatrogenic syndemic.
- Appreciate the potentially significant ability of global warming to produce ecosyndemics.

EMERGENT SYNDEMICS OF A TROUBLED WORLD

The capacity of various emergent diseases to spark new, environmentally based syndemic interactions is exemplified by the sudden 1993 outbreak of cryptosporidiosis in Milwaukee, which infected as many as 400,000 people. Cryptosporidiosis has also