PART

1

INTRODUCING KEY CONCEPTS IN SYNDEMICS

Chapter One, "Learning from Lichen: Reconceptualizing Health and Disease," which makes up Part One of this book, introduces the reader to the concept of syndemics and to the biosocial syndemic perspective on human health. Examining the syndemic perspective in a historical context, it describes the developing recognition that health and illness are shaped by multiple and complex factors, and it shows how the identification and study of syndemics has grown out of these far-reaching shifts in the ways that we conceptualize disease.

CHAPTER

1

LEARNING FROM LICHEN

Reconceptualizing Health and Disease

After studying this chapter, you should be able to

■ Locate the syndemic perspective within the evolutionary history of the scientific understanding of disease, including dilemmas encountered in meeting Robert Koch's criteria when attempting to determine the cause of an infectious disease.

■ Understand the syndemic approach as one that supersedes two limitations of conventional biomedical approaches to disease—reductionism and mind-body dualism.

■ Recognize the fundamental importance of biosocial interconnections and relationships in syndemics theory.

■ Explain why syndemics were often not recognized in the past.

■ Explain how the consideration of social factors, such as social disparity, differentiates syndemic processes from the biomedical conception of comorbidity and also differentiates syndemics among humans from synergistic disease inter-actions among animals.

ON NOT PLANTING CUT FLOWERS: THE WEIGHT OF HISTORY

It was just a few decades ago, in the 1970s, that medical anthropology, the source discipline for the syndemics concept, was a new field. George Foster and Barbara Anderson, in laying out an analytical approach to health-related issues for this new field, suggested a structural division of medical systems into two components: a *disease theory* system and a *health care* system, defining the first component as the "beliefs about the nature of health [and] the causes of illness" that prevail within a particular medical system (Foster & Anderson, 1978, p. 37). It is these beliefs that I am concerned with here.

The historical pathway leading to the contemporary biomedical and public health understanding of disease causation is both long and intricate (Richardson, 1991). On the one hand it is part of the larger historical course leading from prescientific to scientific modes of thought, and on the other hand it runs from simple to more complex scientific understandings of what disease is and how it develops within bodies, within populations, and within social and environmental contexts. The syndemic orientation, although recent in expression, is in fact an outgrowth of the new way of thinking about the causes of sickness that emerged and caught hold in the mid-1800s in a process commonly referred to as *the rise of **germ theory***. This approach led first to the biomedical and public health conception of the nature of both contagious and noncontagious diseases (and more recently to reexamination of the assumed differences between these two broad categories of disease). This point then—the transition to a modern biological understanding of disease—is the starting place for examining the syndemic perspective, in that, as noted by historian Daniel Boorstin, trying to understand the present or plan for the future without a sense of the past is like trying to plant cut flowers (McCullogh, 2005).

GERM THEORY AND THE BIOMEDICAL CONCEPTION OF DISEASE

A critical moment in the evolution of biomedicine occurred during the mid-nineteenth century. During this epoch the healing system that was to evolve into modern biomedicine underwent a profound transformation, as detailed in the following sections.

Health as Balance

Prior to the mid-nineteenth century and dating back to the era of ancient Greece, physicians commonly understood health in terms of the balance among bodily fluids known as **humors.** Most prominent among ancient Greek physicians was Hippocrates (circa 460–370 B.C.), a man often credited in the West with being the father of medicine. Rejecting the notion of disease as a divine punishment for violations of spiritual laws—a disease theory that long predates ancient Greece yet lives on in the modern world (as seen, for example, in some religious interpretations of the HIV/AIDS epidemic as God's punishment of a sinful world)—Hippocrates and his peers believed

that certain human moods, emotions, and behaviors were directly under the influence of blood, yellow bile, black bile, and phlegm (and that these humors were, in turn, linked to the four elements of fire, air, water, and earth in the natural environment). When these four humors were not in balance (a state called *dyscrasia,* or "bad mixture"), a person fell ill and remained so unless balance was restored through medical intervention. As Erickson (2008) notes, Hippocrates rationalized disease, thereby laying the foundation for the "biomedical understanding that diseases—both individual . . . and epidemic . . . are natural processes not supernatural punishments" (p. 25).

The humoral notion of disease causation was elaborated further by another ancient physician, Galen (circa 131–200 A.D.), who stressed that understanding of disease must be based on experiential awareness of human anatomy and physiology. (Owing to a government ban on human dissection, Galen gathered his own knowledge of human anatomy, sometimes inaccurately, from examining the corpses of pigs, primates, and other animals.) Galen's influence spread throughout the Western and Arab worlds and remained a factor in medical approaches to healing through the mid-1800s. As Hays (2000) observes, "Bleedings and purges . . . remained the order of the day for the early nineteenth century physician, however much he might have forsworn allegiance to Galenic humors" (p. 216).

Pollution Theory

Also important in historical thinking about disease causation, and reflective of the naturalistic and environmental understanding found in humoral theory, was the theory about the effects of ***miasma***, or pollution theory. This understanding viewed toxic vapors given off by decomposing organic matter in the environment as the cause of many diseases. One such disease was malaria, believed to be caused by poisonous and foul-smelling environmental vapors arising from bodies of water found at low elevations and filled with particles of decomposed matter. This led European colonists in Africa, for example, to settle at high attitudes, a strategy that proved effective because it located the homes and offices of colonial administrators above the normal (temperature-sensitive) breeding elevation of mosquitoes, the real **vectors** of malarial infection. (Global warming and the resulting breeding of mosquitoes at ever higher elevations would make such a practice less effective today.)

Cholera in London   From 1831 to 1833 and again from 1848 to 1849, London, then the most populous city in the world, experienced several epidemics of cholera. The name *cholera* is derived from the Greek term for bile and reflects that this water-borne disease was originally conceptualized as resulting from an imbalance of humors. However, in mid-nineteenth-century London, William Farr, a doctor who served as the assistant commissioner for the 1851 city census, asserted that cholera was transmitted by bad air and, in London, specifically by a noxious concentration of *miasmata* (a non-living entity of organic origin) found along the banks of the Thames (at that time a heavily polluted industrial river). During this era there was no understanding that a single disease could produce multiple symptoms, and thus the diarrhea caused by

a cholera infection was seen as a totally different disease from the cholera itself (and not, as it was found to be many decades later, an adaptive strategy that creates an intestinal alkalinity favorable to *Vibrio cholerae,* the immediate causative agent of cholera). When a major cholera epidemic again broke out in London, in 1854, Farr was appointed by the General Board of Health to the Committee for Scientific Enquiries in Relation to the Cholera Epidemic. Although not as severe as the epidemic of 1849, the 1854 epidemic—during which about 11,000 Londoners succumbed (Winterton, 1980)—was especially devastating in the Broad Street area of the Soho district, where the death toll reached three times the rate in London as a whole.

Snow on Broad Street   In addition to being the site of numerous cowsheds, animal slaughterhouses, grease-boiling pots, overcrowded working-class dwellings, and decaying sewers, Soho was home to the now infamous Broad Street pump. John Snow, a physician who had initially gained fame in 1846 by successfully administering the anesthesia chloroform to Queen Victoria during the births of Prince Leopold and Princess Beatrice, claimed that this public water station was the source of the local outbreak and that some kind of living entity in the water, an unseen germ of some sort spread by fecal contamination, was the cause. Snow was convinced the pump was a primary source of infection because the surrounding area was so hard hit during the outbreak. Between August 31 and September 10, over 500 people who lived on or near Broad Street (now renamed Broadwick Street) died of cholera (and ultimately 616 people in Soho were victims of the epidemic). People were fleeing the neighborhood in terror. Snow lived nearby, and he began interviewing the family members of those who had died, thereby inventing field epidemiology in the process. Using addresses that Farr had provided (despite his disagreement with Snow's perspective on disease causation), it was not long before Snow realized that families who drew their water from the Broad Street pump were the hardest hit and that most of the deaths were among people who lived only a short distance from the pump. He also found that not one of the seventy workers at the nearby Broad Street brewery had gotten sick; these workers were given free beer everyday and consequently never drank water from the pump.

This sociogeographical patterning of disease cases, Snow concluded, could not be explained by miasma theory. To prove his case he even tried examining samples of water from the pump under a microscope, although not one powerful enough for him to see the microbes they contained. Nonetheless he was convinced by his other findings that the germs were there and the cause of the illness and death occurring around him. He consequently self-published a report for distribution to fellow physicians and friends, followed by an essay published in the *London Medical Gazette* (Summers, 1989). Meanwhile, William Budd, in Bristol, England, who would later gain medical fame by demonstrating that typhoid fever was a waterborne pathogenic disease, had reached a conclusion somewhat similar to Snow's and published his view in a book a month after Snow's essay appeared. The difference was that Budd thought the agent of cholera was a fungus, which he and a group of fellow physicians believed they had observed in the stools of cholera patients, a view that was soon discredited.

Contested Understandings   The initial response of health officials to Snow's assertion is reflected in the tone of the summary of it developed by John Simon, a physician who served as the head medical officer of London at the time: "This doctrine is, that cholera propagates itself by a 'morbid matter' which, passing from one patient in his evacuations, is accidentally swallowed by other persons as a pollution of food or water; that an increase of the swallowed germ of the disease takes place in the interior of the stomach and bowels, giving rise to the essential actions of cholera, as at first a local derangement; and that the morbid matter of cholera having the property of reproducing its own kind must necessarily have some sort of structure, most likely that of a cell" (quoted in Frerichs, 2001). Although Simon plainly understood Snow's theory, lacking direct evidence of the cell in question he found the argument, so to speak, hard to swallow, and rejected the relevance of germ theory to the cholera epidemic.

Similarly, despite Snow's national stature, the Committee for Scientific Enquiries, under Farr's influence, eventually concluded that "[a]fter careful inquiry, we see no reason to adopt [the belief that the Broad Street pump was to blame for the outbreak]. We do not feel it established that the water was contaminated in the manner alleged [by Snow]; nor is there before us any sufficient evidence to show whether inhabitants of that district, drinking from that well, suffered in proportion more than other inhabitants of the district who drank from other sources" (Eyler, 1979, p. 118).

Instead, cleaving to miasma theory, the committee concluded that "on the whole evidence, it seems impossible to doubt that the influences, which determine in mass the geographical distribution of cholera in London, belong less to the water than to the air." Indeed, the committee went so far as to scold those who followed Snow in accepting the germ theory of disease: "Many of the public believe that everything we eat and drink teems with life, and that even our bodies abound with minute living and parasitic productions. This is a vulgar error and the notion is as disgusting as it is erroneous" (quoted in Winterton, 1980, p. 17).

Another well-known proponent of the miasmatic theory at the time (although like others working in medicine she later embraced germ theory) was Florence Nightingale, who had gained an international reputation as a devoted nurse during the Crimean War (no mean accomplishment given the opposition to female nurses caring for wounded male soldiers). Because of her belief in miasma theory (and her statistical calculations showing that seven times as many British soldiers died from diseases contracted in the hospital as died from wounds received on the battlefield), she campaigned for the reform of hospitals, insisting that they be regularly cleaned and scoured until sanitary and fresh smelling. During the 1854 cholera epidemic, while serving as superintendent at the Institute for the Care of Sick Gentlewomen, in Upper Harley Street, London, Nightingale also volunteered at Middlesex Hospital, which received many of the victims of the epidemic. Of the 278 cases of cholera treated at the hospital, 123 died—a fatality rate of 53 percent—including one of the hospital's nurses (Johnson, 2006). Yet to Nightingale's mind's eye what was occurring was not the transmission of living, disease-causing microorganisms but rather the emergence of impurities from foul environments. This view also led her to write about her experience with another

disease (in a footnote in the pamphlet "Notes on Nursing for the Labouring Classes"), "I have seen with my eyes and smelt with my nose smallpox growing up in first specimens, whether in closed rooms, or in overcrowded wards, where it could not by any possibility have been 'caught' but must have begun" (quoted in Penner, 2004, p. 92).

Ending the Epidemic   Although the Committee for Scientific Enquiries was later to reach its conclusion that the cause of the cholera epidemic was bad air, when the members of the London Board of Governors heard Snow's argument, they ordered the closing of the Broad Street pump, and the epidemic soon faded away. Consequently, although no one in London had seen the germ involved in the development of cholera, the 1854 epidemic ultimately gave considerable impetus to the rise to dominance of germ theory within biomedicine. Ironically, Italian biologist Filippo Pacini had already identified the cholera bacterium and had published a scientific paper on his discovery ("Microscopical Observations and Pathological Deductions on Cholera") through the Paris Academy of Sciences. Using a microscope he had purchased with his limited savings while still a medical student, Pacini conducted histological examination of the intestinal tissues of individuals who had died of cholera in Florence and identified a comma-shaped bacillus that he named *Vibrio.* Unfortunately, as sometimes happens in science with findings that are ahead of their time, Pacini's paper was ignored for thirty years, and it is unlikely that Snow had any awareness of it. (Pacini was finally credited with his discovery, eighty-two years after his death, when the International Committee on Nomenclature adopted *Vibrio cholerae Pacini* as the official name of the microorganism that is the proximal cause of cholera.)

The Rise of Germ Theory

Despite their considerable contributions, neither Snow nor Pacini was in fact the first to propose a germ theory of disease. Almost two thousand years earlier, Marcus Varro (116–127 B.C.), the architect whom Julius Caesar had assigned to the task of building a great public library in ancient Rome (a project that went unrealized because of Caesar's assassination), had warned those looking to select hygienic locations for buildings to avoid areas near swamps, because "in swampy places minute creatures live that cannot be discerned with the eye and they enter the body through the mouth and nostrils and cause serious disease" (quoted in Amici, 2001, p. 4). How Varro, lacking the technology to identify them, came to believe in the existence of disease-causing microbes is not clear. The microscope was not invented until the late 1600s, although magnifying glasses and the use of emeralds for magnification purposes are mentioned in *Naturalis Historia,* written by Pliny the Elder, a Roman naturalist and philosopher who lived during the first century A.D. What is known is that Varro's recognition had little impact on the medical perspective of his day or subsequently and is of interest today primarily as an intriguing footnote in the history of disease understanding.

During the sixteenth century, having observed epidemics of bubonic plague, typhus, and syphilis (and having written a 1,300-verse poem in Latin hexameters focused on a fictional shepherd named Syphilus, the source of the latter disease's

modern name), the Veronese physician Girolamo Fracastoro (circa 1478–1553) came to question miasma theory (as well as beliefs about divine retribution) as lacking in evidentiary support. In his major medical treatise, *On Contagion and the Cure of Contagious Diseases,* published in 1546 (and in which he dismisses his poem as a youthful endeavor), he asserted there was better support for the notion that diseases were spread by tiny living or at least lifelike seminaria (seeds) or germs (although his recommended treatment was bleeding the sufferer and administering mercury to return him or her to humoral balance). This view of disease causation has led some to nominate Fracastoro for the title "father of germ theory" (see, for example, Greenwood, 1953), although others question this titling (Magner, 2002) owing to ambiguities in Fracastoro's sixteenth-century narrative and to his speculation that seminaria might arise from poisonous emanations born of planetary conjunctions (a factor in Stephen Jay Gould's [2000] argument that the greatest "poetry" ever composed about syphilis was penned not by Fracastoro but by the scientists who methodically and meticulously developed the elegant map of the 1,041 genes that constitute the genome of the **pathogen** *Treponema pallidum,* now known to be responsible for the disease).

Less conflicted and ambiguous was the contribution of Jacob Henle (1809–1885), a prominent German pathologist after whom various structures within the human body, some of his own discovery, are named. Henle compared alternative explanations of disease in his 1840 book *Misamata and Contagion.* Although the book did not achieve instant recognition as a critical turning point in medical disease understanding, it "was retrospectively recognized as a landmark" (Magner, 2002, p. 256) with the subsequent confirmation of germ theory. This triumph was achieved when Louis Pasteur (1822–1895), demonstrated in the 1860s that specific microbes are responsible for specific fermentations and later linked microbes to disease (initially in silkworms), and when Henle's student Robert Koch (1843–1910) isolated both the microbe causing cholera and the microbe causing tuberculosis.

Aftermath of the Epidemic   Living on the cusp of the great transition from the miasmatic to the germ theory of disease causation, William Farr eventually came to embrace Snow's (and Pacini's) understanding of cholera and of infectious disease generally, as did most of his fellow biomedical physicians. His conversion to the new paradigm marked both the broader transformation going on in medicine—namely its emergence as a bioscience—and the celebration of Snow's role in that process. Indeed, in a March 2003 survey that *Hospital Doctor* magazine conducted of its readers, John Snow was voted the "greatest doctor" of all time, with Hippocrates coming in second (Oleckno, 2008). Although it has been suggested that Farr did not initially accept Snow's ideas about germ theory because, unlike Snow, he was not open to new perspectives, Eyler (2001) stresses that the reverse may have been the case: "Judged by the standards of his time Snow was the dogmatic contagionist and premature reductionist. Farr was the more cautious in weighing all evidence" (p. 230). As a result of Snow's and others' dogged commitment to an idea (and to seeking out the evidence to support it), germ theory evolved from a controversial notion into the cornerstone of biomedical disease

theory. In the process, biomedicine came to privilege understanding cell biology over understanding the social origins of disease and other disorders, an emphasis that has hindered a broad ecological conception of human illness (Singer, 1986).

Separation of the Part from the Whole   The scientific discoveries that propelled the acceptance of germ theory not only undercut older views of disease causation but have also contributed to the development of an understanding that differentiates biomedicine from other ethnomedical systems around the world. Davis-Floyd and St. John (1998) have called this understanding "the principle of separation," and see it as a product of "an overwhelmingly linear mode of thinking" (p. 17). This principle stipulates that each of the world's various entities is best understood when considered independently of the other entities of its natural environment. Davis-Floyd (1994) observes that "the essence of [biomedical] research and description is separation—of elements from the whole they compose, of humans from nature, of mind from body, of mother from child" (p. 1127). Similarly, George Engel (1977), an anthropologically informed physician who during his life specialized in the psychophysiological aspects of human health and illness, argued that the distinctive feature of biomedicine is its embrace of "both reductionism, the philosophic view that complex phenomena are ultimately derived from a single primary principle [in this case, that everything can be explained in terms of chemistry and physics], and mind-body dualism, the doctrine that separates the mental from the somatic" (p. 130).

Reflective of this atomistic approach to knowledge, biomedicine separates the person with an illness from his or her immediate social context and community, diseased organ systems from the whole body, and one disease from another. Further, it breaks every disease into its constituent parts and manages disease treatment through a complex and atomistic array of medical specialties. As Davis-Floyd and St. John (1998) stress, a "drive toward separation" if carried to its logical extreme, as has more or less occurred in biomedicine, can "obscure the many meanings in the non-linear" and "the interconnections and relationships between entities" (p. 17). In his widely cited article quoted earlier, George Engel (1977) maintained that biomedicine's adherence to "a model of disease [that is] no longer adequate for the scientific tasks and social responsibilities" of the discipline has led to a crisis in biomedicine. Herein lies the value of a syndemic perspective, which seeks to clarify the impact of biosocial interconnections and relationships (p. 129).

REVOLUTIONS IN BIOMEDICAL REALITIES

Although biomedicine "purports to be belief- and value free" (Gaines & Davis-Floyd, 2004, p. 100), its understanding of reality—its ontological conception of the nature of nature and of basic life processes—is rooted in a particular construction of the world, an ideology that has both driven and reflected the encompassing Western cultural worldview. Germ theory, and biomedical understanding generally, initially assumed that like cholera each infectious disease is caused by a specific, identifiable pathogen.

Although this view opened the world up to new insights (including the discovery of a wide array of pathogens), it has also carried conceptual restrictions. As Francois Jacob (1988), winner of the 1965 Nobel Prize in medicine, notes: "In analyzing a problem, the biologist is constrained to focus on a fragment of reality, on a piece of the universe which he arbitrarily isolates to define certain of its parameters. In biology [including medicine] any study begins with a 'system.' On this choice depend the experimenter's freedom to maneuver, the nature of the questions he is free to ask, and even the type of answer he can obtain" (p. 16).

When constraint is imposed on biomedical thinking, it limits not only, as Jacob suggests, the nature of the questions that are asked but also the answers that are meaningful, acceptable, even thinkable. As historian of science Thomas Kuhn noted in *The Structure of Scientific Revolutions,* a seminal book on the scientific process, this is the everyday or normal way scientific understanding operates. In any specialized subfield (including biomedicine), science tends to be guided by a **reigning paradigm,** that is, by "an entire constellation of beliefs, values and techniques, and so on, shared by the members of a given community" (Kuhn, 1970, p. xii). In common contemporary parlance, scientific thinking normally takes place "within the box," because unless doubts and uncertainties about basic assumptions have arisen to rattle confidence in that box (for example, through research findings that cannot be explained because they appear to fall "outside the box"), the box defines reality. Thus once germ theory and its "to each disease its own unique pathogenic cause" perspective gained dominance in biomedicine, a *paradigm effect* occurred, canalizing thinking and hindering attention to other ways of seeing the available evidence.

PROBLEMS WITH THE POSTULATES

Despite the compelling force of reigning paradigms, as Kuhn emphasized, science is not stagnant (by design!) and revolutions in scientific thinking are, one might argue, equally a normal part of science. Consequently, since the adoption of germ theory, scientific understanding about the causes of disease has continued, in stages, to evolve. These changes can be seen as stemming in part from problems encountered in implementing a set of postulates developed by Koch (and broadly accepted in the field as the gold standard) for determining whether a particular microbe is the cause of a specific disease. For medical science to affirm that a particular organism causes a disease, Koch (1890/1987) argued:

1. The organism must be present in every case of the disease but not in healthy individuals.

2. The organism must be capable of being isolated from the sufferer and grown in pure culture.

3. The specific disease must be reproduced when a pure culture of the organism is inoculated into a healthy, susceptible host.

4. The organism must be recoverable from the experimentally infected host.

The problems began during the period just after the achievements of Snow, Pasteur, and Koch became generally known, an epoch often thought of as the golden age of breakthroughs in medicine and infectious disease research. For Theobald Smith, a physician turned pathology researcher who was to become a preeminent pioneer in American microbiology, it was an exciting time of regular discoveries of pathogens, vectors, and disease causation. It was also a time during which diseases like hog cholera (now called classical swine fever) "swept through the countryside, causing devastating losses. During the fall months, looking across the prairies of the Middle West, one could often see smoke ascending from perhaps a half-dozen farms where pigs dead of cholera were being burned" (McBryde, quoted in U.S. Department of Agriculture and Agricultural Research Service, 2006). In 1884, as a lowly lab technician in the newly created U.S. Department of Agriculture's Bureau of Animal Industry (BAI), a center established by Congress to respond to costly waves of livestock epidemics, Smith set out to discover the cause of hog cholera, one of the most economically damaging pandemic diseases of pigs in the world.

When Smith came to this line of work, he had two years of medical training and had read, on his own, the papers of Pasteur. At the BAI he was supervised by veterinary pathologist and BAI chief Daniel Salmon (for whom *Salmonella,* the enterobacterium that causes diseases like typhoid fever, paratyphoid fever, and food poisoning, is named, even though it was actually discovered by Smith, a point of enduring tension between the two scientists; Dolmon, 1969). Having confirmed that a microbe, *Salmonella choleraesuis* (now called *Salmonella enterica*), was found consistently in pigs suffering the symptoms of hog cholera and could be isolated and grown in pure culture, having seen the disease after healthy hogs were infected with the microbe, and having recovered the bacterium from animals that had been infected in this way, Smith (although Salmon insisted on first authorship of the published findings) concluded that **Koch's postulates** had been met, and declared that the cause of hog cholera had been discovered. Notably, both Pasteur and Koch accepted this conclusion and assumed that the problem of hog cholera was well on its way to resolution (Zinsser, 1936). This proved not to be the case, as Marion Dorset, another BAI scientist, had no success with a serum made from the *Salmonella choleraesuis* bacterium during an 1897 outbreak of the disease in Iowa. Six years later, Dorset was able to show that the actual cause of hog cholera was not a bacterium at all but a virus (genetically a much simpler entity), a discovery leading to the eradication of the disease in the United States.

Why had Smith, known to be a cautious worker, gone wrong? As it turned out, swine infection with *S. enterica,* which causes its own health problems (salmonellosis and typhoid fever), was secondary to the viral infection. Smith, in other words, had unknowingly stumbled on a synergistic interaction among animal pathogens in which one (a virus) facilitated infection by another (a bacterium) producing frequent coinfection, and he had identified the wrong pathogen as the cause of hog cholera. Because of his many subsequent discoveries (such as the role of a protozoan parasite, babesia, in the development of Texas fever among cattle and the activity of ticks in its transmission),

this mistake did not hurt Smith's career, but its occurrence is instructive about the potential consequences of assuming one has successfully isolated a pathogen.

Disease Carriers   Over time other problems with Koch's postulates also emerged. First, the discovery of asymptomatic **disease carriers** threw into doubt the idea that a known pathogen could not be found in healthy individuals. Mary Mallon, an early twentieth century cook for several New York families, for example, was found to be a carrier for typhoid fever (caused by *S. enterica*). Although she had no disease symptoms, she did pass on the bacterium to twenty-two members of the families she worked for and who did become sick, earning her infamy as Typhoid Mary. As a public health measure, she was forcibly quarantined in an institution on an island in the East River for much of the rest of her life. Mary Mallon was not unique; asymptomatic carriers (sometimes called *well carriers*) are now known to be a common feature in the spread of many infectious diseases, including polio, herpes simplex, and hepatitis. When Koch discovered that there were asymptomatic carriers of disease, he dropped the second half of his first postulate.

Uncultivable Microbes   It has also turned out that some microbes cannot be successfully grown in pure culture. For example, bacteria in the rickettsia family (responsible for an array of diseases, such as Rocky Mountain spotted fever) cannot live in artificial nutrient environments. Similarly, *Mycobacterium leprae,* the bacillus that causes leprosy, has never been cultivated in vitro in its classic rod-shaped form, because it appears to lack the genetic capacity to grow outside the human body. Further, although a significant number of bacteria have been isolated in the human mouth, it is estimated that only about half the species that dwell in the oral cavity can be cultured (Rolph et al., 2001).

Over time other strategies, such as molecular techniques, have been adopted to identify bacteria species that have never been cultivated. It was only with the introduction of molecular genetic strategies, for example, that the uncultivable bacterium *Tropheryma whippelii* was identified as the cause of Whipple's disease, a chronic, systemic infectious disease, most common in middle-aged men, that typically causes malabsorption in the small intestine and a wide range of other clinical manifestations including arthritis. Additionally, new culture methodologies have been invented in recent years, such as chicken tissue and embryo cultures (which can be used to grow rickettsia bacteria). Still, it is assumed by most microbiologists and pathologists that there are many pathogenic microorganisms at play in human health that have yet to be identified because of the limitations of existing technologies. Only in very recent years, as discussed in Chapter Five, have researchers discovered the viral origins of some chronic diseases previously believed not to be contagious (such as peptic ulcer disease, in which *Helicobacter pylori* plays a role, and cervical cancer, which involves several human papillomaviruses).

One consequence of encountering an uncultivable microbe is evident in the case of Lyme disease, which is caused by several species of the hard-to-culture spirochetal

Borrelia bacterium. Although Lyme disease is recognized as the most common tick-borne disease in North America and Europe, as well as one of the fastest-growing infectious diseases in the United States (with over 20,000 cases a year nationally since the turn of the twenty-first century and over 30 cases per 100,000 persons in the ten most heavily infested states), inability to culture its causative agent has led to considerable professional disagreement and intense acrimony over the guidelines to be used in diagnosis. The Centers for Disease Control and Prevention (CDC) has been forced to rely on such potentially ambiguous criteria as presentation of symptoms (which can vary considerably across infected individuals), physical findings (such as a bull's-eye rash, which does not appear in many cases), and the possibility of exposure to infected ticks (such as the black-legged tick in North America and several other tick species in Europe) based on place of residence or visitation (CDC, 2006b).

Masked Infections   Problems with Koch's third postulate began with the fact that some pathogens cannot be cultured, but other difficulties have arisen as well. For various reasons (such as acquired immunity from prior exposure to a pathogen, as occurs with influenza, and genetic immunity, as seen for example in individuals who acquire a sickle cell allele from at least one parent), exposure to a pathogen does not necessarily lead to detectable infection. Of even greater importance for the issues under consideration is the fact that the presence of a second pathogen (for example, one that weakens the immune response to the presence of foreign organisms generally) can increase the likelihood that infection will develop in exposed individuals. In other words, in many instances, without the bodily effects of synergistic interaction among diseases, mere exposure to a single pathogen (including intentional inoculation) does not produce disease. At the same time, if the disease of concern is in fact the consequence of interaction among pathogens or other disease causes, it will not develop (or have the same expression) unless the new host is exposed to all those pathogens or causes.

Immune Response and the Reisolation of Pathogens   In addition to the challenges to Koch's postulates already discussed, there can be difficulties with the reisolation of a pathogen from an inoculated individual because that requires the capacity to segregate and culture the pathogen. This capacity does not exist for some microorganisms (including those that infect only humans and hence, for ethical reasons, can never be tested using Koch's postulates), and it does not exist in cases of a healthy immune system because the body has eliminated the pathogenic agent (although it may exist in cases of coinfection owing to degradation of immune system capacity). Additionally, the modus operandi of some pathogens, as found for example in rheumatic heart disease (which begins with a streptococcal throat infection), involves what might be called a hit-and-run pattern, in which the organism is no longer in the host by the time the disease is evident.

Complexities of Disease Understanding   Multiplying the difficulties encountered in using the postulates was the realization that a single disease might have multiple causes (a situation known as *multifactorial disease*) and that a single pathogen might produce

more than one disease. These issues extend beyond infectious disease to disease with an environmental or genetic origin. For example, with regard to the first point, it is recognized that the chronic eye infection known as trachoma is a common source of blindness in developing countries. This disease has been linked to sexually transmitted infection with the pathogen *Chlamydia trachomatis.* Research by Dean, Kandel, Adhikari, and Hessel (2008), however, has found that trachoma can also be caused by *Chlamydophila psittaci* and *Chlamydophila pneumoniae.* Consequently, these researchers conclude that the existence of multiple agents causing trachoma helps to "explain the failure to detect chlamydiae among active trachoma cases, when only *C. trachomatis* is assayed," "the failure of active trachoma cases to resolve their clinical disease following effective *C. trachomatis* treatment, and the limited effectiveness of the WHO strategy to control trachoma" (p. e14). Also of note in this regard is the potential effect of interaction among copresent trachoma-causing pathogens. Similarly, coinfection with multiple strains of the microbe that causes dengue can significantly change the clinical picture and severity of that disease (a topic to be discussed in Chapter Six).

With regard to the second point, that a single cause can produce multiple diseases, consider the hepatitis B virus, which is the proximal cause of hepatitis, cirrhosis, chronic liver disease, and liver cancer (Merican et al., 2000). Moreover, the actual expression of any disease may vary widely across individuals, including affecting different organ systems in the body. Thus HTLV-1 (human T-cell lymphotropic virus type 1), a retrovirus that was once mistakenly proposed as the cause of HIV/AIDS but that actually causes adult T-cell leukemia, T-cell lymphoma, and other diseases, is expressed in three clinical patterns: cancer, autoimmune disease, and immunosuppression disease. Tuberculosis also has various expressions, depending on how the infection gains access to the body and the host's response to its presence. Although pulmonary infection through breathing in *Mycobacterium tuberculosis* is most common, it is also possible to acquire the disease by drinking infected cow's milk, leading to lesions in the intestinal track. Notably tuberculosis of the gastrointestinal tract is being seen more frequently among people with HIV/AIDS. These are examples of what have come to be called ***spectral diseases***—that is, diseases with a spectrum of alternative clinical manifestations. Again, **disease interactions** may play a role in determining which of an array of possibilities finds actual disease expression in a patient or population.

Roads Less Traveled   In assessing the overall value of Koch's postulates in light of subsequent discoveries, Cochran, Ewald, and Cochran (2000) conclude that the postulates "were useful because they could generate conclusive evidence of infectious causation, particularly when (1) the causative organisms could be isolated and experimentally transmitted, and (2) symptoms occurred soon after the onset of infection in a high proportion of infected individuals. While guiding researchers down one path, however, the postulates directed them away from alternative paths: researchers attempting to document infectious causation were guided away from diseases that had little chance of fulfilling the postulates, even though they might have been infectious" (p. 406).

Modifications of Germ Theory

The result of our growing understanding about disease complexities has been a process of germ theory revision (that is, scientific evolution) at various points in time to accommodate new information. Consequently, despite its obvious power as an explanatory model, germ theory as first articulated by researchers like Snow, Pasteur, Koch, and many more is not the germ theory in vogue a hundred years later. A much more complex and nuanced comprehension about the relationship between pathogens and disease now abides, an understanding that recognizes the importance in contagious disease of **host-pathogen** interactions (and that includes an enormous increase in the appreciation of the intricacies of the immune system and the realization that symptoms may be the result of attempts by the host to rid itself of a pathogen, as occurs, for example, in cystic fibrosis); accepts three primary disease pathways (infectious agents, genetics, and body-environment interactions); includes awareness of coinfection and copresent infectious and noninfectious diseases; is cognizant of the existence of pathogens with multiple strains (with differing capacities to cause bodily damage); and is attuned to the role of biological individuality in population health. In this sense the issue of syndemics, which as indicated earlier has been a usually unanalyzed complicating factor since the earliest efforts to account for specific diseases using germ theory, presents but another stage in the normal scientific evolution of biomedical and public health thinking about disease. To the degree that it encourages a focus not just on disease interactions but on the fundamental importance of the social conditions that foster **disease clustering** and interfaces, syndemics theory also represents a **paradigm shift** in the understanding of what disease is and how it is manifested in complex biosocial feedback environments.

CONFRONTING COMORBIDITY

Over the course of time, biomedicine has encountered many patterns in nature that call into question the assumption that the principle of separation is the most fruitful approach for understanding threats to health. The most notable of these patterns is the frequent co-occurrence of more than one disease or other disorders in the same patient. The term *comorbidity* has traditionally been used in biomedicine to denote this co-occurrence (Feinstein, 1970). Some comorbid disease patterns are sufficiently regular to have acquired their own names. For example, *Austrian's syndrome,* also called *Osler's triad,* is a disease complex consisting of endocarditis, meningitis, and pneumonia caused by *Streptococcus pneumoniae* infection. It is commonly associated with excess alcohol consumption but has also been described among injection drug users. The medical literature on this condition consists primarily of individual case reports rather than focused analyses of the nature of the interactions among the three diseases.

Much of the focus in comorbidity research, in fact, has been the development of schemas (see, for example, Charlson, Pompei, Ales, & McKenzie, 1987) to assist physicians in making treatment decisions (such as whether the benefits of treating one condition in a patient with multiple health disorders will outweigh the negative effects

this treatment is likely to have on the patient's other disorders). Generally speaking, such work has tended to see the diseases involved as independent entities despite their copresence in the same patient.

Health disciplines differ in the emphasis they place on assessing the nature of the relationship between co-occurring diseases and other maladies. In behavioral health, for example, a field that has evidenced a strong interest in comorbidity, a primary concern involves determining whether two apparently different problems are in fact alternate and cycling manifestations of the same condition (as may be the case in bipolar disorder). Also of interest is whether or not one disorder causes another, as happens with the abuse of alcohol or other drugs to self-medicate anxiety or depression (see Chapter Six). Additionally, it is known that there are cases in which a patient is suffering from two distinct behavioral disorders that are independent of each other but that are both the consequence of a third disorder. Finally, coexisting disorders may be traced to common risk factors, such as various injurious experiences early in life (Neale & Kendler, 1995).

Mere awareness of comorbidity as a factor in human health is not the same thing, however, as having a syndemic perspective. The differences between the terms *comorbid* and *syndemic,* as Mustanski, Garofalo, Herrick, and Donenberg (2007) aptly point out, are "not simply semantic—comorbidity research tends to focus on the nosological issues of boundaries and overlap of diagnoses, while syndemic research focuses on communities experiencing co-occurring epidemics that additively increase negative health consequences. For example, it is possible for two disorders to be comorbid, but

---

Why isn't there more research on syndemics? One reason can be found by looking at studies of chronic health problems. Chronic diseases like diabetes, cardiovascular disease, and cancer are recognized as significant threats to patients' quality of life. Further, it is well known that as people age, the number of chronic diseases they are likely to have goes up. However, most research on the life effects of chronic disease focuses on only one disease and its impact on patient well-being, social functioning, and quality of life. Because the co-occurrence of other diseases complicates examination of the specific effects of the chronic disease of interest, patients with comorbid conditions often are excluded from patient samples in chronic disease research. As a result the whole question of disease interaction and its significant impact on patients is often overlooked. Yet as chronic disease researchers at the Netherlands Institute of Health Services Research (Rijken, van Kerkhof, Dekker, & Schellevis, 2005) have found, patients suffering from comorbidity report the lowest levels of physical functioning. These researchers also point out that comorbid chronic diseases appear to have a synergistic effect and cause greater physical disability than would be expected from merely adding up the separate effects of individual chronic diseases.

not represent a syndemic (that is, the disorders are not epidemic in the studied population or their co-occurrence is not accompanied by additional adverse health consequences). Beyond the focus on disease clustering and interaction, the term *syndemic* also implies a focus on health disparities and the social conditions that perpetrate them" (p. 40).

Various co-occurring diseases have been described that do not (as best as is known) appear to interact in adverse ways, although in some cases the findings of different studies conflict. For example, it is known that the *Ixodes* tick that transmits the pathogen (*Borrelia burgdorferi*) identified as the source of Lyme disease can simultaneously pass on several other human pathogens, including both *Anaplasma phagocytophilum,* the bacterial cause of human granulocytic anaplasmosis, a disease that like HIV/AIDS appears to damage the immune system in a way that promotes opportunistic infection (Dumler et al., 2005), and *Babesia microti,* a protozoan parasite that causes the malaria-like disease babesiosis. Some (but not all) human studies have found that co-infection with human granulocytic anaplasmosis increases the severity of Lyme disease (Krause et al., 1996). In contrast, when mice were experimentally co-infected with *B. microti* and *B. burgdorferi,* no change was found in the course of infection of either disease as revealed by a range of measures, including pathogenic load, spleen weight, and blood chemistry (Coleman, LeVine, Thill, Kuhlow, & Benach, 2005). Rather, both diseases proceeded along their normal course of infection and caused readily identifiable, landmark symptoms specific to each disease.

In short, the mere co-presence of two or more diseases and/or other disorders is not the defining feature of a syndemic. Further, disease interaction can produce positive health effects, a condition referred to here as a *countersyndemic* (see Chapter Five). Often, however, when diseases come together in a population and in individual patients, the outcomes are neither neutral nor positive. A primary goal of this book is to advance recognition and understanding of the many instances of adverse health effects arising from connections among epidemic disease clustering, disease interaction, and **health and social disparities.**

TOWARD SYNDEMIC RECONCEPTUALIZATION

The syndemic perspective moves our conception of health beyond the narrow frames of traditional reference. An essential feature of syndemics is revealed by an unexpected life form, the lichen. Although in appearance and structure the often taken-for-granted lichen appears to be a simple plant, it in fact constitutes a symbiotic community. One member of this community is a fungus—most commonly of the Ascomycota phylum (which includes truffles and baker's yeast) but occasionally a member of the Basidiomycota phylum (which includes mushrooms and puffballs but also a human pathogenic yeast of the genus *Cryptococcus,* known in people with AIDS to produce meningitis). The other member of the lichen community is an algae—usually either (although sometimes both) green algae or blue-green algae (actually a phylum of bacteria) but also sometimes yellow-green algae or more rarely brown

algae. Although plant parasitism is not unusual in nature, it is not clear that that is what is occurring with lichen. Rather, both plant "partners" appear to benefit from the relationship (a type of symbiotic connection known in biology as **mutualism**). Thus the fungi derive sugars, their only nutrient, from the algae, and the algae gain the protection of the fungi, allowing them to live in environments that they otherwise could not inhabit. Nature writer Douglas Chadwick (2003) says that because they are not a single organism but an interactive group of species, he thinks of lichens "as kind of a *doorway* between organisms [or individual species] and ecosystems. Look out one direction, and you see individual things; look the other way, you see processes, relationships—things together. This is the new level in understanding biology" (p. 119).

A parallel to the lichen case is seen in the mutualistic relationship between humans and certain lactic acid bacteria, such as *Lactobacillus plantarum.* These bacteria live, for example, on the vaginal epithelia of women. This environment provides the bacteria with a stable habitat, constant temperature, and steady supply of nutrients in the form of glycogen (which is abundant in epithelial cells). In turn, as part of the metabolism of glycogen, the bacteria produce lactic acid, resulting in a normal vaginal pH of 3.5 to 4.5, an acidity level that protects the vagina from colonization by harmful yeasts and other invasive microbes (see Chapter Six). Notably, elsewhere in the body, in the gastrointestinal tract for instance, so-called friendly bacteria (such as *Lactobacillus*) are directly involved in stimulating the immune system to produce white blood cells that are critical to fighting infection. It was recognition of this sort that led Ludwik Fleck, a Polish biologist, who like Chadwick was fascinated by lichen (and for the same reason), to write, "The [biomedical] conception of infectious disease . . . is based on the notion of the organism as a closed unit and of the hostile causative agents invading it. . . . An organism can no longer be construed as a self-contained, independent unit with fixed boundaries, as it was still considered according to the theory of materialism [that is, germ theory]" (Fleck, 1935/1979, p. 60). The bounded unit view of the organism, Fleck stressed, is a historical bias that is unbecoming to modern biology.

The significance of the kind of interspecies relationship seen in the formation of lichen is one of the recent understandings reflected in the reconceptualization of health and disease embodied in the syndemic paradigm. Motivation for the examination of syndemics is guided by Chadwick's (2003) timely warning that "[i]f we continue to focus chiefly on species—even though we embrace all shapes and sizes of them—rather than on connections, our view of nature will remain incomplete. So will our efforts at protection" (p. 125), and our approach to disease!

LOCAL KNOWLEDGE

Anthropologists and others who work with indigenous communities commonly differentiate *expert knowledge* (for example, the findings and pronouncements of scientists, anthropologists included) from *local* or *indigenous knowledge,* which is "knowledge that does not owe its origin, testing, degree of verification, truth status, or currency to

distinctive professional techniques, but rather common sense, casual empiricism, or thoughtful speculation and analysis" within communities (Lindblom & Cohen, 1979, p. 12). All societies have **local knowledge** about their surrounding environment and about the nature of diseases and disease causes and effects, and this knowledge may match, overlap, or be at considerable odds with professional or authoritative knowledge on the same topic. Indeed, Latour (1979) and Wynne (1996) maintain that all knowledge, even authoritative knowledge, is local in the sense that it emerges from a local socioeconomic milieu and is shaped by local sociocultural and historical factors.

This book is primarily concerned with expert knowledge about synergistically related diseases and the sway of social environments on interactive disease processes. Yet it bears noting that local knowledge about disease interactions, or what might be called *folk syndemics,* in the sense suggested by McCombie (1987) in her discussion of *folk flu* in the American Southwest, also exists. McCombie points out that although the expert understanding is that flu is a respiratory tract infection that is caused by a virus belonging to the Orthomyxoviridae family and that results in fever, sore throat, headache, runny nose, and muscle pain, the lay or folk model of flu includes gastrointestinal symptoms. A somewhat different overlap of folk and professional disease lexicons is seen in the account by Muela, Ribera, and Tanner (1998) of *fake malaria,* a folk-labeled disease found in southeastern Tanzania that people believe imitates the symptoms of real malaria (and that would be diagnosed as such by biomedical physicians) but that is interpreted locally to be caused instead by witchcraft, not pathogens.

In the case of folk syndemics, local knowledge about disease includes cultural beliefs about disease interactions with enhanced adverse consequences. For example, Nichter (2008) reports, "In several South and Southeastern Asian countries where I have conducted research, alcohol and tobacco are thought to cause a latent illness such as TB or sexually transmitted disease to flare up or reoccur" (p. 52). From a public health standpoint, awareness of folk syndemic beliefs as a component of broader folk explanatory models of health and illness (Kleinman, 1978) may be of considerable importance in the implementation of socially acceptable and culturally meaningful interventions to address comorbid conditions.

CONNECTIONS: HUMAN AND NONHUMAN

The syndemic orientation is founded on a recognition of the fundamental importance of biosocial connections in health. It is now clear that diseases and other health conditions (such as nutritional status and stress) interact synergistically in various and consequential ways and that the social conditions of people with illnesses are critical to understanding the impact of diseases at the individual and population levels. A syndemic approach, consequently, examines both *disease concentrations* (that is, multiple, coterminous diseases and disorders affecting individuals and groups) and *disease interactions* (that is, the ways in which the presence of one disease or disorder enhances the health consequences of other diseases and disorders, paving the way, for example,

for new infection or enhanced lethality). Thus one concern of the syndemic approach is the nature of the specific pathways through which diseases and other health conditions interact biologically within individual bodies and within populations and thereby multiply their overall health burden.

The syndemic perspective, however, does not stop with the consideration of biological connections (myriad, complex, and fascinating as they may be), because in the human world disease develops within and is significantly influenced by the social contexts of disease sufferers. Human social environments, including the prevailing structures of social relationships (such as social inequality and injustice) and also sociogenic environmental conditions (for example, hazards of the built environment, sales of toxic commodities, pollution, species loss, and climate change) contribute enormously to both disease clustering and interaction.

Without question, disease synergies are not limited to human populations and occur as well in the nonhuman animal world. For example, veterinary pathologists at the Indiana Animal Disease Diagnostic Laboratory at the Purdue University School of Veterinary Medicine have identified patterns of consequential synergistic interaction between a group of viruses called porcine circoviruses, first identified in Europe in 1974, and other pathogens, such as bovine viral diarrhea virus (a disease agent that may have spread to pigs from deer populations), that significantly increase the fatality rate of dually infected pigs. In recent years porcine circoviruses have spread to pig populations around the world. In places where swine are infected with other viruses in addition to the newly introduced porcine circovirus, mortality rates have jumped by 35 to 50 percent. According to Roman Pogranichniy, a Purdue virologist involved in this research, "We think that the new co-factors, including the bovine viral diarrhea virus-like pathogen and other swine viruses, work together with porcine circovirus to attack the animals' systems and become more virulent" (quoted in Steeves, 2008, p. 1). Similarly, research has shown that bacterial, fungal, and viral infections are frequent and generally worse in animals with tick-borne fever. Experimental research with sheep, for example, found not only that animals dually infected with tick-borne fever and louping-ill virus were more susceptible to louping-ill but that almost all of them died of hemorrhagic syndrome involving a systemic fungal infection with *Rhizomucor pucillus.* In contrast, none of the sheep given louping-ill virus alone developed this syndrome (Brodie, Holmes, & Urquhart, 1986). In related research Jolles and Ezenwa (2006) examined the effects of interaction of gastrointestinal worms and tuberculosis in the African buffalo and found that mortality was heightened in coinfected individuals. They noted that this pattern could be explained by the adverse effects of helminth infection on individuals suffering from TB but concluded that this simple disease dynamic model could not explain their mortality findings, and they hypothesized instead that host defenses against one infection might block simultaneous immunity to the other, an explanation that accurately reproduced their finding when tested through computer modeling. Subsequently, Jolles and Ezenwa have begun to examine disease interaction patterns at three distinct levels of biological organization: individuals, populations, and species. Their plan is to scale up their approach using a comprehensive

database of parasites (a term commonly used to refer to protozoa and helminths but applicable to all disease-causing microbes) and other pathogens that infect primates, ungulates, carnivores, and humans to test whether microbe interactions determine patterns of helminth distributions across populations and species.

In light of this research the question must be asked: are disease synergies in non-human animal species syndemics, in that the structure of social relations and the issue of inequality and its health effects are not factors in the spread, clustering, and inter-action of disease entities? Of course in some animal populations, such as those of nonhuman primates, social hierarchy is an important factor in which animals get sick, as Sapolsky (2005) points out in his discussion of the ways in which characteristics of social rank among animals can have adverse adrenocortical, cardiovascular, reproduc-tive, neurobiological, and immunological consequences (and as a result, syndemic-like) effects. Among hierarchical animals, individuals may be highly stressed without being subordinated members of their group. For example, although it is easy "to imag-ine that subordination can produce an excess of physical stressors" in that subordinate animals "may have to work harder for calories, or be calorically deprived," and may be "the subjects of unprovoked displacement aggression" (Sapolsky, 2004, p. 397), among some species being at the top of a dominance hierarchy can also be stressful because of aggressive challenges from up-and-coming and would-be dominant animals or even from teams of genetically related individuals, as occurs in lion prides. Additionally, the health of animals, domestic and wild, is significantly influenced by human activity and human social structures. People regularly move animals to new environments, exposing them to new diseases. Domestic animals, like their human handlers, live in built environments that are intended to serve human needs (such as increasing milk productivity among dairy stock or producing veal by using growing stalls, hormones, and antibiotics) more than the needs of animals. Similarly, anthro-pogenic changes in physical environments can significantly affect their quality (in terms, for example, of air quality or access to food and water) from the standpoint of health (see, for example, Grandin, 1997, 1998). In other words, whatever the natural social patterns of animal species, human social structures and economies are signifi-cant factors in disease interactions that affect animal health, even in wild populations.

Nonetheless, although it is not productive to draw too fine a conceptual line sepa-rating human from nonhuman animal species—or human and nonhuman animal dis-eases, given the zoonotic origin of many human ailments and the role of animals in human therapies (Rock, Buntain, Hatfield, & Hallgrímsson, 2009)—this book under-lines the special influence of unequal social relations on health among humans in its conception of *syndemics* and applies the term *synergies* to human or animal disease interactions that lack (at least as far as is known at any point in time) an important social origin.

The tendency, perhaps accelerated in recent years, for pathogens to jump success-fully from animal to human hosts (and hence the value of paying attention simultaneously to both animal and human diseases); the requirement for the syndemic perspective to focus equally on both biological and social phenomena (and their numerous dimensions);

and the necessity of focusing on diseases of pathogenic, genetic, and environmental origin (and their interactions) all suggest the centrality of multidisciplinarity in syndemics research, an issue addressed in Chapter Two.

SUMMARY

This chapter introduced and explored the interactive, biosocial understanding of human health that underlies the syndemic perspective. It has shown how the syndemic perspective developed in response to the distinct features of earlier stages in the evolution of disease conception, especially (with the rise of germ theory and biomedicine) reductionism and mind-body dualism and the subsequent realization of the limitations of these frames of understanding.

KEY TERMS

comorbidity
disease carrier
disease clustering
disease interaction
folk syndemic
germ theory
health and social disparities
host-pathogen interactions
humors
Koch's postulates

local knowledge
masked infection
miasma
mutualism
paradigm shift
pathogen
reigning paradigm
spectral disease
vector

QUESTIONS FOR DISCUSSION

1. What are the key features of a syndemic? Why are syndemics important in public health?

2. Why are some social groups at greater risk for syndemics than others?

3. How does the emergence of the syndemics model relate to prior transitions in the conception of disease?

4. What are the key differences between the concept of *comorbidity* and the concept of a *syndemic*?

5. What are the problems that arise in describing disease interactions in nonhuman animal species as syndemics?