CHAPTER 3

A Historical-Ecological Perspective on Epidemic Disease

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From the perspective of historical ecology, with its distinct focus on the human species, this chapter examines the history of epidemics by exploring the interrelationships between parasites, human hosts, and their environments. In this process, human societies are considered to be an integral part of the environment. While the emphasis of the chapter is on the ways in which differences in human social organization have affected patterns of epidemic disease through time, the value of a holistic approach is stressed through noting that parasites, their human hosts, and their physical environments have also changed, both singly and together, due to interactions between them and also to their own internal dynamics. The aim of this chapter is not to deny that there are general and long-term correspondences in historical processes, but rather to argue that an understanding of the trajectories of biological and social change and the complexities of the processes at work can be best achieved through viewing epidemics in their specific historical and geographical contexts.

The intimate relationship between humans and the natural world finds perhaps its clearest expression in human disease. Human diseases emerge from interactions between parasites, hosts, and their environments: no understanding of their origins, spread, and impact can be achieved if any of these three elements is excluded. It is necessary to be aware of the biology of parasites and hosts, the manner in which diseases are transmitted, the environmental conditions necessary for their survival, and also the characteristics of the human society affected. The need for a holistic approach is evident.

Infectious disease may be characterized as either chronic or acute. Chronic diseases—such as herpes simplex, tuberculosis, and treponemal infections—are those that persist for long periods, exhibiting little fluctuation in occurrence, and generally without conferring immunity on their hosts. Acute infections, on the other hand—such as measles, rubella, and smallpox—are characterized by short periods

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of infection that result in high morbidity and potential mortality while conferring lifelong immunity on survivors. The persistence of acute infections depends either on the existence of large populations of previously uninfected persons, or on nonhuman hosts, such as rodents or insects, in which the parasites may survive or reproduce. Historically, acute infections took the form of epidemics that resulted in short periods of high mortality—becoming endemic only where human populations were of sufficient size to generate enough susceptibles in the form of children to maintain the disease indefinitely. Chronic infections, on the other hand, are generally endemic, taking a regular toll of the population. Although the distinction between chronic-endemic and acute-epidemic diseases is important, it is not fixed: as already indicated, acute infections may become endemic in large populations, while minor changes in a parasite, host, or environment may occasionally cause a normally endemic disease to erupt as an epidemic (Ramenofsky 1987:140). The primary concern of this chapter is with acute infections that historically took the form of epidemics.

Epidemic diseases appear relatively late in the historical record and are commonly associated with the emergence of agriculture and sedentary communities (Cockburn 1971:50; Fenner 1970:64–65; 1980:14–16). Unidentified epidemics afflicted the Mediterranean, and probably India and China, in the first millennium B.C., but the first epidemics to be linked to measles and smallpox occurred in the second and third centuries A.D. (McNeill 1976:102–113). Within the last two hundred years, however, the incidence of epidemics has generally declined. This has been attributed to acquired immunity; to the isolation of disease organisms and the introduction of preventive measures, both medical and environmental; to improvements in living conditions and nutrition; and to changes in the parasites themselves. The relative importance of these factors, both in general and in the case of specific diseases, has been hotly debated (Cohen 1989:54; McKeown 1976:42–72; 1988:77–87). The fact that epidemics have declined in occurrence does not necessarily mean that humans are more healthy—for they continue to be afflicted by chronic infections, while the incidence of noncommunicable disease such as heart disease, diabetes, and cancer has increased (Boyden 1970:204–207; Mascie-Taylor 1993:14). Furthermore—as attested by Legionnaires' disease, Ebola, Marburg, Bolivian hemorrhagic fever, and AIDS—new transfers from other animal species, or changes in human parasites or the environment, mean that hitherto-unknown diseases continue to emerge.

Viewed from the perspective of the present, epidemic diseases appear to have already had their heyday in history, which makes them a fitting focus of historical ecology. Unfortunately, much of the history of human disease remains obscure, since it predates written records. Apart from the Black Death that afflicted Europe in the fourteenth century, the best-documented epidemics relate to the New World and to isolated communities, often located on islands—especially in the Pacific, where the impact of Old World diseases was late and sporadic (see, e.g., Black 1966; Cliff et al. 1981; Kunitz 1993; Stannard 1989). Because of the shortage of evidence for earlier periods, the discussion below draws on studies of the epidemiological and

demographic consequences of European expansion, particularly in the Americas, and on observations of historically more recent epidemics on which the mathematical models of the spread and impact of disease have generally been based.

It is worth noting at this stage that although European expansion was particularly significant in the spread of human disease, it was not a historically unique event; nor did it affect non-European peoples equally. Prior to European expansion, human populations in large parts of Eurasia and Africa had already been afflicted by newly emerging diseases to which they had not been previously exposed and had suffered levels of disease mortality comparable to those experienced in the Americas after 1492 (Patterson and Hartwig 1978:8); they had also, through centuries of contact, developed various levels of immunity to epidemic diseases, so that their contact with Europeans did not precipitate demographic disasters on a scale comparable to that experienced in the Americas or the Pacific islands (Crosby 1976; Dobyns 1966; Jacobs 1974). This example highlights the significance of the specific epidemic history of a region for understanding the subsequent occurrence and impact of disease at even the most extended geographical and temporal scale.

Present understanding of the spread and impact of human diseases derives in large part from attempts to control them. For such purposes sophisticated mathematical models, generally disease-specific, have been developed that are based largely on levels of susceptibility and patterns of contact. Such models have sometimes taken the age-structure, immunity, and distribution of populations into account, but differences in environmental and social conditions have not figured significantly, nor have biological differences in populations (human or nonhuman) been considered, particularly over a long timescale (Sattenspiel 1990:246–249, 270). The limitations of deterministic models, particularly the simple ones, have become more apparent with recent developments in the science of chaos that have stressed the complexity of systems and demonstrated how minor differences in initial conditions can produce major and unpredictable effects (Gleick 1987). In ecology, population biology, and epidemiology chaotic models have been developed to account for spatial and temporal discontinuities (Anderson and May 1979; Olsen and Schaffer 1990; Schaffer 1987). While these models have attempted to explain unpredictability in the incidence of infections, the range of factors considered has not expanded. In part this reflects the difficulty of handling complex systems mathematically, for in many cases the potential effect of minor differences in host, parasite, or environment on the origin, spread, and impact of a disease has been recognized, if only in passing.

In the qualitative literature there has been a growing recognition of the influence of environmental and social conditions on geographical and temporal variations in the progress of epidemics (Cohen 1989:7; Kunitz 1994:5–7; Milner 1980:46–47; Newson 1985:48). It cannot be assumed that a newly introduced disease will spread unhindered, and that its impact will be uniform; rather, each epidemic has its own history. In this chapter I will examine the significance of the local social context in understanding the origins, spread, and impact of epidemic diseases; but in order to stress the importance of a holistic perspective for an understanding of the historical

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Bioenvironmental Conditions: Variations and Change

Studies of the history of human disease (Cockburn 1971; McKeown 1988) have often recognized the reciprocal relationship between humans and parasites. Humans coexist with parasites as part of the animal world. The human body, especially the lower intestine, is home to hundreds of parasites, and the diversity of species it houses reflects the diversity of the environment in which it exists. Thus African and Malaysian hunter-gatherers living in the tropical rain forest contain twice as many intestinal parasites as Bushmen and Australian Aborigines, who live in a species-poor environment (Dunn 1972:227–228). Since human activity has often resulted in a simplification of the environment, over time the number of species harbored by humans has generally declined (Polunin 1977:16).

Many parasites coexist with humans without causing them any harm. Other parasites are unable to establish themselves in human hosts because when they invade the body either they kill the host, or else they induce an immune response that results in the parasite's death. Occasionally, however, a parasite is transmitted that is able to survive without killing its host, and in this way a new human pathogen emerges. Even then, if it has a short period of communicability it may be unable to reach another human host before it becomes extinct. The establishment of human diseases must have been characterized by many false starts. It is worth noting that most human diseases are thought to have originated in animals, and particularly in domesticated species with which humans have more intense contact; for example, smallpox is related to cowpox, and measles probably to canine distemper or rinderpest (Cohen 1989:7; McNeill 1976:54–56).

Once established as human diseases, parasites continue to interact with humans, sometimes resulting in genetic change. The life cycles of parasites are generally extremely short, being only a fraction of that of humans, so that the chance that a mutation will occur is far greater for them than for humans (Black 1980:42). Since most human diseases did not emerge until sedentary farming communities had become established, too few generations have passed for appreciable genetic changes to have occurred in humans (Boyden 1970:193)—with the obvious exceptions of the sickle-cell and the Duffy negative traits found among African populations, which provide some immunity to different forms of malaria (Dunn 1993:858–859). The former has been associated with *Plasmodium falciparum*, which may have emerged with intensive agriculture about 2,000 years ago; the latter trait, which is linked to the more benign *Plasmodium vivax*, is more widely dispersed, and this probably reflects its greater antiquity (Black 1980:50; Wiesenfeld 1967:1134–1139). Although these traits are associated with African populations, some resistance to malaria has been acquired by New World groups through racial mixing. Most often, however,

the most immediate and effective responses limiting human diseases have been social rather than biological. Vaccination programs, modifications to the environment, and the development of pesticides and insecticides have all been attempts to control or mitigate the impact of disease. However, as in the case of malaria, such actions have sometimes encouraged the emergence of new resistant strains.

While humans have developed little genetic resistance to disease, recent research has suggested that the degree of genetic diversity of human groups may influence disease mortality (Black 1992b). The significance of genetic homogeneity for disease mortality is that pathogens become preadapted to successive hosts who possess similar immune systems, resulting in increased virulence and host mortality. Native South Americans lost genetic traits as they passed through the environmental bottlenecks of the Bering Strait and the Panamanian isthmus, becoming genetically more homogeneous and therefore unusually susceptible to newly introduced diseases (Black 1990:65–68). Francis Black (1992a:4–5) has estimated that in the New World, which he defines as the Americas, Australasia, and Oceania, the minimum probability of a pathogen's encountering a host with a similar immune system is 28 percent, compared to only 2 percent in the Old World. In small isolated groups, however, the tendency toward genetic homogeneity is countered by random genetic drift—though operating against this process may be the seizure of captive women and children, or the incorporation of small groups, if their populations are too low to maintain a supply of suitable spouses (Bodmer and Cavalli-Sforza 1976:392–398). Therefore Black (1990:65) suggests that genetic distances among apparently unrelated tribes are not much greater than among tribes of one cultural group. Even if genetic diversity can be shown to affect disease mortality, its significance relative to environmental and cultural factors in influencing the pattern of infection remains difficult to determine (Jenkins et al. 1989:28–30; Svanborg-Eden and Levin 1990:33–34).

Discussions of the spread and impact of particular diseases in history often draw on the experience of recent epidemics. Yet even when a disease can be identified in the past—and this in itself is often problematic, given the vague descriptions of symptoms in the historical record, which in addition are sometimes based on classification systems that are not easy to interpret—it should not be assumed that the characteristics of the parasite have remained constant. Over a long time period probably no pathogen has gone unchanged, and some have changed considerably (McKeown 1988:77). Many viral diseases—especially influenza, but also smallpox and syphilis—evolve rapidly through mutation, recombination, and reassortment. These processes may produce different strains of the disease, with different latent and infectious periods and levels of virulence, to which previous exposure may not have conferred immunity (Ramenofsky 1987:137–138; Sattenspiel 1990:254).

Until recently it was generally considered that the coevolution of hosts and parasites resulted in reduced virulence, since this was to their mutual benefit (Allison 1982:245; Anderson and May 1991:249–250; Pimental 1968:1437). Indeed, it has been suggested that a number of human diseases, including smallpox and bubonic plague, have through repeated infections lost some of their virulence. In recent his-

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tory scarlet fever seems to have declined because of changes in the character of the disease (McKeown 1976:82–85). It has also been suggested that the sudden disappearance of plague from western Europe in the seventeenth century may have been due to changes in the virus or in the resistance of rat vectors (Appleby 1980:169–173; Slack 1981:471–472). However, on theoretical grounds it has been argued that the coevolution of parasites and hosts may not necessarily trend toward avirulence but may instead follow many paths, depending on the degree to which parasite transmissibility and recovery rates are linked to rates of host mortality (Anderson and May 1991:649–653; Levin et al. 1982:214). The key point is that each disease organism has its history. It cannot be assumed that the smallpox virus that devastated New World populations in the sixteenth century was the same as that which had afflicted China in the fourth century A.D., or was to strike Hawaii in 1853. Variations in the virulence of particular strains of disease may be relatively small, but in particular contexts they might be of considerable significance.

Before embarking on a discussion of societal and cultural influences on the origin, spread, and impact of human diseases, it is worth commenting on the significance of variations in the physical environment, even though this is often recognized, at least at a general level. Each parasite has certain environmental limits beyond which it cannot survive, of which perhaps the most obvious is climate. The influence of climate appears to be greater for diseases spread by aerial transmission—such as colds, influenza, measles, and smallpox—and for vector-borne diseases such as malaria, yellow fever, and sleeping sickness, which require certain temperatures for the reproduction of the parasite. Many diseases are confined to tropical climates. Seasonal variations in temperature and humidity, most notably in temperate climates, may also result in marked seasonal variations in the incidence of disease (Fenner 1982:112–114; Mascie-Taylor 1993:9–11; Upham 1986:119–122). In some cases the seasonal effects are not direct, but are mediated by human activities that often reflect climatic variations. For example, the regular congregation of people at particular times of the year for wider communal purposes, such as harvests or religious festivals, may facilitate the spread of disease, while seasonal food shortages may affect disease susceptibility.

Other environmental variables affecting the habitat of parasites are more closely related to human activity. Any change in habitat will encourage the expansion of some parasites at the expense of others, and the greater the simplification of habitats the greater the opportunity for particular species to multiply (Polunin 1977:15–16). Hence forest clearance may encourage the reproduction of mosquitoes that propagate malaria, but the specific parasites may also vary according to particular conditions—for example, on the coast of Ecuador the more virulent *Plasmodium falciparum* is found in more open environments, whereas the more benign *P. vivax* is prevalent where the vegetation cover has been less disturbed (Newson 1993:1190). Changes in the level of grazing may also influence the incidence of disease. Cattle may constitute alternative blood meals for mosquitoes, thereby encouraging the spread of malaria, but reduced levels of grazing by altering the environment may bring other disease hazards. In the 1890s rinderpest caused a decline

in cattle populations in Africa, which enabled the bush vegetation to recover and thereby encouraged the spread of tsetse flies carrying sleeping sickness (Curtin et al. 1978:553–554). Other forms of human environmental change associated with the increased incidence of disease are irrigation and dam building. These activities create stagnant water bodies that constitute favored habitats for the reproduction of mosquitoes and snails involved in the transmission of malaria and schistosomiasis, respectively (Boyden 1992:204; Cockburn 1971:49–50; Cohen 1989:42–43; Grove 1980:196–198; Kunitz 1994:11; McKeown 1988:190; Wirsing 1985:313). However, the process of human-induced environmental change is not unidirectional or always irreversible. Particular habitats may be abandoned in favor of others as a result of population decline, wars, or technological change (Denevan 1992:381).

Given the significance of environmental conditions for the incidence of epidemic disease, it is important to note that even when the impact of human activities is excluded, conditions have not remained constant. The influence of long-term climatic change has often been recognized, but environments have also experienced short-term perturbations and disturbances, sometimes caused by fire or drought, that have occurred as part of ecological processes (Cronon 1983:11; Demeritt 1994:23–26; Worster 1990:8–11). Ecologists now feel less comfortable with the concept of an ecosystem following a succession toward some sort of "climax" or natural equilibrium; they prefer to view nature as a landscape of patches that are continually changing through time and space as a result of their own dynamic processes. Nature is not only acted upon, but has its own history. Temporal and spatial variations in environmental conditions may not only affect the distribution of parasites, but through influencing the availability of resources also affect subsistence patterns, nutrition, and disease susceptibility. Studies of the subsistence economies of traditional societies show that they are affected, albeit often unconsciously, by relatively small shifts in environmental conditions (Dean et al. 1985:550; Larson, Johnson, and Michaelsen 1994:271, 288).

It has been shown that bioenvironmental conditions have not remained constant, but have changed through time, both individually and through interactions between them. While certain broad generalizations may be made about the nature of those changes, the changes themselves have often been unpredictable. Against this background of variability in hosts, parasites, and environments, the second part of this paper will examine how differences in social conditions affect their interaction and hence influence the origins, incidence, spread, and impact of epidemic disease.

The Origins and Incidence of Human Disease

When human populations subsisted on wild food resources, they were too small and too mobile to enable most parasites to become established as specifically human pathogens. Acute infections such as smallpox, measles, or influenza, which are characterized by short periods of infectivity, could not have survived, because they would have died out before reaching new hosts. The most successful diseases

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were those that had longer infectious periods and did not kill their hosts, and those that could survive on a nonhuman vector or intermediate host. Diseases that can persist for long periods in small populations include herpes simplex, chicken pox, typhoid, dysentery, hepatitis, leprosy, and treponemal infections; and, of those with nonhuman vectors, leishmaniasis, malaria, filaria, and schistosomiasis (Black 1975: 515–518; 1980:45–49; Cockburn 1971:50; Fenner 1980:14–15; Garruto 1981: 560–564; McKeown 1988:38, 49). As human populations increased and began to settle in permanent nucleated settlements, the opportunities expanded for parasites to develop as human pathogens. Thus with the beginnings of agriculture and sedentary life, the major killer diseases emerged (Cockburn 1971:48–51; Fenner 1970:48–68; McKeown 1988:48–56; McNeill 1976:54–57).

Population size is critical for understanding the incidence of infection. Since endemic infections are characterized by latency and recurrence, they can persist in small societies. Herpes simplex and chicken pox can survive in populations of less than 1,000, and even in isolated family units (Fenner 1970:58, 64). Acute infections require much larger populations. In classic papers, Maurice Bartlett (1957) argued that for measles to become endemic in U.S. cities, 7,000 susceptibles are required in an urban population of 250,000–300,000; Black (1966:210), using evidence from island communities, suggested that a threshold exceeding 500,000 may be required for a densely settled population. Since smallpox spreads less rapidly than measles, its threshold population has been estimated at 200,000 (Fenner et al. 1988:118).

Where populations are small and dispersed, such as among tribal groups, the shortage of new susceptibles to infect means that the spread of a disease is slow and "fade-outs" are common (Cliff and Haggett 1988:245–246; Haggett 1994:10–11; Neel 1977:160). Small communities may therefore remain relatively disease-free for long periods, but their lack of exposure to infection leads to a build-up of susceptibles so that when a disease is reintroduced through contact with a larger population it is associated with a higher level of mortality that affects adults as well as children. Whether diseases can become endemic or not is significant, because, as will be elaborated below, adult losses may undermine the functioning of the group and have particularly adverse effects on demographic trends, especially where populations are small. A further characteristic of the pattern of infection among small dispersed populations is that it is highly irregular in space and time (Dobson 1989:280; Haggett 1994:12). This is because contacts are fewer and there are frequent "fade-outs," so that in an epidemic some communities are likely to escape infection. If the same disease is reintroduced within a short period its spatial impact will be significantly affected by the irregular pattern of mortality and immunity produced by the previous epidemic. Within any region of dispersed population, therefore, there are likely to be considerable differences between neighboring communities in terms of their epidemic histories and their demographic trajectories.

Interest in the influence of population size on the spread of disease has focused on the population threshold needed to maintain a pool of susceptibles, but it is increasingly recognized that the latter is dependent not only on the size of the

population, but on a range of other factors, including the birth rate, migration, and the loss of immunity (Cohen 1989:49). Increasingly these variables have been incorporated into epidemic models. The birth rate has often been regarded as central to understanding both endemicity (Anderson and May 1979:366; Black 1966) and levels of recovery from epidemics (Thornton, Miller, and Warren 1991:30–39). In noncontracepting societies the birth rate is largely determined by social practices such as age at marriage, breast-feeding, child spacing, and sexual taboos (Marcy 1981:309–323). Hence a change from polygamous to monogamous practices, often associated with Christian conversion, by encouraging an increase in birth rate (Hern 1992:53–64; 1994:127, 137; Krzywicki 1934:201–202; Reid 1987:39–41) might have implications for the persistence of a disease as well as for demographic recovery. Thus while population size relates to culture in general, the size of the pool of susceptibles may vary widely from community to community according to social custom.

The Spread of Human Diseases

Human diseases are transmitted in different ways. Some are spread by direct face-to-face contact, while others are spread by a vector such as a rodent or insect. In the latter case, human location near the nonhuman reservoirs is essential for disease transmission, and, as already indicated, disease occurrence is often highly dependent on environmental conditions. The following discussion will concentrate on directly transmitted diseases.

Epidemics may break out when changes in the parasite or the environment create more favorable conditions for a pathogen's spread, but historically many were introduced from other regions. Indeed, the incidence of infection is often highly dependent on the intensity of outside contacts or the movement of peoples to new disease environments. In examining the history of epidemics, William McNeill (1976) has identified three major periods of disease outbreaks that followed significant population movements. These movements included the establishment of contact between Europe and Asia in the early Christian era, the expansion of the Mongol empire in the thirteenth century, and the beginning of European overseas expansion in the fifteenth century. Over time the improvements in methods of transport have enabled epidemic diseases to spread more quickly and widely. The development of ocean navigation and European colonial expansion from the fifteenth century is particularly significant in the history of disease, since it vastly expanded contacts and introduced infections to previously unexposed populations (Fenner 1970:65–66; 1980:19–20; McNeill 1976:185–216; 1979:97). Colonial expansion did not only bring epidemic diseases, it also established new trading networks and patterns of migration that facilitated their spread, while it transformed native economies and societies in ways that affected their impact (Dobson 1989:287–294; Jackson 1994:161; Kunitz 1993:130, 132, 157; Newson 1985:49–66; 1993:1188, 1193–1194). Because colonial empires often sought to capture sources of labor, their efforts were concentrated

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where there were dense native populations; since epidemics could spread more easily among these groups, it was they who bore the initial impact of disease. Subsequently, more remote (and often smaller) populations were affected—but, as will be shown later, here the effects differed.

Epidemic models developed to explain the spread of infections have tended to calculate the probability of contacts based on population size, without considering the geographical distribution and mobility of the population and the nature and intensity of human contacts. In small, mobile, dispersed populations, contacts are generally fewer, irregular, and unsustained, and the build-up of parasites is more limited (Cohen 1989:39–41; Coimbra 1988:90–91; Polunin 1977:8–13; Wirsing 1985:311). This contrasts with large, permanent, nucleated settlements, which engender unhealthy conditions by encouraging the build-up of wastes and parasites, and also facilitate the rapid spread of directly transmitted diseases. Historically, towns and cities could not sustain their populations independent of migration from the countryside, which replenished urban losses from disease and famine (McNeill 1976:65; 1979:96). More recent historical processes involving the congregation of formerly dispersed peoples in nucleated settlements—to achieve greater administrative control or Christian conversion, or to facilitate major development projects such as dam building—have encouraged the spread of disease (Cook 1943:30–34; Curtin et al. 1978:554; Wirsing 1985:313). The significance of population concentration may be extended to the scale of individual households. In sixteenth-century Ecuador, Spanish attempts to control promiscuity by replacing extended-family or multifamily households with nuclear family residences were recognized at the time as a factor reducing the levels of disease mortality (Newson 1992:109).

It is not only population size and distribution per se that are important in understanding the spread of disease, but also the geographical location of communities and the character and intensity of contacts between them (Milner 1980:47; 1992:110–111). Rugged terrain or adverse climatic conditions are likely to hinder the spread of disease, while easy communications are likely to facilitate it (Shea 1992:160–161). Ann Ramenofsky (1990:41–42) has proposed a model relating the impact of disease to settlement types—specifically, to their location, duration (sedentary or mobile), and form (nucleated or dispersed). She hypothesizes that nucleated settlements (whether sedentary or mobile) located along primary drainage systems have the lowest probability of persistence because of more frequent and regular contacts, both between their members and with outside groups. In general, friendly contacts are likely to be more frequent and sustained. Indeed, trading contacts have generally been recognized as important channels for the spread of disease, and in the African context it has even been suggested that there might be variations in patterns of infection among ethnic and occupational groups according to their participation in trade (Hartwig 1978:25–43; Patterson and Hartwig 1978:12). In addition, where friendly relations exist, native responses to health crises may involve visiting the sick, the convening of communal gatherings, or the provision of hospitality for those fleeing from epidemics—all of which might actively promote their spread (Crosby 1976:297; Dobyns 1983:16; Krech 1978:715–716; Wirsing

1985:311). Hostile relations, on the other hand, generally discourage contacts and the spread of acute infections, even though these might be transmitted during a brief raid. In some cases uninhabited buffer zones between hostile groups, such as probably existed in the Amazon Basin and parts of North America, may have acted as effective disease barriers (De Boer 1981:365; Myers 1976; Snow and Lanphear 1988:17; Thornton, Warren, and Miller 1992:192–193).

The Impact of Epidemic Disease

Patterns of disease mortality will vary in the first instance with the particular pathogen responsible and the level of immunity acquired by a population, the latter depending in part on the size of the population and whether the disease has become endemic. Endemic diseases persist among human populations, taking a small but regular toll of infants as they are weaned and lose the immunity acquired from their mothers. (At this stage the nutritional status of an individual may play a significant role in determining susceptibility. The relationship between nutrition and susceptibility to disease will be considered further below.) Where diseases fail to become endemic, communities may remain disease-free for long periods, but this leads to a build-up of susceptibles, so that when an infection is reintroduced from the outside it results in high levels of mortality among adults as well as children.

Mathematically it can be shown that high death rates from infrequent epidemics have a less-significant effect on the general level of mortality and rate of population growth than do endemic infections that kill a significant proportion of children within ten years of birth (Dobson 1989:288; McKeown 1976:69). However, this generalization is not particularly helpful in predicting the overall impact of epidemics, for it takes no account of the indirect effects of child and adult losses on the continued functioning of the society and on demographic trends, which would vary according to specific social and environmental conditions. Where losses occur among children they may retard demographic recovery, but they do not threaten the functioning or survival of the group. Adult losses, however, not only result in an immediate loss of reproductive capacity, but may also undermine a wide range of activities necessary for the community's maintenance and recovery (Kunitz 1984:560; McNeill 1979:96; Whitmore 1991:479–480). This pattern is most common among small populations, which commonly possess other features (to be elaborated upon below) that can increase the effect of adult losses and raise mortality levels even higher. In order to understand the impact of an epidemic it is therefore necessary to be aware of the particular individuals or social groups affected by the disease.

Adult losses may undermine, or even destroy, subsistence patterns, create food shortages or famines, and thereby enhance disease susceptibility. Falling numbers may result in decreased production, weaken reciprocal social obligations, and cause a shift in emphasis on different activities, sometimes closing off subsistence options altogether and rendering communities more vulnerable in the face of environmental perturbations (Larson, Johnson, and Michaelsen 1994:276). The devastating impact

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of Old World epidemics in the Americas led to the collapse of many forms of intensive production—such as irrigation canals, terracing, and raised fields—that could not be maintained with declining populations (Denevan 1992:375–381). In some cases it led to the abandonment of agriculture altogether (Balée 1992:51).

Significant differences appear to exist between state and nonstate societies in their ability to sustain food supplies and economic production during epidemics. State societies are generally more productive, have forms of storage and food distribution systems that may overcome temporary shortages, and are characterized by political organizations that can mobilize labor to maintain production in times of crisis. Even though, in general, state societies may be better able to cope with epidemics, food supplies will vary between social groups (class, race, gender, and age) according to their status and access to resources. The economies of nonstate societies, especially those dependent on wild food resources, are often highly adapted to specific environments. Food production prospects are generally dependent on whether subsistence strategies or cooperative activities can remain viable with a reduced population. In small societies the loss of only a small number of those with special skills, such as hunting, may be a serious threat to food supplies—particularly in regions of marked seasonality, or where groups are dependent on a limited range of resources (Dobyns 1983:16, 332; Hill 1989:12; Krech 1978:717; McGrath 1991). The vulnerability of these groups is often enhanced by the limited surpluses produced, and by their lack of familiarity with methods of food storage. Furthermore, their leaders may lack the authority to organize practical responses to epidemic crises that might aid survival (Stannard 1991:531; Zeitlin 1989:57–60). During the measles epidemic that afflicted the Yanomama in the 1960s, James Neel and others observed that village life collapsed completely: only a few members remained capable of providing food and water or tending the sick, and the concern for well-being seldom extended beyond the immediate family (Neel et al. 1970:427).

In assessing the impact of epidemics on food production, nutritional levels, and disease susceptibility, it is important to recognize that the nutritional status of a community is not determined solely by food supplies: it is also dependent on the members' energy requirements (Walter and Schofield 1989:17–21). These are greater in colder environments and where individuals are involved in arduous labor or are constantly fighting infection. The harsh labor conditions often introduced by colonial regimes not only affected the health of native peoples directly but also contributed to their declining nutritional status, which in many cases was already threatened by diminished food supplies resulting from new external demands on their lands, labor, and production (Newson 1985:62–66). According to Stephen Kunitz (1994:51), a significant factor explaining the greater depopulation of Hawaii and New Zealand compared to western Polynesia (Samoa and Tonga) was the more extensive expropriation of land, which destroyed native subsistence production and increased disease susceptibility. Clearly the study of variations in environmental and social conditions is essential to understanding nutritional status.

Epidemics may bring famine, starvation, and death, but in many cases the greatest impact of reduced levels of production is through declining nutritional levels

that may enhance disease susceptibility (McKeown 1988:52–55). However, the direct relationship between malnutrition and infection is difficult to substantiate, since malnourished individuals are also likely to experience poor living conditions, where crowded accommodation and inadequate sanitation may favor the spread of disease. Furthermore, deficiencies in protein or in particular vitamins and minerals may actually provide some resistance to infection (Cohen 1989:167); for example, many disease organisms need iron to thrive, so that short-term iron deficiency may assist the body to fight infection. In general, the relationship between disease and nutrition is now thought to be less clear, and certainly more complex, than previously envisaged.

In regard to disease mortality, the link with malnutrition appears stronger for some diseases than for others (Cohen 1989:167; Livi-Bacci 1991:35–39; McKeown 1988:52–53; Rotberg and Rabb 1983:305–308). In the case of measles and most respiratory and intestinal infections, levels of morbidity and mortality appear to increase with poor nutrition; whereas smallpox, plague, yellow fever, and malaria seem to be relatively unaffected by nutritional status, and have in the past killed both affluent and poor (McKeown 1988:52). Also, nutritional status is probably insignificant in the case of particularly virulent strains, and where individuals have not been previously exposed to the disease. However, where microorganisms become endemic, malnutrition may exert a greater influence on disease mortality, particularly infant and child mortality (Harpending, Draper, and Pennington 1990:257–258). Class and gender differences also may be of significance at this stage.

The importance of local conditions for understanding the impact of epidemic disease becomes particularly evident when social reactions are considered. Epidemics may result in social disorganization, as marriages break down due to death or flight and as political authority and religious beliefs are questioned for their failure to explain or cope with disaster (McGrath 1991:417; Stannard 1991:531; Zubrow 1990:761). These processes of social disintegration may lead to the biological or cultural extinction of the group, but they may also result in the emergence of new forms of social organization that may aid recovery. The particular demographic path followed by a group is closely linked to existing social practices and demographic regimes, while the degree of any recovery achieved will affect the size of the population, the numbers of susceptibles, and the future pattern of disease spread.

Epidemics that result in high adult mortality result in greater social disorganization. However, the impact of the loss of equal numbers of adults will differ in different societies. Adult losses are more difficult to sustain in small communities, where the formation of new unions may be more difficult to achieve because the availability of partners is by definition more limited and sex ratios are often more volatile (Early and Peters 1990:137, 140; Kunitz 1994:9). The problem of unbalanced sex ratios may be aggravated in the case of particular diseases—such as smallpox, influenza, malaria, and dysentery—that cause high levels of mortality among pregnant women (McFalls and McFalls 1984:60–61, 130, 533–534; Stannard 1990:336–347). The ability t
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In conditions of declining numbers and limited marriage pools, in order to ensure biological survival groups may have to modify their population policies and social attitudes. As Charles Wagley (1951) showed for two Tupi-Guaranian groups, even within the same cultural-linguistic group differences in social practice may mean that some populations are preconditioned to cope more effectively with epidemic stress. Groups with rigid social practices that function to control population numbers may be significantly disadvantaged in demographic crises, whereas those that are more flexible in their population policies or in their attitudes toward outsiders may be able to survive. Francis Johnston and his coworkers (Johnston et al. 1969:33) observed that the Peruvian Cashinahua were able to recover from a severe epidemic in 1951 by abandoning birth control practices. It is also noteworthy that many surviving indigenous societies are exogamous and in past periods have been prepared to absorb or be absorbed by outsiders (Dobyns 1983:306, 310–311; McGrath 1991:414; Milner 1980:47; Thornton 1986:128–129). Why some groups opt for change and others do not is unclear. Henry Dobyns (1983:303–306, 310–311) has suggested that amalgamation with other cultural groups occurs where native populations have fallen below their conception of the ideal size of a community. In other cases, decisions appear to be more pragmatic. For example, Black (1975:516) notes how two small societies in northern Brazil joined the Tiriyó when faced with the problem of finding marital partners not forbidden by incest taboos. Anthony F. C. Wallace (1956:269–272) doubts that such decisions emerged from community deliberations; rather, he thinks they were directed by individuals—particularly native leaders and shamans, perhaps guided by vision experiences brought on by physical and psychological stress. In the case of the Cashinahua noted above it was the shaman who proscribed contraceptives and abortifacients (Johnston et al. 1969:33). Just as the spread of epidemics may be highly variable in time and space, so social practice and action may enhance that variability, resulting in some communities' becoming extinct while their neighbors survive.

Social structures and belief systems can also influence a group's response to an epidemic crisis. Individuals' ability to cope with stress, which in itself can enhance disease susceptibility (Cassell 1976:107–123), may be affected by the actions of their leaders and by the way the epidemic is explained. As previously noted, leaders may play critical roles in shaping community reactions, instilling a "will to survive," and organizing practical responses to epidemic crises, such as ensuring continued production, introducing public health measures, or providing social support (McGrath 1991:417; Stannard 1991:531; Zeitlin 1989:57–60). Even though the

medical treatments employed may be of little intrinsic value, the provision of basic needs is clearly essential to survival; and it is less commonly recognized that nursing care can reduce mortality levels significantly (Carmichael 1983:59–60; Crosby 1976:294; McCaa 1995:420–422). Indeed, Mark Cohen (1989:39) argues that one of the few advantages of sedentism in regard to the impact of disease is the ability to care for the sick.

The limited political power exercised in nonstate societies might render them less able to cope with epidemic crises than those with strong leadership. Any authority the leaders may possess could be weakened further by their apparent inability to control an epidemic, especially where leadership has a functional rather than a hereditary basis. Yet while, in general, state societies may be better able to cope with disasters (and it might even be suggested that highly centralized polities would be most effective in crisis situations), when leaders succumb to the epidemic or the political organization fails to contain the crisis, dissension over policies and procedures can lead to the rejection of authority—leading to social disorder, and sometimes violent conflict. This was the case in the Andes between 1524 and 1527 when the death of the Inca ruler, Huayna Capac, probably from smallpox, precipitated civil wars (Newson 1992:88). Conflicts between groups and within groups may not only enhance mortality levels directly, but by disrupting subsistence production and social functioning may aggravate the impact of epidemics (McGrath 1991:412, 417–418; Milner 1992:111).

A successful response depends not only on effective leadership but also on a correct identification of the cause of the epidemic and the means of its transmission. Quarantining might effectively isolate a disease spread by face-to-face contact, such as smallpox and measles, but it would have little effect on reducing the incidence of a disease spread by a vector, such as malaria or typhus; in the latter case, changes to environmental conditions would be more appropriate (McGrath 1991: 410–417). Only within the last two hundred years have the causes of most diseases been accurately identified. Previous success, particularly with unfamiliar infections, was largely a matter of chance, trial and error, and perceived effectiveness, without an understanding of the process of transmission or the biology of parasites (Boyden 1970:197–200; 1992:207–208). Where the cause of an epidemic is not understood it may be ascribed to a supernatural agent. This may result in a fatalistic attitude toward death, or in the placing of blame on a particular individual, group, or class, which may lead to social conflict (Jenkins et al. 1989:37, 44–45; McGrath 1991:411, 417). Increased intergroup warfare following epidemics has been commonly noted in Amazonia, where sickness and death are often attributed to sorcery—which requires revenge (Early and Peters 1990:80; Ferguson 1990:241–242; Hill 1989:12–13; Newson 1995:318). Misidentification of the cause of a sickness and inappropriate responses may thus enhance the levels of mortality that can be attributed directly to disease.

Although specific environmental and social contexts are important in understanding the effect of epidemic disease on individual communities, the preceding discussion suggests that a disease's incidence and impact are significantly affected by the size of the population and its political economy. In state societies, the spread

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of disease may be facilitated by the relatively large size of the population and its nucleated settlement patterns; but in the longer term, the effect of epidemics on these societies may be moderated as the relatively large size of their populations enables diseases to become endemic and ensures an adequate supply of spouses to maintain the fertility rate. Furthermore, their production systems and sociopolitical organizations generally enable them to cope more effectively with epidemic crises and achieve a level of demographic recovery. Conversely, a settlement pattern of small dispersed populations may retard the spread of diseases and moderate their direct impact; but in these circumstances diseases fail to become endemic, so that when infections are reintroduced from outside they cause high levels of adult mortality that may have particularly adverse effects on fertility levels and food production. Furthermore, such groups often lack the strong leadership needed to foster a "will to survive" and mobilize community efforts to cope with disaster. Thus while disease mortality might be moderated (though this would depend on the frequency of reinfection), the indirect impact of epidemics might threaten any demographic recovery and raise mortality rates to levels greater than those suffered by state societies.

These broad differences in the pattern of infection may partially account for the continued decline of native peoples in lowland areas of Latin America during the colonial period, while most former chiefdom and state societies experienced a degree of demographic recovery, the timing of which varied from region to region but began in some areas in the early seventeenth century (Newson 1985:43–45). This contrast in demographic trends was characteristic of colonial Ecuador, where there are suggestions that in the highlands some diseases were becoming endemic in the early seventeenth century when the Indian population began to recover (Alchon 1991:57–58, 76–77), whereas in the eastern lowlands and on the Pacific coast epidemics and population decline continued throughout the colonial period. Although it may be hypothesized that such differences in demographic trends may be related to differences in the pattern of infection, it is difficult to generalize about differences in their aggregate effects—for clearly the impact of epidemics, particularly on small populations, would depend, among other things, on the frequency of reinfection.

This chapter has focused on epidemics, which historically have exerted a powerful influence on demographic trends. Nevertheless, even in these circumstances disease mortality can provide only a partial explanation for demographic changes, which also reflect changes in fertility rates as well as the effect of other factors on mortality levels. Epidemics have often occurred during periods of economic crisis and political change that in many cases have been characterized by conflict, major social upheavals, economic hardship, the questioning of belief systems, and even environmental change. These processes, perhaps most evident in the expansion of colonial rule, may not only affect demographic trends directly, but also, through interacting with biological processes, may influence patterns of infection and mortality. The coincidence of epidemics with periods of change is often critical in understanding their demographic impact (Kunitz 1993:135; 1994:13; Patterson and Hartwig 1978:10–13). The complexity of the interaction is such that one cannot be understood without the other.

Conclusion

Historical studies of epidemics often assume that, once introduced, diseases have spread unhindered, and that their impact has been uniform. In this chapter I have stressed that the origins, spread, and effect of epidemic disease cannot be understood without acknowledging the significance of differences in the character of the parasite, the host, and the physical and social environment in which they interact over time. In particular, I have shown how the impact of epidemic disease is influenced by the size, distribution, and character of human populations—especially their settlement patterns, subsistence systems, sociopolitical organization, and ideology. This means that the effect of even a single disease is likely to be highly variable in time and space. Although I have here stressed the importance of particular environmental and social circumstances, my aim has not been to argue for a relativistic approach, but rather to demonstrate the value of adopting a holistic framework that acknowledges human variability and the dynamic quality of interactions. In essence, I am advocating a fuller recognition of the complexity of the processes at work—for it is only from this perspective that the highly variable temporal and spatial patterns in the spread and impact of epidemic disease, which are unexplained by simple deterministic models, can be understood, and that long-term correspondences can be identified.

References

Alchon, S. A. 1991. *Native Society and Disease in Colonial Ecuador*. Cambridge: Cambridge University Press.

Allison, A. C. 1982. Coevolution between hosts and infectious disease agents and its effects on virulence. Pp. 245–267 in Anderson and May (eds.), *Population Biology of Infectious Diseases*.

Anderson, R. M. and R. M. May. 1979. Population biology of infectious diseases: Part I. *Nature* 280:361–367.

———. 1991. *Infectious Diseases of Humans: Dynamic and Control*. Oxford: Oxford University Press.

———, eds. 1982. *Population Biology of Infectious Diseases*. Heidelberg: Springer-Verlag.

Appleby, A. B. 1980. The disappearance of plague: A continuing puzzle. *Economic History Review* 33(2): 161–173.

Balée, W. 1992. People of the fallow: A historical ecology of foraging in lowland South America. Pp. 35–57 in K. H. Redford and C. Padoch (eds.), *Conservation of Neotropical Forests: Working from Traditional Resource Use*. New York: Columbia University Press.

Bartlett, M. S. 1957. Measles periodicity and community size. *Journal of the Royal Statistical Society*, ser. A, 120:48–70.

Black, F. L. 1966. Measles endemicity in insular populations: Critical community size and its evolutionary implications. *Journal of Theoretical Biology* 11:207–211.

———. 1975. Infectious diseases in primitive societies. *Science* 187:515–518.

———. 1980. Modern isolated pre-agricultural populations as a source of information on prehistoric epidemic patterns. Pp. 37–54 in Stanley and Joske (eds.), *Changing Disease Patterns and Human Behaviour*.

———. 1990. Infectious disease South American forest tribes. I *Populations in Transition*.

———. 1992a. Low polymorphisı risk from variant pathogens. Ur

———. 1992b. Why did they die?

Bodmer, W. F. and L. L. Cavalli-S cisco: Freeman.

Boyden, S. 1970. Cultural adaptatic den (ed.), *The Impact of Civilis* tional University Press.

———. 1992. *Biohistory: The Inte and Present*. Paris: UNESCO an

Carmichael, A. G. 1983. Infection, Rabb (eds.), *Hunger and Histor*

Cassell, J. 1976. The contribution c *Journal of Epidemiology* 104(2):

Cliff, A. and P. Haggett. 1988. *Atlas*

Cliff, A. D., P. Haggett, J. K. Ord, and *graphy of Epidemics in an Island*

Cockburn, A. T. 1971. Infectious d 12:45–62.

Cohen, M. N. 1989. *Health and the F*

Coimbra, C. E. A. Jr. 1988. Human s lowland Amazonia: The case of C

Cook, S. F. 1943. *The Conflict Betw The Indian Versus the Spanish Mi* geles: University of California Pre

Cronon, W. 1983. *Changes in the La gland*. New York: Hill and Wang.

Crosby, A. W. 1976. Virgin soil epic America. *William and Mary Quar*

Curtin, P., S. Feierman, L. Thompson, a

Dean, J. S., R. C. Euler, G. J. Gumerm Human behavior, demography, anc *can Antiquity* 50(3): 537–554.

De Boer, W. 1981. Buffer zones in the historical approach. *American Anti*

Demeritt, D. 1994. Ecology, objectivit eties. *Journal of Historical Geogra*

Denevan, W. M. 1992. The pristine my *the Association of American Geogr*

Dobson, M. J. 1989. Mortality gradient gland and colonial America. *Social*

Dobyns, H. F. 1966. Estimating aborigi with a new hemispheric estimate. *C*

———. 1983. *Their Number Become Eastern North America*. Knoxville:

———. 1990. Infectious disease and evolution of human populations: The example of South American forest tribes. Pp. 57–74 in Swedlund and Armelagos (eds.), *Disease in Populations in Transition*.

———. 1992a. Low polymorphism in the immune system puts New World populations at risk from variant pathogens. Unpublished MS.

———. 1992b. Why did they die? *Science* 258:1739–1740.

Bodmer, W. F. and L. L. Cavalli-Sforza. 1976. *Genetics, Evolution, and Man*. San Francisco: Freeman.

Boyden, S. 1970. Cultural adaptation to biological maladjustment. Pp. 190–218 in S. Boyden (ed.), *The Impact of Civilisation on the Biology of Man*. Canberra: Australian National University Press.

———. 1992. *Biohistory: The Interplay Between Human Society and the Biosphere: Past and Present*. Paris: UNESCO and Parthenon.

Carmichael, A. G. 1983. Infection, hidden hunger, and history. Pp. 51–66 in Rotberg and Rabb (eds.), *Hunger and History*.

Cassell, J. 1976. The contribution of the social environment to host resistance. *American Journal of Epidemiology* 104(2): 107–123.

Cliff, A. and P. Haggett. 1988. *Atlas of Disease Distributions*. Oxford: Blackwell.

Cliff, A. D., P. Haggett, J. K. Ord, and G. R. Versey. 1981. *Spatial Diffusion: An Historical Geography of Epidemics in an Island Community*. Cambridge: Cambridge University Press.

Cockburn, A. T. 1971. Infectious diseases in ancient populations. *Current Anthropology* 12:45–62.

Cohen, M. N. 1989. *Health and the Rise of Civilization*. New Haven: Yale University Press.

Coimbra, C. E. A. Jr. 1988. Human settlements, demographic patterns, and epidemiology in lowland Amazonia: The case of Chagas's disease. *American Anthropologist* 90:82–97.

Cook, S. F. 1943. *The Conflict Between the Californian Indian and White Civilization: 1. The Indian Versus the Spanish Mission*. Ibero-Americana, vol. 21. Berkeley and Los Angeles: University of California Press.

Cronon, W. 1983. *Changes in the Land: Indians, Colonists, and the Ecology of New England*. New York: Hill and Wang.

Crosby, A. W. 1976. Virgin soil epidemics as a factor in the aboriginal depopulation in America. *William and Mary Quarterly* 33:289–299.

Curtin, P., S. Feierman, L. Thompson, and J. Vansina. 1978. *African History*. Harlow: Longman.

Dean, J. S., R. C. Euler, G. J. Gumerman, F. Plog, R. H. Hevly, and T. N. V. Karlstrom. 1985. Human behavior, demography, and paleoenvironment on the Colorado plateaus. *American Antiquity* 50(3): 537–554.

De Boer, W. 1981. Buffer zones in the cultural ecology of aboriginal Amazonia: An ethnohistorical approach. *American Antiquity* 46:364–377.

Demeritt, D. 1994. Ecology, objectivity, and critique in writings on nature and human societies. *Journal of Historical Geography* 20(1): 22–37.

Denevan, W. M. 1992. The pristine myth: The landscape of the Americas in 1492. *Annals of the Association of American Geographers* 82(3): 369–385.

Dobson, M. J. 1989. Mortality gradients and disease exchanges: Comparisons from Old England and colonial America. *Social History of Medicine* 2: 259-295.

Dobyns, H. F. 1966. Estimating aboriginal American population: An appraisal of techniques with a new hemispheric estimate. *Current Anthropology* 7:395–416.

———. 1983. *Their Number Become Thinned: Native American Population Dynamics in Eastern North America*. Knoxville: University of Tennessee Press.

Dunn, F. L. 1972. Epidemiological factors: Health and disease in hunter-gatherers. Pp. 221–228 in R. B. Lee and I. DeVore (eds.), *Man the Hunter*. Chicago: Aldine-Atherton.

———. 1993. Malaria. Pp. 855–862 in K. F. Kiple (ed.), *The Cambridge World History of Human Disease*. Cambridge: Cambridge University Press.

Early, J. D. and J. F. Peters. 1990. *The Population Dynamics of the Mucajai Yanomama*. New York: Academic Press.

Fenner, F. L. 1970. The effects of changing social organization on the infectious diseases of man. Pp. 48–76 in Boyden (ed.), *The Impact of Civilisation on the Biology of Man*.

———. 1980. Sociocultural change and environmental diseases. Pp. 7–26 in Stanley and Joske (eds.), *Changing Disease Patterns and Human Behaviour*.

———. 1982. Transmission cycles and broad patterns of observed epidemiological behaviour in human and other animals. Pp. 103–119 in Anderson and May (eds.), *Population Biology of Infectious Diseases*.

Fenner, F. L., D. A. Henderson, I. Arita, Z. Jezek, and I. D. Ladnyi. 1988. *Smallpox and Its Eradication*. Geneva: World Health Organization.

Ferguson, R. B. 1990. Blood of the Leviathan: Western contact and warfare in Amazonia. *American Ethnologist* 17:237–257.

Garruto, R. M. 1981. Disease patterns of isolated groups. Pp. 557–597 in H. Rothschild (ed.), *Biocultural Aspects of Disease*. New York: Academic Press.

Gleick, J. 1987. *Chaos*. London: Heinemann.

Grove, D. L. 1980. Schistosomes, snails, and man. Pp. 187–204 in Stanley and Joske (eds.), *Changing Disease Patterns and Human Behaviour*.

Haggett, P. 1994. Prediction and predictability in geographic systems. *Transactions of the Institute of British Geographers*, n.s., 19:6–20.

Harpending, H. C., P. Draper, and R. Pennington. 1990. Cultural evolution, parental care, and mortality. Pp. 251–265 in Swedlund and Armelagos (eds.), *Disease in Populations in Transition*.

Hartwig, G. W. 1978. Social consequences of epidemic diseases: The nineteenth century in eastern Africa. Pp. 25–45 in G. W. Hartwig and K. D. Patterson (eds.), *Disease in African History*. Durham, N.C.: Duke University Press.

Harvey, H. R. 1967. Population of the Cahuilla Indians: Decline and causes. *Eugenics Quarterly* 14:185–198.

Hern, W. M. 1992. Polygyny and fertility among the Shipibo of the Peruvian Amazon. *Population Studies* 46(1): 53–64.

———. 1994. Health and demography of native Amazonians: Historical perspective and current status. Pp. 123–149 in A. Roosevelt (ed.), *Amazonian Indians from Prehistory to the Present: Anthropological Perspectives*. Tucson: University of Arizona Press.

Hill, J. D. 1989. Ritual production of environmental history among the Arawakan Wakuénai of Venezuela. *Human Ecology* 17(1): 1–25.

Jackson, R. H. 1994. *Indian Population Decline: The Missions of Northwestern New Spain, 1687–1840*. Albuquerque: University of New Mexico Press.

Jacobs, W. R. 1974. The tip of the iceberg: Pre-Columbian Indian demography and some implications for revisionism. *William and Mary Quarterly* 31:123–132.

Jenkins, C., M. Dimitrakakis, I. Cook, R. Sanders, and N. Stallman. 1989. Culture change and epidemiological patterns among the Hagahai, Papua New Guinea. *Human Ecology* 17(1): 27–57.

Johnston, F. E., K. M. Kensinger, ture of the Peruvian Cashina *Human Biology* 41:29–41.

Krech, S. III. 1978. Disease, starva *ican Ethnologist* 5:710–732.

Krzywicki, L. 1934. *Primitive Soc*

Kunitz, S. J. 1984. Mortality ch 559–582.

———. 1993. Historical and conte Mascie-Taylor (ed.), *The Anthr*

———. 1994. *Disease and Social Europeans*. Oxford: Oxford Un

Larson, D. O, J. R. Johnson, and J. Chumash of central California *thropologist* 96(2): 263–299.

Levin, B. R., A. C. Allison, H. J. Br and H. R. Thieme. 1982. Evolut May (eds.), *Population Biology*

Livi-Bacci, M. 1991. *Population a tory*. Cambridge: Cambridge Ur

McCaa, R. 1995. Spanish and Nahu Mexico. *Journal of Interdisciplin*

McFalls, J. A. and M. H. McFall Press.

McGrath, J. W. 1991. Biological in ease. *American Journal of Physi*

McKeown, T. 1976. *The Modern Ri*

———. 1988. *The Origins of Huma*

McNeill, W. M. 1976. *Plagues and*

———. 1979. Historical patterns of

Marcy, P. T. 1981. Factors affectin *Journal of Family History* 6:309-

Mascie-Taylor, C. G. N. 1993. The b Mascie-Taylor (ed.), *The Anthrop*

Milner, G. R. 1980. Epidemic disease *of Archaeology* 5(1): 39–56.

———. 1992. Disease and sociopoli J. W. Verano and D. H. Ubelak Washington, D.C.: Smithsonian I

Myers, T. P. 1976. Defended territ 78:354–355.

Neel, J. V. 1977. Health and disease i in K. Elliott and J. Whelan (eds.), tion Symposium 49. Amsterdam:

Neel, J. V., W. R. Centerwall, N. A. C measles and measles vaccine in *American Journal of Epidemiolog*

Johnston, F. E., K. M. Kensinger, R. L. Jantz, and G. F. Walker. 1969. The population structure of the Peruvian Cashinahua: Demographic, genetic, and cultural relationships. *Human Biology* 41:29–41.

Krech, S. III. 1978. Disease, starvation, and northern Athapaskan social organization. *American Ethnologist* 5:710–732.

Krzywicki, L. 1934. *Primitive Society and Its Vital Statistics*. London: Macmillan.

Kunitz, S. J. 1984. Mortality change in America, 1620–1920. *Human Biology* 56(3): 559–582.

———. 1993. Historical and contemporary mortality patterns in Polynesia. Pp. 125–166 in Mascie-Taylor (ed.), *The Anthropology of Disease*.

———. 1994. *Disease and Social Diversity: The European Impact on the Health of Non-Europeans*. Oxford: Oxford University Press.

Larson, D. O, J. R. Johnson, and J. C. Michaelsen. 1994. Missionization among the coastal Chumash of central California: A study in risk minimization strategies. *American Anthropologist* 96(2): 263–299.

Levin, B. R., A. C. Allison, H. J. Bremermann, L. L. Cavalli-Sforza, S. A. Levin, R. M. May, and H. R. Thieme. 1982. Evolution of parasites and hosts. Pp. 213–243 in Anderson and May (eds.), *Population Biology of Infectious Diseases*.

Livi-Bacci, M. 1991. *Population and Nutrition: An Essay on European Demographic History*. Cambridge: Cambridge University Press.

McCaa, R. 1995. Spanish and Nahuatl views on smallpox and demographic catastrophe in Mexico. *Journal of Interdisciplinary History* 25(3): 397–431.

McFalls, J. A. and M. H. McFalls. 1984. *Disease and Fertility*. New York: Academic Press.

McGrath, J. W. 1991. Biological impact of social disruption resulting from epidemic disease. *American Journal of Physical Anthropology* 84:407–419.

McKeown, T. 1976. *The Modern Rise of Population*. London: Edward Arnold.

———. 1988. *The Origins of Human Disease*. Oxford: Basil Blackwell.

McNeill, W. M. 1976. *Plagues and Peoples*. Oxford: Oxford University Press.

———. 1979. Historical patterns of migration. *Current Anthropology* 20(1): 95–98.

Marcy, P. T. 1981. Factors affecting the fecundity and fertility of historical populations. *Journal of Family History* 6:309–326.

Mascie-Taylor, C. G. N. 1993. The biological anthropology of disease. Pp. 1–25 in C. G. N. Mascie-Taylor (ed.), *The Anthropology of Disease*. Oxford: Oxford University Press.

Milner, G. R. 1980. Epidemic disease in the postcontact Southeast. *Mid-Continental Journal of Archaeology* 5(1): 39–56.

———. 1992. Disease and sociopolitical systems in late prehistoric Illinois. Pp. 103–116 in J. W. Verano and D. H. Ubelaker (eds.), *Disease and Demography in the Americas*. Washington, D.C.: Smithsonian Institution Press.

Myers, T. P. 1976. Defended territories and no-man's-lands. *American Anthropologist* 78:354–355.

Neel, J. V. 1977. Health and disease in unacculturated Amerindian populations. Pp. 155–177 in K. Elliott and J. Whelan (eds.), *Health and Disease in Tribal Societies*. CIBA Foundation Symposium 49. Amsterdam: Elsevier.

Neel, J. V., W. R. Centerwall, N. A. Chagnon, and H. L. Casey. 1970. Notes on the effects of measles and measles vaccine in a virgin-soil population of South American Indians. *American Journal of Epidemiology* 91:418–429.

Newson, L. A. 1985. Indian population patterns in colonial Spanish America. *Latin American Research Review* 20(3): 41–74.

———. 1992. Old World epidemics in early colonial Ecuador. Pp. 84–112 in N. D. Cook and W. G. Lovell (eds.), *Secret Judgments of God: Old World Disease in Colonial Spanish America*. Norman: University of Oklahoma Press.

———. 1993. Highland-lowland contrasts in the impact of Old World diseases in early colonial Ecuador. *Social Science and Medicine* 36(9): 1187–1195.

———. 1995. *Life and Death in Early Colonial Ecuador*. Norman: University of Oklahoma Press.

Olsen, L. F. and Schaffer, W. M. 1990. Chaos versus noisy periodicity: Alternative hypotheses for childhood epidemics. *Science* 249:499–504.

Patterson, K. D. and G. W. Hartwig 1978. The disease factor: An overview. Pp. 3–24 in G. W. Hartwig and K. D. Patterson (eds.), *Disease in African History*. Durham, N.C.: Duke University Press.

Pimental, D. 1968. Population regulation and genetic feedback. *Science* 159:1432–1437.

Polunin, I. 1977. Some characteristics of tribal peoples. Pp. 5–20 in K. Elliott and J. Whelan (eds.), *Health and Disease in Tribal Societies*. CIBA Foundation Symposium 49. Amsterdam: Elsevier.

Ramenofsky, A. F. 1987. *Vectors of Death: The Archaeology of European Contact*. Albuquerque: University of New Mexico Press.

———. 1990. Loss of innocence: Explanations of differential persistence in the sixteenth-century southeast. Pp. 31–48 in D. H. Thomas (ed.), *Columbian Consequences*, vol. 2. Washington, D.C.: Smithsonian Institution Press.

Reid, A. 1987. Low population growth and its causes in pre-colonial Southeast Asia. Pp. 33–47 in N. G. Owen (ed.), *Death and Disease in Southeast Asia: Explorations in Social, Medical, and Demographic History*. Oxford: Oxford University Press.

Rotberg, R. I. and T. K. Rabb, eds. 1983. *Hunger and History*. Cambridge: Cambridge University Press.

Sattenspiel, L. 1990. Modeling the spread of infectious disease in human populations. *Yearbook of Physical Anthropology* 33:245–276.

Schaffer, W. M. 1987. Chaos in ecology and epidemiology. Pp. 233–248 in H. Degn, A. V. Holden, and L. F. Olsen (eds.), *Chaos in Biological Systems*. New York: Plenum.

Shea, D. E. 1992. A defense of small population estimates for the central Andes in 1520. Pp. 157–180 in W. M. Denevan (ed.), *The Native Population of the Americas in 1492*. 2d ed. Madison: University of Wisconsin Press.

Slack, P. 1981. The disappearance of plague: An alternative view. *Economic History Review* 34(3): 469–476.

Snow, D. R. and K. M. Lanphear. 1988. European contact and Indian depopulation in the Northeast: The timing of the first epidemics. *Ethnohistory* 35(1): 15–33.

Stanley, N. F. and R. A. Joske, eds. 1980. *Changing Disease Patterns and Human Behaviour*. London: Academic Press.

Stannard, D. E. 1989. *Before the Horror: The Population of Hawaii on the Eve of Western Contact*. Honolulu: University of Hawaii.

———. 1990. Disease and fertility: A new look at the demographic collapse of native populations in the wake of Western contact. *Journal of American Studies* 24(3): 325–350.

———. 1991. The consequences of contact: Toward an interdisciplinary theory of native responses to biological and cultural invasion. Pp. 519–539 in D. H. Thomas (ed.), *Columbian Consequences*, vol. 3. Washington, D.C.: Smithsonian Institution Press.

Svanborg-Eden, C. and B. R. Levi
populations: A critical reexan
Disease in Populations in Tran
Swedlund, A. C. and G. J. Armela
thropological and Epidemiolo
Thornton, R. 1986. History, structu
and Tolowa (Hush) Indians of
Thornton, R., T. Miller, and J. Wa
ing smallpox epidemics. *Ameri*
Thornton, R., J. Warren, and T. Mi
187–196 in J. W. and D. H. Ub
Washington, D.C.: Smithsonia
Upham, S. 1986. Smallpox and cl
gist 88:115–128.
Wagley, C. 1951. Cultural influenc
Wallace, A. F. C. 1956. Revitalizat
Walter, J. and R. Schofield. 1989.
ciety. Pp. 1–73 in J. Walter ar
Order in Early Modern Society
Whitmore, T. M. 1991. A simulat
basin of Mexico. *Annals of the*
Wiesenfeld, S. L. 1967. Sickle-cell
157:1134–1140.
Wirsing, R. L. 1985. The health of
rent Anthropology 26(3): 304–3
Worster, D. 1990. The ecology of
1–18.
Zeitlin, J. F. 1989. Ranchers and In
change and indigenous survival
view 69(1): 23–60.
Zubrow, E. 1990. The depopulation

Svanborg-Eden, C. and B. R. Levin. 1990. Infectious disease and natural selection in human populations: A critical reexamination. Pp. 31–46 in Swedlund and Armelagos (eds.), *Disease in Populations in Transition*.

Swedlund, A. C. and G. J. Armelagos, eds. 1990. *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. New York: Bergin and Garvey.

Thornton, R. 1986. History, structure, and survival: A comparison of the Yuki (Ukomno'm) and Tolowa (Hush) Indians of northern California. *Ethnology* 25:119–130.

Thornton, R., T. Miller, and J. Warren. 1991. American Indian population recovery following smallpox epidemics. *American Anthropologist* 93(3): 28–45.

Thornton, R., J. Warren, and T. Miller. 1992. Depopulation in the Southeast after 1492. Pp. 187–196 in J. W. and D. H. Ubelaker (eds.), *Disease and Demography in the Americas*. Washington, D.C.: Smithsonian Institution Press.

Upham, S. 1986. Smallpox and climate in the American southwest. *American Anthropologist* 88:115–128.

Wagley, C. 1951. Cultural influences on population. *Revista do Museu Paulista* 5:95–104.

Wallace, A. F. C. 1956. Revitalization movements. *American Anthropologist* 58:264–281.

Walter, J. and R. Schofield. 1989. Famine, disease, and crisis mortality in early modern society. Pp. 1–73 in J. Walter and R. Schofield (eds.), *Famine, Disease, and the Social Order in Early Modern Society*. Cambridge: Cambridge University Press.

Whitmore, T. M. 1991. A simulation of the sixteenth-century population collapse in the basin of Mexico. *Annals of the Association of American Geographers* 81(3): 464–487.

Wiesenfeld, S. L. 1967. Sickle-cell trait in human biological and cultural evolution. *Science* 157:1134–1140.

Wirsing, R. L. 1985. The health of traditional societies and the effects of acculturation. *Current Anthropology* 26(3): 304–322.

Worster, D. 1990. The ecology of order and chaos. *Environmental History Review* 14(1–2): 1–18.

Zeitlin, J. F. 1989. Ranchers and Indians on the southern isthmus of Tehuantepec: Economic change and indigenous survival in colonial Mexico. *Hispanic American Historical Review* 69(1): 23–60.

Zubrow, E. 1990. The depopulation of native America. *Antiquity* 64:754–765.