Fernando Casanueva

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Unraveling the Web of Disease

Noble David Cook
and W. George Lovell

The Snuffles (*Florentine Codex*)

There was then no sickness; they had then no aching bones; they had then no high fever; they had then no smallpox; they had then no burning chest; they had then no abdominal pains; they had then no consumption; they had then no headache. At that time the course of humanity was orderly. The foreigners made it otherwise when they arrived here. They brought shameful things when they came.

11 Ahau was when the mighty men arrived from the East. They were the ones who first brought disease here to our land, the land of we who are Maya, in the year 1513.

Like a servant of God who bends his back over virgin soil, they recorded the charge of misery in the presence of our Lord God: the introduction of Christianity occurs; blood-vomit, pestilence, drought, a year of locusts, smallpox are the charge of misery, also the importunity of the devil.

The Book of Chilam Balam of Chumayel

The essays collected in this volume focus on the impact of epidemic disease on native peoples throughout colonial Spanish America. Our spatial range, from central Mexico to southern Chile, is vast, and the time span

covered by our inquiries stretches from the era of conquest to the eve of independence. Whether our attention is drawn to the beginning of the sixteenth century or the end of the eighteenth, from Aztec core to Mapuche periphery it is incontestable that Old World infections markedly shaped New World destinies. Epidemics struck early and lingered late. In some instances disease outbreaks even preceded direct physical contact between natives and newcomers. The effects of disease transfer, furthermore, continued throughout the nineteenth and well into the twentieth century. There evolved in colonial Spanish America what might best be considered a web of disease. This web, and its underlying dynamics, are what we attempt to unravel in this concluding essay.

We tend, in the present century, to view pronounced population growth as commonplace, an almost natural state of affairs. In rapidly industrializing western societies, demographic expansion began in the late eighteenth century and generally continued until control of births slowed the process. Growth continues at a dramatic rate in the nations of the so-called Third World, where modern public health measures have sharply lowered the death rate without providing for a corresponding reduction in the number of births. For several generations we have experienced almost continuous population expansion, leading many to assume that the demographic characteristics of the world today represent the normal pattern (Hollingsworth 1969; Wrigley 1969; Sánchez-Albornoz 1974).

In preindustrial times, population growth tended to be slow and relatively continuous, until the approximate carrying capacity of the social and economic environment was reached, or until an alien element was introduced. At this point, conditions were propitious for a sudden catastrophe, or "die-off," of a sizeable portion of people. There were often sudden "spurts" of growth when changed circumstances prevailed, as occurred

when plants and animals were domesticated (Sauer 1952) or when a new and valuable food resource was exploited (Crosby 1972). As populations neared a peak, however, the natural checks of war, famine, and pestilence, or some combination of all three, acted to limit further expansion. Social variables such as age at marriage, nursing patterns, spacing of children, the practice of infanticide or (in extreme cases) human sacrifice and cannibalism could depress demographic growth. These societal factors tended, in the short term, to be less significant as checks than the Malthusian trinity of conflict, hunger, and disease (Boserup 1988; Zubrow 1975; Hollingsworth 1969).

There are abundant examples of the process, especially from Europe, where the record of history is more complete. A plague beset the people of Athens during the Peloponnesian Wars. It led to the city's downfall and the ultimate victory of the Spartans and their allies. A series of epidemics, coming from the East, penetrated the Roman Empire beginning about A.D. 180, and another series struck in the middle of the third century. Mortality was highest in the urban complexes (Grmek 1989; McNeill 1976; Hopkins 1983). Towards the end of the fifth century the population of many cities was only a fraction of what it had been before the onset of sickness. Founded on the principles of orderly town life, Roman civilization failed to survive in its original form. The Black Death, also coming from the East, struck Europe in the mid-fourteenth century. City populations were again cut by half or more by outbreaks of the plague. The economic and demographic upswing of the previous century was reversed. European confidence was shattered as thousands of people, rich and poor alike, faced the specter of sudden death. In many parts of Europe, it took a century and a half for the population to recover from the devastation. These epidemics, and their social and economic consequences, have attracted close scholarly attention (Gottfried 1983; Ziegler 1976).

It is in the New World, however, and not in the Old, that disease outbreaks probably caused the greatest loss of life known to history. In all likelihood, the severest and most protracted human mortality ever to occur did so in the years following overseas expansion by Europeans in the late fifteenth century. Technological improvements in shipbuilding, advances in navigation and mapmaking, and increasing sophistication of the machinery of war, as well as fundamental changes in the way economic, political, and social life were organized, made possible long-range sea voyages and facilitated the conquest and colonization of lands new to Europeans. Non-European peoples who before had dwelled in virtual isolation became part of a wide and widening world. As Crosby (1986) has shown, European expansion brought about, across the globe, an unprecedented exchange of people, plants, animals, weeds, pests, and pathogens. From the standpoint of the native peoples of the Americas, who had been shielded from Old World diseases for a long period of time, contact with Europeans was catastrophic (Borah 1976). Within a century, perhaps 90 percent of the Indians of the New World had succumbed because of the ravages of disease, famine and warfare (Borah 1964; MacLeod 1973: 1–20).

The Agents of Death

The diseases that appear to have been most destructive of Native American lives are smallpox and measles. We will review the salient pathogenic features of these illnesses as they relate to recorded outbreaks in colonial Spanish America. Afterwards we will treat, in similar summary fashion, influenza, plague, typhus, yellow fever, malaria, leishmaniasis, syphilis, and tuberculosis. By so doing, various strands of the web of disease will be isolated and illuminated. We point out in advance, however, that the web is a complex gestalt, one in

which the whole was decidedly more ruinous than the sum of its lethal parts.

Smallpox

As several contributors have observed, accurate diagnosis of disease symptoms reported by nonspecialists for nonmedical purposes is fraught with difficulty. Only during the past one hundred years has the germ theory of infection supplanted the medical belief systems of the ancient and medieval worlds (Cumston 1987). Compounding the problem of accurate diagnosis from scant, sketchy, or contradictory evidence is the fact that when a new disease agent infects a virgin population, abnormal symptoms often occur (Crosby 1976a). Explicit identification of smallpox provides a clear example of the difficulties we encounter when working with early colonial testimony.

Smallpox occurs in two principal forms: *variola major*, usually with mortality rates of 30 to 50 percent, and *variola minor*, with much lower mortality levels. Complicating the business of identification is the existence of five different types of *variola major*, all with varying levels of mortality: episodes of benign semiconfluent smallpox result in 10 percent mortality, benign confluent in 20 percent, malignant semiconfluent in 25 percent, malignant confluent in 70 percent, and fulminating smallpox in almost 100 mortality (Dixon 1962). Even with the advantages of twentieth-century health care, before the elimination of smallpox in the 1970s, people who contracted malignant or fulminating strains could not be significantly assisted. Mortality of pregnant women was especially high, some 50 percent and more.

The clinical symptoms of smallpox as we know it are well described in standard references: fever, malaise, and then a generalized eruption usually on the third day, which progresses from papules to vesicles and, finally, to pustules (Dixon 1962). In the past, those who

survived an attack were often marked by pitted skin (Hopkins 1983:1–21; Anderson and Arnstein 1956:301–11). The problem is that other diseases could have been confused with smallpox as it passed through progressive stages.

Smallpox is transmitted among human beings by individuals who are ill with the infection or convalescing from it. The virus is passed through secretions in the throat and the nose, and from the lesions themselves. It is communicable until lesions are completely healed and the scabs covering them have fallen off. Direct contact with the material containing the virus was necessary for the spread of smallpox. The virus enters the human body through the respiratory tract and has an incubation period of eight to ten days (Dixon 1962:68, 88; Joralemon 1982:120; Anderson and Arnstein 1956: 301–4).

Given the high communicability of smallpox, we might well ask why the disease did not, as far as is known, reach the Americas before 1518. One answer is probably the high percentage of immune Europeans who crossed the Atlantic during the first voyages. Smallpox in sixteenth- and seventeenth-century Europe primarily affected children, so most adults who undertook the journey had developed an immunity by being exposed to the disease during childhood. The virus could remain active for several weeks in the scab, but intense heat and solar radiation, common conditions during the Atlantic crossing, usually destroyed it. The early crossings were generally slow and subject to delay, the trips taking several weeks. Finally, however, the smallpox virus made the fateful passage. It may have done so via scabs hidden away in bundles of clothing or textiles, which would provide the virus with ample protection. Alternately, a ship may have sailed across with enough infected and nonimmune passengers to keep the virus alive through human transmission. The latter situation certainly prevailed during the peak of the slave trade,

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when large cargoes of young, susceptible blacks taken on board ships on the African coast provided ideal carrying conditions for the transfer of smallpox to port cities in the New World. The disease struck Santo Domingo in December 1518 or January of the following year, the time and place most contemporary scholars (Crosby 1972:35–40, 40–47; Dobyns 1983:11–16; McNeill 1976:183) assign to the origins of smallpox in the New World. Smallpox, however, may have made an earlier American landfall, for around the time of the expedition led by Francisco Hernández de Córdoba to the Yucatán, in 1517, a cruel and unfamiliar sickness had already devastated the peninsula. Writing in the 1560s, the Franciscan missionary Diego de Landa (1941:42) recorded the testimony of Maya survivors in his *Relación de las cosas de Yucatán*, which speaks of "great pustules, which rotted bodies with a great stench, so that the limbs fell in pieces in four or five days." Although no mention is made of pock marks, Inga Clendinnen (1987: 19) thinks the sickness "was almost certainly smallpox." She reaches this conclusion in sound scholarly company, for Ralph Roys (1967:138) in his translation of the *Book of Chilam Balam of Chumayel* observes that "an epidemic of smallpox swept through Yucatán in Katun 2 Ahau, and it may have been brought by the party of Spaniards who were shipwrecked and cast on the east coast in 1511."

What we do know, as Prem and others have observed, is that smallpox was said to have been introduced into Mexico by a black servant of Panfilo de Narváez who had been stricken by the disease. From the Gulf Coast, sickness then spread west and south. Many scholars, Lovell among them, note that a well-known passage in the *Annals of the Cakchiquels* tells of terrible sickness in Guatemala between 1519 and 1521. Diagnosis is problematical, but designation as smallpox has its supporters, including medical doctors. Newson, as have several researchers before her, establishes the pres-

ence of smallpox in the Andes in the 1520s, where it took a heavy native toll. According to Borah, the first outbreaks of smallpox in the Andean highlands, one of which killed the Inca ruler Huayna Capac, may have originated not as diffusions from Mexico and Central America but as overland transfers from the Río de la Plata region, far to the south. The first fully documented epidemic of smallpox to reach New Granada was recorded in 1558. It came from Hispaniola, and mortality associated with it was high. Smallpox flared up again in New Granada in 1588. This may have been the origin of part of the devastating sickness that swept Peru at this time. Evans, in Chapter 5, examines the impact of smallpox on the community of Aymaya, in present-day Bolivia, in 1590. Of 194 deaths that critical year, 147 were caused by smallpox. About a quarter of these deaths were among children under the age of five. This presents us with an unusual piece of evidence, for smallpox mortality in Europe in the sixteenth century would have evinced a far different pattern, with a much larger percentage of deaths among children than occurred in the Andes. Smallpox reappeared in Aymaya between 1609 and 1610. Thereafter, even in distant parts of the empire, smallpox occurred at fairly regular intervals throughout the colonial period, as Casanueva demonstrates in Chapter 7. By the early nineteenth century, however, scientific initiatives such as the royal expedition led by Francisco Xavier de Balmis had positive and beneficial effects (S. F. Cook 1941; M. M. Smith 1974; Lovell 1988), although smallpox enjoyed a post-Jenner existence in some areas that reflects poorly on republican-era governments.

Measles

Measles is an acute, short-term viral infection. Its classic symptoms include the onset of fever, the appearance of a spotted rash, and the development of a cough. The disease is highly communicable but has a relatively low

level of mortality for people who have been exposed to it for generations. Measles is most serious for children under three years of age, fetuses, and secondarily for adults. Complications involve infections of the middle ear, pneumonia, and sometimes encephalitis. The last two can cause death. Measles is transmitted by direct contact with infected droplets via the respiratory passage. Incubation varies from eight to twenty-one days, the characteristic rash appearing around the third day of illness. Temporary immunity lasting about six months is passed from a mother who has previously contracted measles to her infant. The greater the population density, the more rapid the spread of disease. In large urban clusters, epidemics usually take place at intervals of two or three years. In rural districts, measles epidemics occur infrequently, only after infection from the outside (Anderson and Arnstein 1956:287–94).

A study of disease outbreaks during the late seventeenth and eighteenth centuries in Santa Ana de Triana, a parish of Seville, indicates that measles was virtually endemic in the port city, from which most official trade with the Indies was conducted (N. D. Cook et al. 1988). Almost all those who contracted the disease, as in the case of smallpox, were children. Given its relatively short incubation period and its brief span of being acutely infectious, it would be necessary to have a group of children on board a fleet to transfer measles across the Atlantic. This situation might not have prevailed early on, but by the 1530s surely did. In Chapter 1, Prem suggests that measles hit sixteenth-century Mexico in cycles of approximately thirty years, with a presumed first appearance in 1531. Lovell, in Chapter 2, discusses a measles pandemic in Central America between 1532 and 1534, one that occurred throughout Guatemala as well as in Honduras and Nicaragua. Both contributors, however, caution about the problems of accurate diagnosis. In Chapter 3, Newson argues that the measles present in Nicaragua in 1533

also reached Panama. From there, the human traffic ferried to Peru as part of the Nicaraguan slave trade (Radell 1976) could afterwards have transmitted measles to the Andes, certainly by the mid or late 1530s. A combination of measles and smallpox, according to Newson, reached Peru in 1558. By far the most devastating outbreaks of sickness, however, swept the Andes from 1585 to 1591, when several diseases, measles among them, were present. The Villamaríns, in Chapter 4, indicate that measles took a heavy toll in the Sabana de Bogotá in 1617–18. In Chapter 6, Alchon reports great devastation in Ecuador in 1785. Measles also hit the Sabana de Bogotá in 1788 and is recorded in Peru a year later. These eighteenth-century outbreaks appear to have struck Indian peoples with the same severity as earlier episodes.

Measles, then, proved a deadly sickness for Native Americans who came in contact with it. It is difficult for us to imagine what destruction this common childhood disease of the Old World produced in the "virgin soil" context of the Americas. Mortality must have been very high indeed. Dobyns (1983:270, 284–85) estimates 50-percent mortality during a hypothesized epidemic of 1531–33 in Florida, a 25-percent rate in 1596, and a 16-percent figure for 1727 or 1728. Measles, he contends, may have caused the death of more Native Americans than any other disease except smallpox.

Influenza

Another communicable airborne disease is influenza, a condition characterized by fever, a general feeling of malaise, and prostration. We now know the agent is a virus, divided into two major strains, type A and type B, both of which are comprised of several substrains. Susceptibility is high. The virus is transmitted via droplets or secretions from the infected host. Influenza becomes deadly usually as a result of complications, the most common of which is the invasion of pneumono-

cocci and streptococci organisms in the weakened respiratory tracts. Incubation appears to be two or three days for most strains. Although children are most susceptible, adults, especially the ill or elderly, suffer from a higher death rate (McGrew 1985:150).

Influenza occurs in waves, normally during the winter months. There are sharp variations in severity from one epidemic wave to the next. In fact, the influenza virus is extremely unstable and mutates with great frequency. Temporary immunity following one attack does not provide immunity against exposure to other strains. Accurate diagnosis of cases on the basis of colonial documentation is almost impossible because it is easy to confuse outbreaks of influenza with those of other viral infections (Crosby 1976b; Dobyns 1983:19). Guerra (1985, 1986) contends that the first major epidemic to reach America was influenza, or swine flu, which swept Hispaniola in 1493. Prem (Chapter 1) and Lovell (Chapter 2) mention that literature for Mexico and Guatemala correlates some early epidemics with influenza (McBryde 1940). Fevers, nosebleeds, severe coughs, and stiffness of the neck are some of the vaguely described symptoms. The last major pandemic occurred in 1918, when global mortality exceeded that of the recently ended Great War. During this outbreak young adult males were hit with exceptional virulence (Crosby 1976b).

Infections Spread by Arthropods

Several diseases affecting Native Americans were spread by arthropods, especially lice and fleas, and also by dipterans. These diseases warrant close examination as a group because of their complex etiology. Bubonic plague, typhus, yellow fever, malaria, and leishmaniasis are examples. The pool of infection is often in other mammals, and the epidemics usually appear in *Homo sapiens* as the result of an accidental explosion of the disease (epizootic) in the normal host population.

Environmental factors play a major part in the dis-

semination of these infections. International migrations of humans and rodents on ships contributed to the diffusion of these diseases during European expansion. If a suitable vector were not available in new territory, then diffusion was cut short, as was the case with leishmaniasis. Climatic variations obviously play a role in the passage of arthropod-borne disease. Yellow fever, which periodically entered the northern part of the United States, retreated with the onslaught of winter. On the other hand, in Europe during the Middle Ages epidemics of typhus most often took place in winter months because people tended to concentrate in buildings, where the body louse found an ideal setting. Infrequent bathing and the rare washing of woolen garments provided near perfect conditions for the propagation of lice and, in turn, the rapid transmission of typhus (Zinnser 1935; Anderson and Arnstein 1956:433–35).

Plague. Plague (*Pasteurella pestis*) was one of the major killers in medieval Europe. Various pandemics swept the continent, including the most famous of all, the Black Death, which caused heavy mortality in the mid-fourteenth century. Plague is spread from host rodent populations through the bite of the rat flea (*Xenopsylla cheopis*), or directly from person to person. There are two forms of the plague, bubonic and pneumonic, or pulmonary. The bubonic form, spread after infection by the flea, is characterized by swollen lymph nodes, often with fatal septicemia. Pneumonic or pulmonary plague is the more deadly, and mortality levels before the advent of antibiotics reached 100 percent. This highly communicable form is spread from person to person via sputum infected with *Pasteurella pestis*. Incubation requires from two to three days. Body temperature falls, there is a severe cough, then a bloody discharge. Coma and death follow.

Reexamining the Black Death in Europe, Gottfried (1983) reports sharp variations in mortality levels from

one region to another. Total mortality from the plague that began in 1348, in an epidemic series that lasted in some locales up to eighteen months, was high. The range for Florence is from 45 to 75 percent, for Genoa 30 to 40 percent, for Milan only about 15 percent, but for Venice as high as 60 percent. Gottfried (1983:8, 42–76) calculates a mortality of from 33 to 50 percent for the whole of Italy, and for the entire Mediterranean basin some 35 to 40 percent. He estimates 35 to 50 percent for London, but only 20 to 25 percent for eastern Europe. Dobyns (1983:18–20, 30–31) argues that bubonic plague reached the New World in the 1540s, in a pandemic that included all of Mesoamerica, the Andean region of South America, and possibly Florida. Drawing on the evidence of Cieza de León, Newson suggests (Chapter 3) that pneumonic or pulmonary plague probably reached Ecuador in 1546 and spread from there throughout the Andes. This may have been an extension of the sickness noted by Prem and Lovell for, respectively, Mexico and Guatemala. Other plague epidemics broke out in 1576–80, 1612–19, and 1707. Mortality during these episodes, especially the one of 1545 to 1548, was high. Further research on the impact of plague in the New World is needed, however, before definitive conclusions can be reached (Dobyns 1983: 18–20, 30–31).

Typhus. Typhus is an acute infection manifest, like measles, by the onset of fever and the appearance of a rash. Three main forms are known: (1) epidemic or classical typhus, transmitted by the body louse (*Pediculus humanus*) and resulting in heavy mortality; (2) endemic or murine typhus, spread by the flea and producing lower rates of mortality; and (3) tsutsugamushi disease, carried by a mite and common in parts of Asia. The epidemic form is the most dangerous, usually occurring when people are densely concentrated without adequate sanitation. Periods of war and famine have pro-

vided ideal conditions for the spread of typhus. Incidence of typhus tends to be highest during the colder months of the year.

The typhus agent is *Rickettsia prowazekii*, a microorganism living in cells lining the gut of the body louse. The agent is expelled in the feces, and the louse survives the infestation for about twelve to eighteen days. The microorganism can live for several days in the dried feces. *Rickettsia* usually enters human beings through cuts or abrasions in the skin, such as scratched insect bites. Incubation in human beings lasts anywhere from ten to fourteen days. The early symptoms include headache, loss of appetite, fever, and general malaise. Body temperature peaks at the end of the first week, remains elevated until about the twelfth day, then drops to normal among survivors between the fourteenth and sixteenth days. A rash appears on the fourth to sixth day and shows up as red or dark-red spots some two to five millimeters in diameter. Some spots rise slightly above the skin. In severe cases the rash might cover most of the body. Fatal episodes produce marked prostration followed by delirium and end in coma and cardiac arrest. Those who survive face a long and slow convalescence. Mortality can vary from approximately 5 to 25 percent. The level is much higher for the elderly, about 50 percent. Children, on the other hand, face mortality levels of less than 5 percent (Anderson and Arnstein 1956:449–51).

Typhus is a disease the Spaniards apparently knew well. One of the clearest European accounts of the illness comes from the period of conflict in which the kingdom of Granada was enmeshed in 1489–90. Spaniards called the disease *tabardillo*, in reference to its characteristic symptoms: spots covering the body like a tabard, or sleeveless cloak. It struck the Iberian peninsula repeatedly during the sixteenth century and could have accompanied the troops led by Hernán Cortés during the conquest of Mexico (Dobyns 1983:21, 31; McNeill

1976:194–95). Prem considers typhus a possible explanation of the dreadful epidemic that hit Mexico in 1545 and may have carried off more than 60 percent of those taken ill. Lovell connects this sickness with one that swept Guatemala that same year. Typhus seems to have reappeared in Mexico and Guatemala between 1576 and 1581, in combination with other serious ailments. Certainly the demographic consequences of sickness lasted well into the 1580s. Another wave of typhus beset Guatemala in 1607–8 and again in 1631–32. The 1630s outbreak may have been pan-American in scale, for the Villamarins report it from 1630 to 1633 in the Sabana de Bogotá, where possibly one-fifth or more of the population succumbed.

Yellow fever. Spread by the *Aedes aegypti* mosquito in normal epidemic form, yellow fever is a fatal viral infection mostly found in the tropics but which, during the months of high summer, can move into temperate zones. In the nineteenth century, for instance, a bout of yellow fever extended into the United States as far north as Boston and the upper Mississippi basin. The etiology of the disease is complex. An infected human being passes the virus to the mosquito. After ten to fourteen days' incubation in the mosquito, the virus can be passed until the death of the insect. In tropical areas of Africa and South America, it appears that yellow fever is endemic in certain primate populations and may be transmitted by other mosquito vectors. Symptoms include a sudden illness, fever, a slowed pulse, and finally jaundice—hence the name "yellow fever." Vomiting of blood of a dark, almost black hue also occurs. Tolerance among blacks suggests that yellow fever was endemic along the African coast for some time (Anderson and Arnstein 1956:468–74).

It is difficult to date precisely when yellow fever first entered the New World. One serious outbreak, acknowledged by Dobyns (1983:279–80) as the first, be-

gan in Barbados in 1647, reached the Yucatán in 1648, and is recorded for Guadeloupe, Cuba, and Saint Kitts in 1648–49. Another severe outbreak swept the northeast coast of Brazil from 1686 to 1694. Yellow fever reached Boston in 1693, having been brought there by the British fleet returning from Barbados. It hit Charleston and Philadelphia in 1699, with high mortality (Marks and Beatty 1976:149–50). Yellow fever epidemics were common in the eighteenth century. An epidemic lashed New York City in 1702, when 570 people died in a population of fewer than 8,000. Other epidemics hit the city in 1743, 1745, and 1748. Newson notes yellow fever as occurring, probably for the first time, in 1740 in Guayaquil, where it reappeared three years later.

Charleston was ravaged by yellow fever in 1706, perhaps also in 1711, then again in 1728 and 1732. There was a serious outbreak in 1745, a mild outbreak in 1758, then a major series in 1790, 1791, 1792, 1795, 1798, and 1799. Philadelphia was hit in 1741 and 1747. In 1762 the disease entered the city again, having been carried from Veracruz to Cuba in 1761, there infecting British troops who, after attacking Havana, sailed to Philadelphia. Another severe epidemic hit Philadelphia in 1793. Between 1794 and 1805, yellow fever contaminated the port cities of Charleston, Norfolk, Baltimore, New York, and Boston. Smaller cities were also affected. New Orleans suffered in 1796 and throughout much of the nineteenth century (Duffy 1968:100–112). Baltimore, Philadelphia, and Boston were struck by a devastating series in 1819. Mortality was often high. Of a population of about 50,000 in the 1790s, New York City lost 732 in the 1795 outbreak and more than 2,000 in 1798. Epidemics of yellow fever abound in the nineteenth century. A classic example of the effect of yellow fever is seen in Haiti. There Napoleonic troops attempted, in 1802, to suppress an independence movement led by Pierre Toussaint L'Ouverture. Over 40,000 Europeans died from the disease, thus destroying Napoleon's plans for poli-

tical aggrandizement in the New World (Marks and Beatty 1976:150–60).

Malaria. The etiology of malaria is even more complex than that of yellow fever. Malaria is caused by three strains of plasmodium: (1) *Plasmodium vivax*, called "tertian malaria" because chills come every third day; (2) *Plasmodium malariae*, or quartan type, characterized by chills at four-day intervals; and (3) *Plasmodium falciparum*, which is the cause of most deaths from malaria and is associated with irregularly spaced, nearly daily occurrences of chills. Malaria is transmitted by several species of the anopheles mosquito. In *Homo sapiens*, the plasmodium invades red blood cells, becoming a mature trophozoite. At this point, it ruptures both itself and the membranes of the red blood cells, becoming a large number of merozoites. Chills are associated with the rupture of the cell membranes. The merozoites then enter new red blood cells, becoming trophozoites, having multiplied themselves asexually. Some of the trophozoites in the human body become sexually differentiated gametocytes. If the gametocytes enter the mosquito, they can reproduce sexually. The gametocytes, drawn into the mosquito's stomach, finally penetrate the insect's body cavity, forming an oocyst. After ten to fourteen days, these break into sporozoites, invade the mosquito's salivary glands, and then may be injected into another person. The mosquito is infectious for its entire life. Infection may be carried in hibernating mosquitos from one season to the next. Malaria persists in afflicted individuals for an indefinite period, during which time there may be spells of chills and anemia. Populations in regions where malaria is endemic can be greatly weakened and debilitated (Anderson and Arnstein 1956:468–74).

Leishmaniasis. Also called *uta*, *espundia*, or *jukuya*, leishmaniasis is brought on by the action of the protozoan *Leishmania braziliensis*, found in the foothills of the An-

des. The infection is passed to *Homo sapiens* or a mammal that can act as host (dogs and rodents) by the bite of an infected sand fly of the *Lutzomyia* genus. According to Gade (1979: 271), leishmaniasis "was one of the few ecopathogenic diseases found in South America before the arrival of the Europeans." It is endemic to the American tropics and subtropics from 22 degrees north to 30 degrees south. The impact of leishmaniasis, in contrast to diseases that sicken and cause distress almost immediately, is long term. In that sense it resembles tuberculosis. Gade (1979:269) describes succinctly the normal course of the disease:

> The syndrome begins with a primary skin lesion where the bite occurs, usually on the arm or leg, which later heals. One to six months later, pathological organisms may appear in the nasal mucosa, but more typically, a long period—sometimes many years—of dormancy passes before the characteristic facial lesions develop. The nose, palate and upper lip may become ulcerated; the underlying cartilage may also be destroyed. If the larynx becomes infected, the vocal chords may fail, the necrosis of the trachea can limit food intake. Ultimately the disease may result in death through gangrene, bronchopneumonia or starvation.

Fortunately, the sand-fly vector responsible for leishmaniasis has a restricted altitudinal range on the slopes of the Andes. Dense populations lived above that range, well in excess of 2,500 meters, and were consequently less endangered.

Syphilis and Tuberculosis. Like leishmaniasis, syphilis and tuberculosis are not ordinarily acute, communicable diseases, but they can shorten life and debilitate their victims. It is possible, however, for a person to carry any one of these three diseases for a long period of time and still survive. In his introduction, Borah touched briefly on the possible New World origins of syphilis, transmitted by *Treponema pallidium* during sexual intercourse. Syphilis appears to be communicable for up to five years

from spirochetes present in open lesions often not visually detectable. A primary lesion may appear within three to four weeks, but sometimes as short as eight days or as long as eight weeks after exposure. The secondary stage may consist of malaise, fever, a generalized rash, and a sore throat. This stage can go undetected easily, and a period of latency follows. This latent condition might last as long as the life of the host. The final stage consists of a concentrated attack usually on one of the major systems of the victim: the central nervous, skeletal, or cardiovascular system. At this point the disease becomes fatal. Congenital infections are often fatal before the birth of an infant (Anderson and Arnstein 1956:376–80; Dobyns 1983:34–35).

Especially acute during the nineteenth century, tuberculosis is another disease that erodes physical welfare slowly. Caused by the tubercle bacillus *Mycobacterium tuberculosis*, the illness seems to be particularly influenced by population density, general economic and nutritional levels, prevalence of milk-borne infection, and ethnic background. Mortality rates vary significantly. Death from tuberculosis in the United States fell from 153.8 per 100,000 in 1910 to 26.3 in 1949. The disease can attack most parts of the human body. The response is to replace areas of tissue destroyed by the disease with fibrous tissue, or calcification. Anderson and Arnstein (1956:354) state that "the outcome of any case depends on the tissue involved and whether or not the fibrous repair processes develop more rapidly than the necrotic action." Tuberculosis is manifest in three different strains: human, bovine, and avian. The bovine strain tends to concentrate in the bones and joints, whereas the human strain is primarily pulmonary. Bacillus is expelled in lung infections in the sputum, where it can exist for up to several weeks, protected by mucin covering. Human beings with active progression of the disease are potential carriers. The bacilli are sensitive to heat, hence the success of pasteurization of milk with

regard to eliminating the bovine strain as a major threat (Anderson and Arnstein 1956:354–71). S. F. Cook (1946: 324) and Dobyns (1983:34–36) suggest that in pre-Columbian times Native Americans enjoyed an existence relatively free of infectious diseases, suffering mostly from respiratory disorders such as tuberculosis and a number of gastrointestinal disturbances.

The Key Variables

Epidemiologists have long been interested in the study of the causal chain of disease, as best it can be discerned from past outbreaks of sickness. For them, a major purpose of such research is to establish the basis for predicting the course of future epidemics. For scholars interested in past epidemics and their impact on human societies, the reverse is true: a better understanding of history is attained by present knowledge of disease characteristics.

Mass outbreaks of sickness are predicted by analyzing various factors, including the nature of the causative agent, the exposure of the individual to that organism, the disposition of the host (immunity, resistance, susceptibility), and the environment that influences the process (Sinnecker 1976:23). Susceptibility of the human host is a key epidemiological factor. Individual susceptibility determines the spread of a disease, but it is the susceptibility of the population as a whole that differentiates a series of ill people from a mass outbreak. Population density and communication patterns are also important variables. Resistance to a disease may be genetic or acquired. A mother passes to her infant temporary immunity or resistance to certain diseases if she has herself experienced them. It is also possible that slightly longer protection is afforded by the breast-feeding of infants.

Invasion of an infant's system by an infectious agent provokes a response to contain and ultimately destroy the foreign organism. This response varies in different

individuals and depends again on a number of factors, especially nutrition. In some illnesses, if the dose of the infectious agent is small, the infant will be able to suppress the agent. Increasing doses leads to stronger internal defenses against the infection. This may go on until the mass infection is too great for the body response to handle, or if the internal defenses are weak, as might occur when a person is suffering from exhaustion or is malnourished. At that point, the disease temporarily wins the battle, and clinical manifestations become evident. In the case of most viral diseases, the size of the dosage or inoculum does not appear to be a significant factor, and the disease tends to run its course with no subclinical infections (Anderson and Arnstein 1956:13–41).

Genetic factors also play a role in the individual's response to infection. Precise measurement of the role of genetic variation, however, is difficult to obtain. Blood factors influence the ability of some African groups to survive in malarial areas, but that same blood factor has had one detrimental side-effect: the tendency to acquire sickle-cell anemia (Sinneker 1976:63–64). The response of Native Americans to what are often considered European childhood diseases is a subject the contributors of this volume have addressed. Perhaps the sharpest mortality differences between Europeans and Amerindians lie in the experience of measles and smallpox, where the exposure of Europeans, subjected to the ravages of both diseases over many generations and centuries, led to a mortality level that allowed the continuation of both the virus and its host.

Epidemiologists have studied the relationship between host and parasite with regard to the introduction of an alien species into a new environment (Sinnecker 1976:55–58). The individual's response to the onslaught of an infection also depends on the virulence of that particular outbreak, for the severity of a disease organism for a host population can and does vary over time.

Here the mechanism is probably natural selection, for a disease that destroys an entire host population ultimately destroys itself. There is thus a premium for the selection of less fatal forms of the disease agent, as Zinsser (1935) recognized. The classic example of this process was the introduction of the myxomatosis virus into Australia in an attempt to control the explosive growth of the wild rabbit population. In the first epizootic, rabbit mortality reached 97 to 99 percent. In the second epizootic, it registered 85 to 95 percent. By the third epizootic, mortality had fallen to 40 to 60 percent. Natural selection might favor rabbits resistant to the myxomatosis virus, but given the duration of rabbit generations, this advantage must have been minimal. It is more likely that natural selection of less virulent forms of the virus played a role in the lower levels of mortality in the later epizootics.

Human generations, of course, are chronologically much longer than rabbit generations. The factor influencing an advantage for less virulent infections has probably always been at play in human epidemics. Problems arise if there is a human population that can acquire and transmit a disease with a low mortality experience, living side by side with a population that lacks this resistance. In such cases, there may be virtually no premium for natural selection of weaker forms of the infection, and so the new or "virgin" population may be completely destroyed (N. D. Cook 1981:72–73, 268; Sinnecker 1976:59–61).

Other biological factors enter into the disease equation. Mortality levels are related to both age and sex in certain illnesses. Old people and infants commonly suffer highest mortality during certain epidemic outbreaks. Heightened mortality during influenza epidemics, for example, is evident among elderly folk, who often succumb to ensuing pneumonia infections. On the other hand, direct deaths from influenza in the 1918 pandemic were, in the isolated Maori, highest among young,

adult, working males, a mortality pattern that holds also for the same pandemic in the United States (Crosby 1976b). For women, mortality levels due to infectious diseases tend to be highest during pregnancy. Some infections, including smallpox, measles, and syphilis, affect the fetus as well as the mother (N. D. Cook 1981: 66–67; Sinnecker 1976:135–47).

The latent and infectious periods are clear-cut factors regulating the speed of progression of an epidemic. Once the disease agent has entered the human body, there is a period of development until the body's defenses are overwhelmed and clinical symptoms appear. As we noted in our survey of disease agents, the incubation period varies from one illness to another, and there are fluctuations also in the latency period within the disease itself. The longer the chronological time involved in the latency period, the greater the possibility that the disease can be spread without detection from one locale to another. The extent of transmission is dependent on the speed of communications. Also related is the period of infectiousness. Once again, the longer the period of infectiousness, the more likely it is that disease will spread over vast regions because infected persons carry it to a point where sickness can be transmitted to another individual. With arthropod-spread epidemics, the feeding cycle of the vector is also a factor (Sinnecker 1976:180–82).

The latency and infectious periods, essential biological factors involved in epidemic propagation, relate directly to social factors, particularly communication networks and population densities. The latter is in fact both a biological and cultural variable. It can be controlled by societal decisions: concepts of ideal family size, spacing of infants, age at marriage, a decision of allowable population concentration, colonization efforts to limit high densities, celibacy, and even cannibalism, infanticide, and human sacrifice. How quickly disease is transmitted is directly related to population density:

the greater the number of inhabitants in a confined, crowded space then the greater the degree of contact between individuals, resulting in the rapid spread of infection from one person to another. Some epidemiologists maintain that major epidemics as we know them would not have existed in hunting, fishing, and gathering societies that had little outside contact and low population densities (McNeill 1976; Boserup 1988).

When dealing with communication networks, technological developments are crucial. With modern air travel, virtually all major world regions are less than twelve hours flying distance one from another. This condition allows an acute communicable epidemic in one region almost immediately to reach each other one. The rapid spread of influenza epidemics to almost all sections of the globe is a consequence of this transportation revolution. Although the speed of ships in the era of Columbus was slow, velocity was fast enough to have significant demographic consequences. Indeed, as Parry (1963) so vividly demonstrates, technological innovations in shipbuilding, navigation, and armaments made possible the creation of a global network dominated by Europeans in the Age of Reconnaissance. From the late fifteenth to the end of the nineteenth century, passage across land and water accelerated rapidly as a result of further improvements in transportation (Crosby 1972: 35–63; McNeill 1976:176–207). Native Americans, as we have seen, paid a high price during this period of European domination.

Hygiene and sanitation are other factors that influence the spread of infections, especially the transmission of water-borne epidemics such as cholera and typhoid. Contaminated sewage leads to the rapid dissemination of the infectious agent and the outbreak of an epidemic. Recognition of the relationship between polluted water supplies and disease led public officials in many nations during the late nineteenth century to provide purified drinking water. The consequence was

a rapid fall in the incidence of water-related epidemics. During the colonial period, however, such was not the case, and so water-borne infections, although not much discussed, must have taken a toll in human lives.

Sanitation goes beyond merely providing purified water. Diseases spread by contact with droplets suspended in the air are also amenable to control by hygienic measures. Societal customs with regard to sneezing and disposal of nasal secretions can have a marked effect on the spread of epidemic infections. Attempts to deflect a sneeze into a handkerchief, for example, can reduce drastically the number of disease agents that can enter the air and thus be transmitted to other individuals. Likewise, careful washing with a disinfectant can help to reduce the number of disease agents that are transferred by direct contact.

Towards the end of the nineteenth century, before scientific recognition of the process by which infectious diseases are transferred, most sanitary measures were a result of social custom and existed in large part by chance. Attempts by sixteenth-century Europeans to restrict bathing in tropical and subtropical regions reduced the cleanliness of aboriginal residents and directly contributed to spreading disease and death (Anderson and Arnstein 1956:43–58). In some cases, however, native healing practices were detrimental to the sick during times of illness. Indians in Mesoamerica, for example, as well as in North America, used sweat treatments followed by quick cold baths or swims in frigid waters to cure fevers. Such treatments only served to increase the mortality associated with measles and smallpox, for the weakened cardiovascular system was just not able to stand the shock (Dobyns 1983:16).

Societal practices for the care of the ill can also influence the recovery rate. Sickness in the New World before the arrival of Europeans was in general not life threatening for aboriginal inhabitants, a circumstance that Old World diseases changed markedly. Age-old

cures prescribed by knowledgeable shamans were simply no longer effective, breeding wariness and fear and provoking new forms of reaction. In some instances, after the lethal nature of Old World infections was recognized, Indians abandoned sick persons to die or to fend for themselves, as friends and relatives fled their homes to avoid contagion. We even have evidence from late in the colonial period of full-scale abandonment of settlements, where the living did not bury the dead, but instead left them behind to rot (Lovell 1988). In such a situation, all semblance of normality disappeared. Crops were neither sown nor harvested, animals roamed untended, chores and routines were forgotten. Communal life disintegrated. When, months later, hungry survivors returned to their villages, Bosch-like scenes of horror awaited them.

Seasonal factors are yet another consideration, for fluctuations in temperature and humidity affect both the conditions diseases operate in and how human beings decide to live. Measles, for example, occurs usually in the late autumn and winter, appearing perhaps because of increased indoor crowding and a higher incidence then of respiratory infections brought on by the cold and damp. Smallpox in Europe tended to peak during the drier summer months, when people and goods moved around more. Cholera seems also to occur most often in the summertime. In disease transmitted by arthropods, seasonal variation is clearly marked. Yellow fever and malaria are restricted in temperate zones to the warmer months, when mosquitoes are most active. Plague in Eurasia had two peaks, one in January and another in summer. The summer epidemics are related to the greater activity of the flea and rodent vectors as well as people working outdoors. The January peak represents the maximum concentration of humans and rodent pests indoors. The common cold is most prevalent in winter. Even minor cooling of the body can alter resistance to infection. Lowest resistance also seems to be

associated with changes in human physiological activities, which in Europe occur in the months of August and February (Sinnecker 1976:203–15).

Warfare and pilgrimages have a major impact on the spread of disease. Armies mobilize comparatively large numbers of men. Living at close quarters, often malnourished, ill-clad, and seldom if ever clean, warriors have frequently suffered higher disease mortality preparing to fight than in actual combat. Soldiers conducting sieges have been especially subject to crowd diseases, foremost of all typhus and plague, which can then infect civilians. Similarly, famous pilgrimages, such as the ones to Mecca, Jerusalem, and Santiago de Compostela, have been associated with sickness and contagion (Omar 1957; Sinnecker 1976:203).

The role of the environment is also crucial. A number of variables clearly influence the spread of disease, among them altitude, temperature, and the presence or absence of water. Seasonal variations in the distribution of malaria are based on the activity of the mosquito vector. Malaria would not be expected, even in areas near the equator, where the elevation is high enough for water to freeze during certain times of year. Humidity, too, is an important consideration. Floods, of course, influence how people and commodities circulate. Some diseases slow down during months of heavy rainfall, when transportation lines are disrupted. At the same time, other diseases flourish in times of flood because of water contamination. The complexity of relationships between the environment and the passage of sickness makes it difficult to ascertain accurately how all of these variables interact (Sinnecker 1976:206–210).

Caught in the Web of Disease

A communicable disease is simply what the term suggests. Diseases are "communicated," or transferred, from one person to another along established routes of

transportation. Even diseases carried by arthropod vectors must run parallel to lines of communication. A network, a web of disease is soon established, but it depends at all times on human traffic. The spread of the first smallpox pandemic helps us visualize the process. If we leave aside the possibility of smallpox in the Yucatán prior to 1518, a pattern of pandemic spread is reasonably well documented, even allowing for chronological imprecision or spatial gaps: from Caribbean islands (Hispaniola and Cuba) late in 1518 or early in 1519, Mexico in 1520, throughout Guatemala that same year and the next, then showing up some five years later in Peru. A mainland course south from Mexico as far as Central America is evident. Far less so, but likely, is diffusion north from Mexico, perhaps even across large areas of North America, as Dobyns (1983:11–16; 1989) has asserted. Such claims, whether smallpox reached Florida overland from movement along the Gulf Coast or from native canoes or Spanish ships approaching the peninsula from Cuba, demand a meticulous appraisal of the information at hand, as Henige (1985-86; 1986; 1989) pointedly cautions and as Borah, in this volume, more charitably reiterates.

That the first diseases introduced from the Old World to the New found ideal conditions for the rapid transmission of sickness across vast distances is indisputable. Sizeable populations existed that were immunologically defenseless against the quick work of unknown pathogens. Diseases passed back and forth as long as the chain of vulnerability was unbroken. After a century or so, during which time depopulation in many regions of the order of 90 percent or more had occurred, pandemic activity abated, probably because both the size and density of Indian populations had been reduced to a level at which the possiblity of the spread of new diseases was curtailed. Epidemics that originated as "visiting people" (Greek *epidemos*) became endemic, ones that stayed among or "in people" (Greek *endemos*). The

dynamics of the web of disease adapted to a new reality, one in which patterns of sickness changed both in impact and manifestation, with more and more small-scale incidents and fewer and fewer large-scale outbreaks. Certainly by the eighteenth century, as Lovell (1988) indicates in the context of Guatemala, it was possible for disease to break out in some communities without necessarily spreading to neighboring ones only a short distance away. Serious epidemics, however, did still occur during late colonial times, as the chapters by Alchon and Casanueva clearly attest.

In retrospect, an aura of inevitability surrounds the demographic collapse suffered centuries ago by Native American populations, decidedly the most tragic feature of the colonial experience in Spanish America. Given the limited state of knowledge, then, about what epidemic disease was, how it was transmitted, and what possible measures could restrict its spread, once Europeans reached the New World the fate of native peoples was effectively sealed. Several eyewitnesses drew a direct correlation between outbreaks of sickness and Indian depopulation, but most Spaniards did not understand the reasons behind aboriginal demise, even if they soon became aware of what it would mean for their chances of material enrichment or their desires for religious converts (Phelan 1970:92–96). Non-Spaniards who witnessed or were informed about goings on, the English in particular, attributed the loss to demoniacal acts of cruelty on the part of Spanish conquerors and colonists, a view that was enhanced by the disturbing accounts of Fray Bartolomé de Las Casas. The controversial Dominican, however, conveniently overlooked the role disease played in shaping the colonial experience, even among the native groups he knew and grew to love best. Regardless of how we choose to rank the key elements of survival, the fact remains that when Spaniards (even those moved by the most enlightened of intentions) set out to conquer Indians, unforeseen

things happened. The year 1492, for all Native Americans, came to represent disaster. A Maya plaint tolls a collective epitaph: "There was then no sickness. The foreigners made it otherwise when they arrived here. They brought shameful things when they came."