

Epidemics and Society

From the Black Death
to the Present

F R A N K M . S N O W D E N

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CHAPTER 3

Overview of the Three Plague Pandemics

541 to ca. 1950

Bubonic plague is the inescapable reference point in any discussion of infectious diseases and their impact on society. In many respects plague represented the worst imaginable catastrophe, thereby setting the standard by which other epidemics would be judged. In later centuries, when societies experienced new and unfamiliar outbreaks of disease, they waited anxiously to see whether they would equal plague in their devastation. Particularly feared diseases, such as cholera in the nineteenth century and both the Spanish influenza and AIDS in the twentieth, were said by some to be “the return of the plague.” Similarly, tuberculosis, the leading killer of the nineteenth century, was widely referred to as “the white plague.” Indeed, the term “plague” has become a metonym for societal calamities in general, even when the crisis is not infectious, as in such phrases as “a plague of accidents” or a “plague of bank robberies.”

What features of bubonic plague, and of the responses of communities to it, marked it as so distinctive and so fearful? The most striking aspect of plague is its extraordinary virulence. “Virulence” is the capacity of a disease to cause harm and pathological symptoms. It is the measure of the ability of a pathogen to overcome the defenses of the human body to induce sickness, suffering, and death. Plague in that sense is extremely virulent. It strikes rapidly, causes excruciating and degrading symptoms, and, if untreated, invariably achieves a high case fatality rate (CFR), which simply

means the kill rate of a pathogen—the ratio of deaths to illnesses. In the era before the discovery of antibiotics, plague normally killed more than half of the people it infected, for a CFR of at least 50 percent—a rate attained by few other diseases. Furthermore, its progress through the body was terrifyingly swift. As a rule, plague killed within days of the onset of symptoms, and sometimes more quickly.

Other fearful characteristics of this disease were the age and class profiles of its victims. Familiar endemic diseases primarily strike children and the elderly. This is the normal experience of a community with infectious diseases such as mumps, measles, smallpox, and polio. But the plague was different: it preferentially targeted men and women in the prime of life. This aspect made plague seem like an unnatural or supernatural event. It also magnified the economic, demographic, and social dislocations that it unleashed. In other words, plague left in its wake vast numbers of orphans, widows, and destitute families. Furthermore, unlike most epidemic diseases, the plague did not show a predilection for the poor. It attacked universally, again conveying a sense that its arrival marked the final day of reckoning—the day of divine wrath and judgment.

Another distinguishing feature of plague is the terror it generated. Communities afflicted with plague responded with mass hysteria, violence, and religious revivals as people sought to assuage an angry god. They also looked anxiously within their midst to find the guilty parties responsible for so terrible a disaster. For people who regarded the disease as divine retribution, those responsible were sinners. Plague thus repeatedly gave rise to scapegoating and witch-hunting. Alternatively, for those inclined to the demonic interpretation of disease, those responsible were the agents of a homicidal human conspiracy. Frequently, vigilantes hunted down foreigners and Jews and sought out witches and poisoners.

This chapter presents an introductory overview of plague as a disease, including the public health policies deployed against it, the general impact of plague, and the three major plague pandemics that span some fifteen hundred years of history (fig. 3.1).

Plague and Public Health

Plague was also significant because it gave rise to a critically important societal response: the development of public health. Bubonic plague inspired the first and most draconian form of public health policy designed to protect populations and contain the spread of a terrible disease, that is, various

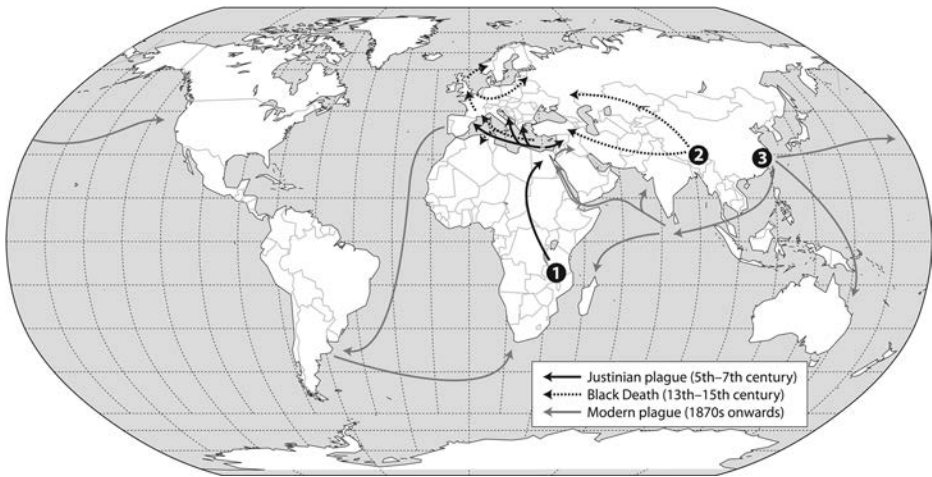


Figure 3.1. The three pandemics of bubonic plague. (Drawing by Bill Nelson.)

forms of enforced isolation of the sick (we omit Hansen's disease and the relegation of its victims to leper colonies on the grounds that leprosaria were noncurative institutions and did not contribute to the development of public health strategies). Anti plague measures of public health involved reliance on the military for their implementation. They mandated, first of all, sanitary cordons—military lines intended to isolate a population by preventing all movements of people and goods. In addition, defenses against the plague included pesthouses, known as lazarettos, and quarantine; health authorities variously known as health magistrates or boards of health were equipped with emergency powers to enforce the regulations. In some places stocks and scaffolds were erected to remind the population of the powers of these agencies.

Renaissance Italian city-states played a special role as the pioneers of these anti plague responses. This responsibility was thrust upon them by their vulnerable positions at the center of trade routes in the Mediterranean, where they received passengers and goods—and stowaway rats—from the Middle East and North Africa. Florence and the port cities of Venice, Genoa, and Naples were pioneers in the development of these policies, which were widely imitated.

In later centuries, another pattern emerged. When new and deadly diseases struck, such as cholera, yellow fever, and AIDS, one of the first responses of health authorities was to attempt to reactivate anti plague measures. It is said of military generals that they tend to fight the last war

over again, thereby confronting new enemies with inappropriate strategies from the past. Much the same can be said of public health authorities over the centuries. This temptation is all the greater because the instruments of antiplague defense give the impression of a forceful and energetic response, thereby providing the population with some sense of protection.

The Impact of Plague

A leading feature of the plague as a disease is its enormous social impact. Certainly bubonic plague makes a strong case for not regarding infectious diseases as a narrow and specialized interest. Plague was part of the “big picture,” as essential for understanding history during the periods in which it afflicted society as the study of war, religion, economics, and high culture. The point is not, of course, to make a case for disease determinism, or what one might call “microbial Marxism.” The argument is more simply that certain diseases do have a transformative effect on society, and plague is one of them. Most other diseases, even massive killers such as influenza or polio, have not had a similar influence. A major task of our exploration of plague is to examine why such major differences exist in the realm of infectious diseases—why some leave a major cultural, political, and social imprint, and others do not.

Bubonic plague is one of the best examples of a disease that affects every aspect of society. It transformed the demography of early modern Europe. Its recurring cycles, with an epidemic every generation, constituted a major brake on population growth between the fourteenth and eighteenth centuries. It had devastating effects as well on economic life and development. And it substantially influenced religion and popular culture, giving rise to a new piety, to cults of plague saints, and to passion plays. Bubonic plague also deeply affected the relationship of people to their mortality, and indeed to God.

The plague in Europe led to an outpouring of sermons and religious pamphlets in which a central theme was theodicy—that is, the vindication of an omnipotent God’s goodness in the face of evil and suffering. It was relatively easy to accept that God could be angry and would punish those who turned away from him and disobeyed his commandments. But how could one explain the gruesome suffering and mass death of innocents, especially children? It is true that plague led to an upsurge of piety, but it also generated a powerful undertow pulling in the opposite direction. For some, the experience of bubonic plague led to the terrifying conclusion that there might

be no God. A loving and all-powerful being would not take the lives of half the population of a great city, indiscriminately slaying men, women, and children. The result was not so much atheism as a mute despair that was most often barely articulated—a psychological shock that, with historical hindsight and anachronism, one might call posttraumatic stress.

Plague also had major effects on the arts and culture. In literature, an entire genre of plague literature arose, including works by Giovanni Boccaccio, Daniel Defoe, Alessandro Manzoni, and Albert Camus. It also transformed the iconography of European painting and sculpture, and it deeply affected architecture, with the construction of major cathedrals and churches dedicated to the redeemer, the Virgin Mary, and the plague saints Sebastian and Roch. Plague columns, often built to celebrate the ending of plague in a city, appeared in Vienna and throughout central Europe to remind the population of God's mercy.

As late as the mid-twentieth century, the disease inspired Ingmar Bergman's 1957 film *The Seventh Seal*. At the height of the Cold War, Bergman was deeply concerned about the possibility of nuclear war. As a way of imagining the apocalypse, bubonic plague presented itself naturally as the ultimate experience of human calamity and therefore as a metaphor for atomic catastrophe.

Similarly inspired by the experience of plague in the seventeenth century, the city of Oberammergau in Bavaria initiated a German tradition of passion plays. The survivors of an epidemic there in 1630 took a vow: if the people were spared, the city council promised to perform a passion play involving the whole population of the town and to continue to do so at regular intervals in perpetuity. This vow gave rise to an ongoing, and controversial, series of plays that enacted the passion of Christ and sometimes incited viewers to anti-Semitic violence.

Plague also had a major intellectual impact on the medical paradigm of disease by profoundly testing the humoral explanatory framework. The doctrines of Hippocrates and Galen had difficulty in satisfactorily explaining the passage of bubonic plague. How was it possible that vast numbers of people experienced the same humoral imbalance at almost precisely the same time? Hippocrates and his followers invoked the possibility of what might be called, in present-day parlance, environmental insults. The orthodox explanation was that the atmosphere in a given locality had been "corrupted," creating an "epidemic constitution." Its cause was a deadly fermentation arising from decaying organic matter either in the soil or in nearby marshes and swamps. This poisonous effusion contaminated the air

and sickened large numbers of susceptible people when they inhaled the poison or absorbed it through their pores.

A medieval variant of this view was propounded by astrologers, who suggested that the trigger to plague and other epidemic diseases was a dangerous alignment of the stars and planets. Disorder in the cosmos was then reflected in disorder in the microcosm of the body. Even those who did not regard the appearance of a comet or the conjunction of planets as the direct causes of an epidemic often believed that such celestial events could serve as portents. Similarly, unusual climatic events—earthquakes, floods, and fires—could presage a crisis in public health.

Girolamo Fracastoro, a sixteenth-century Italian physician, confronted the problem of explaining epidemics in a fundamentally different manner. He eliminated the mediation of the humors altogether, suggesting instead that epidemic disease was caused by a poisonous chemical transmitted—in ways that he did not understand—from one person to another. In the seventeenth century the German Jesuit Athanasius Kircher developed the idea further, suggesting that the plague was spread by what he called “animalcules” that somehow passed from an infected person to a healthy one. Fracastoro and Kircher were thus pioneers in developing the concept of contagion.

The idea of contagion appealed at first far more to the popular imagination than it did to the mind of elite, university-trained physicians who could find the suggestion nowhere in the classic texts. Only in the late nineteenth century did microbiology validate the heretical etiology of Fracastoro and Kircher through the work of Louis Pasteur (1822–1895) and Robert Koch (1843–1910), as discussed in Chapter 12.

A History of Three Plague Pandemics

It is important to distinguish among three closely related terms. Infectious diseases are normally placed along a continuum according to their severity in terms of the numbers of sufferers and the extent of their geographical reach. An “outbreak” is a local spike in infection, but with a limited number of sufferers. An “epidemic,” by contrast, normally describes a contagious disease that affects a substantial area and a large number of victims. Finally, a “pandemic” is a transnational epidemic that affects entire continents and kills massive numbers of people. All three terms, however, are loose approximations, and the boundaries separating one from another are imprecise and sometimes subjective. Indeed, a contagious disease confined to a single

locality is occasionally termed a pandemic if it is sufficiently virulent to afflict nearly everyone in the area.

In accordance with this terminology, humanity has experienced three pandemics of bubonic plague. Each consisted of a cycle of recurring epidemic waves or visitations, and the cycle lasted for generations or even centuries. So recurrent were these outbreaks of plague that they provided writers with a logical and convincing device to drive the plot of a story forward. A notable example is Shakespeare's tragedy *Romeo and Juliet*, which unfolds against the background of an outbreak of plague in the Italian city of Verona. The tale reaches its tragic denouement as a result of the disease, which interrupts communication between Verona and Mantua. When Friar John attempts to deliver Juliet's crucial letter to Romeo, who is exiled in Mantua, he is forcibly detained: "The searchers of the town, suspecting that we . . . were in a house where the infectious pestilence did reign, sealed up the doors and would not let us forth. So that my speed to Mantua there was stayed. . . . So fearful were they of infection" (Act 5, scene 2, lines 8–12, 17). Bubonic plague thus furnished an entirely plausible artifice for structuring the tale of the star-crossed lovers and their double suicide. As Shakespeare's audience well knew, plague in early modern Europe was an ever-present danger that could strike at any time and without warning.

The cyclical pattern of plague was marked also by a pronounced seasonality. Plague epidemics usually began in the spring or summer months and faded away with the coming of colder weather. Especially favorable were unusually warm springs followed by wet, hot summers. The modern explanations for these propensities are the need of fleas, which carried the disease, for warmth and humidity to enable their eggs to mature, and the inactivity of fleas in cold and dry conditions. Although this pattern predominated, the disease has also been known to erupt mysteriously in Moscow, Iceland, and Scandinavia in the depth of winter. These atypical eruptions of the disease posed serious epidemiological puzzles.

The First Pandemic, or the Plague of Justinian

The first appearance of bubonic plague in world history was the so-called Plague of Justinian, or Justinianic Plague, named after the Byzantine emperor Justinian I, under whose reign it first appeared. Some people held his alleged misdeeds, according to the historian Procopius, to be responsible for incurring divine wrath. The pandemic is thought by present-day geneticists, however, to have originated as a zoonosis, or epidemic disease that can be

transmitted from animals to humans, in an endemic African “focus” (or local area of infection). In 541 CE it first erupted as a human affliction at Pelusium in the Nile Delta. Thereafter it lasted through eighteen successive waves over a period of two hundred years until 755, when it disappeared as suddenly and mysteriously as it had arrived.

This round of pestilence afflicted Asia, Africa, and Europe, and it left a dreadful but unquantifiable mortality in its wake. Few direct accounts of this disaster have survived, but the extant reports of such eyewitnesses as Gregory of Tours, John of Ephesus, Bede, and Procopius agree on the magnitude of the calamity. In Procopius’s words, this was “a pestilence by which the whole human race was near to being annihilated.”¹ Recent impressionistic assessments suggest a total fatality of 20–50 million.

This extensive mortality and the descriptions of the classic symptoms of bubonic plague—the hard bubo in the armpit, groin, or neck—are clear diagnostic indicators of the identity of the disease. In recent years, moreover, paleopathologists have been at work exhuming bodies from the cemeteries of late antiquity, extracting DNA from their dental pulp, and confirming the presence of the bacterium *Yersinia pestis*, which causes the disease. Scientists working in Bavaria in 2005, for example, identified the plague bacillus in skeletal remains from a sixth-century cemetery at Aschheim, strongly suggesting that the traditional diagnosis of bubonic plague is accurate (fig. 3.2)

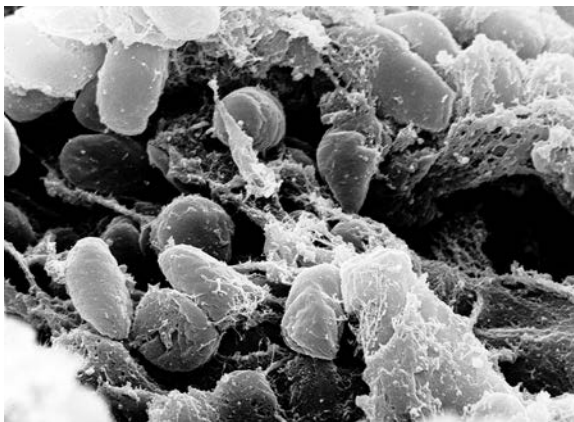


Figure 3.2. Scanning electron micrograph of a mass of *Yersinia pestis* bacteria, which cause bubonic plague, in the foregut of the flea vector. (Rocky Mountain Laboratories, NIAID, NIH.)

The Second Pandemic, or the Black Death

The second plague pandemic began in Central Asia in the 1330s, reached the West in 1347, and persisted for five hundred years until it disappeared in the 1830s. Its first wave in Europe, from 1347 until 1353, is often called the Black Death today, although this restricted terminology began only in the eighteenth century. Various fourteenth-century accounts instead refer to the disaster as the “great pestilence,” the “plague of Florence,” “the mortality,” and the “plague.” Partly for this reason, and partly because of the dark buboes and gangrene that are plague symptoms, many scholars still employ the original and more extensive meaning of Black Death—as a synonym for the whole of the second pandemic.

This pandemic is conventionally thought to have arrived aboard Genoese galleys that sailed from the Black Sea in the summer of 1347 and docked at Messina in Sicily. It then spread rapidly to the rest of the island, onward to Sardinia and Corsica, and then in a more leisurely manner to mainland Italy. There its work was assisted by ships bearing plague that docked at Genoa. Mainland Italy and the whole of mainland Europe were engulfed in the general epidemic carnage. That Italian cities were the first in Europe to be ravaged by plague was no coincidence: their early devastation reflected the geographical vulnerability of Italy’s position at the center of Mediterranean trade.

At the time the Black Death arrived, Europe was in the throes of a lengthy period of social and economic hardship that favored the advance of the disease. The thirteenth century had been a period of economic expansion, demographic growth that doubled the numbers of Europeans between 1100 and 1300, and urbanization. Substantial towns with populations in excess of fifteen thousand inhabitants multiplied, and within them crowding and unsanitary housing became significant urban problems. Then, after about 1270, an economic recession set in as output stagnated, producing lower wages and contributing to poverty. Agricultural production fell sharply, leading to a classically Malthusian crisis—that is, population growth exceeded output, leading to famine.

Unprecedentedly sustained bad weather then fatally undermined a system that was already beginning to stall. Driven by persistent torrential rain occurring at critical times over successive years, and worsened by unseasonably cool temperatures that shortened the growing season, the production crisis culminated in a series of catastrophic crop failures. Widespread flooding, windstorms, and brutal winters compounded the impact, with “seedbeds sodden, crops and pastures under water, grain rotting, fish traps

wrecked, dikes washed away, meadows too wet to be mown, turf too soggy to be cut, and quarries too swollen with the overflow to be worked for stone or lime.”² Contemporaries feared the need for another Noah and his ark.

Rivaling the Egyptian famine predicted by Joseph in the book of Genesis, but without the Pharaoh’s storage facilities or modern distribution networks, the late medieval “Great Famine” lasted—with ever-escalating impact—from 1315 to 1322. Causing the death of millions, it afflicted the whole continent north of the Alps, and it was followed by additional years of severe shortage and high prices between 1345 and 1348. Furthermore, in 1319 and 1320 a devastating disease thought to be rinderpest decimated cattle across northern Europe, seriously reducing access by most of the population to meat and milk and crippling production by destroying draft animals and their manure. This “Great Bovine Pestilence” therefore combined with recurring crop failures to undermine human nutrition, growth, and development.

The starkly unequal power relationships of late medieval society deepened the economic depression and enormously exacerbated poverty. Writing with specific reference to Sweden but in terms that applied to the whole of western Europe, paleoecologist Per Lagerås makes this point forcefully:

The impoverishment of the population was also due to the inequality of medieval society. Even under normal conditions, heavy burdens of taxes, rents, tithes, and labour duties left little surplus for ordinary people. The number-one priority for the upper classes and the central power was to keep up their luxury consumption and life style while little resources were invested back into the agricultural system. When they experienced declining incomes due to poor yields their immediate reaction was to compensate by further raising taxes and rents. This counterproductive reaction is partly to blame for the stagnating economy and the non-sustainable agriculture. For the above-mentioned reasons people were on the edge of starvation.³

The results seriously compromised resistance to disease among people who were born after 1315. They suffered malnourishment in their developmental years and had become immunocompromised adults by the time plague-carrying Genoese ships docked at Messina.

During its relentless progress from Sicily across the whole of Europe, the Black Death outstripped even the Great Famine in its impact. During its first wave, between 1347 and 1353, it is estimated to have killed as much as

half the population of the continent, creating what Lagerås calls “the worst disaster that has ever hit Europe.”²⁴ One of the most famous local tragedies was the epidemic that decimated Florence in 1348 and is vividly portrayed by Boccaccio’s *Decameron*. Other famous epidemics include Milan in 1630, which resulted in two major works of plague literature by Alessandro Manzoni—*The Column of Infamy* and *The Betrothed*; Naples in 1656; and the “Great Plague” of London during 1665–1666, which is the subject of Daniel Defoe’s influential account *A Journal of the Plague Year*.

Then, for reasons that are much debated and that we discuss in Chapter 4, bubonic plague receded from western Europe between the end of the seventeenth century and the middle of the eighteenth. The last epidemics to strike Scotland occurred in 1640, England in 1665–1666, the Netherlands in 1710, France in 1720–1722, and Italy in 1743. Interestingly, outbreaks at Messina form convenient bookends for the second pandemic: it was the first place in the West to suffer in 1347, and the last in 1743.

Although, understandably, the demographic disaster so graphically described by contemporary writers and historians at the outset of the second pandemic has captured the imagination, it is clarifying to recognize that the virulence of the plague did not decline over the centuries. Some of the final epidemics of the second pandemic were among the most devastating and dramatic, such as the great disasters in London (1665–1666) and Marseille (1720–1722). What had changed was that these final attacks were localized events that failed to achieve the continental reach of the first invasions.

The Third Pandemic, or Modern Plague

The third and final pandemic of bubonic plague originated, like the second, from a Central Asian focus, erupting in the wake of social unrest and warfare in China in 1855. It attracted global attention when it attacked Canton and then Hong Kong in 1894 and later moved on to cities that were nodal centers in international trade such as Buenos Aires, Honolulu, Sydney, Cape Town, Naples, Oporto, and San Francisco. Unlike its predecessors, which devastated nearly every place they touched, the third pandemic was radically uneven in its impact as it followed instead the international fault lines of inequality, poverty, and neglect.

Bubonic plague during the third pandemic overwhelmingly scourged Third World countries while largely sparing the industrial nations of Europe and North America. Above all, this third round of plague ravaged India, where it caused as many as 13 to 15 million deaths between 1898 and 1910.

Before finally receding, it ultimately killed approximately 20 million people, according to conservative estimates, and touched five continents, though largely sparing the industrial West. Furthermore, in India and China plague was not a universal affliction as it had been during the second pandemic. In India, it had a predilection for the infamous tenements (*chawls*) of Bombay (now Mumbai) and the hovels (*bastis*) of Calcutta while barely affecting Europeans or the wealthy.

Europe experienced brief flare-ups in 1899 at Naples, Oporto, and Glasgow but lost a total of seven thousand people to plague for the entire half-century after 1899. Central and South America suffered thirty thousand plague deaths during the third pandemic. The United States recorded a mortality of approximately five hundred victims in minor outbreaks at San Francisco, New Orleans, and Los Angeles.

In the Americas the third pandemic had a restricted impact on humans, but it did leave a major environmental imprint by establishing stable reservoirs of infection among sylvatic rodents in the Southwest of the United States, the northeast of Brazil, and the south of Argentina. In those locations it persists to the present amidst massive cyclical die-offs of rodents and an ongoing trickle of cases of bubonic plague among humans who venture into the wrong place or whose pets exchange fleas with ground squirrels and gerbils. The Centers for Disease Control and Prevention (CDC) reports that between 1900 and 2016, there were just over one thousand cases of plague in the United States, concentrated in New Mexico, Arizona, Colorado, and California, mostly among hunters and campers.

The reservoirs in the Americas thus formed additions to the preexisting plague reservoirs on every other continent. As a result, the World Health Organization (WHO) reports that 3,248 cases of plague and 584 deaths occurred between 2010 and 2015, spread over four continents, but with concentrations in the Democratic Republic of the Congo, Madagascar, and Peru. The official statistics are, however, almost certainly significant underestimates because of misdiagnosis, concealment by communities and governments, and the absence of laboratory facilities in many settings.

Most influentially, the third pandemic marked the moment when the complex etiology of the disease with its interaction between rodents, fleas, and humans was unraveled. As a result, beginning in the early twentieth century new public health policies based on this knowledge were implemented. Authorities targeted fleas and rats with insecticides, traps, and poison, thereby replacing the draconian antiplague measures that characterized the second pandemic and the early years of the third.

CHAPTER 4

Plague as a Disease

The Etiology of Plague

The “etiology” of a disease refers to its origins—the path it follows to afflict human beings. Plague has a complicated etiology normally involving four protagonists. The first is the pathogen itself—the oval bacterium originally named *Pasteurella pestis*, but now universally termed *Yersinia pestis*. It was discovered simultaneously in 1894 in Hong Kong by Alexandre Yersin, a Swiss student of Louis Pasteur, and by the Japanese physician Shibasaburo Kitasato, a protégé of Pasteur’s rival Robert Koch.

By 1898 Paul-Louis Simond discovered that, in addition to the bacterium, two vectors were normally responsible for conveying the disease to humans. These were commensal rodents, especially rats, and their parasitic cargo of fleas. Simond’s insights were largely ignored until they were confirmed a decade later, as we will see in Chapter 16, by the exhaustive investigation conducted by the Indian Plague Commission into the epidemiology of the disease. The findings were conclusive for the third pandemic on the subcontinent, but both Simond and the commission made the mistake of assuming that the mode of transmission that prevailed in the third pandemic was the same in all epidemics of the disease. Since this view became the orthodox understanding of plague, the result was a misunderstanding of the Black Death. So much of the epidemiology of the second pandemic was incomprehensible in terms of the rat-flea nexus that, as we will see, it even led to skepticism that the disaster that swept Europe for four centuries after 1347 was plague at all.

There is, however, a universal consensus that plague epidemics begin as “epizootics”—unnoticed epidemics in permanent animal reservoirs of the disease. Especially important are wild rodents—marmots, prairie dogs, chipmunks, and squirrels in their burrows, where underground disasters unknown to humans take place. Plague, therefore, is best understood as a disease of animals by which humans are afflicted by accident and by way of exception. Human involvement can begin if hunters invade the reservoir of infection, contracting the disease directly while skinning their prey and providing a portal of entry for the bacterium to their bloodstream through a cut or other lesion. Warfare, ecological disaster, and famine can multiply the numbers of displaced persons entering the rodents’ habitats. Alternatively, environmental changes, such as flood or drought, can send the animals scurrying over long distances closer to human settlements and above all bringing them into closer contact with the peridomestic rat. Pivotal in the second pandemic was the black rat or “ship rat,” *Rattus rattus*, which lived in close proximity to humans and with whom it shared the same dietary preferences.

The bacterial exchange from wild rodent to domestic rat, from rat to rat, and from rat to human is mediated by the final plague protagonist—the flea. Two species are deemed to play key roles. The first is the so-called Oriental rat flea, *Xenopsylla cheopis*, which is naturally parasitic on warm-blooded animals and is a highly efficient vector for bubonic plague, enabling it to cross the species barrier from rodent to *Homo sapiens*. The other is the widespread “human flea,” *Pulex irritans*, which does not colonize rodents but only people. It provides transmission from person to person.

In a single blood meal a flea of either species sucks up a quantity of blood equal to its own weight—an amount that contains millions of bacteria. Once engorged with blood and *Y. pestis*, the infected flea does not survive. The bacilli obstruct a valve regulating the movement of food into the insect’s stomach, so the flea slowly dies of starvation and dehydration. This foregut blockage is consequential not only in the life of the flea, but also in disease transmission to humans. By causing the flea to regurgitate ingested blood mixed with plague bacteria, the obstruction ensures that each bite is infective. It also causes the flea to feed ravenously and repeatedly in a frenzied bid to survive. Before dying, an infected *X. cheopis* is a lethally efficient vector.

Furthermore, when a rat carrying infected fleas in its fur sickens and dies, the fleas leap to the warm body of another mammal—whether rodent or human being. The fleas’ sensors—which are highly susceptible to warmth, vibration, and carbon dioxide—enable fleas to locate new hosts; their famous

jumping prowess enables them to make the transfer successfully; and their ability, when they are uninfected, to wait for six weeks between blood meals explains the erratic appearance of cases during the course of an epidemic.

Xenopsylla cheopis demonstrates a pronounced preference for rats, and it moves to humans only in the absence of surviving rodents. For this reason, the mass die-off of a rat colony leads to the sudden availability of swarms of famished fleas to parasitize humans in the absence of their preferred alternative. This behavior explains the explosiveness of plague epidemics. A graph of plague mortality and morbidity exhibits a steep spike at the outset rather than the bell-shaped curve characteristic of most epidemic diseases.

After the crossing of the species barrier between rats and humans, foci are established when infected men and women share their fleas and their disease with family members and neighbors. Plague then begins, not as a disease of isolated individuals, but of households, neighborhoods in cities, and entire villages in rural areas. Living conditions, and especially population density and hygiene, are decisive. Overcrowding, with numerous individuals pressed close together in a single room and perhaps whole families sharing a bed, greatly facilitates the exchange of fleas. As the plague spreads, particular moments are especially dangerous, such as the laying out of and final attentions to the dead. As a corpse cools, the fleas infesting it grow desperate to escape to the next warm body.

To give rise to an epidemic, the early foci in Africa and Central Asia needed links to wider networks of contact, trade, religious faith, and commerce. One such link involved the garments of the sufferers. An item of clothing in the early modern world was precious, so the clothes of the dead, as well as bed linens, were reused by others or packed in crates and sold at markets and fairs, often with fleas still alive among the folds. Certain professions also came into regular and direct contact with the sick, the dying, and the dead, as well as with their ectoparasites. Street vendors, physicians, priests, gravediggers, and washerwomen were at serious risk in time of plague, and they conveyed disease from place to place as they moved about their duties. Other key figures in the transmission of plague were millers and bakers, because grain attracted rats.

Monasteries also played a significant part in the first pandemic and the late medieval outbreaks of the second, explaining the ability of the disease to decimate the thinly settled countryside as well as urban centers. Monasteries acted as nodes in the grain trade, linking settlement to settlement and village to village; they were substantial communities of people living in close quarters; and during times of disaster monasteries often served as places of

refuge for people fleeing plague-ridden localities. On monastic grounds the healthy, the ill, and the bearers of infected fleas commingled. In such a context the flea readily established circuits of transmission for plague.

Fleas are severely restricted in their range, however, as opposed to rats, which are wonderful travelers. They hide in shipments of grain and are transported overland in carts or down waterways on barges and boats. But the rat also went much farther afield—by sea. Infected rats climbed aboard ships via ropes and gangplanks and were lifted aboard in crates of wheat and rice. In this way, shipping was essential to the spread of plague over long distances; this helps to explain the epidemiology of the disease—that is, its tendency to arrive in a country by ship and then to move inland by road and river traffic. For the black rat, the Mediterranean was not a barrier but a highway.

Istanbul (known as Constantinople between 330 and 1453) was a vital hub of trade and disease. It linked the whole of the Mediterranean—overland via the Balkans and by sea to Venice, Naples, Corfu, Genoa, Marseille, and Valencia. Sometimes there was havoc at sea when plague destroyed entire crews, and ghost ships drifted on the waves. More frequently, however, a ship would dock, and its cargo of rats would disembark via the same hoists, ropes, and gangplanks that had first brought them aboard. At the same time infected passengers and crew would go ashore, together with their fleas. Procopius already noted in the sixth century that the plague “always took its start from the coast, and from there invaded the interior.”¹

Not surprisingly, then, the first indication of plague was frequently a dramatic die-off of rats in the streets. This onset of pestilence is dramatically apparent in various works of plague art, such as Albert Camus’s novel *The Plague*. In this book the emergence of innumerable sick and dying rats in the streets of the Algerian city of Oran is the prologue to the epidemic disaster, which serves as a metaphor for evil as embodied in the rise of Nazism and fascism.

Similarly, the neoclassical painter Nicolas Poussin (1594–1665) included rats in his painting *The Plague at Ashdod* (1630) (fig. 4.1). The biblical book 1 Samuel relates how the Philistines triumphantly displayed the stolen ark of the covenant in their temple to the pagan god Dagan, thereby declaring the superiority of Dagan over the God of the Israelites. God punished the Philistines by scourging them with plague and destroying the city. To heighten the terror of the scene, Poussin prominently depicted rats on the streets of doomed Ashdod. The seventeenth-century painter understood that for the viewer, the mass appearance of rats aboveground was a familiar premonitory sign of plague and impending disaster.



Figure 4.1. In his painting of 1630 *The Plague at Ashdod* Nicolas Poussin included rats, which were known to be a premonitory sign of impending disaster. Musée du Louvre, Paris.

Twentieth-century scientific investigations have carefully confirmed the link between plague and rats. Archeologists have recovered the bones of rats at plague burial sites dating from the Black Death, and as already mentioned, the Indian Plague Commission extensively documented the rat-flea nexus and its role in the modern plague.

Until the turn of the twentieth century, however, the link between the rat and bubonic plague was not deemed to be causal. It was thought that the rat was infected before humans because of its low physical stature. With its nose close to the soil or the floor, the rat was more quickly susceptible to poisonous effluvia rising from the earth and to plague-infested dust underfoot. Suddenly, before people were stricken, rats began to appear in the streets and in the middle of rooms indoors. In a dazed condition and with uncertain balance, these rodents demonstrated no concern for their natural predators and enemies. Instead, in a frenzy of thirst, they desperately sought water until they lost their strength and collapsed. They died where they fell with telltale buboes on their necks, their limbs splayed, and rigor mortis upon them even before death.

Miasmatic theory provided an explanation, since this doctrine held that plague originated in the soil, rising slowly from it and progressively killing different animals as it reached the air they inhaled. Thus it was logical that rats, with their snouts to the ground, should fall victim first, and that humans, with their superior height, should be stricken only later. Such an etiology indicated that human plague followed the disease in rats but was not caused by it.

Symptomatology and Pathology

Attention to the effect of a disease on the individual human body is not a matter of ghoulish curiosity. Epidemic diseases are not simply transposable causes of suffering and death. On the contrary, the history of each high-impact infectious disease is distinct, and one of the major variables is the specific manner in which it affected its victims. Indeed, a feature of bubonic plague was that its symptoms seemed almost purposefully designed to maximize terror; they were excruciating, visible, dehumanizing, and overwhelming.

After being bitten by an infected flea, a person experiences an incubation period varying from one day to a week, and then the classic symptoms of the disease appear, launching the first stage of bubonic plague. At the site of the flea bite, a black blister, or carbuncle, arises, surrounded by red pock marks. Along with the carbuncle, the infected person experiences high fever, shivering, violent headache, nausea, vomiting, and tormenting thirst, after which the second stage of the disease begins. Unlike malaria-transmitting mosquitoes, for example, fleas do not inoculate bacteria directly into the bloodstream but instead deposit them into the skin. As few as ten bacilli are now thought to be sufficient to produce an infection. The reason is a stealth mechanism, or “virulence factor”—the production of an enzyme that enables the bacteria to elude the body’s defense mechanisms. Rapidly multiplying, *Y. pestis* invades the lymph system, drains into the regional lymph nodes nearest the infective bite, and causes the appearance of a bubo.

A bubo is an inflammation and swelling of a lymph node of the arm-pit, neck, or groin to form a hard mass sometimes as large as an orange beneath the skin. Its location varies according to the site of the infective flea bite, and it is not uncommon for more than one to erupt. The bubo is familiar as the classical symptom that appears almost infallibly and gives the disease its name as “bubonic” plague.

Buboes are a source of agony for patients. The sixteenth-century French surgeon Ambroise Paré explained that the bubo generates great heat and

causes a “pricking pain, as it were with needles, burning and intolerable.”² Defoe reported in his *Journal of the Plague Year* that the pain caused by the bubo was so violent that some victims in London dove into the Thames to escape it—an observation that Paré corroborated, writing from Paris that patients there hurled themselves naked from their windows. In the more measured parlance of the modern physician, the inflamed and suppurating bubo is described as “exquisitely tender.”³ There is also a consensus that the body and all of its excretions—pus, urine, sweat, the breath—have an overpowering stench, as if putrefaction precedes the individual’s demise. Surviving accounts by attendants in pesthouses during epidemics describe the intolerable smell of the bodies of the sufferers as the worst aspect of their employment. The historian Jane Stevens Crawshaw, for instance, thus summarizes Father Antero Maria’s account of his experience at Genoa, where he served in the plague hospital in 1575:

the sick within the *lazaretti* stank horribly—so much so that a single patient could render a room uninhabitable. He wrote that individuals in the *lazaretti* fled the company of others because of the smell and confessed himself to having hesitated many times before entering rooms—not, he says, for fear of infection but because the smell was so foul. This was made worse by the vomiting brought on by the illness. This, he said, was so disgusting that it turned the stomach. He recorded it as the most difficult aspect of the conditions in the *lazaretto*, too abominable to describe in words.⁴

Meanwhile *Y. pestis* bacilli—still “the most feared pathogen of the bacterial world”—continue to proliferate exponentially, doubling their numbers every two hours.⁵ In evolutionary terms, such expansion is positively selected because a bacteremia level of 10 to 100 million bacilli per cubic milliliter of blood is needed to ensure that a flea biting a human is infected. With the flea as a vector, extreme virulence was necessary to the transmission and survival of plague bacilli. An immediate consequence is that the ever-replicating bacteria rapidly overwhelm the body’s defenses by preferentially targeting and destroying the cells—dendritic cells, macrophages, and neutrophils—that provide the body’s immune response. In the words of a 2012 US Geological Survey report:

Y. pestis uses a needlelike appendage to target a host’s white blood cells [and] injects proteins . . . directly into the host white

blood cells. These proteins act to destroy immune functions of the host and prevent it from developing an inflammatory response that would inhibit or prevent further growth of the bacteria. . . . *Y. pestis* can also inject into the host a different protein . . . that prevents the host from producing two of its own proteins that would be used to stimulate the formation of a mass of immune cells to surround the bacteria and prevent its growth. . . . In the case of plague, the host cells get a false message that tissue damage is under control, when, in fact, *Y. pestis* bacteria are rapidly taking over visceral organs, particularly the liver and spleen, leading to loss of function.⁶

Escaping from the lymphatic system into the bloodstream, the multiplying bacteria initiate the third stage of the disease: septicemia. Having gained access to the blood, the bacteria release a powerful toxin that is normally the cause of death. It attacks tissues, causing blood vessels to hemorrhage, giving rise to purple, subcutaneous spots—the so-called tokens of plague. They earned this name because many people thought them to be signs, or “tokens,” of God’s anger.

By producing degeneration of the tissues of the heart, liver, spleen, kidneys, lungs, and central nervous system, the systemic infection initiates multiple organ failure. At this point patients have wild bloodshot eyes, black tongues, and pale, wasted faces with poor coordination of the facial muscles. They experience general prostration, teeth-shattering chills, respiratory distress, and a high fever that normally hovers in the range of 103° to 105°F, but in some patients reaches 108°F. In addition there is progressive neurological damage manifested by slurred speech, tremors in the limbs, a staggering gait, seizures, and psychic disturbances ending in delirium, coma, and death. Pregnant women, who are especially vulnerable, invariably miscarry and hemorrhage to death. Sometimes there is also gangrene of the extremities. This necrosis of the nose, fingers, and toes is one probable source of the terms “Black Death” and “Black Plague.”

During the first European epidemic of Black Death at Messina in 1347, the Franciscan chronicler Michael of Piazza wrote a vivid description of the sufferings of the victim:

The “burn blisters” appeared, and boils developed in different parts of the body: on the sexual organs, in others on the thighs, or on the arms, and in others on the neck. At first these were of

the size of a hazelnut and the patient was seized by violent shivering fits, which soon rendered him so weak that he could no longer stand upright, but was forced to lie on his bed, consumed by a violent fever and overcome by great tribulation. Soon the boils grew to the size of a walnut, then to that of a hen's egg or a goose's egg, and they were exceedingly painful, and irritated the body, causing it to vomit blood by vitiating the juices. The blood rose from the affected lungs to the throat, producing a putrefying and ultimately decomposing effect on the whole body. The sickness lasted three days, and on the fourth, at the latest, the patient succumbed.⁷

As soon as anyone was seized with headache and shivering during a visitation of the plague, he or she anticipated a fatal outcome. The minority of patients who recovered from their ordeal faced a lengthy convalescence and an array of lasting or permanent sequelae. These included deafness, impaired vision, paralysis of the muscles of one or more limbs, inability to speak as a result of laryngeal paralysis, and loss of memory. Psychological trauma also persisted after so arduous an ordeal. The experience did not even confer an acquired immunity, as a survivor from an epidemic in one year could die from plague the next. Against this background of sudden onset, a fulminant course, and a cascade of excruciating symptoms that usually ended in death, it is small wonder that the very word "plague" came to be synonymous with calamity and the worst imaginable disaster. In the Islamic world, it was even widely known as the "great annihilation."

Types of Plague

Bubonic Plague

Bubonic plague arises as an infection of the lymphatic system, it is transmitted primarily by flea bites, and it has the symptomatology described above. It is also the most common form of plague and has had the largest historical impact in all three of the plague pandemics. But the disease can also occur in two other forms, known as septicemic and pneumonic plague. It is vital to stress that these are not three distinct diseases, but simply three different manifestations of the single disease of plague, and all three are caused by *Y. pestis*.

Septicemic Plague

Primary septicemic plague is the fulminant and rarest of the three forms of plague. Like bubonic plague, it is transmitted by flea bite; but unlike bubonic plague, primary septicemic plague begins with inoculation of *Y. pestis* into the bloodstream, without prior inflammation of the lymph nodes and the formation of buboes. The bacteria immediately metastasize, with rapidly fatal consequences. In some cases, the progress of the disease is so fast that the patient dies within hours even before the onset of symptoms. More commonly, however, the sufferer endures organ failure, severe nausea, fever, and abdominal pain followed within hours by death through multiple causes. In septicemic plague, the CFR approaches 100 percent.

More common is secondary septicemic plague, which simply denotes a stage in the normal progression of bubonic plague untreated with antibiotics. In this stage the bacteria, having already caused the classic symptoms of the disease, escape the lymph system and enter the bloodstream. There they initiate the multiplication, diffusion, and toxicity that lead inexorably to death.

Pneumonic Plague

Pneumonic plague is a severe infection, not of the lymphatic system or the blood, but of the lungs, so historically it has sometimes been called “pestilential pneumonia.” The etiology may be the spread of the plague bacteria from the lymphatic to the respiratory system—a condition known as “secondary pneumonic plague.” Alternatively and more important historically, pneumonic plague can be transmitted directly from person to person by inhaling droplets from the coughs and sneezes of a plague victim whose respiratory system is the original site of infection, resulting in “primary pneumonic plague.”

Since the site of infection is the lungs, the symptomatology is significantly different from that of bubonic or septicemic plague. The portal of entry of *Y. pestis* via the lungs significantly affects its symptoms and, above all, its lethality and time course through the body. One reason is the different temperature of the flea gut as compared with that of the human body immediately before infection. In bubonic flea-to-human transmission, the temperature environment in which *Y. pestis* develops is that of the flea gut, 26°C, whereas in pneumonic human-to-human transmission the plague pathogen develops at 37°C. Recent research suggests that at the molecular

level, the development of the bacterium at the higher temperature activates genes that express virulence. These genes cause the production of antigens that destroy phagocytes (white cells) and chemical reactions that enable the pathogen to elude detection by the receptors of the large white blood cells known as macrophages. The result is “an immunosuppressive environment in the lungs” that leads to “rapid bacterial proliferation” in the lung alveoli—the tiny sacs where the vital exchange of oxygen and carbon dioxide in the bloodstream takes place.⁸

Thus attacked, the pneumonic plague sufferer experiences symptoms similar to those of acute pulmonary pneumonia. With destruction of the lung alveoli, edema, and hemorrhage, the patient manifests severe respiratory distress, fever, chest pain, cough, nausea, headache, and frothy, bloody sputum. Furthermore, the condition is universally fatal, often within less than seventy-two hours.

A significant historical corollary of the mode of transmission of primary pneumonic plague is that it does not depend on the rat and the flea. Here is the probable solution to the epidemiological puzzles that led to a current of “plague deniers,” such as Graham Twigg and Samuel K. Cohn. They postulate that the disease responsible for the second pandemic was not plague at all but anthrax or a combination of anthrax and an unspecified comorbidity. If plague was responsible, they ask, why did massive die-offs of rats not figure more frequently in plague literature, paintings, and chronicles of the Black Death? How could a disease dependent on the gradual movement of rats sweep so quickly across the European continent? How could bubonic plague burst out in Moscow or Iceland during frigid winters, when fleas are inactive? Why were crucial features of the epidemiology and virulence of the third pandemic so different from descriptions by contemporary observers of the second?

In this context, the example of Iceland appears especially confounding. With a slight delay due to its distance and isolation, Iceland suffered the first wave of the Black Death in 1402–1404, when perhaps 50 percent of the population perished. But there is a conundrum that adds significantly to the puzzlement already engendered by the onset of plague despite the island’s climate. A feature of Icelandic fauna in the late medieval period was that it lacked a population of rats. How could a disease transmitted by rats and Oriental rat fleas be widely and rapidly diffused in the absence of both?

Osteoarchaeology—the systematic excavation and scientific examination of human bones from archeological sites—has provided conclusive findings with regard to the presence of plague across northern Europe, and

genetic research has provided at least partial answers to most of the outstanding questions. Examination of bones and dental pulp extracted from bodies exhumed from plague burial sites has irrefutably established the presence of *Y. pestis*. In the terse comment of a recent researcher, "Finally, plague is plague."⁹ These findings do not exclude the possibility of a second epidemic pathogen as well, but they provide robust proof that bubonic plague was a factor. Furthermore, to date, the DNA of anthrax or other epidemic pathogens has not been detected in plague burial grounds.

In addition, genomic research has identified mechanisms that resolve many of the difficulties confronting those who consider plague as almost certainly responsible for the medical events of the second pandemic. It is now known that there are different strains of *Y. pestis*; that these strains vary in their proclivity to produce bubonic or pneumonic plague; and that the strain implicated in the Black Death was highly virulent, in part because of its tendency to produce the pneumonic form of the disease.

These data shed new light on the Black Death. Person-to-person dissemination by droplets allows a far more rapid propagation of the disease than the comparatively slow long-distance movements of rats over land and sea. Pneumonic plague also spreads more easily in winter than bubonic plague because it does not depend on the activity of fleas but rather on the human behavior of congregating indoors during cold weather within close range of coughs and sneezes. It would therefore thrive in the winter environment of northern and eastern Europe.

Furthermore, droplets were not the only vehicle for direct human-to-human transmission without the mediation of the rat. The human flea *Pulex irritans* can also play a significant role apart from the rat flea *Xenopsylla cheopis*. Investigation of the small but famous sample of the Derbyshire village of Eynsham in its experience of plague in 1665–1666 revealed that human-to-human propagation by the human flea was far more prevalent than by the rat flea. Such direct human-to-human diffusion occurring during the second pandemic as a whole would help to explain the rapid spread of the disease, which far outran the onward progress of rats and the fleas they concealed in their fur.

Since the Black Death appeared in Europe as a sudden and unfamiliar invader from abroad, the population possessed no immunity against it. Genetic findings demonstrate that although the Plague of Justinian was also caused by *Y. pestis*, it was a strain that was genetically distant from the strains responsible for the second and third pandemics. Crossover immunity from one to the other is therefore unlikely. Consequently, the Black Death probably

spread as a “virgin soil epidemic” analogous to smallpox in the Americas—a factor that also helps to explain its extraordinary virulence and rapid spread. Furthermore, a pneumonic disease spreading through air droplets would account for the paucity of contemporary references—in art, literature, and chronicles—to rodent die-offs that preceded the affliction of humans.

Meanwhile, the contrasting predominance of bubonic rather than pneumonic plague in the third pandemic clarifies some of the features that distinguish it from the second: the constant references to swarms of dying rats by all observers, in clear contrast to the Black Death; the slow and erratic movement of the disease; its tendency to persist even for years in the localities it invaded; and its exclusive preference for warm climates and mild seasons. Pending further research and greater clarity, the preponderance of current evidence suggests that the traditional attribution of all three pandemics to *Y. pestis* is correct—provided that allowance is made for different strains and due account is taken of the balance between bubonic and pneumonic plague.

The specific features of pneumonic plague also explain the interest of bioterrorists and germ warfare laboratories in this disease. It spreads rapidly, is readily aerosolized, and is nearly 100 percent lethal. Furthermore, it begins with mild flulike symptoms that delay recourse to diagnosis and treatment, and it frequently runs its course within the human body in less than seventy-two hours. The opportunity to deploy curative strategies is therefore exceptionally brief. This situation is rendered even more critical by the recent appearance of antibiotic-resistant strains of *Y. pestis*. By virtue of these characteristics, the CDC has classified *Y. pestis* as a “Tier 1 select agent”—a pathogen with the highest appeal for use as a weapon of biological warfare or bioterror.

Conclusion

In practice, plague sufferers often received no medical attention. In the early waves of the Black Death in particular, societies were caught unprepared by an unknown “emerging disease.” No facilities—administrative, religious, or medical—were in place to cope with the tidal wave of disease and death. Physicians and attendants recognized that they were powerless to understand or treat the invading new affliction and that they were far too few in number to cope with the catastrophe that engulfed them. Their profession also exposed them disproportionately to risk, and they perished in great numbers during outbreaks.

Furthermore, overcome with terror like the population at large, many doctors joined patients' relatives and friends in the general flight from plague-threatened cities. Indeed, one of the horrors of an epidemic of plague was that it broke the common bonds of humanity. As a result, sufferers were often abandoned to face agony and death alone. The most famous and perhaps most harrowing plague testimony is that of Giovanni Boccaccio in the *Decameron*, which was based on the experience of Florence in 1348:

Tedious were it to recount, how citizen avoided citizen, how among neighbors was scarce found any that shewed fellow-feeling for another, how kinsfolk held aloof, and never met, or but rarely; enough that this sore affliction entered so deep into the minds of men and women, that in the horror thereof brother was forsaken by brother, nephew by uncle, brother by sister, and oftentimes husband by wife: nay, what is more, and scarcely to be believed, fathers and mothers were found to abandon their own children, untended, unvisited, to their fate, as if they had been strangers. Wherefore the sick of both sexes, whose number could not be estimated, were left without resource but in the charity of friends (and few such there were), or the interest of servants, who were hardly to be had at high rates and on unseemly terms.¹⁰

An aspect of the dread felt by those threatened with plague was that it broke the framework developed in the Middle Ages for dealing with death. The historian Philippe Ariès has explained that populations across Europe had devised a series of beliefs, practices, and rituals to provide support in dealing with mortality. These strategies helped people to cope with loss, to heal the tear caused in a family or community by the death of one of its members, to express grief, and to pay their last respects to the dead. Taken together, the practices constituted an “art of dying” (*ars moriendi*) that was codified in instructions elaborated in paintings, engravings, sermons, and books that all explained how to die properly in accord with Christian doctrine. A work of the kind—called a *memento mori* (reminder of death)—explained who should be present at the last hour, detailed the last sacraments to be administered by the clergy, and dictated the proper funeral rites—the laying out of the body, the wake, the procession, the funeral service and burial ceremony, the interment in consecrated ground, and the funeral meal for surviving friends and relatives. In all of these rituals the objective was to allow communities to express the values of solidarity and human dignity.

The most famous of all writers on the theme of *ars moriendi* was the ascetic seventeenth-century Anglican Bishop Jeremy Taylor. His major works were *The Rule and Exercises of Holy Living* (1650) and *The Rule and Exercises of Holy Dying* (1651). The aim of both books, which were widely read in both Britain and America, was to remind the faithful that life on earth is unsafe and ultimately unimportant; therefore, believers should primarily spend their time preparing for eternal life. It was vitally important to die with one's worldly affairs in good order and with one's soul in a state of grace ready to confront judgment day. Taylor's books were instructional manuals on how to achieve both ends—the material and the spiritual. Taken together, they indicated the means to “tame death” (using Ariès's term) so that believers faced mortality confident in the knowledge of being properly prepared to confront it.

What made bubonic plague especially fearful was that it presented communities with the antithesis of the “art of dying” and robbed individuals of the opportunity to achieve a “tame death.” It caused believers to face sudden death, or *mors repentina*, in which victims could be caught with wills unwritten in this world and unconfessed souls in a state of sin that would lead to damnation in the next. Death from plague was sudden; people died alone and without the attention of the clergy; and often they were denied funeral rites and proper burial.

Fear of sudden death from plague is therefore analogous to the terror described by Drew Gilpin Faust in her book *This Republic of Suffering: Death and the American Civil War* (2008). Faust places the terror of sudden death at the center of her account because it was widespread among soldiers on both sides of the conflict. The dread appears repeatedly in their letters home to loved ones and friends. In this respect, bubonic plague resembles total war, as both present unlimited possibilities for the sudden death that can come to us “as a thief” (Rev 3:3).

The New Testament book of Revelation provides a vivid account of the end times—with a day of wrath, a great apocalypse, and plagues and suffering. During the plague centuries, the visual arts made the depiction of “death unleashed” as described in Revelation central to their iconography. Many artists portrayed the “triumph of death,” which takes the form of a universal plague complete with the Four Horsemen of the Apocalypse. Perhaps no better example of this terrifying genre can be found than the painting *The Triumph of Death* (1562–1563) by the Flemish master Pieter Bruegel the Elder. In the foreground and center of the picture Death himself drives a great ox-cart while wielding a scythe to reap his grim harvest. Before him the Angel

of Death sets forth blowing a trumpet while people are dying all around and graves are opening to yield up their skeletons.

A second major aspect of plague iconography was “vanitas,” that is, a symbolic expression of the idea that earthly life is fleeting and insignificant (fig. 4.2). The vanitas theme gained widespread currency in the wake of the first wave of the Black Death and then faded in the eighteenth century with the coming of the Enlightenment and the end of the second pandemic. The traditional Christian view of the transitoriness of life was expressed in the book of Ecclesiastes (1:2–4): “vanity of vanities, all is vanity. What profit hath a man of his labour which he taketh under the sun? One generation passeth away, and another generation cometh.” Such paintings often display temporal goods embodying the hubris of human aspirations—gold, musical instruments, scholarly tomes, globes, and elegant garments. These were juxtaposed to striking symbols of the underlying truth that human



Figure 4.2. Harmen Steenwijck, *Vanitas stilleven* (ca. 1640), which symbolically depicts the transience of life and certainty of death.

The *vanitas* was a popular art form during the Black Death.

Museum De Lakenhal, Leiden.



Figure 4.3. German painter Lukas Furtenagel painted this *vanitas*, *The Painter Hans Burgkmair and His Wife Anna*, in 1529. Kunsthistorisches Museum, Vienna.

accomplishment is minimal and life is short—skulls, candles whose flame has just gone out, hourglasses marking the passage of time, crossbones, skeletons, and shovels. One example by the German painter Lukas Furtenagel shows the faces of a middle-aged couple reflected in a hand mirror as skulls (fig. 4.3).

Another artistic motif that coincided with the era of the plague was the *danse macabre*. Such works of art portray Death as a skeleton summoning people of all ages, ranks, and sexes to join in a merry dance. Sometimes Death is armed with a scythe, arrow, or dart, and he leads the dance while playing a musical instrument. Churches often enacted such a performance, transposing the representation of the fragility of life to the medium of theater. More recently, Bergman's plague film *The Seventh Seal* reaches its denouement when Death summons the protagonists to join in a jolly dance.

We have considered the cultural and physical impacts of plague on the populations it afflicted, but how did the authorities of church and state seek to contain the disaster? What administrative strategies and medical therapies did they deploy? We now turn to the collective responses of societies to the emergency.