The Bubonic Plague

A bacterial disease carried by fleas that feed on rats, it has afflicted human beings for more than 1,000 years. The factors responsible for its alternate rise and fall remain a mystery

by Colin McEvedy

In the year 1346 Europe, northern Africa and nearer parts of the Middle East had a total population of approximately 100 million people. In the course of the next few years a fourth of them died, victims of a new and terrifying illness that spread throughout the area, killing most of those unfortunate enough to catch it. The disease put an end to the population rise that had marked the evolution of medieval society: within four years Europe alone suffered a loss of roughly 20 million people. The disease responsible for such grim statistics was the bubonic plague, and this particular outbreak, lasting from 1346 to 1352, was known as the Great Dying or the Great Pestilence. Later it was appropriately referred to as the Black Death, a name that has come down through history.

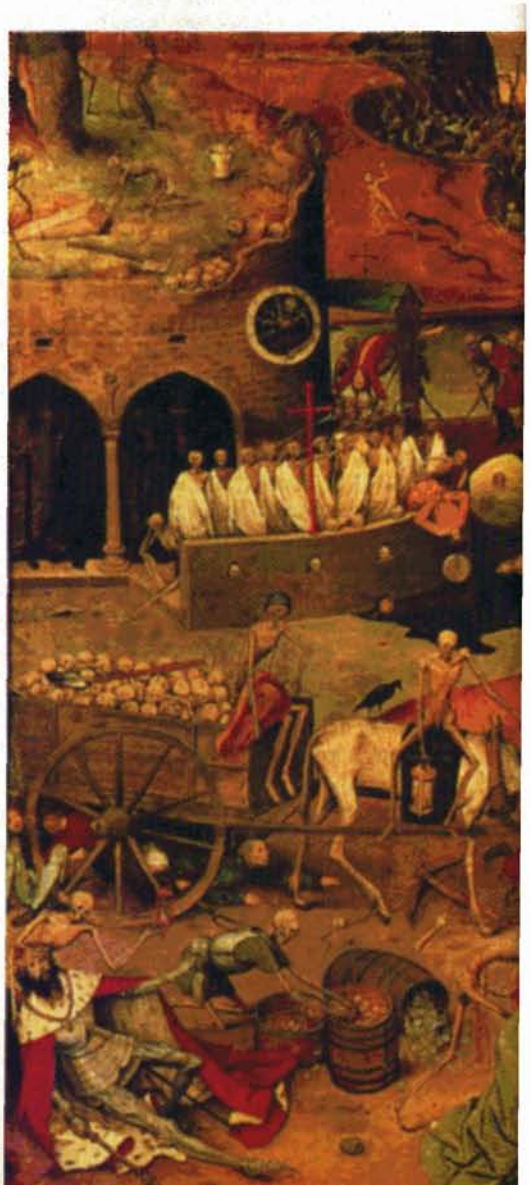
Although the effects of the Black Death may have been particularly catastrophic, striking as it did after a long period in which the disease had been unknown in the West, this was not the first time the plague had ravaged Europe. Some 800 years earlier, during the reign of the emperor Justinian in the sixth century, there was an epidemic of similar proportions. There were also repeated, if less widespread, epidemics in the two centuries following the plague of Justinian's time, and for four centuries after the Black Death. The disease has undergone a precipitous decline since that time, but it still occurs sporadically in various parts of the world today, including the U.S.

From 70 to 80 percent of those who contracted the plague in the 14th century died from it. Indeed, the symptoms usually presented themselves with a ferocity that presaged death within five days. The name, bubonic plague, derives from one of the early signs of the disease: the appearance of large, painful swellings called buboes in the lymph nodes of the armpit, neck or groin of the victim. Three days after the appearance of the buboes people were characteristically overwhelmed by high fever, became delirious and broke out in black splotches that were the result of hemorrhaging under the skin. As the disease progressed, the buboes continued to grow larger and more painful; often they burst.

The bursting is said to have been particularly agonizing, capable of arousing even the most moribund patients to a state of frenzy. Yet physicians always regarded the bursting as a good sign, if only because it indicated that the patient was still capable of putting up a fight a week or so after the onset of the illness. Of those who were going to die, probably half were already dead by this stage.

In some cases a person's bloodstream was directly infected, which led to septic shock, massive hemorrhaging and rapid death, a form of the disease known as septicemic plague. In other cases plague was transmitted as a type of pneumonia; in pneumonic plague the victims collapsed, spit blood and were almost always dead within a few days.

Strange as it may seem, in view of the frequency of the disease and the toll it exerted on the population, no one at the time had an inkling of its fundamental nature, its ultimate cause or how it was spread. During the period of the Black Death people were inclined to attribute the disease to unfavorable astrological combina-



HORROR OF THE PLAGUE is captured in Pieter Brueghel the Elder's painting "Triumph of Death," where death, in the form of roving skeletons, overwhelms a kingdom of the living. Neither the king with his piles of gold nor the young revelers at their table can escape the relentless army of the dead. Behind the king a corps of skeletons pushes victims into a water-filled mass grave; a barren landscape already robbed of life can be seen in the distance. Apocalyptic visions of this kind were common in the centuries when the plague ravaged Europe and the healthiest could be wiped out in a few days.

tions or malignant atmospheres ("miasmas"), neither of which could be translated into a public-health program of any kind. More paranoid elaborations blamed the disease on deliberate contamination by witches, Moslems (an idea proposed by Christians), Christians (proposed by Moslems) or Jews (proposed by both groups).

It was not until 1894 that the French bacteriologist Alexandre Yersin discovered that bubonic plague is caused by a gram-negative bacterium, *Yersinia pestis,* belonging to a group of bacteria known as rod-shaped bacilli, many of which are pathogenic. Plague bacilli are found at low frequency in many wild rodent populations throughout the world and are transmitted from one rodent to another by fleas. In the case of the bubonic plague the flea often responsible for transmitting the disease is the oriental rat flea, *Xenopsylla cheopis.* When a flea bites an infected rat, it ingests the bacilli, which proceed to replicate within its digestive tract, forming a solid mass that obstructs the flea's gut; the flea is unable to ingest blood and becomes ravenously hungry. In a feeding frenzy it repeatedly bites its animal host, regurgitating plague bacilli into the host's bloodstream every time it does so. These injection sites then act as foci for the spread of bacilli. If the host animal dies, as it is likely to do, the flea moves to the next available live rodent. The disease spreads rapidly in this manner; as the number of live rats decreases, the fleas move to warm-blooded hosts on which they would not normally feed, such as human beings and their domesticated animals, and so an epidemic is launched.

Once the disease enters the human population it can sometimes spread directly from human to human through the inhalation of infected respiratory droplets. The normal mode of spread is by the bite of rat fleas, however; the disease does not persist in the absence of rodents, which are the primary hosts for both the plague bacillus and the rat flea.

The essential requirement for an epidemic (an outbreak in a human population) is a rodent epizootic (an outbreak in an animal population). This is necessary both to initiate and to sustain the disease in human beings. Of course, the two populations must be in close contact for the transmission to be successful, but it is unlikely that this was ever a significant variable in medieval times. In rural as well as urban areas humans lived surrounded by rats.

The Black Death is thought to have migrated along the Silk Road, the trans-Asian route by which Chinese silk was brought to Europe. There are two reasons for believing this was the case. The first is that outbreaks of the plague were recorded in 1346 in Astrakhan and Saray, both caravan stations on the lower Volga River in what is now the U.S.S.R. The second

is that during the years 1347 and 1348 the Arab traveler and scholar Ibn Battuta, returning along the Spice Route from a stay in India, first reported hearing news of the plague when he reached Aleppo in northern Syria, not before. That clue excludes transmission by way of the Indian Ocean and Persian Gulf ports.

Most likely the disease erupted first among marmots, large rodents native to central Asia (they are related to woodchucks but belong to a different species) whose fur was an important article of trade throughout that part of the world. According to this historical reconstruction, trappers coming across dead or dying animals collected their furs, delighted to find such an abundant supply, and sold them to dealers who in turn (without worrying about reports of illness among the fur trappers) sold them to buyers from the West. When the bales of marmot furs sent west along the Silk Road were first opened in Astrakhan and Saray, hungry fleas jumped from the fur, seeking the first available blood meal they could find.

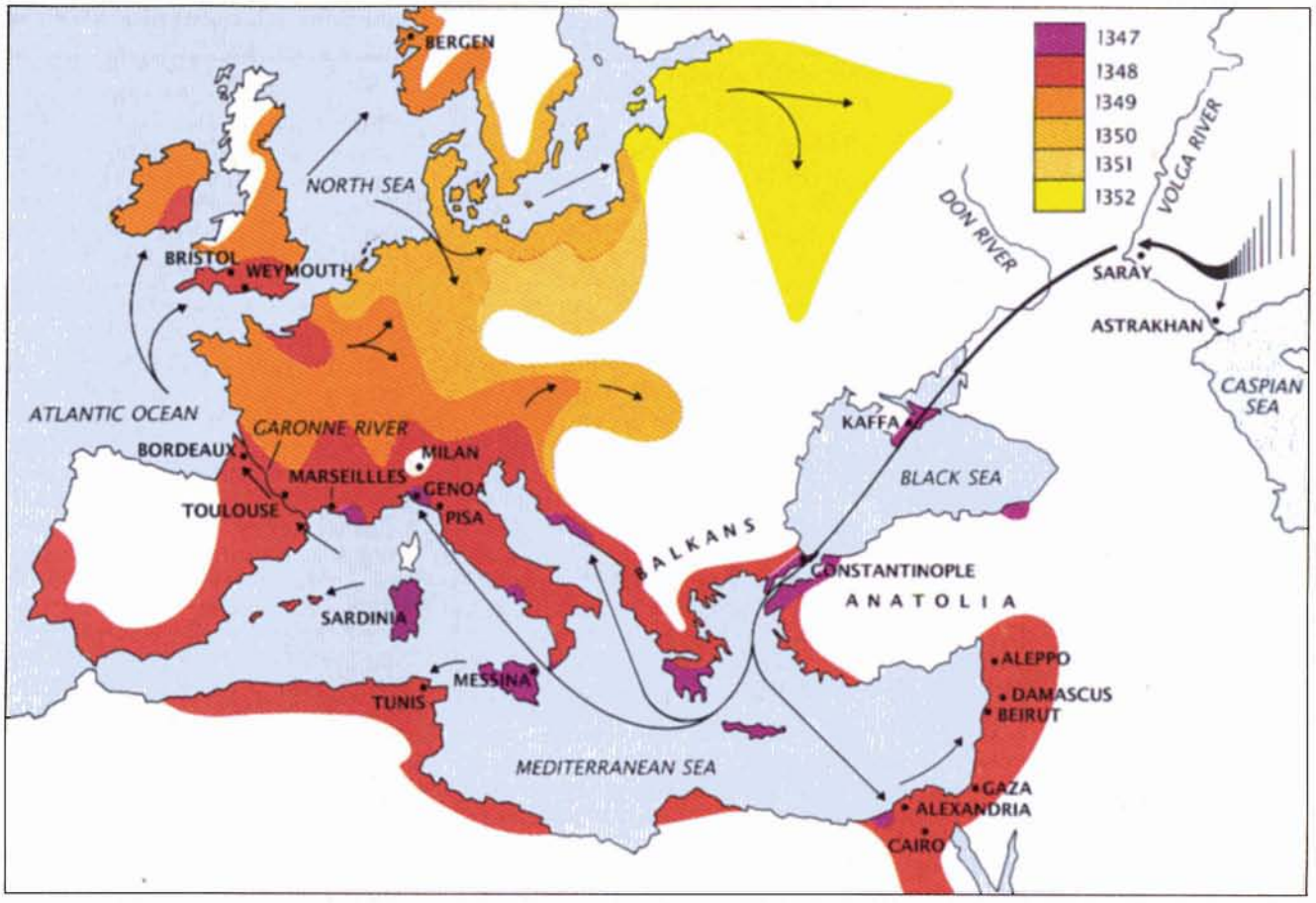
PHYSICIAN'S GARB that was worn during a plague outbreak in Marseilles in 1720. The birdlike costume, made of leather, covered its wearer from head to toe and was believed to provide protection from contagion. The large beak contained sweet-smelling herbs to filter airborne contagion; the wand contained incense that was thought to ward off impurities. Even the eyeholes, which held crystalline lenses, were protective.

From Saray the disease is thought to have traveled down the Don River to Kaffa, a major port on the Black Sea, where a large rat population provided the perfect breeding ground for the plague bacillus. Because many of the rats in Kaffa were living on sailing vessels bound for the ports of Europe, the disease had a ready means of transport to that part of the world.

Indeed, it would be difficult to design a more efficient means of disseminating the plague than a medieval ship. The holds of these ships were generally crawling with rats; when the crew slept, the rats took over, running through the rigging and dropping fleas onto the decks below. The cycle of infection, from flea to rat and rat to flea, would be maintained until the rat population was so reduced by the disease that it could no longer sustain the fleas and the plague bacteria they were carrying. Hungry fleas, seeking any host they could find, would then carry the disease into the human population. It is small wonder that by the end of 1347 plague had broken out in most ports on the route linking Kaffa to Genoa in northern Italy.

The two most important ports along this route were Pera, a suburb of Constantinople, and Messina in Sicily. Both places were stopover points for ships crossing the Mediterranean and became major foci for further dissemination of the plague. The initial impact on the population of Constantinople was graphically described by the emperor Cantacuzenus, who lost a son to the disease in 1347. He recounts how it spread throughout the Greek islands and along the coasts of Anatolia and the Balkans, killing "most of the people." In Messina the first outbreak was recorded in October of 1347, launching an epidemic that quickly spread to include the entire island.

From there in early 1348 the Black Death crossed over to Tunis on the north coast of Africa and then spread by way of Sardinia to Spain. By the time it reached Spain the Black Death had also spread to the heart of Europe, a fact that can be blamed at least in part on the Genoese, who are said to have heartlessly turned away ships from the east carrying their sick countrymen. Not only did such hardheartedness have little effect (the city was as badly hit as any in Europe) but also the diversion of ships to other ports, such as Marseilles and Pisa, hastened the spread of the plague throughout Europe.



BLACK DEATH came from central Asia to Europe via the Silk Road, arriving in Kaffa in about 1347. From there it was carried by ship to the major ports of Europe and northern Africa. Most of Europe was affected before the epidemic finally subsided in 1352. Milan, the largest city to escape the plague, is believed to have done so because it is the farthest Italian city from the sea.

By this time the epidemic was raging throughout the Mediterranean. Ships carrying silk, slaves and fur brought it to Alexandria before the end of 1347; from there it spread south to Cairo, east to Gaza, Beirut and Damascus and finally along the north coast of Africa to Morocco.

By 1348 the Black Death had jumped from the Mediterranean region to the Atlantic coast of Europe. It crossed southwestern France by way of the regional capital, Toulouse, and rapidly passed down the Garonne River to Bordeaux on the west coast. From there it is likely that one of the ships loading claret for the British market brought the Black Death to Great Britain. In 1348 it was first recorded at Weymouth on the south coast of England, and it is believed to have spread to Ireland from Bristol.

From England the plague crossed the North Sea to envelop Scandinavia in its deadly grasp. According to one story, the invasion of Scandinavia can be blamed on a ship that left London in May, 1349, bound for Bergen with a full crew and a cargo of wool. The ship is reported to have been seen some days later, drifting off the coast of Norway. Local people who rowed out to investigate found the crew dead and returned to shore, carrying the wool and—unwittingly—the plague with them. That started a chain reaction as village after village along the Norwegian coast succumbed to the disease.

The following year the Black Death ravaged the populations of Denmark and Germany before entering Poland in 1351 and Russia in 1352. This in effect completed the circle; not only had the disease returned to within a few hundred miles of its entry into Europe on the Volga steppe but also after four long and devastating years mortality rates in western Europe had finally returned to normal.

The society that emerged from the period of the Black Death became quite prosperous; the survivors had inherited the fortunes of their deceased relatives and many were able to move into positions of prominence once closed to them. Their good fortune did not necessarily last for long, however. In 1356 a second outbreak of the plague appeared in Germany and spread rapidly throughout Europe. It exacted a particularly heavy toll among the children born since the end of the Black Death.

Thereafter the plague returned to Europe with mournful regularity; indeed, the continent never seemed free of it for more than a few years at a time. Although the later epidemics never matched the Black Death in terms of overall mortality, they nonetheless continued to have a negative impact on population growth in Europe through the end of the 14th century.

At this point an equilibrium was reached between plague and people, and in the 15th century the population began to recover. In particularly hard-hit regions it took more than a century for numbers to return to their original levels, but by the end of the 16th century populations all over were higher than they had been prior to the onset of the Black Death.

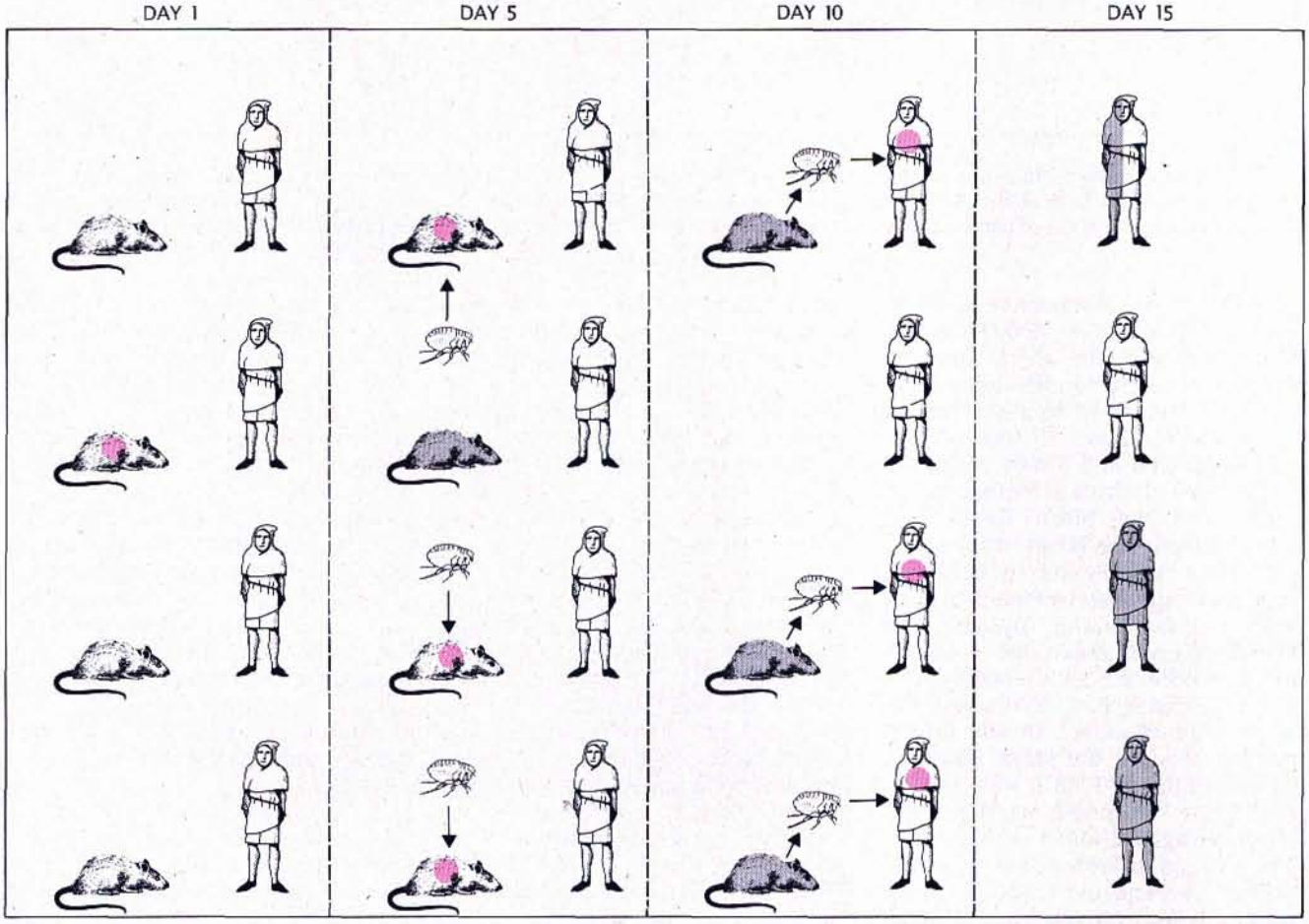
Strangely, when the plague did reappear (which it continued to do, albeit less frequently), it often did so with a ferocity equal to any recorded in previous outbreaks. In the last epidemic in France, from 1720 to 1722, half of the population of the city of Marseilles died, together with 60 percent of the population in neighboring Toulon, 44 percent at Arles and 30 percent at Aix and Avignon. Yet the epidemic did not spread beyond Provence, and the total number of deaths was less than 100,000.

By the 16th century it was widely believed the plague spread as a result of contagion: a toxic factor that could be transferred from the sick to the healthy. Human-to-human transmission was thought to take place either directly through physical contact with a sick person or indirectly by the clothes or bed sheets. In response, many towns and villages instituted quarantine regulations. The authorities in England, for example, recommended that plague victims be locked up in their homes or transferred to special "pest houses." An extreme example of adherence to public policy is the famous case of William Mompesson, rector in the small village of Eyam in Derbyshire, who persuaded the entire community to enter into quarantine when the plague erupted there in 1666. One by one the parishioners who remained faithful to their contaminated hearths fell victim to the disease. A mortality rate of 72 percent indicates that the community probably had a morbidity (infection) rate of 100 percent, an extraordinary price to pay for a misconceived theory.

Locking people in their homes is, of course, one of the worst possible ways to fight the plague. The plague is a disease of "locality," most likely to manifest itself when rats, fleas and people are kept in close contact with one another. To confine people is to maximize their chance of being bitten by a plague-carrying flea or infected through close contact with another human being.

Officials recognized that quarantines were dangerous to healthy individuals confined with sick relatives, but they imposed them nonetheless in the belief that some lives must be sacrificed in order to stop further spread of the disease. Because it is rats that carry the plague (and the rats were free to travel), the entire quarantine effort was a waste of time—and lives.

Attempts were also made to quarantine passengers and goods arriving in boats from overseas. When sickness suggestive of the plague was observed among crew members or passengers, the ships were diverted to lazarettos (quarantine stations) until the authorities deemed it safe to release them. At Marseilles in May of 1720, for example, the sailing ship *Grand Saint Antoine* was placed in quarantine for three weeks because eight of its crew had died in the course of the voyage from the Near



PROGRESSION OF THE PLAGUE through a medieval household could be very rapid once the black rats that lived there became infected. An infected rat, marked with a red dot on day 1, is shown to die from the disease by day 5. When that rat dies, its fleas leave, carrying the plague with them to other rats in the house. By day 10 these rats have also died from the disease and the fleas turn at that point to human beings, infecting almost 75 percent of them. By day 15 approximately half of the humans in the house will have died from the plague; a fourth of them will have recovered from it and a fourth will have escaped it.

East. In spite of these efforts to limit the spread of the plague, the disease broke out in Marseilles—first among the dockworkers who unloaded the ship's cargo when it was released from quarantine and then in the population at large.

There is little evidence that quarantines of this type were ever very effective. Venice was one of the first seaports to introduce quarantine regulations, early in the 15th century, enforcing them by imposing the death penalty on anyone who broke the rules. Yet Venice suffered from the plague as much as any city in Italy, presumably because it was impossible to prevent rats aboard quarantined ships from jumping ashore, carrying the plague with them.

Finally, after innumerable cycles of onslaught and retreat, the plague disappeared from Europe. London's last experience with the disease, the Great Plague, began in 1665 and ended in spectacular fashion with the Great Fire of 1666. At that time it was natural for Londoners to believe they owed their deliverance to the purifying conflagration. Later it was suggested Londoners owed their resistance to the plague to the reconstruction that followed the fire and the fact that the rebuilt city boasted brick houses and wide, rubbish-free streets in place of the higgledy-piggledy structures and malodorous alleys of medieval times.

This explanation is attractive but does not hold up under scrutiny. One reason is that the fire destroyed only the central part of London, the area least affected by any of the outbreaks of plague earlier in the century, leaving untouched the overcrowded suburbs that had provided the disease with its main lodging in previous times. A second reason is that other cities in Europe, such as Paris and Amsterdam, became plague-free during the same period—a phenomenon that could not be linked to the Great Fire of London.

A somewhat more convincing (but still flawed) theory suggests that the disappearance of the plague coincided with a slow rise in prevailing standards of health and hygiene. Although hygiene cannot be eliminated as a factor, it does not explain why subsequent outbreaks followed the standard course, complete with high rates of mortality, but were farther and farther away from the center of Europe each time they appeared. It was almost as if Europe were developing some form of resistance to the plague that kept the infection from propagating in the usual way. In the north the path of retreat was to the east; in the Mediterranean it was to the south. The later the epidemic, the less it seemed to be capable of spreading. This, moreover, was at a time when, according to every available index, traffic by land and by sea was increasing.

When the role of rats was finally established late in the 19th century, it was suggested that the subsidence of the plague could be explained by changes in the population dynamics of the black rat, *Rattus rattus*. During the 18th century it had been observed that the black rat, the historic carrier, had been largely displaced by a new species, the brown rat (*Rattus norvegicus*), which would have been a much poorer vector of the plague: the brown rat is as susceptible to the plague bacillus as the black rat but does not normally live in close proximity to humans. Brown rats typically live in dark cellars or sewers, whereas black rats overrun the upper rooms and rafters of a house. Because the oriental rat flea has a maximum jump of 90 millimeters (a little more than 3.5 inches), the difference in preferred habitats may have been enough to isolate humans from plague-infested fleas.

The brown-rat theory seems plausible but does not fit the geography: the brown rat spread across Europe in the 18th century from east to west, whereas the plague retreated from west to east. The brown rat was in Moscow long before the city experienced a particularly severe epidemic of the plague in the 1770's; it did not reach England until 1727, more than 60 years after that country's last bout of the plague.

The late Andrew B. Appleby of San Diego State University suggested an alternative theory, namely that a certain percentage of black rats became resistant to the plague over the course of the 17th century and that the resistant animals would have increased in number, spreading across Europe during the next 100 years. Although these rats might still be infected by the plague bacillus, they would not die from it and therefore could support a large population of fleas, rendering it unnecessary for the fleas to seek other hosts. This theory, however, does not conform to what is known about resistance to plague in animal populations. As Paul Slack of the University of Oxford has pointed out, rat populations often develop resistance when exposed to a pathogenic bacterium or virus, but such resistance is short-lived and is therefore unlikely to have been responsible for broad-based immunity to the plague.

A more plausible theory suggests that a new species of plague bacillus, *Yersinia pestis*, may have evolved that was less virulent than the previous strain. Being less virulent, it might have acted as a vaccine, conferring on infected animals and humans a relative immunity to more virulent strains of the bacterium.

The bacteriological theory is acceptable on several grounds. First, it conforms to the dictum, proposed by the American pathologist Theobold Smith, that "pathological manifestations are only incidents in a developing parasitism," so that in the long run milder forms of disease tend to displace more virulent ones. Second, it explains why the decline of the plague is associated with a failure to spread beyond local outbreaks: a disease cannot travel far when the number of people susceptible to it is low. Third, it is supported by the existence of a close relative of the plague bacillus, *Yersinia pseudotuberculosis*, which does not induce visible illness in rats but does confer on them a high degree of immunity to the plague.

Did *Y. pseudotuberculosis*, or a relative with similar properties, gradually spread through the rodent population of early modern Europe, making it impossible for *Y. pestis* to gain a foothold there? Although no direct evidence exists to support that hypothesis, it seems more reasonable than any other.

The discovery and widespread use of antibiotics has conferred on human beings a different form of protection from the plague. Although the disease still occurs with regularity throughout parts of Africa, South America and the southwestern U.S. (in 1986, 10 cases were reported in the U.S.), it is never again likely to reach epidemic levels now that we know how it spreads, what public-health measures are appropriate and how to treat plague cases as they occur. Nevertheless, many questions about the plague are as yet unanswered. For example, the mode of transmission in rural areas, where rat populations are discontinuous, is entirely unclear. And what explains the distribution of the plague throughout the world today? Why are only certain rodent populations reservoirs of the disease whereas others are entirely free of it?