

## Introduction

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During the fifteenth century plagues assaulted cities in northern Italy more frequently than during the late fourteenth century. In the earlier century, pestilences that were described as bubonic plague visited major cities once in every fifteen to twenty years. In the 1400s plagues hit some cities, for example, Venice and Florence, at least once a decade. In contrast to the devastating effects of plague on rural areas in the fourteenth century, the countryside was usually spared this scourge during the fifteenth century. Thus the basic experience confronting fourteenth- and fifteenth-century observers on the frequency and extension of plague led them to different conclusions about how plague spread.

There were many other differences between the fourteenth- and fifteenth-century epidemics that reshaped the attitudes of Italian city dwellers toward plague in the fifteenth century. Fourteenth-century plague survivors could not have defended the conclusion that plague was a contagious disease of the poor. Too many prominent citizens died in the outbreaks before the fifteenth century. But wealthy Florentines of the 1430s could be reasonably sure that flight from the city would protect them from plague. When they returned the records would show clearly the heavy mortality in working-class districts of the city and the loss of whole families to plague; at the same time prosperous districts were relatively spared. Florentine secular and religious leaders were certain by the 1450s that all the evidence supported the tenet that this was a contagious disease. By midcentury, Florentine legislators were beginning to consider serious changes in traditional sanitary policy in order to accommodate the general conviction that one must control the contagious spread of disease. The next generation of leaders would articulate clearly the further “experience” that plague and poverty were close companions.

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Not only were the Florentines' stated motives for adopting new plague controls similar to those of their northern Italian neighbors, but they also adopted containment measures against plague similar to and nearly simultaneously with legislation in Venice, Mantua, Siena, Perugia, Milan, and other areas from which we possess some record. Novel plague legislation appeared throughout northern Italy during the second half of the fifteenth century. But when new sanitary policies were elaborated, during the middle third of the fifteenth century, professional medical opinion did not concur with the legislators' model of plague spread. In fact most of the serious medical concern with a contagion theory did not surface until the sixteenth century. Nevertheless the absence of a scientifically defended contagion theory did not stop policymakers from outlining strategies for the isolation of plague sufferers and plague "carriers" far beyond the originally passive, defensive quarantine devised in the fourteenth century. By the 1460s northern Italian leaders in many different cities all agreed that plague hospitals were necessary to decrease the spread of a contagious disease.

To be so convinced, the leaders of the mid-fifteenth century must have been persuaded that their experience of plague was unambiguous despite what doctors were saying and that the new policies could be justified on the grounds of plague contagion alone. This book will show that from our twentieth-century perspective the misdiagnosis of plague – attributing to plague many deaths that could have been caused by other infectious diseases spread by human contact – was prevalent during the fifteenth century and effectively supported the legislators' stance. But even without the problem of determining just who died from plague, fifteenth-century legislators found the new measures attractive because the legislation successfully addressed social disruptions created by plagues. The eventual adoption of the contagion principle of plague defense served the need both to solve the problem of recurrent epidemic disease and to confront the growing burden of urban poverty.

My concern with plague will involve two main questions. First, I will tackle the problem of the extent to which true plague (*Yersinia pestis*) epidemics may have occurred by considering what other infectious diseases contributed significantly to outbreaks of "pes-

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tilence.” Rather than elaborate mechanisms whereby plague could have behaved as the fifteenth-century cases present themselves, I have chosen to explore the historical significance of the probability that other ordinary, nonplague causes of death were common during the plague years. Historical demographers have often acknowledged heavy losses among infants and children during mortality crises. Some scholars even have suggested that epidemic crises due to causes other than plague certainly contributed to the demographic collapse throughout Europe from 1400 to 1450. This study will examine in detail, and with surprisingly good fifteenth-century evidence, the causes of death other than plague. We will not be as concerned here, however, with the number who died and the demographic effects of epidemics as we will with the social and historical significance of different causes of death in the fifteenth century. The records of causes of death do have social significance even if they are not demographically precise. Furthermore differentiating victims of true plague from victims of other diseases during a plague year is more important in historical epidemiology than in historical demography because attention to these details is the only way to pinpoint the changes in the behavior of human infections over time.<sup>1</sup>

Second, I will discuss the historical effects of changes in plagues and in plague controls. Northern Italian city-states led the rest of Europe in adopting measures for dealing with recurrent plague: the quarantine, the lazaretto (pest house), and health boards. Yet these and other means of segregating and isolating the sick, with very few exceptions, were not adopted until fully a century after the Black Death of 1348. Most cities saw ten or eleven plagues before they decided that this was a contagious disease, best interrupted by controlling the sources of contagion. The controls adopted could not have been either rational or successful in dealing with the disease caused by *Y. pestis*. They would have been very effective, however, in controlling other contagious diseases that have escaped historians’ attention.

Many questions have been overlooked in the attempts to remember who built the first lazaretto, devised the first quarantine, created the first permanent health boards, and wrote the first medical treatise on contagion. There are few studies of how a diagnosis was made; there are even fewer studies of other diseases besides plague that affected the lives of early Renaissance Italians. Many

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Excerpt

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scholars have asserted that differential mortality – between rich and poor, between young and old, between male and female – was important, but few have actually attempted to analyze such phenomena. Here I will present evidence that all do not die equally before epidemic infections, and I will discuss how differential mortality could be historically important. I believe that differential mortality in plague years justified many of the changes in legislative policy.<sup>2</sup>

In discussing both the biological aspects of past plague and their historical effects, I will focus on a case study of Florentine mortality during the early fifteenth century, made possible by the survival of early urban death registers. The Florentine Grain Office's Books of the Dead allow us to examine the mortality in Florence from 1385 to 1458 in detail not usual with records this early. I selected for analysis a manageable sequence from 1385 to 1458, hand-counting over 60,000 cases. The series originally was little more than a register of all burials performed by communal gravediggers, but by 1424 included diagnoses of causes of death during epidemic years. With deaths alone one could show the decline in absolute mortality from plague during the early fifteenth century. Because burial place, and often residence of the deceased, were registered as well, we can document the pronounced clustering of deaths during fifteenth-century epidemics. But with the inclusion of diagnoses, we can derive a rare glimpse of both ordinary and epidemic mortality that cannot be inferred from good demographic studies. The Florentine registers are unique, as far as I know, in providing this diagnostic information before 1450.<sup>3</sup> But Florentine experience with plague was probably typical of other Italian cities in the early fifteenth century.

Thus this study seeks to show that there is much we do not know about epidemic diseases in the two centuries after plague appeared in Europe. There are two separate histories here, that of *Y. pestis* and other infections that have been mistaken for it, and that of the importance of epidemics in shaping human history, unconnected to the identification and description of diseases familiar in twentieth-century medicine. Plagues changed; and plagues changed medical and social history.

Throughout this book we will be concerned with what is true plague and what merely assumes the label “plague” as a matter of

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historical convenience. Although the most important issues this study seeks to address concern how and why fifteenth-century Florentines came to revise their procedures for dealing with epidemics, these issues cannot be discussed without an analysis of the actual disease environment that conditioned fifteenth-century perceptions. In other words, I have of necessity undertaken an epidemiological study that depends on twentieth-century models and understanding of infectious diseases.

Some readers will argue that such concerns are present-minded, distorting the fifteenth-century Florentine view of epidemics. Others may find that shifting back and forth between Renaissance usage of the word “plague” and modern meanings of the term is confusing. Thus I offer the following brief summary of my assumptions about how “true” *Y. pestis* plague is caused, and the extent to which I consider this twentieth-century material useful in understanding fifteenth-century “plague.”

The ecology of plague is extremely complex. It is not normally a disease of humans; plague cannot become “endemic,” no matter how frequently it afflicts human populations. The disease is almost always acquired indirectly by an arthropod vector, or directly from an infected mammal. There are no human carriers of plague like a “Typhoid Mary,” continually shedding the bacterium.<sup>4</sup> Thus it is inappropriate to discuss first the signs, symptoms, and effects of human infection with this disease. Human cases of plague are in fact uncommon, a fact fundamental to understanding the biological and historical significance of recurrent plague in Europe.

*Yersinia pestis*, the microorganism that causes plague, commonly infects the rodents of several large families. The disease can become enzootic, chronically affecting animal communities, and can reach epizootic proportions when susceptible animal hosts have contact with the organism. Most known cases of human plague have resulted from direct or indirect consequences of epizootic plagues; human plague rarely occurs without the presence of plague-infected mammals.<sup>5</sup>

But the black commensal rat, *Rattus rattus*, so popular in historians’ descriptions of epizootic plague, is by no means at the beginning of the story; it is only the proximal transmitter of plague to humans. In all the research conducted on plague during the past

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thirty years, the domestic black rat has played no part in maintaining plague in any area studied. Plague zoologists distinguish “wild plague,” sometimes called “sylvatic plague,” from “domestic plague.” Both “natural” or permanent plague foci and temporary foci can exist, the latter depending on

a wide variety of circumstances primarily involving epizootic hosts (domestic rats or susceptible wild rodents and their constituent fleas). Such temporary infected areas may persist for decades or, under certain circumstances, become plague-free only to be reinfected from permanent foci (via contiguity, long-range transport, etc.).<sup>6</sup>

There are no natural plague foci in Europe today, and even the historical plagues may have depended on the periodic reintroduction of the organism from Middle Eastern or Balkan regions.<sup>7</sup>

Since 1928 plague researchers have suspected that the infection was separately maintained in wild rodents, and since the 1950s that concept has predominated in plague surveillance and control. In wild plague *Y. pestis* displays a periodic pattern of epizootic extension depending on the rodent reservoirs and on the flea vectors, and thus on the seasons favoring these two hosts. Latent infection may occur, though rarely, in hibernating animals, and rodent warrens can preserve live plague bacillus for months, even years, because the microclimate there is favorable to the survival of both insect vector and microorganism. Plague is spread by the movements of rodents, not humans, into affected areas and warrens as well as out of them, and by the transfer of infected fleas. Plague survives in any region because a fairly wide variety of resistant rodents survive with minimal plague infection. Susceptible species, such as *R. rattus*, cannot perpetuate the disease. As a species, *R. rattus* does not become resistant to plague infection.<sup>8</sup>

Plague is thus a disease of field rodents, advancing slowly and regularly “field to field, . . . burrow to burrow, in thin epizootic trails winding across the countryside, infecting village rats in passing.”<sup>9</sup> Investigators in Iran, India, Java, Madagascar, and Kenya all found the same complex of resistant rodents and highly susceptible rodents supporting permanent plague in natural foci. Violent epizootics break out among susceptible rodents, and the dispersal of infected animals in turn spreads the plague to new, resistant rodents. Epizootics occasionally lead to human infection.

The bacillus responsible for plague, *Y. pestis*, was renamed in

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1971 from the older *Pasturella pestis*, a name still seen occasionally in historical literature. The international committee on biological nomenclature changed the name not only to honor Alexandre Yersin, the student of Pasteur working in Southeast Asia who first isolated the organism, but also because other members of the *Pasturellae* are relatively harmless to humans. Not so *Yersinia* species. Among this genus “enterocolitica” and “pseudotuberculosis” species are human pathogens. Among animals *pestis* is by far the most stable and widespread of the *Yersinia* species.<sup>10</sup>

*Yersinia pestis* always retains its bacteriological identification as a Gram-negative, bipolar-staining, facultative anaerobic, non-lactose-fermenting, non-spore-forming bacterium. The full microbiological details are available elsewhere and need not detain us here.<sup>11</sup> *Y. pestis* can vary in virulence and in infectivity, independently of levels of host nutrition, immunity and host ecodemography, whether that host be human, rodent, other mammal, or flea. Many different antigens are included in the genetically determined makeup of the organism, and variations among these can elaborate antigens more or less effective in first infecting a cell and then reproducing the microorganism in the face of the hosts’ normal immunological defenses. Pathogenicity, or the relative lethality of the microorganism may, moreover, vary among different population groups of the same host species, as well as between members of what historian Emmanuel Le Roy Ladurie called the *ménage à quatre*, the complex of bacillus, rodent, flea, and other mammal.<sup>12</sup> Each member population involved in this long chain of transmission could conceivably contribute to separate and dramatic variations in the pathogenicity of *Y. pestis*. Natural selection of one or more specific antigen types in the rat may not enhance the reproduction of the bacillus in the flea.

Adding to the complexity of plague, we could consider the role of the arthropod vector, usually a flea.<sup>13</sup> The insect must feed on an animal with plague septicemia (organisms in the bloodstream), and then successfully transmit that infection to another animal before it dies. Entomologists derive elaborate formulas for determining both “vector efficiency” and “vector capacity” based on flea counts and speciation of all the fleas involved in wild plague, and on the ability of each species to become “blocked” (unable to feed without infecting the host) with the growth of a bolus of plague organisms in the forestomach. Both the transfer of fleas

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from one species of mammal to another and the blockage of a flea more than temporarily are fairly rare biological events.

In the western United States, for example, plague is enzootic among ground squirrels and meadow voles, but transfer of fleas, infected or not, to rats has not been observed, even where rats are abundant.<sup>14</sup> Massive interspecies transfer of plague may, however, occur during epizootics. This is not well documented, but it appears to be the most likely circumstance for transferring plague to commensal rodents, and ultimately to humans.

This somewhat technical review of plague ecology emphasizes to the historian that biological details can occasionally impede useful analysis of past plagues. One might easily surmise that virtually any event external and exogenous to human societies may explain the disappearance, as well as the many appearances, of plague in Europe. The judicious selection of relevant ecological conditions could defend a retrospective diagnosis of *Y. pestis* for any given outbreak of pestilence. When we consider the variety of clinical forms human plague can assume, as we shall do throughout this book, a diagnosis of plague could be defended for almost any epidemic in early modern Europe. Since anything can be plague, it is difficult to gain a confident sense of the change in the disease over time, or to be sure that true plague behaved differently five hundred years ago than it does today.

In order to escape this logical trap, which potentially undermines any venture in historical epidemiology, the present study will make two assumptions at the outset. First, unless there is very persuasive, unassailable evidence to the contrary we must begin from the position that infectious diseases, including *Y. pestis*, in human communities of the European late Middle Ages are similar in both epidemiological and individual clinical presentation to analogous twentieth-century infections. Second, no study of epidemic crises in early modern Europe should overlook a very simple epidemiological maxim that common things occur commonly. The general level of technological and socioeconomic development in fifteenth-century cities helped determine the causes and circumstances of human illness and death in both normal years and years of high mortality. Many epidemic crises of the late Middle Ages could not possibly have been due to plague alone.

Many researchers have of course acknowledged that different infectious diseases could contribute to mortality during plague years.



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This observation is one of common sense. However, many researchers assume that details about all the different individual causes of death in a mortality crisis merely qualify generally valid, population-level phenomena of past human plagues. The supramortality from plague itself is enormous, they argue, and the overall demographic and epidemiological effects of a plague thus give a faithful picture of the behavior of *Y. pestis* at the population level.<sup>15</sup>

Since this book is not devoted to a case study exploring the demographic effects of plague in early modern Europe, it examines the phenomena of nonplague causes of death during plague years, as well as nonplague epidemics, as events having historical significance quite apart from the demographic context in which they are usually discussed. “Accessory” causes of death in an epidemic of plague reveal much about the circumstances in which a diagnosis of plague was made. The original diagnoses of plague, in individual cases and in epidemics, were made by persons who saw plague’s significance quite differently than we do. Furthermore the diagnoses made in the past were not necessarily based on unchanging criteria. A fifteenth-century diagnosis of plague had consequences in the fifteenth century that need historical examination, if only because those consequences involved altering the ecological conditions of plague occurrences.

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## Recurrent epidemic diseases: plague and the other plagues

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True plagues, the bubonic plagues caused by the microorganism *Y. pestis*, began in late medieval Europe with the extraordinary mortality of 1348. Very little has been written about nonplague epidemics of the fourteenth and fifteenth centuries. Other pestilences seem to have little significance when seen in the chilling light of a scourge that returned at least once every fifteen years and claimed thousands, even millions, of lives. Yet the other plagues significantly shaped both patterns of death and attitudes toward death in Renaissance Italy. Along with plague, epidemics of dysentery, smallpox, and influenza also influenced mortality and morbidity patterns in both plague and nonplague years. They contributed to every observer's notions of who was most likely to die and where, when, and how one might expect death to come. Although the names of the other plagues disappeared from chronicles and diary records once the recurrent threat of bubonic plague became apparent, their symptoms persisted in the records, indiscriminately mingled with those of bubonic plague.

### PERIODIC EPIDEMICITY

One of the most important features of epidemics in the early Renaissance is that many of them returned regularly.<sup>1</sup> Someone surviving to the age of twenty-five would almost certainly have witnessed several different epidemics, although plague was the only disease he or she was likely to view as distinctive and recurrent. In the fourteenth century chroniclers were particularly aware of the recurrence of bubonic plague as a discrete disease and often mentioned at least one past outbreak. Five major plagues struck northern Italy in the fourteenth century and all were described as *anguinaia*, *giandussa*, and *glandulorum*, words that note the prominent inguinal swellings many plague victims show when close to death.