Journal of Interdisciplinary History, XXXVII:3 (Winter, 2007), 371–393.

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A Plague of Plagues: The Problem of Plague Diagnosis in Medieval England

Beginning in 1348, an epidemic of massive proportions struck Western Europe, spreading from Italy northward. Contemporary accounts referred to it as the *pestis* or a *pestilentia generali,* terms translated as plague. Although it has generally been accepted that this great epidemic of the mid-fourteenth century, often referred to as the Black Death, was the plague, this view has been challenged by scholars from various disciplines.

Although the term *plague* has, at times, been used to describe an epidemic of great magnitude, this article will use it in the biological sense. Alexander Yersin identified the bacterial agent of the disease during the Hong Kong epidemic of 1894. First labeled *Pasteurella pestis,* the bacteria would later be renamed *Yersinia pestis* in Yersin's honor. There are three related Yersinia species: *Yersinia pestis, Yersinia pseudotuberculosis,* and *Yersinia entercolitica.* Only the first one poses a major threat to humans. Dating from the early nineteenth century, scholars have equated the great pestilence of the fourteenth century with the Black Death and, by extension, a plague. In this case, the term *plague* was merely a label for a disease of extreme virulence.[1]

In one sense, the question of whether the Black Death was *Yersinia pestis* or some other ailment is a moot point, because only laboratory testing can provide conclusive evidence for a clinical diagnosis. The only available evidence derives from chronicles and medical treatises often fragmentary and even misleading; a few mortality records, often oblique in origin; and scant DNA evidence.

In another sense, the question of whether the outbreak in the fourteenth century was indeed the plague is relevant to under-

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1 Justus Friedrich Carl Hecker (trans. B.G. Babington), *The Black Death in the Fourteenth Century* (London, 1833).

standing the impact of disease on medieval society and to understanding the potential impact of the plague today. The publication of three recent works reopens the question of plague diagnosis in a fashion that warrants re-examination of the evidence.[2]

This article examines this vexed question using England as a case study—England having been the focus of much of the debate concerning the viability of the plague diagnosis. This approach presents both difficulties and advantages concerning evidence. It takes into account medieval descriptions of the plague; early twentieth-century, pre-antibiotic plague epidemics; and various scientific analyses of plague, including efforts to analyze medieval DNA. This interdisciplinary perspective produces better results than a study centered on history alone.

Doubts concerning the diagnosis of plague for the great epidemic of the fourteenth century center on the following points of contention: timing—the speed with which the epidemic spread; mortality rate—the percentage of the population that died during the epidemic, in toto and by gender; seasonality—the months of the year that exhibited the highest mortality; the agents of infection—the manner in which the disease spread; and symptoms—the extent to which the indications at the time match those of *Yersinia pestis*. Critics of the plague diagnosis advance arguments concerning these points to show that the disease of late medieval England could not have been the plague because the evidence does not fit the nomenclature of the plague in the twentieth century.

This study falls into the pro-plague camp, arguing that the epidemic that spread across mid-fourteenth-century England was *Yersinia pestis,* though with an important caveat: The plague did not by itself cause the high mortality in mid- and late fourteenth-century England. No one disease alone could have caused that mortality rate. Plague, however, played a major role in the large number of deaths.

THE PROGRESS OF THE PLAGUE Although not all of the chronicles agree, the Black Death seems to have arrived in England at the end of June 1348 through the port of Melcombe Regis in Dorset. An

2 Susan Scott and C.J. Duncan, *The Black Death: A Biological Reappraisal* (New York, 1984); *idem, Return of the Black Death* (Chichester, 2004); Samuel K. Cohn, Jr., *The Black Death Transformed* (London, 2002).

entry point in the southwest, probably connecting with France, makes the most sense, but both the *Anonimalle Chronicle* and the *Eulogium Chronicon* indicate that the plague arrived via Bristol, whereas Henry Knighton indicated that it was Southampton. The disease spread across the south of England, remaining in Hampshire in early 1349. It had reached London by November before moving north. Robert of Avesbury indicates that the plague cut a swath through London, striking particularly hard from February through May 1349. Late in 1348 and early in 1349, the plague arrived in the north, either by land or by sea through Hartlepool or Newcastle. Gradually the plague spread throughout the north of England, apparently peaking in Lincolnshire in July 1349, bringing high mortality to Lincoln and to the hilly and remote districts of the diocese.[3]

The chronicle accounts are uniform in indicating high mortality, but they do not provide a definition of *high.* Robert of Avesbury and Henry Knighton maintained that death generally occurred within two to four days after symptoms were noticed and indicated that the disease spread quickly. Geoffrey le Baker provides the fullest account, indicating that this first outbreak often killed the young and strong. He also provides one of the few English descriptions of the symptoms, describing what he calls boils and black pustules on various parts of the body.[4]

Evidence for the mortality of the disease is second-hand, pri-

3 Antonia Gransden, "A Fourteenth-Century Chronicle from the Grey Friars of Lynn," *English Historical Review,* LXXII (1957), 274; Edward Maunde Thompson (ed.), *Adae Murimuth continuate chronica Robertus de Avesbury de gestis mirabilibus regis Edwardi Tertii* (Rolls Series, 93, 1889), 406; Ranulf Higden (ed. C. Babington and Joseph Rawson Lumby), *Polychronicon Ranulphi Higden monachi Cestrenis* (Rolls Series, 41, 1866), II, 213; J. M. J. Fletcher, "The Black Death in Dorset (1348–49)," *Proceedings of the Dorset Natural History and Antiquarian Field Club,* XLIII (1922), 1–14. V. H. Galbraith (ed.), *The Anonimalle Chronicle, 1333–1381* (Manchester, 1927), 30; F. Scott Haydon (ed.), *Eulogium Chronicon sive temporis* (Rolls Series, 9, 1858), I, 344; Geoffrey Howard Martin (ed.), *Knighton's Chronicle, 1337–1396* (New York, 1995), 98–99; C. E. Boucher, "The Black Death in Bristol," *Transactions of the Bristol and Gloucestershire Archaeological Society,* LX (1938), 31–46; Thomas Beaumont James, "The Black Death in Hampshire," *Hampshire Papers,* XVIII (1999), 9; E. M. Thompson (ed.), *Avesbury,* 406–407, 409. Richard Lomas, "The Black Death in County Durham," *Journal of Medieval History,* XV (1989), 127–140; A. Hamilton Thompson, "The Pestilence of the Fourteenth Century in the Diocese of York," *Archaeological Journal,* LXXI (1914), 97–154; Edward Augustus Bond (ed.), *Chronica monasterii de Melsa* (Rolls Series, 43, 1868), III, 36–37; A. H., Thompson, "Registers of John Gynewell, Bishop of Lincoln, for the Years 1347–1350," *Archaeological Journal,* LXVIII (1911), 300–360.

4 E. M. Thompson, *Avesbury,* 407; Martin, *Knighton,* 98–99; E. M. Thompson (ed.), *Chronica Galfridi le Baker de Swynebroke* (Oxford, 1899), 98–99.

marily from accounts of clerical vacancies, Inquisitions Post Mortem, and tenancy records. As Hatcher demonstrates, court rolls and manorial records are not without flaws; converting heriots into death rates or replacement rates from inheritance proceedings poses substantial difficulties. Nonetheless, as he also indicates, such records can give an indication of population trends. Death rates varied widely from region to region. In the diocese of York, for example, the average mortality rate among the secular clergy ranged from 27 percent in some areas to a high of 61 percent. The disease hit the secular clergy particularly hard, as it did the inhabitants of such regions as East Anglia. Based on an analysis of the London elite, Megson argues that at least 35 percent of London's population died in the first outbreak and probably more when the mortality of all classes was taken into account.[5]

Death rates among the peasantry are more scarce and more variable. Razi computes a death rate of between 40 and 46 percent, with a peak in June, July, and August 1349, for the parish of Halesowen in Worchestershire. Benedictow argues that using clerical vacancies understates mortality rates; the poor were even more likely to die from the plague than other Englishmen because of greater exposure to the disease, poor living conditions, and weakened immunity from poor diet. He sets England's mortality rate at 62.5 percent, although he does not ascribe all deaths directly to the plague. Benedictow's seeming precision is suspect but as many as half of the English population might have died in the plague outbreak.[6]

The Black Death remained endemic in England throughout the fourteenth century and into the fifteenth century. An outbreak

5 Ole J. Benedictow, *The Black Death* (Woodbridge, 2004), 27, 32, provides an extensive discussion regarding the various sources of medieval mortality information. John Hatcher, "Mortality in the Fifteenth Century: Some New Evidence," *Economic History Review,* XXXIV (1986), 20; E. H. Thompson, "Pestilence in York," 111–113. The situation in York contrasts with a clerical mortality rate of 36% among the clergy of Coventry and Lichfield diocese. R. A. Davies, "The Effect of the Black Death on the Parish Priests of the Medieval Dioceses of Coventry and Lichfield," *Historical Research,* LXII (1989), 87. Robert S. Gottfried, *The Black Death* (New York, 1983), 65; J. L. Fisher, "The Black Death in Essex," *The Essex Review,* LII (1943), 12–20; John Aberth, "The Black Death in the Diocese of Ely: The Evidence of the Bishop's Register," *Journal of Medieval History,* XXI (1995), 275–287; Barbara Megson, "Mortality Among London Citizens in the Black Death," *Medieval Prosopography,* XIX (1998), 125.

6 Zvi Razi, *Life, Marriage and Death in a Medieval Parish* (New York, 1980), 102–104; Benedictow, *Black Death,* 262, 368, 377.

in 1361 was labeled "the children's plague" due to the high death rate of children. Other outbreaks followed in 1374 and 1390 when boys and adolescents were reportedly most affected.[7]

The extant plague treatises came from the continent; several of them seem to have derived from John of Burgundy's work of 1365. John of Burgundy was a physician from Liege who provided a short manual specifying causes of the plague, as well as various forms of treatment. The later accounts based on it are not always forthcoming about symptoms, concentrating more on prevention and remedy. In any case, no specifically English plague treatises appear to exist.[8]

English sources present an imperfect record of the symptoms and progress of the disease known as the great pestilence. Not even the addition of continental accounts provides much direct information about it. The extent of our knowledge is that the disease was perceived as killing large numbers of people in a few months, that it did not discriminate by social class or gender, and that it often took the apparently healthy, though at times it appeared to strike the young or the aged. Contemporary descriptions indicate that the disease spread widely and rapidly, in both urban and rural areas; outbreaks were reported throughout England in less than a year. The first outbreaks were reported in July 1348; by July 1349, the plague was present in Yorkshire and Durham.[9]

THE PLAGUE WAS NOT THE PLAGUE Recent assaults on the identification of the great pestilence of the fourteenth century (and later) with the plague, beginning with Twigg's in 1984, derive, in part, from the observation of outbreaks clearly identified as the plague in the late nineteenth century and in the twentieth century. Although medieval accounts were inconsistent, they all agreed that the 1348/49 epidemic produced a high mortality rate,

7 Martin, *Knighton,* 184–185; Gransden, "Chronicle of the Grey Friars," 275; Galbraith, *Anonimalle Chronicle,* 77; Thomas Walsingham (ed. H.T. Riley), *Historia Anglicana* (Rolls Series, 28, 1863;1864), I, 411; II, 163–164; L. C. Hector and Barbara F. Harvey (eds.), *The Westminster Chronicle, 1381–1394* (New York, 1982), 438–439.

8 Dorthea W. Singer, "Some Plague Tractates (Fourteenth and Fifteenth Centuries)," *Proceedings of the Royal Society of Medicine,* IX (1916), 159–214. In addition to Singer, a more complete account may be found in Anna M. Campbell, *The Black Death and Men of Learning* (New York, 1931).

9 Today, most historians of medicine question the viability of retrospective diagnoses using historical evidence. Accordingly, this article approaches the question from a biological perspective that takes the documentary evidence into account.

from one-third to 60 percent of the population. Examining late nineteenth- and early twentieth-century accounts of plague epidemics in India, China, and Manchuria, the plague critics argue that the plague could not possibly have killed so many people. Today, bubonic plague, the most common variant, normally produces a case fatality rate of 14 percent. Both primary and secondary pneumonic plague, however, are far more lethal, even today. The critics maintain that if plague deaths had been reported only for such cities as London, the mortality rate could have been higher, though not in the range generally alleged for the great pestilence. Because high mortality rates in rural areas were also ascribed to the plague, the critics argue that the later evidence refutes the idea that the fourteenth-century disease was the plague.[10]

Furthermore, Benedict's 1996 examination of the plague in southern China during the nineteenth century indicates that it spread only a few miles a year. This geographical argument again appears to cast doubt on an outbreak of plague in medieval England. Whatever the disease that beset England in the fourteenth century happened to be, it spread too far too fast to be the plague.[11]

Modern bubonic plague tends to be a summer and early fall ailment, with most of its deaths occurring in these months. Pneumonic plague often causes a high death rate in winter months when people are crowded together indoors. However, according to the plague critics, pneumonic plague is mainly a secondary ailment, following upon an earlier bubonic plague outbreak. The

10 Graham Twigg, *The Black Death: A Biological Reappraisal* (New York, 1984); Scott and Duncan, *Biology of Plagues; idem, Return of the Black Death;* Cohn, *Black Death Transformed.* In a review of Twigg's *Black Death,* Gottfried, *Speculum,* LXI (1986), 217–219, finds numerous historical and biological errors. In a review of Cohn's *Black Death Transformed,* Daniel Lord Smail, *Journal of Interdisciplinary* History, XXXIV (2004), 622–623, finds conclusive evidence that the great pestilence was not the plague. On the other hand, Ann Carmichael, "Review Essay: Plagues and More Plagues," *Early Science and Medicine,* VIII (2003), 253–266, is sharply critical of the books by Scott and Duncan and by Cohn. Josiah Cox Russell, "Effects of Pestilence and Plague, 1315–1385," *Comparative Studies in Society and History,* VIII (1966), 464–473, concludes that by 1385, the population of England had declined by 40 to 50%. P. Jeremy Goldberg, "Introduction," in W. Mark Ormrod and Philip G. Lindley (eds.), *The Black Death in England* (Stamford, 1996), 4, indicates a 45% mortality rate; Thomas V. Inglesby et al., "Plague as a Biological Weapon," *Journal of the American Medical Association,* CCLXXIII (2000), 2282. Today, various antibiotic treatments are available for plague (2286).

11 Carol Benedict, *Bubonic Plague in Nineteenth-Century China* (Stanford, 1996); Cohn, *Black Death Transformed,* 133–134.

great pestilence appeared to sweep across England in unstoppable fashion regardless of the season. As Twigg put it, "The seasonal mortality pattern during the Black Death must be one of the most telling arguments against it being bubonic plague." Shrewsbury also stated that pneumonic plague was not a possible explanation and that bubonic plague alone could not have produced the mortality rate of the fourteenth century.[12]

Seasonality is thus linked to the vectors of disease. The plague critics assume that bubonic plague is spread by rat fleas, most notably *Xenopsylla* cheopis. A flea that bites a rat infected with the plague becomes "blocked," unable to satisfy its hunger. If such an increasingly ravenous flea were to bite a human, it would regurgitate the plague bacillius into the new human host. Although no one suggests that fleas were not omnipresent in medieval England, plague critics argue that for such infection to occur, the rats that carry them must be in close proximity with people, but since *X. cheopis* prefers rat, not human, hosts, the rats would have to die before fleas would begin to look elsewhere. The plague critics also argue that the human flea, *pulex irritans,* was unlikely to carry the plague. Furthermore, the chronicles do not mention rats at all, and the most common medieval rat, the black rat, was too timid routinely to share space with humans and thus infect them. The brown rat that now dwells close to humans apparently was not present in the fourteenth century. Moreover, *X. cheopis* is a tropical flea, unlikely to be active enough in winter months to disseminate plague so quickly and extensively as alleged.

Based on a hypothesis of bubonic plague carried by rats, Cohn holds that a disease produced through transmission by fleas would have produced a mid-summer mortality peak. London and Lincoln would confirm it to some extent but not everywhere else.[13]

12 Twigg, *Black Death,* 185; J. F. D.. Shrewsbury, *A History of the Bubonic Plague in the British Isles* (New York, 1970), 123–125.

13 Cohn, *Black Death Transformed,* 43–44, 186; Koenraad Bleukxx, "Was the Black Death (1348–49) a Real Plague Epidemic? England as a Case Study," in Welmer Verbecke et al. (eds.), *Serta devote in memoriam Guillehemi Lourdaux* (Louvain, 1992), II, 64–113; Twigg, *Black Death,* 75; Scott and Duncan, *Biology,* 357. David E. Davis states that rats were not responsible for the spread of the plague in the fourteenth century, contending that most deaths came from pneumonic plague (Davis, "The Scarcity of Rats and the Black Death: An Ecological History," *Journal of Interdisciplinary History,* XVI [1986], 461). Megson, "Mortality among London Citizens," 133; E. M. Thompson, "Pestilence in York,"152; *idem.,* "Registers of Gynewell," 358.

Although the great pestilence appeared to kill indiscriminately, some chroniclers maintained that it targeted people in the prime of life and in later outbreaks, most notably that of 1369, male children and adolescents. Judging from infection and mortality statistics for recent outbreaks, the critics view the reported incidence of disease as all wrong for plague. Because humans have no natural immunity to plague, no particular age group should fall victim to it.[14]

Checking symptoms is one way to diagnose a disease. As noted above, the chroniclers are decidedly unhelpful in this area. Only Geoffrey le Baker provides an extensive description of the symptoms of the disease to rival, say, Boccaccio's description in *The Decameron.* The medieval accounts sometimes speak of buboes, a common plague symptom, but when they do, they describe them as occurring on all parts of the body. The critics take a two-fold approach to this issue—first, that no mention of buboes means no plague and, second, that plague buboes commonly occur in the groin, typically as high as fleas can jump, not in lymph nodes throughout the body. Moreover, multiple buboes do not occur. Critics accept that three to four days from onset to death can be characteristic of pneumonic plague but add that this time span can occur with other ailments.[15]

Cohn accepts the evidence indicating that the great pestilence of the fourteenth century was not the plague, although he advances no alternative. Scott and Duncan also accept it, particularly with the regard to the rapid spread and the mortality rate of the disease, venturing that the great pestilence and later reputed plague outbreaks must have been a hemorrhagic fever, much like ebola. Twigg originally posited anthrax as the likely culprit, although his later work advances no alternative. Even Shrewsbury, who did not dissent from the bubonic-plague theory, had doubts concerning the mortality rate that led him to cite typhus as also responsible for the high mortality rate in medieval England. In light of the collec-

14 Cohn's argument, *Black Death Transformed,* 212, that humans have no immunity to plague is not entirely valid; limited immunity is possible in certain circumstances. Stephen R. Ell, "Immunity as a Factor in the Epidemiology of Medieval Plague," *Reviews of Infectious Diseases,* VI (1984), 866–879; A. Wake, H. Morita, and M. Wake, "Mechanisms of Long and Short Term Immunity to Plague," *Immunology,* XXXIV (1978), 1045–1052.

15 Gionvanni Boccaccio (trans. M. Rigg) *The Decameron* (London, 1921; orig. pub. 1353), I, 5–11; *Chronica Galfridi le Baker,* 98–99; Cohn, *Black Death Transformed,* 64–65, 77–78.

tive evidence, the critics have had the last word that the great pestilence was not the plague.[16]

Yet, arguing from negative evidence, as do all of the critics at some point, is always tricky business. Did the chroniclers, for example, simply assume that rats were present in medieval society, so there was no need to mention them? Nor can the lack of discussion about particular symptoms be taken as solid proof that the plague was not present in medieval society.

The critics also build their case on a comparison between modern plague and the great pestilence. They would appear to be on safer ground on that score, except that the evidence concerning the plague is rapidly expanding, especially as access to knowledge concerning plague in the former Soviet Union increases. Recent advances might raise the question of whether the plague of the third pandemic was similar enough to that of the Middle Ages to permit exact comparisons.

THE COMPLEXITY OF PLAGUE Scholars who assumed that the great pestilence was the plague traditionally devoted scant attention to defending the position. Although Orent's recent discussion of plague includes a defense of the plague hypothesis for the Middle Ages, the most convincing arguments for the plague hypothesis come from recent scientific work that describes the biology of the plague. Starting with the work of Yersin and Kitaro in the late nineteenth century, an increasingly full description of the disease has developed. More recently, microbiologists have examined the genetic structure of *Yersinia*. This work has been augmented by examination of DNA taken from remains of reputed medieval plague victims.[17]

Death Rates and Plague Virulence One of the ostensibly telling arguments against a diagnosis of plague for the fourteenth-century epidemic derives from the mortality rate. Twentieth-century plague outbreaks did not approach the mortality rate of

16 Cohn, *Black Death Transformed,* 247; Scott and Duncan, *Biology,* 356, 384–389; *idem, Return,* 173; *idem,* "What Caused the Black Death?" *Postgraduate Medical Journal,* LXXXI (2005), 317; Twigg, *Black Death,* 213–218; *idem,* "The Black Death: A Population-Wide Infection," *Local Population Studies,* LXXI (2003), 40–52; Shrewsbury, *History of Plague,* 224–225.

17 Wendy Orent, *Plague* (New York, 2004). Orent provides a good introduction to the debate between American and Russian scientists (33–60).

medieval outbreaks. Leaving aside the issue of better medical care, even in outbreaks before antibiotics, death rates during the pestilence of the fourteenth century and those of, say, the plague outbreaks in late nineteenth-century India or China show substantial differences. Shrewsbury tried to escape from the usual mortality rate of bubonic plague by arguing that bubonic plague in England was often accompanied by typhus. Cohn questions this reasoning, indicating that such a synergy was unlikely per se and unlikely to produce the high mortality rates usually reported for medieval Europe if it occurred.[18]

The magnitude of the death rate from the mid-fourteenth-century English epidemic has been disputed because of the difficulties inherent in medieval demographic material and the question of whether the population was already in decline. The second question, which concerns the long-term impact of the famine of 1315 to 1317, is highly controversial. Using records compiled from the parish of Halesowen, Worcestershire, Razi argues that in spite of reverses, especially in the years from 1310 to 1319, the population of Halesowen continued to grow until 1348. Yet, Smith warns that this trend may not have been universal. He contends that replacement rates used by Razi are far from perfect because of the difficulty in obtaining data concerning adult life expectance and mean age at death. As Bolton indicates in his examination of the impact of the first outbreak, the evidence for sustained population decline before 1349 is scant. He settles on a 30 to 40 percent mortality rate for a population between 5 and 6 million. Whatever the cause, scholars agree that the population of England in 1350 had decreased substantially.[19]

The virulence of the plague bacillus is a complex issue. Environmental factors such as temperature and humidity are important. So too is the presence of a host and how the bacillus is

18 Shrewsbury, *History of Plague,* 197, 124–125; Cohn, *Black Death Transformed,* 62.

19 Jim Bolton, "'The World Upside Down': Plague as an Agent of Social Change," in Ormrod and Lindley (eds.), *Black Death in England,* 26; Razi, *Life, Marriage and Death,* 22–34; R. M. Smith, "Demographic Developments in Rural England, 1300–48: A Survey," in Bruce M. S. Campbell (ed.), *Before the Black Death* (Manchester, 1991), 44–47. The debate concerning the use of parish records to obtain medieval demographic information is complex and unresolved. Razi argues for their value, and Smith and Poos are highly critical of them. See Lawrence R. Poos, Razi, and Smith, "The Population History of Medieval English Villages: A Debate on the Use of Manor Court Records," in Razi and Smith (eds.), *Medieval Society and the Manor Court* (New York, 1996), 298–368.

transmitted. At the microbiological level, four determinants of virulence are plasmid-mediated. Genetic alterations have an effect on flea mortality or can either retard or promote the distribution of human antigens. Depending on the type of mediation, some hosts might be able to develop short- or long-term immunity to plague. Ell also points out that people with an iron deficiency are more susceptible to the plague. Contraction of other infections might be a mixed blessing; in certain circumstances, other infections, like salmonella, might confer a measure of immunity to the plague. Because food supplies were more likely to be contaminated in the fourteenth century, salmonella infections were probably more common in medieval Europe than today. Humans infected with *Y. pseudotuberculosis* or *Y. enterocoliticea* might also gain immunity to *Y. pestis*. The microbiology of *Yersinia pestis* is proving to be exceedingly complex and scientists are still unraveling the characteristics of the plague bacillus.[20]

The Evolution of Y. pestis A partial explanation for the high mortality rates of the Middle Ages concerns the nature of *Yersinia pestis*. At present, seventy-six strains of three biotypes (biovars) of *Y. pestis* have been identified. Biovars are local variants of a species that have adapted to specific environmental conditions. Despite genetic differences among them, biovars can interbreed successfully. One biovar may evolve from another in an adaptation to local environmental conditions. Different biovars can lead to epidemics of differing virulence and even differing symptoms. The existence of multiple plague biovars increases the difficulty in understanding the relationship between plague epidemics at different times and places.[21]

20 D. M. Ferber and Robert R. Brubaker, "Plasmids in *Yersinia pestis*," *Infection and Immunity*, XXXI (1981), 839–841; Kathleen A. McDonough et al., "Mutations in the pla Gene of *Yersina pestis* Alters the Course of the Plague-Flea Siphonaptera: Geratophyllidae Interaction," *Journal of Medical Entomology*, XXX (1993), 772–780; Guy R. Cornelius et al., "The Virulean Plasmid of *Yersina*, an Antihost Genome," *Microbiology and Molecular Biology Review*, LXII (1998), 135–152; Brubaker, "Factors Promoting Acute and Chronic Diseases Caused by Yersiniae," *Clinical Microbiology Reviews*, IV (1991), 315–320; Ell, "Immunity as a Factor," 873–876, 868; Wake, Morita, and Wake, "Mechanisms of Immunity," 1049–1050; E.-R. Brygoo and S. Rajenison, "Recerche d' une immunité paraspécifique pour le bacilli de Yersin chez des souris inoculées avec Salmonelles," *Archives de l' Institut Pasteur de Madagascar*, LXI (1972), 13–16.

21 Mark Achtman et al., "*Yersinia pestis*, the Cause of Plague, is a Recently Emerged Clone of *Yersinia pseudotuberculosis*," *Proceedings of the National Academy of Science*, XCVI (1999), 14043–14048; Andrey P. Anisimov, Luther E. Lindler, and Gerald B. Pier, "Intraspecific Diversity of *Yersinia pestis*,' *Clinical Microbiology Reviews*, XVII (2004), 436–442.

The three biovars that researchers have identified are biovar Antiqua—responsible for the fourth-century plague of Justinian; biovar Medievalis—responsible for the fourteenth-century plague; and biovar Orientalis—responsible for the nineteenth-century plague pandemic. Although the three biovars share much of the same genome, there are important differences, and each variant appears to be endemic to a particular region of the world. Biovar Medievalis is endemic to the Caspian Sea region of the former Soviet Union, and biovar Orientalis is endemic to parts of southeast Asia, India, South America, and North America. Until recently much of our information about the behavior of *Y. pestis* has come from studies of biovar Orientalis. The other two biovars are postulated to be older than biovar Orientalis, which is assumed to have evolved from them.[22]

The older plague biovars appear to have evolved from *Yersinia pseudotuberculosis*. The authors of a study of *Y. pseudotuberculosis* report that its virulence can be increased through mutation and that the same scenario can apply to the genetically related *Y. pestis*. The authors of a study of the genetic variability of *Y. pestis* find a great deal of genetic exchange between the older plague strains. This exchange could have led to the appearance, and subsequent disappearance, of a more virulent strain. In any case, the two older strains are assumed to be more virulent than biovar Orientalis.[23]

One view concerning the evolution of diseases, particularly plague, sees them evolving into less virulent strains in order to survive; McNeill espoused it for the plague. A more nuanced view, grounded in evolutionary biology, is that some diseases can evolve into more virulent strains, especially if this process enhances trans-

22 Achtman et al., "Cause of Plague," 14043–14048; Anisimov, Lindler, and Pier, "Intraspecific Diversity of *Yersinia pestis,* 436–442.; V. V. Suntsov and N. I. Suntsova, "Ecological Aspects of the Evolution of the Plague Microbe *Yersinia pestis* and the Genesis of Natural Foci," *Biology Bulletin of the Russian Academy of Sciences,* XXVII (2000), 541–552.

23 Achtman et al., "Cause of Plague," 14043, 14047; P. S. G. Chain, "Insights into the Evolution of *Yersinia pestis* through Whole-Genome Comparison with *Yersinia pseudotuberculosis,*" *Proceedings of the National Academy of Sciences,* CI (2004),13826–13831; Roland Rosqvist, Mikael Skurnik, and Hans Wolf-Watz, "Increased Virulence of *Yersinia pseudotuberculosis* by Two Independent Mutations," *Nature,* CCCXXIV (1988), 525. Christopher Wills, *Yellow Fever, Black Goddess* (Reading, Mass., 1996), 4, provides a more general discussion of the evolution of plague. It is almost necessary for *Y. pestis* to kill its host to be transmitted to a new host via fleabite (Brubaker, "Factors Promoting Diseases Caused by Plague," 320). Vladimir L. Motin et al., "Genetic Variability of *Yersinia pestis* Isolates as Predicted by PCR-Based IS*100* Genotyping and Analysis of Structural Genes Encoding Glycerol-3-Phosphate Dehydrogenase (*glpD*)," *Journal of Bacteriology,* LXXXIV (2002), 1026.

mission. *Y. pestis* seems to have taken the latter path, at least in the Middle Ages. The biovar Orientalis may have evolved into a less virulent strain, lowering the mortality rates in nineteenth- and twentieth-century epidemics.[24]

This evidence makes some important points. First, *Yersinia pestis* is able to mutate readily and has done so in the past. Second, mutations can introduce a more virulent form of the plague. Third, discussion about medieval plague should center more on biovar Antiqua and biovar Medievalis than on biovar Orientalis.

Septicemic Plague and Pneumonic Plague Another factor may have also influenced the mortality of the great pestilence. Bubonic plague is the most common form of *Yersinia pestis,* but two other major forms exist. Septicemic plague is rare, usually occurring as part of a bubonic plague epidemic; even today it is generally fatal. Recent research indicates that septicemic plague could conceivably have contributed to the virulence of the fourteenth-century epidemic, since fleas can carry this form of *Y. pestis* and cause septicemic plague infections independent of bubonic plague infections.[25]

Pneumonic plague infections are generally secondary to infections of bubonic plague but can also occur as a primary infection once an initial case of bubonic plague occurs. In this case, the bacillus passes from one person to another, skipping the flea/rat stage necessary for the transmission of bubonic plague. If treated in timely fashion with massive antibiotics, pneumonic plague is rarely fatal. Before the use of antibiotics, it was often fatal. Benedictow and Scott and Duncan deny that pneumonic plague could have spread rapidly enough and could have infected rural areas extensively enough to have been a factor in the great pestilence. Their dismissal is probably correct for cases of secondary pneumonic plague, in which afflicted people are already too ill and immobile to spread the disease elsewhere. Primary pneumonic plague, however, displays no symptoms during its one-to-three-day incubation. A person with primary pneumonic plague could travel as

24 William McNeill, *Plagues and People* (Garden City, 1976), 173–174; Paul W. Ewald, *Evolution of Infectious Disease* (New York, 1994), 34–55; B. Joseph Hinnebusch, "Bubonic Plague: A Molecular Case History of the Emergence of an Infectious Disease," *Journal of Molecular Medicine,* LXXV (1997), 649.

25 Florent Sebbane et al., "Role of the *Yersinia pestis* Plasminogen Activator in the Incidence of Distinct Septicemic and Bubonic Forms of Flea-Borne Plague," *Proceedings of the National Academy of Sciences,* CIII (2006), 5526–5530.

many as thirty miles during that period, spreading the infection to new places. Coupled with the likelihood of several initial cases of primary pneumonic plague arising from bubonic cases, crossing lines of infection might have increased the likelihood of a wider population being infected. Gani and Leech, for example, have developed a Markov-chain model tracing the relationship of the infection to the spread of the infection to show how delay in dealing with cases of pneumonic plague can lead to its spread.[26]

Outbreaks of pneumonic plague cannot be attributed to any secular property of the plague bacillus. Instead, extrinsic factors, such as environmental conditions, are more likely to determine whether primary pneumonic plague develops. The plague outbreak in fifteenth-century Iceland is alleged to have involved pneumonic plague alone because Iceland had no rats to serve as hosts for bubonic plague-carrying fleas until the late eighteenth century at the earliest. Recent plague outbreaks in Ecuador and Madagascar included several cases of primary pneumonic plague, raising the possibility that the medieval outbreaks, especially the outbreak of 1348/49, included a larger pneumonic component than the plague critics accept.[27]

26 A 1998 outbreak of bubonic and pneumonic plague in Madagascar proved to be somewhat resistant to treatment with antibiotics. R. Migliani et al., "Résurgence de la peste dans le District d'Ikongo à Madagascar en 1998," *Bulletin de la Societe de pathologie exotique,* XCIV (2001), 118; Mahery Ratsitorahina et al., "Epidemiological and Diagnostic Aspects of the Outbreak of Pneumonic Plague in Madagascar," *Lancet,* CCCLV (2000), 113; R. Galimand et al., "Multidrug Resistance in *Yersinia pestis* Mediated by a Transferable Plasmid," *New England Journal of Medicine,* CCCXXXVII (1997), 677–680; L.-T. Wu, "First Report of the North Manchurian Plague Prevention Service," *Journal of Hygiene,* XIII (1913–1914), 237–290; *idem,* "Plague in the Orient with Special Reference to the Manchurian Outbreaks," *Journal of Hygiene,* XXI (1922–1923), 62–76; Bendictow, *Black Death,* 30; Scott and Duncan, *Biology of Plagues,* 70; Raymond Gani and Steve Leach, "Epidemiologic Determinants for Modeling Pneumonic Plague Outbreaks," *Emerging Infectious Diseases,* X (2004), 608–613.

27 Karl F. Meyer, "Pneumonic Plague," *Bacteriological Reviews,* XXV (1961), 249–260; Gunnar Karlsson, "Plague without Rats: The Case of Fifteenth-Century Iceland," *Journal of Medieval History,* XXII (1996), 263–284. Benedictow, *Plague in the Late Medieval Nordic Countries* (Oslo, 1992), 214–227, denies the possibility of a solely pneumonic plague outbreak in Iceland. One reason why a flea-borne aliment might not have had been able to establish itself in Scandinavia was the religious practice of burning the bedstraw of the deceased, which would have destroyed an important flea habitat (Thomas A. DuBois, *Nordic Religions in the Viking Age* [Philadelphia, 1999], 103). Jean-Marc Gabastou et al., "An Outbreak of Plague Including Cases with Probably Pneumonic Infection, Ecuador, 1998," *Transactions of the Royal Society of Tropical Medicine and Hygiene,* XCIV (2000), 387–391; Migliani et al., "Résurgence de la peste," 115–118; Ratsitorahina et al., "Epidemiological and Diagnostic Aspects," 111–113. Gani and Leach, "Epidemiological Determinants," 613, indicate that stochastic effects can produce a significant outbreak of primary pneumonic plague.

Major outbreaks of pneumonic plague occurred in early twentieth-century Manchuria—the first in 1910/11, followed by a second in 1917/18, and a third in 1920/21. These outbreaks are important to plague history because they provided a setting in which the plague struck in a pre-antibiotic society, yet one in which scientists were able to study the symptoms and reach a diagnosis that was verified in a laboratory. Comparison of the Manchurian outbreaks with the fourteenth-century outbreak provides further support for the argument that plague was present in fourteenth-century England.

The three Manchurian plague outbreaks began as bubonic plague, but secondary, and possibly primary, pneumonic plague also ensued. Wu Lien Teh (Wu Liande) indicated that tarbagans, or marmots, played a large role in providing the initial hosts for *Y. pestis,* inferring that plague spread from tarbagan fleas—*Ceratophyllus silatievi.* A tarbagan can survive nine to twelve days once infected, allowing the plague to spread readily from one colony to another and thence to trappers, who spread the disease to a wider human population. Both Chinese and Russians succumbed, especially the railroad workers crowded together in close living quarters. The average mortality rate was as low as 2 percent in some towns but as high as 25 percent in others. The second plague episode produced a slightly lower mortality rate than the first because authorities were better prepared to fight it. By the time of the third outbreak, the Chinese authorities had learned enough to lower death rates even further.[28]

The plague spread easily from town to town along the railroad. Its spread was halted to some extent when passengers became subject to routine examination while boarding and exiting trains, and those infected were immediately quarantined. The close living conditions of the infected railroad workers also helped to check mortality, since individuals with the plague passed it to other infected men, allowing the epidemic to burn itself out more

28 Wu Lien-Teh (Wu Liande), *Plague Fighter* (New York, 1959), 81, 83, 95; Wu, "Plague in the Orient," 63, 66; *idem,* "The Second Pneumonic Plague Epidemic in Manchuria, 1920–21," *Journal of Hygiene,* XXI (1922–23), 265–266. Wu Liande's account of the 1910/11 outbreak should now be supplemented by Mark Gamsu, "The Epidemic of Pneumonic Plague in Manchuria 1910–1911," *Past & Present,* 190 (2006), 147–183. Wu Liande's figures, particularly for the third epidemic, must be viewed with caution; he had an incentive to underestimate the death rate in order to show the efficacy of the Plague Prevention Service, which he headed. Gamsu, "Epidemic of Pneumonic Plague in Manchuria," 162.

quickly. Once the infected railroad workers died, no new hosts were available for the disease. The highest death rates occurred in winter months, usually in January and February, contrary to the summer mortality peaks of bubonic plague epidemics. Typical pneumonic plague symptoms, such as drowsiness and dizziness, a 102° F temperature lasting about twenty-four hours, and a cough usually occurring within two to three days, were common. The cough was dry at first but soon accompanied by a "liquid, frothy sputum" tinged with blood. This stage held the greatest danger of infection. If it was left untreated, death often ensued.[29]

The Manchurian plague outbreaks show how easily the disease could spread, as well as how virulent it could be during the winter months. Manchuria's death rates did not reach those of fourteenth-century England; its medical care and its ability to quarantine the sick and reduce casualties stemming from complications was more advanced. As in Ecuador and Madagascar, plague might have developed as primary cases or as secondary infections. An outbreak of bubonic plague complicated by primary and secondary pneumonic plague would have been even more disastrous.

Other Diseases Other ailments might also have contributed to the mortality rate of the plague outbreak in the fourteenth century. Pneumonic plague can be misdiagnosed as several diseases, among them influenza, anthrax, Q fever, and tularemia. Indeed, that public health officials initially diagnosed an outbreak of pneumonic plague in Oakland, Calif., in 1919 as influenza is hardly surprising in light of the massive influenza epidemic then sweeping the country. Although most influenza epidemics are mild, the 1918/19 epidemic caused a high death rate worldwide. A similar influenza epidemic could certainly have swept across Western Europe in 1348/49, adding to the plague death toll.[30]

A plague epidemic was likely to cause many secondary deaths. If a family breadwinner died, a food shortage for the family was likely to ensue. Children, in particular, were more susceptible to the secondary effects of a plague epidemic, leading to an increased

29 Wu, "Plague in the Orient," 66, 67; *idem,* "Second Pneumonic Plague Epidemic," 265, 271.

30 Mohamad Daya and Yoko Nakamora, "Pulmonary Disease from Biological Agents, Anthrax, Plague, Q. Fever, and Tularemia," *Critical Care Clinics,* XXI (2005), 761; Jack D. Poland and David T. Dennis, "Diagnosis and Clinical Manifestations," in *Plague Manual: Epidemiology, Distribution, Surveillance and Control* (Geneva, 1999), 48; Gani and Leech, "Epidemiologic Determinants," 610.

mortality rate for them. Even if plague did not kill directly, it left its survivors in a weakened condition, subject to other ailments.[31]

Y. pestis Hosts One tenet of the anti-plague argument is the seasonality of medieval plague. Modern bubonic plague outbreaks tend to peak in late summer and early fall, corresponding with the activity of the rat flea *X. cheopis*. Cohn maintains that northern European outbreaks, like those in England, should have peaked in mid-summer because of the weather patterns. Instead, on the basis of not always complete evidence, epidemics seem often to have peaked in the late fall when temperatures would have been too low for *X. cheopis* to survive.[32]

Yet, even though *X. cheopis* is considered the classic plague vector, 31 of 1,500 types of fleas have been identified as plague vectors, including the northern rat flea, *Nosopsyllus fasiatus*. The human flea, *pulex irritans* (which is not confined just to humans) can also carry the plague bacillus. Numerous *Pulex irritans* were found in the houses of Ecuadorian plague victims in 1998, lending credence to the argument that human fleas are plague carriers. Despite substantial disagreement, some scholars maintain that *Pulex irritans* is an important plague vector. Ell goes so far as to argue that *P. irritans* fleas were a major source for plague transmission from one human to another, negating a need for rats as flea carriers. *Y. pestis* has mutated over time, and one mutation may have involved the vectors of disease and its hosts. If *P. irritans* was the carrier or if the outbreak had become primary pneumonic plague, a large number of cases in the late fall or even in February through March, as reported by Robert of Avesbury, is possible. Moreover, because the blockage of fleas, and thus their ability to affect other beings, decreases at higher temperatures (greater than 28° to 30° C), plague epidemics subside in conditions of high temperature and low humidity.[33]

31 Benedictow, *Black Death,* 22–24, points out the numerous secondary effects on children stemming from an epidemic disease, such as loss of care and malnutrition.

32 Cohn, *Black Death Transformed,* 186.

33 Robert D. Perry and Jacqueline D. Fetherson, "Yersinia pestis: Etiological Agent of Plague," *Clinical Microbiology Review,* X (1997), 53, 51; Burroughs, "Sylvatic Plague Studies," 387, 393; Gabastou et al., "Outbreak of Plague, Ecuador," 389; Orent, *Plague,* 121; M. Baltazard and B. Seydian, "Enquête sur les conditions de à peste au Moyen-Orient," *Bulletin of the World Health Organization,* XXIII (1960), 157–167. Jean-Noël Biraben, *Les homme et la peste en France et dans les pays européens et méditerranéens* (Paris, 1975/76), I, 13, argues that human fleas played an important role in the transmission of plague in the Middle Ages. In a study of sylvatic plague, Burroughs,"Sylvatic Plague," 391, found that *X. cheopis* was the ideal

Cohn maintains that an epizootic of rats would have been a step toward the migration of blocked fleas to humans but that the chroniclers mention no massive rat deaths. The chroniclers, however, generally paid so little attention to the plague that their failure to mention rats is hardly a surprise. Because of the differences in various strains of *Y. pestis,* some types of rats can be hosts for plague-carrying fleas without dying themselves. Most rats die quickly when infected by the plague. Yet, as Perry and Fetherstone point out, animals infected with plague just prior to hibernation survive the winter to provide hosts the following spring. Such a situation would have helped the plague to survive and reappear throughout the fourteenth and fifteenth centuries. Hinnebush also suggests that microgenetic changes that affect the routes of plague transmission can have an impact on the appearance and decline of human disease agents. If *Pulex irritans* was the plague-carrying flea, as Ell indicates, or if primary pneumonic plague was at work, as Karlsson indicates was the case in Iceland, the question of dead rats becomes a moot point.[34]

Medieval accounts of the great pestilence are not forthcoming about symptoms. A few accounts describe how long it took for the disease to run its course, but these accounts must be treated with care. The authors did not intend to provide an exact chronology of the disease. Over-reliance on chroniclers' time frames, as Cohn and Scott and Duncan do on occasion, to argue that the medieval disease moved more rapidly than modern plague may be unwarranted. In addition, the numerous English ports made it easy for plague to gain a foothold throughout the island and then move steadily inland. As the critics point out, other descriptions of symptoms—such as large boils, shortness of breath, spitting of blood, or high fevers—could just as easily have been any number of other ailments. Descriptions of physical symptoms neither confirm nor disprove the presence of plague, particularly given the scant details that the chronicles provide.[35]

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plague carrier but that other varieties of fleas, including *P. irritans,* could become blocked and transmit the plague bacillus. Ell, "Interhuman Transmission of Medieval Plague," *Bulletin of the History of Medicine,* LIV (1980), 502–503; E. M. Thompson, *Avesbury,* 467.

34 Cohn, *Black Death Transformed,* 43; Anisnov, Lindler, and Pier, "Intraspecific Diversity of *Yersinia pestis,*" 443, 446; Perry and Fetherson, "Etiologic Agent of Plague," 54; Hinnebusch, "Bubonic Plague," 651; Ell, "Interhuman Transmission of Medieval Plague," 502–503; Karlsson, "Plague without Rats," 281–284.

35 Martin (ed.), *Knighton's Chronicle,* 98–99; E. M. Thompson, *Avesbury,* 409.

The Promise of DNA Evidence The discussion above neither confirms nor disproves the presence of plague in fourteenth-century England. The medieval symptomatic evidence is so weak that the great pestilence could have been almost any illness. As Cunningham says, symptoms are not the final evidence of the presence of a disease; only laboratory analysis can provide a clinical diagnosis.[36]

One promising avenue has recently opened up through polymerase chain reaction (PCR) analysis of dental pulp from corpses found in reputed medieval plague cemeteries in southern France. Researchers extracted DNA from the pulp of teeth found in cemeteries where plague victims were known to have been buried, subjected it to DNA sequencing using PCR, and compared the results with samples known to come from *Y. pestis* DNA. Southern France is not England, but because the plague critics maintain that plague was nonexistent throughout Europe, the presence of plague DNA at this location is telling, even if not fully convincing. However, subsequent excavations in five other reputed European sites failed to produce *Yersinia pestis*-specific DNA in the laboratory. The debate swirls around the difficulty of avoiding contamination of the samples and of sequencing DNA from the past and around the cemeteries selected for sample acquisition. At present, the arguments and counter-arguments regarding the presence of *Yersinia pestis*-specific DNA from medieval samples do not produce a conclusive argument on either side.[37]

36 Andrew Cunningham, "Transforming Plague: The Laboratory and the Identity of Infectious Disease," in *idem* and Perry Williams (eds.), *The Laboratory Revolution in Medicine* (New York, 1992), 209, 242.

37 Michel Drancourt et al., "Detection of 400-Year-Old *Yersinia pestis* DNA in Human Dental Pulp: An Approach to the Diagnosis of Ancient Septicemia," *Proceedings of the National Academy of Sciences,* XCV (1998), 12637–12640; Didier Raoult et al., "Molecular Identification by 'Suicide PCR' of *Yersinia pestis* as the Agent of Medieval Black Death," *Proceedings of the National Academy of Sciences,* XCVII (2000), 12800–12803; Raoult and Drancourt, "Cause of the Black Death," *Lancet: Infectious Diseases,* II (2002), 459; Drancourt and Raoult, "Molecular Detection of *Yersinia pestis* in Dental Pulp," *Microbiology,* CL (2004), 263–264. PCR—polymerase chain reaction—is a technique that enables researchers to replicate accurately and efficiently copies of a segment of nucleic acid (DNA or RNA) for further comparison and study. Care must be taken to avoid contaminating the sample at all stages. James Wood and Sharon DeWitte-Avina, "Was the Black Death Yersinial Plague?" *Lancet: Infectious Diseases,* III (2003), 327–328; M. Thomas Gilbert et al., "Absence of *Yersinia pestis*-specific DNA in Human Teeth from Five European Excavations of Putative Plague Victims," *Microbiology,* CL (2004), 341–354; Michael B. Prentice, Tom Gilbert, and Alan Cooper, "Was the Black Death Caused by *Yersinia pestis?*" *Lancet: Infectious Diseases,* IV (2004), 72; Carsten M. Pusch et al.,

A PLAGUE OF PLAGUES Barring conclusive laboratory evidence, which is unlikely, the plague debate will have to rely on often circumstantial medieval evidence, fragmentary and conflicted demographic records, examination of more recent outbreaks clearly identified as the plague, and laboratory analysis of *Yersinia pestis*. Naphy and Spicer are correct that a clinical identification is not possible, and, as they say, even if it were, it would not alter the enormous impact that the great pestilence had on medieval Europe. Nonetheless, determining more accurately the disease(s) that struck fourteenth-century England can provide valuable information about medieval people's relationship to the natural world, as well as about the manner in which epidemic disease can spread in different times and places.[38]

The plague critics are successful on one level. They have made a convincing case that the great pestilence exhibited characteristics different from those of the third plague pandemic. Accounts, such as Zeigler's or Gottfried's, which blithely accepted the epidemic of the fourteenth century as the plague, largely on the basis of the word *plague* appearing in descriptions of the disease, must now be treated with care. Medieval authors used the word plague to signify a disease of great proportions with a high mortality rate, not in a clinical sense.

The plague critics' arguments are helpful in indicating the possibility that more than one epidemic disease was present in fourteenth-century England. As Twigg indicated, anthrax exhibits plague-like symptoms and could have produced high mortality rates. It was unlikely, however, to have spread rapidly. Typhus carried by human lice and by rat fleas could also have produced fever, delirium, and inflammation. Scott and Duncan propose that the great pestilence was some sort of hemorrhagic fever. Although their position is intriguing, little evidence supports it as yet, especially when compared to the evidence supporting the plague thesis. Part of the difficulty concerning other potential diagnoses is that plague, anthrax, Q fever, tularemia, and influenza all display

"Yersinial F1 Antigen and the Cause of the Black Death," *Lancet: Infectious Diseases,* IV (2004), 484–485. Tests for the F1 antigen in seventeenth-century DNA samples raise the question of the accuracy of PCR results. Alan Cooper and Hendrik N. Poinar, "Ancient DNA: Do It Right or Not at All," *Science,* CCLXXXIX (2000), 1139, provide a useful approach for testing premodern DNA.

38 William G. Naphy and A.W. Spicer, *The Black Death* (Stroud, 2000), 56.

similar pulmonary symptoms that can lead easily to a mistaken diagnosis.[39]

Although people were more likely to identify influenza correctly, it could produce severe fevers and, in some cases, the spitting of blood, thus emulating the plague. The possibility arises, though it will be difficult to explore, that a highly virulent influenza epidemic was coupled to the first plague epidemic, increasing the mortality rate. As the rapid and deadly progress of the 1918/19 epidemic indicates, influenza can move rapidly through a large area, causing a high mortality rate in the right circumstances.[40]

All of these arguments for a disease other than the plague are predicated on the thesis that the great pestilence was a mystery disease much like the English "sweate" of the sixteenth century. Although the sweating sickness was not the killer that the great pestilence had been, it clearly confounded the medical practitioners of the day. One explanation is that its causative agent was similar to an aborvirus, perhaps a hantavirus. When subjected to closer scrutiny, the hantavirus diagnosis falls apart. Subjecting Scott and Duncan's hemorrhagic plague diagnosis to the same level of scrutiny might well produce a similar result.[41]

Once an epidemic started, contemporaries were likely to conjoin it and other diseases into one great pestilence. In a sense, preclinical disease was socially constructed. People reduced all illnesses with common symptoms into a single diagnosis. Moreover, they tended to attribute all deaths, even accidental deaths, to one cause, thereby inflating the totals ascribed to the plague. On occasion, notably 1369, famine may have helped to produce higher death rates.[42]

39 Scott and Duncan, *Biology of Plagues,* 384–389; Daya and Nakamora, "Pulmonary Disease from Biological Agents," 761.

40 A standard account of the 1918/19 influenza epidemic remains Alfred W. Crosby, *America's Forgotten Pandemic* (New York, 1985).

41 Alan Dyer, "The English Sweating Sickness of 1551: An Epidemic Anatomized," *Medical History,* XLI (1999), 382–383; Mark Taviner, Guy Thwaites, and Vanya Gant, "The English Sweating Sickness, 1485–1551: A Viral Pulmonary Diease?" *Medical History,* XLII (1998), 96–98; Thwaites, Taviner, and Gant, "The English Sweating Sickness of 1485–1551," *New England Journal of Medicine,* CCCXXXVI (1997), 580–582; Eric Bridson, "The English 'Sweate' (*Sudor Anglicus*) and Hantavirus Pulmonary Syndrome," *British Journal of Biomedical Science,* LVIII (2001), 1–6.

42 Regarding fourteenth-century Florence, Ann Carmichael, *Plague and the Poor in Renaissance Florence* (New York, 1986), 26, observes that the interaction between clinical information and epidemiological information is as difficult for lay persons today to understand as it

The chroniclers' silence about the medical aspects of the disease, except for a sentence or two indicating that many people died, is surprising. They often had their own agendas, and providing extensive medical information was not among them. The lack of medical knowledge concerning epidemic disease made it difficult for medical writers to provide exact information, particularly when they may have believed that the great pestilence was caused by sin, by Jews, or by bad air. Epidemic disease in a time before laboratory diagnosis, when any disease was liable to cause death, did not merit the scrutiny that it does now. People often were more concerned about the business of living than the business of dying. Even the great influenza epidemic of 1918/19 that swept across the globe and killed more than 20 million people had passed from the public consciousness by the mid-1920s.

What is the present state of knowledge about the great pestilence of 1348/49? First, the outbreak of the great pestilence in England did not match all of the characteristics of the third plague pandemic in India or China during the nineteenth century or in Vietnam or the United States during the twentieth century. This difference does not mean, however, that the medieval epidemic was not plague. Second, it is impossible conclusively to identify in clinical fashion the causative agent of the great pestilence. Third, other tentative identifications of the great pestilence—anthrax, hemorrhagic fever, or typhus—are even less likely than plague.

Until proven otherwise, the great pestilence will remain the plague, but with an important qualification. The high mortality rate of the fourteenth century was often the result of the plague and other diseases. Both bubonic and pneumonic plague contributed to the fourteenth-century epidemic. Evidence from the early twentieth-century Manchurian epidemic and other instances suggests that primary pneumonic plague may have been a major killer in 1348/49, accounting for the speed with which the epidemic spread across the countryside. The openness of England to the entry of plague-carrying rats from several points also made it easier for the disease to spread quickly.

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was for people in the Middle Ages. See also *idem,* "Contagion Theory and Contagion Practice in Fifteenth-Century Milan," *Renaissance Quarterly,* XLIV (1991), 229. Bolton, "World Upside Down," 27

Recent biological analysis indicates that *Yersinia pestis* is capable of mutation that affects its virulence and that it has mutated into at least three major varieties. It is reasonable to suppose that the biovar Medievalis strain of *Y. pestis* from the Caspian Sea region mutated into an unusually virulent strain, probably one capable of finding a host other than *X. cheopis*. It is also probable that the strain of plague that invaded medieval Europe eventually mutated into a less virulent one.[43]

The argument, however, that the plague was often credited with deaths caused by other diseases and that other epidemic diseases may have also been at work during the great pestilence will be difficult to prove or disprove. Although a final conclusion about the medieval plague might not be forthcoming, an approach to it that combines historical sources and scientific work will benefit scholars more than the one-sided arguments of either plague champions or their critics.

43 Anisimov, Lindler, and Pier, "Intraspecific Diversity of *Yersinia pestis*," 434–464; Achtman et al., "*Yersinia pestis*, the Cause of Plague," 14043–14048.