Black Death Bodies

SHARON N. DEWITTE
University of South Carolina
dewittes@mailbox.sc.edu

MARYANNE KOWALESKI
Fordham University
kowaleski@fordham.edu

Abstract: The fourteenth-century Black Death was one of the most important and devastating epidemics in human history. It caused or accelerated important demographic, economic, political, and social changes throughout the Old World and has therefore been the subject of scholarly research in a variety of fields, including history, anthropology, demography, and molecular biology. In this paper, we examine the Black Death (specifically, the first and second outbreaks of fourteenth-century plague, c. 1347–1351 and 1361–1362) from bioarchaeological and historical perspectives, focusing on attempts to reconstruct mortality patterns and addressing the questions: Who died in England during the Black Death? How did they die, where and when? We evaluate how historical and bioarchaeological sources are uniquely informative about these questions and highlight the limitations that are associated with each type of data. The combination of the two bodies of evidence, when possible, can provide insights that are not possible when each is analyzed in isolation.

Introduction

The fourteenth-century Black Death was one of the most important epidemics in human history, as it caused or accelerated important demographic, economic, political, and social changes throughout the Old World. This epidemic was devastating not only because of its extremely high mortality levels, but also because the deaths it caused were concentrated within a brief period. In the four years that the Black Death was at its height in Europe (between 1347 and 1351), the epidemic killed 30 to 60 percent of the population, amounting to tens of millions of people. This essay argues that a full understanding of the epidemiology of this devastating plague—that is, its levels of mortality; the distribution of deaths by age, sex, wealth, and location; and the susceptibility of victims to disease—can only be attained by bringing together archaeological and documentary evidence. We focus on the people who succumbed to the Black Death in medieval England and make a deliberate effort to compare and contrast

not only what the different types of documentary and material evidence say, but also the analytic questions and methodological approaches that different disciplines have adopted to discuss plague during the fourteenth century, particularly the first (1348–1349) and second (1361–1362) waves of the Black Death.

The Black Death is increasingly recognized as a semi-global phenomenon, likely starting in East Asia and spreading throughout Central Asia, Europe, and North Africa.[1] It is often described as the second of three known pandemics caused by *Yersinia pestis*, the bacterium associated with plague. According to this view, the First Pandemic was the Plague of Justinian, which began in the sixth century C.E., with recurrent outbreaks for about two centuries thereafter.[2] The Black Death of 1347–1351 and subsequent visitations of the plague, including the Great Plague that hit London in 1665, constituted the Second Pandemic. The Third Pandemic began in China in the late eighteenth century, spread from there throughout much of the world, and ended by the mid-twentieth century.[3] Outbreaks of plague continue to this day, although modern antibiotics and public health measures have greatly reduced the mortality rate and spread of the disease. Scholarly disagreement about whether the Black Death was the same bubonic plague of the Third Pandemic or some other disease has raged for some time, but the recent discovery of *Y. pestis* DNA in the dental pulp of victims of the mid-fourteenth-century plague has settled this question.[4] We are still unable to explain fully, however, the different clinical manifestations and epidemiological patterns of *Y. pestis*, including the significantly higher mortality and faster spread of medieval plague, and the likelihood that medieval plague did not follow the same transmission path from animal host to flea to humans often identified in modern bubonic plague.

The documentary sources employed by historians to analyze the Black Death have been known for some time. They include chronicles, which offer direct statements from contemporaries about the symptoms, pace, and mortality rates of the plague, as well as appointments of parish priests, wills, and especially manorial records, which reflect the experience of the rural majority. In the last twenty-five years, a new source of material evidence has become available. Bioarchaeology investigates human skeletal remains excavated from archaeological sites to determine the ages at which people died, as well as their sex, health, diet, migration, experience of interpersonal violence, and other behaviors. The bioarchaeological analysis of skeletal remains is vital for a complete understanding of life in the past because it can yield data often missing from historical documents and can be employed to test assumptions and inferences based on documentary evidence.[5]

How the Black Death Killed: Symptoms, Spread, and Duration

Most of what we know about the symptoms of the Black Death comes from medieval chroniclers, many of them eyewitnesses, and from plague treatises written years and often decades after the first outbreak of plague. Their descriptions are broadly similar, pointing to fever, headache, lethargy, dark patches or small black pustules on the skin, and buboes or swollen lymph glands in the armpit, groin, or neck. Other symptoms included nausea, diarrhea, vomiting, and the coughing up of blood.[6] In *The Decameron*, Boccaccio describes the "plague-boils" of the Black Death as "certain swellings, either in the groin or under the armpits, whereof some waxed of the bigness of a common apple, others like unto an egg, some more and some less."[7] The English chronicler Geoffrey le Baker wrote that victims "were tormented by boils which broke out suddenly in various parts of the body, and were so hard and dry that when they were lanced hardly any liquid flowed out. . . . Other victims had little black pustules scattered over the skin of the whole body."[8] Medieval observers stressed how quickly the disease progressed, noting that death usually came within three days.[9]

Scientists have identified several clinical forms of plague in humans: bubonic, septicemic, pneumonic, gastrointestinal, and pharyngeal.[10] The bubonic form is predominant in modern outbreaks of plague. Its main symptoms are exactly those described by medieval sources: headache, chills, nausea, and fever followed by hard and increasingly painful swellings near lymph nodes in the neck behind the ears, armpits, and groin.[11] Septicemic and pneumonic plague usually develop secondarily to bubonic plague, but can rarely occur as primary diseases if the plague bacillus infects the bloodstream or lungs, respectively, without first infecting the lymphatic system.[12] All forms of plague are different clinical manifestations of infection with *Y. pestis*, and the proportions of infected individuals who die from different forms of untreated plague are high, ranging from 30 to 60 percent for bubonic and septicemic plague and approaching 100 percent for pneumonic plague.[13]

Several medieval accounts recognized the distinct symptoms of pneumonic plague, which included respiratory distress such as shortage of breath and coughing up of blood or sputum, as well as a quick death.[14] To explain the apparent greater virulence of historic plague compared to modern plague, some scholars have suggested that the Black Death was an outbreak of pneumonic plague, which has very high case-fatality rates and can spread from person to person.[15] However, although pneumonic plague can develop in an area after bubonic plague has taken hold,[16] evidence from the pre-antibiotic era suggests that most primary cases fail to transmit the disease to other people.[17] Fatality rates for those who contract pneumonic plague are very high, but during a modern pre-

antibiotic outbreak of bubonic plague, pneumonic plague accounted for only a small fraction of total cases.[18] At present, therefore, it is not clear whether attributing the Black Death to pneumonic plague satisfactorily explains the patterns of the medieval epidemic.

Medieval documentary sources also offer information on the spread, seasonality, and duration of the disease. Chroniclers identified the first English victims during the summer of 1348 in port towns, emphasizing its arrival on ships from the Continent, though they disagree on the exact dates (sometime between late June and late September) and which ports it struck first (Bristol, Southampton, or Melcombe).[19] Some chroniclers offer more precise trajectories for individual locales, as did Robert of Avesbury, a London clerk who wrote that the plague arrived in London on All Saints Day (1 November 1348), was especially virulent from Candelmas (2 February) to Easter (12 April), and ended around Pentecost (31 May 1349).[20] A few medieval observers venture an estimate of the Black Death's duration in England in noting that the 1348–1349 plague "lasted for a whole year" in England although at least one tracked it at two years.[21] There were no such estimates by chroniclers for subsequent visitations of the plague in fourteenth-century England.[22]

Lists kept in bishop's registers recording the appointments of parish priests to newly vacant posts are a more reliable source for studying the chronological and geographical distribution of the Black Death in England. The dates and location of these "institutions to benefices," offer a rough guide to the pace and spread of the plague, indicating that around 45 percent of the beneficed parish clergy died in the course of just over twelve months.[23] Various problems, particularly in accounting for the lag between the incumbent's actual death (which is rarely known) and the appointment of a new priest, mean that these studies tend to underestimate the speed at which the Black Death spread.[24] The timing of the plague in particular villages can also be discerned in scattered references to the "pestilence" in the dated court rolls of specific manors, which sometimes contain countable references to the large numbers of heirs who came to court to claim properties from deceased tenants.[25] Historians employ wills to date when the plague raged in specific towns, such as London, though here too the lag between the actual death of the will-maker and the enrollment of the will can skew estimates of the progression of the disease, while the exclusion of all but the wealthy who left wills narrows the cohort to a very small and biased sample of the population.[26]

Despite their drawbacks, these sources can provide considerable data on when and exactly where the Black Death raged, phenomena that have largely been inaccessible to bioarchaeologists and archaeologists, though recent work that counts pottery fragment as a proxy for human demographic trends represents a promising approach.[27] By combining evidence

from a range of complementary records, such as has been done recently for several towns and regions in medieval England, scholars can offer a fairly comprehensive outline of the chronology of the 1348–1349 epidemic.[28] The documentary sources agree that the Black Death in fourteenth-century England was most virulent in the summer months; outbreaks of modern bubonic plague also show marked seasonality.[29] These sources also indicate the speed at which the Black Death spread in England, taking about a year and a half to cover the length of the country and averaging about 1 km per day, a pace possible because plague probably entered the British Isles via several different seaports from where it spread inland (Figure 1).[30] In contrast, modern bubonic plague moved much more slowly. It took over one hundred years for bubonic plague to spread from Yunnan Province in southwest China to Hong Kong in the late eighteenth and nineteenth centuries.[31]

This contrast raises questions about how plague was transmitted in the medieval period. Medieval chroniclers such as Baker and others articulated a fear of contagion; they believed that the disease could be spread merely by touching the clothing or bedding of the sick, going so far as to declare that anyone "who touched the dead or the sick were immediately infected themselves and died."[32] Medieval people, however, had no understanding of how the disease was contracted or transmitted by different vectors (the organisms such as fleas or ticks through which the disease was carried). Until recently, most scientists have assumed that medieval plague was transmitted in the same fashion as modern bubonic plague, which is a zoonosis, a disease among animals spread by an insect vector.[33] *Y. pestis* is an obligate parasite that can reproduce only by exploiting its mammalian or insect host, so bubonic plague in humans is dependent upon the cyclic transmission between animal hosts and the ectoparasites that live and feed on them.[34] Humans often fall ill when they come into contact with infected animals and are bitten by the animal's fleas or other ectoparasites. This dependence on animals for transmission has been used to explain why modern bubonic plague spread so slowly, but ignores the potential effect of human transportation of infected animals on how quickly and widely the disease spread. It also assumes that plague is primarily spread by rats, an assumption undermined by the biased nature of the investigations of early twentieth-century plague in India that have influenced studies since, and more recent evidence that over 200 animals can serve as hosts for plague.[35] Recent scientific research has shown that *Y. pestis* can be transmitted to humans via not only the rat flea, but also by other insect vectors such as the cat flea, human flea, human body louse, and human hair louse.[36] The application of mathematical models of plague transmission suggests that lice may have been involved in the spread of the disease during the Black Death.[37] In other words, much of what people

read about the epidemiology of the Black Death—particularly that it was spread by rats and fleas—has not been conclusively established using evidence from the medieval period but rather reflects the imposition of a rat-centric view of plague dynamics that is problematic even for modern plague.[38] Further work needs to be done to clarify the transmission dynamics during the medieval period.

How Many the Black Death Killed

Historians have expended enormous effort estimating the mortality rates of the Black Death in 1348–1349. English historians have, in fact, probably done more to advance data collection of these rates than scholars elsewhere because of the variety and quality of documentary material that survives for England, particularly for peasants, who comprised over 80 percent of the total population.[39] Although estimates of mortality rates calculated for well-off groups such as wealthy tenants-in-chief of the king and beneficed clergy are useful, mortality assessments for the rural population bring us closer to how many people the Black Death actually killed.[40] Such estimates for the rural population are not unproblematic given the innovative methodologies that scholars have imposed on recalcitrant sources to extract mortality rates. One method involves counting the number of heriots or death-duties paid by peasants on a manor during a specified period, and comparing the number with the total manorial tenants liable to heriot, an exercise that has produced death rates for many different settlements across England (Table 1).[41] Another method relies on a customary head-tax due from a particular class of manorial tenants. On the manors of Glastonbury Abbey, landless men who represented the poorest sector of village society (called *garciones* and over the age of 12) had to make an annual payment (called *chevagium*, or head tax), which allows mortality rates to be calculated by subtracting the total for the plague year from the total for the previous year (Table 1).[42] A similar tax was the tithing penny due on some Essex manors from all male villeins (serfs) over age 12; the total sums collected, at the rate of one or one-half penny per head, survive for many years, thus theoretically allowing changes in the size of a village's population to be determined.[43] Unfortunately, few Essex tithing lists survive for all the years of the Black Death, so the mortality rates are calculated as the percentage decline in those paying tithing penny between a three- to five-year period.[44] Detailed analyses of manors with especially good court rolls and account rolls have also allowed historians to calculate plague mortality rates of at least 40 percent in Halesowen and over 50 percent on the small but heavily populated manor of Hakeford Hall in the Norfolk village of Coltishall.[45]

The mortality rates derived from these different methods show variations ranging from 20 to 100 percent (Table 1), but the overall average

mortality of adult males in the countryside was about 50 percent. Because landless males had a generally higher mortality rate of 56–57 percent according to the drop in annual payments made by the poor *garciones* on Glastonbury Abbey estates (distributed over four counties), and because mortality rates from heriots exclude women, children, and landless men, there have also been attempts to push this overall figure above 60 percent to account for the many poorer peasants and women left out of the heriot and tithing-penny figures.[46] Whether the figure is 40, 50, or even higher than 60 percent, the Black Death of 1348–1349 had an extremely high mortality, in stark contrast to modern plague mortality, which is rarely more than 2–3 percent even in the absence of antibiotic treatment.[47] During the late 1890s and early 1900s, bubonic plague killed approximately one million people per year in India.[48] Though this is a disturbingly high number of people, it does not approach the high levels of mortality that occurred during the Black Death in terms of the *proportion* of the population killed.

Questions about why the Black Death of 1348–1349 was so deadly remain unanswered by both historians and scientists, although there is some hope that bioarchaeology and paleomicrobiology may provide solutions one day.[49] We now have clear evidence that the Black Death was caused by *Y. pestis* and in particular by a strain of the bacterium ancestral to most extant strains. Comparison of *Y. pestis* DNA from skeletons of individuals who died during the Black Death with modern references has so far failed to reveal any significant functional differences in sections of the genome associated with virulence.[50] Comparisons between *Y. pestis* DNA from different historical epidemics suggest that the First (the Plague of Justinian in the sixth century) and Second (the Black Death) Pandemics represented independent emergences of plague from animals into humans and that the strain responsible for the Plague of Justinian might have subsequently gone extinct.[51] It is also noteworthy that a recent successful analysis of a tooth sample from a Black Death victim, which identified dozens of pathogenic, environmental, and microbiomic taxa, indicates that we have the capability to examine the diversity of pathogens that were circulating at the time of the Black Death.[52] We can thus potentially test the hypothesis that co-infection with multiple pathogens, and resulting within-host competition, drove mortality during the Black Death to extreme levels. If molecular studies of Black Death remains do not reveal any features of the pathogen (or pathogens) that can fully explain the epidemiology of the epidemic, we must look carefully at the characteristics of the human populations that first encountered these pathogens in 1348 to understand the behavior of the fourteenth-century disease.

Recent research suggests that health in general declined before the Black Death emerged in the fourteenth century, which might have contributed to the extraordinarily high mortality of the epidemic.[53] Bioar-

chaeological analysis of human skeletons dating from A.D. 1000 to 1250 reveals declining life expectancies in London across this period, during which there were repeated famines in England resulting from climatic changes.[54] Since longevity (life expectancy) is a commonly used measure of a population's general health,[55] these demographic trends estimated from the skeletal data suggest a decline in health prior to the Black Death that might have exacerbated vulnerabilities to a new disease and rendered the epidemic more deadly than it would have been had it struck a more resilient population. These studies have not yet included skeletal samples from late thirteenth century through the mid-fourteenth century, so it remains to be seen whether these declines in health were sustained right before the Black Death.

Who the Black Death Killed: Wealth, Status, Sex, Locale, and Age

Together historical and bioarchaeological sources can offer a great deal about differential mortality, that is, how wealth, position, age, sex, or frail health might have influenced an individual's susceptibility to plague. These data not only improve our understanding of how the Black Death shaped demographic, economic, political, and social conditions of post-plague medieval populations, but they can also clarify our understanding of the dynamics of emerging infectious diseases today, particularly how the human context can shape the epidemiology and evolution of these diseases.

Contemporary documents tell us precious little about who was infected by the Black Death, although most medieval chroniclers claimed that the Black Death of 1348–1349 was indiscriminate in attacking those of all conditions, age, and sex. Only a few contemporaries noted contrary sentiments, such as: "few, virtually none, of the lords and great men died in this pestilence."[56] Other documentary sources, such as royal genealogies and inquisitions post mortem for well-off tenants-in-chief of the crown, do show that there were very few casualties among the English royal family, that the higher nobility had a mortality rate of perhaps 4.5 percent in 1348 and 13 percent in 1349, and that the mortality rate among wealthy landowners may have been a relatively low 27 percent.[57] English bishops also had a better chance of survival (fewer than 18 percent died in 1348–1349) than the secular and monastic clergy, about 45 percent of whom died in the Black Death.[58] The clerical mortality rates were on average lower than those of the rural majority. Wealth may have mattered since the poorest sector of rural society, those who could neither lease nor own land (marked as "LL," "landless men" in Table 1), died in proportionately greater numbers than more well-off peasants.

Until recently, neither historians nor bioarchaeologists could offer firm evidence that the Black Death of 1348–1349 hit one sex harder than the other, though some medieval chroniclers of later plagues, such as that of 1360–1361, believe the plague killed more men.[59] The documentary evidence relates largely to men, although one study of rural manors in the northern county of Durham found that 52 percent of female tenants died in the Black Death compared to 50 percent of the total tenant population[60] More convincing evidence of greater female mortality during outbreaks of late medieval plague, including the Black Death, has been found in a recent analysis of thousands of death duties (called mortmains) paid in the Hainaut region of the southern Netherlands. Mortmain payments differed from heriots in taxing not the landholding of a tenant, but individuals, including women and young adults who were not yet heads of households. The study found an overall average sex ratio for mortality of 107 in the period 1349–1450, but 94 in plague years and 89 during the Black Death. Further tests on these data suggest that women were disproportionately affected by plague because they biologically had less resistance to the disease than men, rather than because of their differential exposure given their greater numbers in towns or the time they spent at home as caregivers.[61] This exciting new evidence will hopefully stimulate more historical and bioarchaeological studies to help resolve the conflicting evidence about sex-selective mortality during periods of plague.

Even less certainty surrounds the extent to which the Black Death was more virulent in urban than rural areas. One leading historian of plague maintains that rural regions suffered greater mortality than towns, but most historians argue the reverse based on solid data showing high mortality in pre-modern towns and their reliance on migration to maintain and grow their populations.[62] Unfortunately, there is no systematic source of documentary data to measure urban plague mortality such as we have for the clergy, wealthy landowners, and rural dwellers. The urban evidence tends to be indirect and peculiar to individual towns. A comparison of tax returns from Canterbury, for instance, shows that two-thirds of the tax-paying population vanished between 1346/7 and 1351/2, while monastic records show that one-half of the tenants of Rochester Priory in Rochester died and two-thirds of those residing at Durham Priory in Durham.[63] Wills have also been exploited to study trends in urban mortality. A careful study of wills in medieval London, for example, shows that the mortality rate in 1349 was seventeen times higher than in other years, which translates into a mortality rate of about 51 percent if we accept the average of a 3 percent annual mortality rate calculated for early modern populations.[64] It is likely that the actual mortality rate in London was higher, given the relative wealth of will-makers.

Finally, even more debatable is whether age made any difference to susceptibility to plague. Chroniclers occasionally declared that plague targeted the young or old or those in the prime of life, but age was rarely mentioned in accounts of the first wave of the plague in 1348–1349.[65] There is some questionable evidence that older men faced higher risks of mortality compared to younger men[66] (hardly surprising given their greater frailty), which bioarchaeological research has shown did make individuals more susceptible to the plague.

Bioarchaeology can provide especially useful insights into selective plague mortality because it is possible to determine the sex, age, and general health of skeletal remains, even though it cannot establish whether an individual died from plague without using molecular methods, most of which are expensive and destructive. A good deal of this work is statistical and involves the application of methodologies designed to compensate for the limitations of bioarchaeological data. As with historical documents, not every person who lived in the past is included in the observable bioarchaeological record. Certain individuals might be excluded from the samples analyzed because they were buried in unexcavated areas or because their bones were badly preserved, a particular problem for the smaller and thinner bones of infants, who are very often under-enumerated in skeletal samples.[67]

Partial excavation of a site, which is a common result of time and financial constraints, can produce biased skeletal samples if burial locations within the cemetery were influenced by factors like age, sex, socioeconomic standing, or disease status. Another problem relates to the traditional methods of aging skeletons, which tend to underestimate older adult ages,[68] or to group them into broad age intervals, such as "50+ years."[69] These methods are not capable of making distinctions between, for example, a 50- and a 90-year-old, two ages with very different morbidity and mortality regimes. Compounding the problems with age estimation is the standard use of mean age-at-death or life tables to infer mortality patterns, both of which implicitly assume the population under consideration was stationary: that is, closed to migration, with zero population growth, and unchanging age-specific mortality and fertility rates.[70] Departures from these assumptions—which occur, for example, if the population was growing or contracting—means that skeletal age patterns might not accurately represent past trends in mortality patterns.[71]

Human bones often contain specific signs (such as lesions, pitting, thickening, and cracks) that reflect exposure to physiological stress in general or particular health problems, such as malnutrition, tuberculosis, previously broken bones, and other conditions. These health problems can shed light on selective mortality in terms of what might have made certain individuals more susceptible to death from plague. This type of

analysis cannot be taken too far, however, because not all of the genetic, physiological, social, and environmental variations affecting frailty (that is, susceptibility to disease and death) leave discernable signs on the skeleton.[72] Reconstructing health from skeletons is further complicated because skeletal lesions, which form in response to disease, malnutrition, or trauma, might not always indicate poor health. Lesions could actually point to relatively good health because they reflect the ability of individuals to survive the associated stressor long enough for the lesions to form. Similarly, the absence of a lesion might suggest relatively poor health if individuals without them were so frail that they quickly succumbed to stressors and died before lesions had a chance to form.[73] In the absence of other supporting evidence, we thus cannot interpret the presence or absence of skeletal lesions in a straightforward manner, a problem articulated as the Osteological Paradox.[74]

Many of the published bioarchaeological analyses of the fourteenth-century plague have been done using skeletons buried in the East Smithfield cemetery in London.[75] The documentary history of the East Smithfield cemetery indicates that it was used only during the Black Death, so most if not all of the over 600 individuals recovered from the cemetery were victims of the 1348–1349 epidemic.[76] Sharon DeWitte previously compared East Smithfield to a non-epidemic, pre–Black Death assemblage to assess whether risks of death during the Black Death varied by age, sex, or pre-existing health condition (frailty). The results indicate that elderly adults faced higher risks of death compared to younger adults during the Black Death, as occurs under normal mortality conditions.[77] The mortality pattern for children in East Smithfield is less clear, however, perhaps as a result of the small sample of infants and children available from the cemetery.[78] Assessment of sex differences in mortality during the Black Death failed to reveal a significant difference in risk between men and women during the epidemic.[79] Finally, DeWitte and colleagues found that the Black Death disproportionally killed people with skeletal lesions compared to those without them.[80] These results suggest that people who experienced physiological stressors before the Black Death were subsequently more likely to die during the epidemic compared to their peers who lacked the lesions, indicating that the epidemic was selective with respect to frailty.

In summary, the combined evidence from the historical and bioarchaeological sources indicates that wealth, status, health and perhaps age affected risks of death during the Black Death. The elderly, those who were impoverished, and those who had suffered relatively poor health faced higher risks of death during the epidemic than their younger, wealthier, and healthier peers. Neither source of information for England has yielded substantial evidence that one sex fared better than the other, although recent work on the southern Netherlands offers strong evidence that plague

more negatively affected women than men. Further documentary work on towns would be especially welcome to address the extent to which urban mortality might have exceeded rural mortality; this question is more difficult to answer through bioarchaeology since plague cannot be diagnosed from skeletal remains without expensive DNA analysis and since the population at risk cannot be determined in cemeteries. Bioarchaeology does, however, have the potential in the future to clarify the risk of mortality for the youngest individuals in the population during the Black Death once additional Black Death cemeteries are excavated, thereby increasing the available sample sizes for analysis. Isotopic analysis of carbon and nitrogen will also be able to reveal more clearly how diet or nutritional stress, two factors that can produce skeletal lesions, affected risks of death during historical plague epidemics.

Who the Black Death Killed in 1361–1362

In contrast to their remarks that the Black Death of 1348–1349 killed indiscriminately, chroniclers were far more emphatic in describing the age- and sex-specific mortality of the plague of 1361–1362, a topic that has received relatively little attention from historians and bioarchaeologists alike. This "second pestilence," differed first of all in targeting the young. Medieval English chroniclers called it "the mortality of children" or commented on the "death of children and adolescents," or that "children and adolescents were the first to die, and then the elderly," and they claimed that "both rich and poor died, but especially young people and children." Secondly, the chroniclers emphasized the second pestilence's effect on men, saying that it "devoured men rather than women"; noting, for example, that "for this year the mortality was particularly of males." Several spoke of both age and gender, labeling it "a mortality of men, especially of adolescents and boys, and as a result it was commonly called the pestilence of boys."[81] This visitation of the plague also seems to have focused more on southern England, and was said to have started in London where it was so devastating in May and June of 1361 that the royal courts had to be adjourned, a timing confirmed by the peak in wills during the summer.[82] This was the plague referred to in the well-known graffiti inscription in the Hertfordshire church of Ashwell: "wretched, fierce, violent . . . [only] the dregs of the populace live to tell the tale."[83] Although the 1361–1362 plague was the second deadliest in medieval England, we know far less about it in part because the chroniclers had less to say about it, but mostly because historians have not fully exploited what sources are available.

Analysis of the institutions to benefices for the better-off secular clergy shows a mortality rate of about 10–30 percent in 1361–1362, compared to their 45 percent average in 1348–1349.[84] In 1361–1362, evidence from heriots show that the mortality rate on the Hampshire manor of Bishop's

Waltham was 13 percent and 12–18 percent on the Dorset manor of Titchfield, while court roll references to deaths at the manor of Halesowen indicate a mortality rate of a little over 9 percent.[85] The evidence from wills in London suggests a mortality rate of almost 20 percent.[86] This range of mortality rates in the "second pestilence" appear insignificant when compared to the death rates in 1348–1349, but they were higher than for any subsequent visitation of the plague.[87] There is less evidence that wealth—and the better diet, health, and sanitary conditions associated with it—had the impact in the 1361–1362 plague that it may have had in the earlier plague. Although references to the lower mortality of the rich are not particularly prominent among observers of the 1361–1362 plague, inquisitions post mortem suggest that the morality rates of the nobility and tenants-in-chief were relatively higher than in 1348–1349 and closer to the mortality levels suffered by the general population.[88]

We have some suggestive evidence about the sexual bias of the 1361–1362 plague in a study of entry fines paid by new villein tenants on the estate of the bishop of Winchester, which included almost sixty manors located mainly in Hampshire, but also in the West Midlands and West Country.[89] The fines provide a good record of all post-mortem property transactions, while also offering enough evidence on the parties involved to establish the relationship between the deceased and the individual who took over the property. The fines reveal a rise in the numbers and proportions of women inheriting in 1361–1362 compared to previous years, and a marked downturn in the percentage of sons and other male kin who inherited, suggesting greater mortality for men, since women inherited only in the absence of male heirs. It might also be telling that the percentage of widows who inherited rose the most, while potentially younger women (such as sisters, daughters, nieces, and granddaughters) did not inherit any more than they had in earlier decades, implying that older women may have better survived the second plague.

Bioarchaeological analysis reveals some different answers to the question of who was killed by the second wave of the Black Death in 1361–1362. For this essay, DeWitte analyzed data from burials associated with the Cistercian Abbey of St. Mary Graces in London, which overlies the earlier East Smithfield cemetery. The St. Mary Graces assemblage includes burials within the Abbey church and chapels and a larger lay cemetery; within the cemetery, there are nearly 200 burials attributed to the 1361 plague.[90]

DeWitte assessed mortality patterns in the second outbreak of plague, specifically whether it was selective with respect to sex, age, and frailty. The age-at-death distributions (Table 2) from the two sets of plague burials in 1348–1349 (East Smithfield cemetery) and in 1361–1362 (St. Mary Graces) do not differ significantly from each other, but each is different from the normal mortality (non-plague) distribution from St. Mary Graces.[91] There

is some evidence that children and people in the prime of life were more likely to die during outbreaks of plague since the plague burials had more people below the age of 40 and fewer adults above the age of 70 than the non-plague burials in St. Mary Graces. The non-plague burials might be biased toward older individuals, however, since they include individuals who were buried within the Abbey of St. Mary Graces itself, such as monks and wealthy lay people. Of note, there is a higher proportion of children (under age 10) in the 1361 plague burials compared to both the 1348–1349 and the non-plague burials (Figure 2). The 1361–1362 burials contain 10 percent more children than the non-plague burials, and nearly 6 percent more children compared to the earlier Black Death burials (though the latter difference is not statistically significant).

The higher proportion of children in the 1361–1362 plague burials is consistent with the descriptor "mortality of children" for the second outbreak of plague, which could reflect age-related patterns of vulnerability following the Black Death.[92] Individuals who survived the Black Death (and were above the age of 10 during the second wave) might have acquired immunity to resist the disease whereas those born since the epidemic lacked acquired immunity and thus were susceptible to the disease.[93] The (statistically) non-significant difference in age distributions between the two plague burials might mean that the second outbreak was not more lethal to children than the first. These results, however, could also be an artifact of relatively small sample sizes or the poor preservation of infants and young children that affects skeletal assemblages in general. To further examine mortality patterns, the age-at-death data from the 1361 plague burials were used to estimate the parameters of a model of the age pattern of adult mortality.[94] The results indicate that the risk of death increased with adult age during the second outbreak of plague (Figure 3), as was previously observed for the Black Death and for conditions of non-epidemic mortality.[95]

Though there are more adult males than adult females in the 1361–1362 burials (52.7 percent vs. 47.3 percent), as is true for the 1348–1349 and non-plague burials, results from an assessment of differences in risk of mortality between males and females during the 1361 plague do not corroborate the historical evidence that men may have been more susceptible to the 1361–1362 plague.[96] The 95 percent confidence interval for the estimated effect of sex on adult mortality includes both positive and negative values, which indicates a lack of significant difference in risk of death during the 1361 plague between men and women, as was earlier reported for the Black Death and pre–Black Death normal mortality samples. It is possible that (as noted for previous analyses of East Smithfield), this result might be an artifact of the way in which the effect of sex was modeled. Nonetheless, this result raises the question of how we might resolve discrepancies

between historical and bioarchaeological evidence. The most conservative approach is to, at least temporarily, assign greater weight to the positive evidence (provided in this case by the historical evidence) and allow the tension between the two lines of evidence to stimulate further analysis in the event that additional data become available.

There was, however, good evidence that the 1361 outbreak of plague was selective for frailty as was the case during the Black Death.[97] For this essay, DeWitte focused on the risk of death associated with the presence of periosteal new bone formation, a lesion that can form in response to stimuli that traumatize the tissue that covers the surfaces of bone, which in turn results from local or systemic infection or inflammation associated with a variety of factors.[98] The analysis suggests an elevated risk of mortality for those with the skeletal lesion among the 1361 plague victims, as has been found for normal mortality samples and among Black Death victims.[99] In summary, the bioarchaeological evidence indicates that age and frailty, but not sex affected risks of mortality during the second wave of plague. These results demonstrate the ability of bioarchaeological data to confirm or refute historical evidence as well as expand the lines of inquiry beyond those possible with existing historical evidence.

Conclusion

Scholars of medieval plague have only recently combined bioarchaeological, historical, and paleomicrobiological information to achieve a fuller understanding of the epidemiology of the disease and its relationship to modern plague. Until a few years ago when *Y. pestis* was definitively identified as the bacterium responsible for both medieval and modern plague, efforts to compare historical and scientific data on the symptoms, pace, duration, and mortality of plague tended to generate huge controversies in which some scholars adamantly claimed the Black Death was not bubonic plague while others just as ardently argued the opposite. New discoveries in bioarchaeology and paleomicrobiology have in particular moved these debates toward a resolution, and are opening up more fruitful avenues regarding how health, age, social status, wealth, and sex may or may not have affected chances of survival.

Chronicles, along with data drawn from institutions to benefices for the secular clergy, inquisitions post mortem for well-off landowners, wills, and, most importantly, manorial sources about the rural majority reveal much about the temporal and spatial spread of the Black Death, as well as mortality rates, especially the far higher death rates in the 1348–1349 plague compared to the second pestilence in 1361–1362. These historical sources also point to how wealth and social status influenced risks of mortality during the Black Death, and how age and sex might have affected risks during the plague of 1361. The bioarchaeological evidence uniquely

reveals that health status affected risks of death during both outbreaks, which historical evidence simply cannot reveal in any systematic way. By combining the historical and bioarchaeological data, it becomes clear that risks of mortality during at least the first two outbreaks of medieval plague varied along several dimensions including social status, wealth, age, and health status.

Neither bioarchaeological data nor historical evidence provides a flawless perspective on patterns of mortality during medieval plague epidemics, or more generally of life, health, and death in the past. Historical data from the medieval period tend to be biased toward wealthy men, blinding us to the experiences of the majority of the population that was at risk of disease and death during the Black Death. Medieval chroniclers and other record keepers did not take note of many variables modern epidemiologists consider essential to understanding disease processes, so we do not have access to details about the age, sex, and health status of medieval plague victims and survivors from written records.

Cemeteries, on the other hand, tend to be more inclusive than written records, yielding bioarchaeological data on women, children, and the poor, and thus allowing for reconstructions of health and death for a broader sample of once-living populations. Bioarchaeological samples are, however, subject to their own sources of bias, such as differences in preservation, inaccuracies in age estimation, and incomplete excavation. Further, there are relatively few burials clearly associated with medieval plague epidemics, particularly from rural areas, and social status is largely invisible in the burials that do exist.

Ideally, an interdisciplinary approach to Black Death epidemiology allows the strengths of each line of evidence to compensate for the weaknesses in the other. The combination of the bioarchaeological and historical evidence, when possible, can provide insights that are not possible when each is analyzed in isolation. As shown here with respect to sex differences in plague mortality, this approach might highlight discrepancies in the patterns revealed by the two lines of evidence. Such tensions will ideally motivate further study.

Appendix

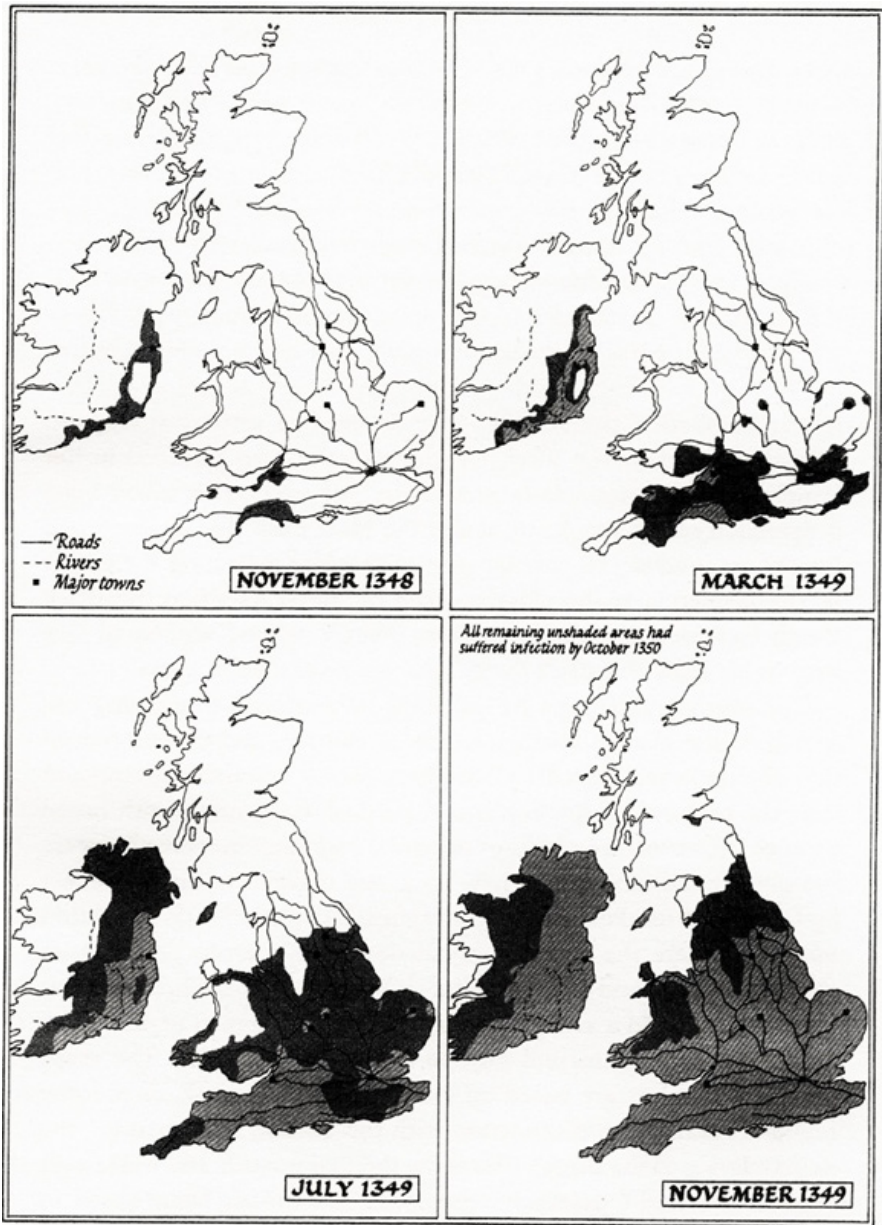

Figure 1: The spread of the Black Death in England, 1348–1349
The dark shading indicates when the Black Death was prevalent in a region; light shading marks areas where the plague had waned; no shading points to areas to which the plague had yet to spread. From *The Scourging Angel: The Black Death in the British Isles*, by Benedict Gummer (The Bodley Head and Vintage, 2009). Reprinted by permission of The Random House Group Limited.

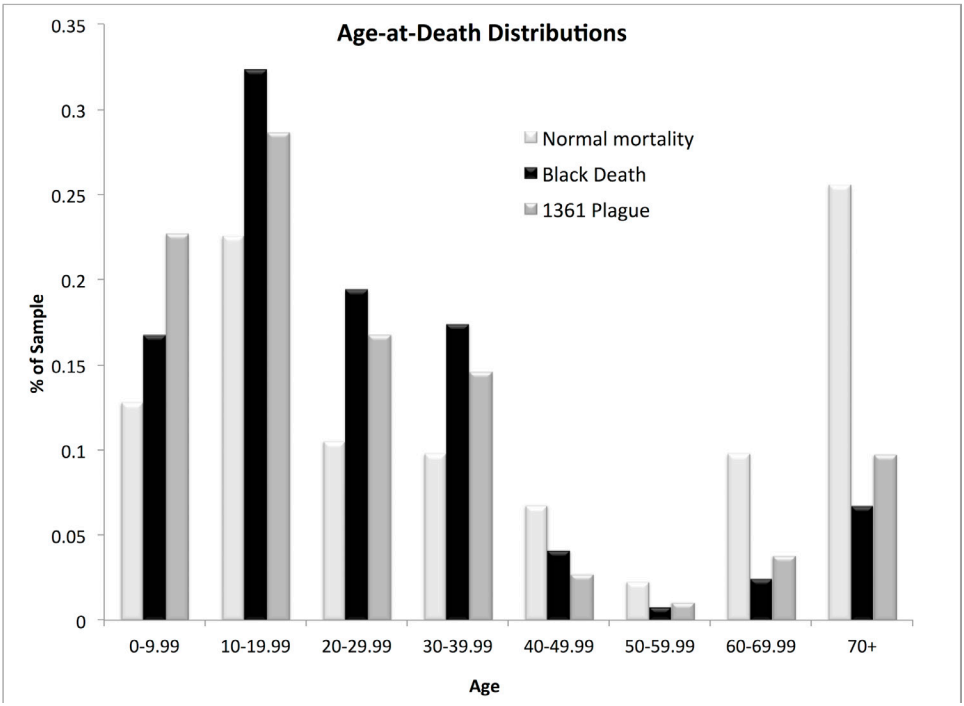

Figure 2: Age-at-death distributions from the East Smithfield Black Death (1348-9) cemetery, and St. Mary Graces plague of 1361-2 and non-plague (normal mortality) burials. Produced by Sharon DeWitte.

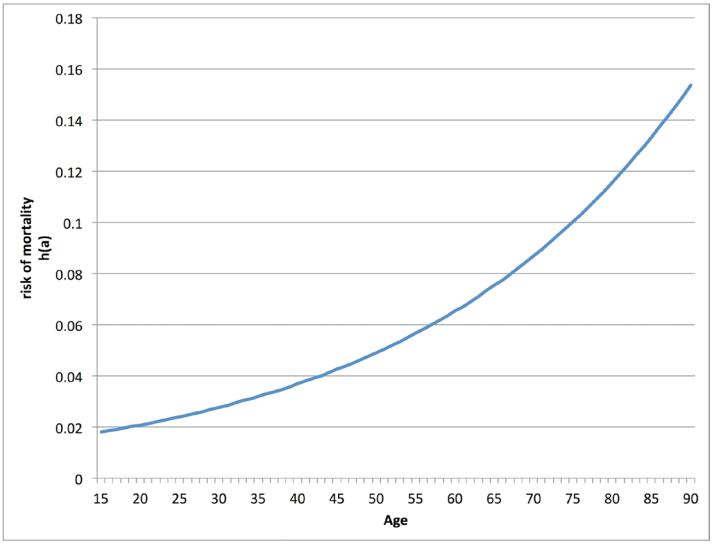

Figure 3 : Hazard of adult mortality for the 1361-2 plague burials in St. Mary Graces cemetery. Produced by Sharon DeWitte.

Table 1: Mortality rates of English rural tenants (mostly men), 1348–1349

Manors	County	Mortality rate	Source type	Source
SOUTH-WEST				
Calstock	Corn.	42%	S: Heriots	Hatcher, 105
Climsland	Corn.	62%	S: Heriots	Hatcher, 105
Marnhull	Dorset	36%	M: Head-tax (LL)	Ecclestone, 26
Ashcott	Soms.	44%	M: Head-tax (LL)	Thompson, 168
Batcombe	Soms.	54%	M: Head-tax (LL)	Ecclestone, 26
Ditcheat	Soms.	54%	M: Head-tax (LL)	Ecclestone, 26
Greinton	Soms.	47%	M: Head-tax (LL)	Thompson, 168
Ham	Soms.	42%	M: Head-tax (LL)	Ecclestone, 26
Mells	Soms.	58%	M: Head-tax (LL)	Ecclestone, 26
Pilton	Soms.	61%	M: Head-tax (LL)	Ecclestone, 26
Street	Soms.	55%	M: Head-tax (LL)	Thompson, 168
Walton	Soms.	52%	M: Head-tax (LL)	Thompson, 168
Walton	Soms.	61%	M: Head-tax (LL)	Ecclestone, 26
Badbury	Wilts.	76%	M: Head-tax (LL)	Ecclestone, 26
Christian Malford	Wilts.	66%	M: Head-tax (LL)	Ecclestone, 26
Damerham	Wilts.	64%	M: Head-tax (LL)	Ecclestone, 26
Downton	Wilts.	66%	E: Heriots (incl. some LL)	Ballard, 213
Grittleton	Wilts.	63%	M: Head-tax (LL)	Ecclestone, 26
Idmiston	Wilts.	50%	M: Head-tax (LL)	Ecclestone, 26
Kington	Wilts.	56%	M: Head-tax (LL)	Ecclestone, 26
Nettleton	Wilts.	48%	M: Head-tax (LL)	Ecclestone, 26
SOUTH-EAST				
Alresford	Hants.	59%	M. Heriots	Arthur, 61
Alverstoke	Hants.	95%	M. Heriots	Arthur, 61
Ashmanworth	Hants.	71%	M. Heriots	Arthur, 61
Beauworth	Hants.	80%	M. Heriots	Arthur, 61
Bishop's Waltham	Hants.	65%	E: Heriots	Titow, 70
Cadland	Hants.	100%	M: Heriots	Watts, 27
Cheriton	Hants.	63%	M. Heriots	Arthur, 61
Corhampton	Hants.	61%	M: Heriots	Watts, 27
Crawley	Hants.	71%	M. Heriots	Arthur, 61
Ecchinswell	Hants.	68%	M. Heriots	Arthur, 61

Manors	County	Mortality rate	Source type	Source
Funtley	Hants.	100%	M: Heriots	Watts, 27
Gosport	Hants.	100%	M. Heriots	Arthur, 61
Hambledon	Hants.	94%	M. Heriots	Arthur, 61
North Waltham	Hants.	54%	M. Heriots	Arthur, 61
Stubbington	Hants.	41%	M: Heriots	Watts, 27
Swanwick	Hants.	64%	M: Heriots	Watts, 27
Titchfield	Hants.	72%	M: Heriots	Watts, 27
Wallsworth	Hants.	62%	M: Heriots	Watts, 27
Woodhay	Hants.	82%	M. Heriots	Arthur, 61
EAST				
Cottenham	Cambs.	49%	M: Heriots	Ravensdale, 198
Oakington	Cambs.	64%	M: Heriots	Page, 121
Dry Drayton	Cambs.	48%	M: Heriots	Page, 121
Chatham Hall	Essex	45%	S: Tithing penny	Poos, *Rural Society*, 107n20.
Fingrith	Essex	63%	S: Heriots	Fisher, 13–20
Great Waltham	Essex	44%	S: Tithing penny	Poos, *Rural Society*, 107n20
High Easter	Essex	54%	S: Tithing penny	Poos, *Rural Society*, 107n20
Margaret Roding	Essex	26%	S: Tithing penny	Poos, *Rural Society*, 107n20
Hakeford Hall	Norf.	56%	S: Heriots	Campbell, "Population Pressure," 96
Walsham-le-Willows	Suff.	45–55%	S: Heriots	Lock, 320–01
MIDLANDS				
Ashbury	Berks.	55%	M: Head-tax (LL)	Ecclestone, 26
Buckland	Berks.	60%	M: Head-tax (LL)	Ecclestone, 26
Brightwell	Berks.	29%	E: Heriots	Ballard, 207–08
Kibworth Beauchamp	Leic.	67%	S: Tithing penny	Postles, 47
Kibworth Harcourt	Leic.	40%	C: Tithing penny	Postles, 46
Witney	Oxon.	66%	E: Heriots	Ballard, 195–96, 208
Cuxham	Oxon.	65%	C: Heriots	Harvey, 135
Higham Ferrers	Northm.	57%	S: Court roll list	Groome, 310
Halesowen	Worc.	40%	M: Entry fines	Razi, 103

Manors	County	Mortality rate	Source type	Source
NORTH				
Aycliffe	Durham	61%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Billingham	Durham	45%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Burdon	Durham	64%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Dalton-le-Dale	Durham	69%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
East Rainton	Durham	29%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Fulwell	Durham	56%	M: Heriots	R. Lomas, 129
Harton	Durham	45%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Hedworth	Durham	27%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Jarrow	Durham	78%	M: Heriots	R. Lomas, 129
Middlestone	Durham	70%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Monk Hesleden	Durham	44%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Monkton	Durham	21%	M: Heriots	R. Lomas, 129
Monkwearmouth	Durham	67%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Moorsley	Durham	45%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Nether Hedworth	Durham	72%	M: Heriots	R. Lomas, 129
Newton Bewley	Durham	48%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)

Manors	County	Mortality rate	Source type	Source
Newton Ketton	Durham	42%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
North Pittington	Durham	56%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Over Hedworth	Durham	36%	M: Heriots	R. Lomas, 129
South Pittington	Durham	52%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
South Shields	Durham	50%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Southwick	Durham	53%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
West Rainton	Durham	34%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Westerton	Durham	61%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Westoe	Durham	56%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Wolviston	Durham	47%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Wallsend	North.	43%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)
Willington	North.	20%	M: Heriots	Benedictow, *Black Death*, 367 (citing R. Lomas)

Note: This table only includes figures calculated directly from 1348–1349 evidence for actual deaths, in which the population at risk was at least twenty people. Unless noted otherwise, these figures relate almost entirely to male customary tenants, that is, villein or "unfree" peasants who owned enough land to feed a family, usually at least fifteen to eighteen acres.

Sources: Under Source Type, the initial letter refers to the owner of the manor. C = College or other corporate owner; E = Episcopal; M = monastic; S = Seigniorial; LL = landless men.

County abbreviations: Berks. (Berkshire); Cambs. (Cambridgeshire); Corn. (Cornwall); Hants. (Hampshire); Leic. (Leicestershire); Norf. (Norfolk); North. (Northumberland); Northm. (Northamptonshire); Oxon. (Oxfordshire); Soms. (Somerset); Suff (Suffolk); Wilts. (Wiltshire); Worc. (Worcestershire).

Table 2: Age-at-death distributions from the East Smithfield Black Death cemetery and the St. Mary Graces cemetery (1361–1362 plague and non-plague). Percentages refer to proportion of total sample.

Age at death		Non-plague		Black Death		1361–1362 plague
0–9.99		17 (12.78%)		82 (16.8%)		42 (22.7%)
10–19.99		30 (22.56%)		158 (32.3%)		53 (28.6%)
20–29.99		14 (10.53%)		95 (19.4%)		31 (16.8%)
30–39.99		13 (9.77%)		85 (17.4%)		27 (14.6%)
40–49.99		9 (6.77%)		20 (4.1%)		5 (2.7%)
50–59.99		3 (2.26%)		4 (0.8%)		2 (1.1%)
60–69.99		13 (9.77%)		12 (2.5%)		7 (3.8%)
70+		34 (25.56%)		33 (6.7%)		18 (9.7%)
Total		133		489		185

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We would like to thank Judith Bennett, Monica Green, and two anonymous reviewers for their helpful comments on an earlier version of this paper. The views and any errors in this paper are our own. Funding from the National Science Foundation (BCS-1261682), the Wenner-Gren Foundation (#8247), and the American Association of Physical Anthropologists supported collection and analysis of data from the St. Mary Graces cemetery. Thanks to Jelena Bekvalac and Rebecca Redfern at the Museum of London Centre for Human Bioarchaeology for providing access to the skeletal samples used in this study and for generously providing the physical facilities for this work. Lastly, we thank Robin Fleming and Katherine French for organizing the Things of the Past Workshops at the Radcliffe Institute at Harvard University in 2014 and the University of Michigan in 2015 where we presented an earlier version of this paper.

Notes

1. Campbell, *Great Transition*, 289–313; Green, "Editor's Introduction," and the essays in Green, ed., *Pandemic Disease*.

2. M. Ziegler, "Black Death," 260; Little, ed., *Plague and the End of Antiquity*; Perry and Fetherston, "*Yersinia pestis*," 36.

3. M. Ziegler, "Black Death, 260.

4. Those who argued that the Black Death was not bubonic plague include Twigg, *Black Death*; Scott and Duncan, *Biology of Plagues*; Cohn, *Black Death Transformed*. For the recent discoveries, see Drancourt et al., "Detection"; Raoult et al., "Molecular Identification of *Yersinia pestis*." These early results were viewed with skepticism because of the use of small, poorly dated samples and because of the possibility of contamination by modern *Y. pestis*; see Twigg, "Black Death"; Wood and DeWitte-Aviña, "Was the Black Death Yersinial Plague?"; Gilbert et al., "Absence," 348, 352. More recent DNA and immunological studies have yielded more

convincing evidence: Bianucci et al., "Rapid Diagnostic Test"; Bos et al., "A Draft Genome"; Haensch et al., "Distinct Clones"; Kacki et al., "Black Death"; Pusch et al., "Yersinial F1 Antigen"; Schuenemann et al., "Targeted Enrichment"; Tran et al., "Co-Detection." In 2011, the entire genome of fourteenth-century *Y. pestis* was reconstructed from DNA fragments isolated from the East Smithfield Black Death burial ground in London; Bos et al., "A Draft Genome." For the East Smithfield cemetery, see also below.

5. Perry, "Is Bioarchaeology a Handmaiden?" 489.

6. For contemporary descriptions, see Horrox, *Black Death*, 14–92. See also the descriptions of symptoms culled from an impressive array of medieval sources in Cohn, *Black Death Transformed*, 57–139, although marshalled to argue that the Black Death was not modern bubonic plague.

7. Boccaccio, *Decameron*, 50–51.

8. Horrox, *Black Death*, 81.

9. Chroniclers often noted the days victims were ill before dying, as in Horrox, *Black Death*, 19 (three days), 25 (three to five days), 27 (three days, some more rapid), 35 (one to three days), 36 (three days), 42 (two days), 55 (two to three days), 64 (three to four days), 74 (three days), 77 (two to three days), 84 (two days).

10. Perry and Fetherston, "*Yersinia pestis*," 57–58; M. Ziegler, "Black Death," 266.

11. Some (Cohn, *Black Death Transformed*,64–65, 68–69, 77–81) argue that buboes or boils arose in the neck and armpits in medieval plague and more often in the groin in modern plague.

12. Higgins, "Emerging or Re-Emerging," 576–77; Cleri et al., "Plague Pneumonia," 12. Pharyngeal and gastrointestinal plague are caused by ingestion of contaminated meat; M. Ziegler, "Black Death," 266.

13. Perry and Fetherston, "*Yersinia pestis*," 58; Lathem et al., "Progression," 17787.

14. Horrox, *Black Death*, 4–5, 43, 63.

15. For critical (and tendentious) discussions of this view, see Benedictow, *Black Death*, 27–31, 236–41; Benedictow, *What Disease Was Plague?* 491–92. Person-to-person transmission occurs when infected individuals cough droplets containing the plague bacteria into the air, which are then inhaled by others in close proximity (see Begier et al., "Pneumonic Plague Cluster," 464; Hinckley et al., "Transmission Dynamics," 554; Pechous et al., "Pneumonic Plague," 191.

16. For example, see Richard et al., "Pneumonic Plague."

17. Hinckley et al., "Transmission Dynamics."

18. Nishiura, "Epidemiology," 1061.

19. P. Ziegler, *Black Death*, 122–27; Benedictow, *Black Death*, 126–29.

20. Horrox, *Black Death*, 64–65.

21. Ibid., one year: 62, 63; more than a year: 81; two years: 64.

22. The other main outbreaks of plague in late fourteenth-century England were in 1361–1362, 1369, 1374–1379, and 1390–1393; their exact dates are open to question since they seem to have become increasingly regional. See ibid., 85–92; Shrewsbury, *Bubonic Plague*, 126–56 (although he asserts these pestilences were not bubonic plague).

23. Previous studies are discussed in Benedictow, *Black Death*, 123–26, 343–59.

24. Wood, Ferrell, and DeWitte-Aviña, "Temporal Dynamics"; Benedictow, *Black Death*, 123–26, 342–48.

25. For example, Lock, "Black Death."

26. For examples, see Megson, "Morality"; Röhrkasten, "Trends of Mortality," 184–89; Sloane, *Black Death in London*, 43–86.

27. Lewis ("Disaster Recovery") assessed thousands of datable potsherds from test-pit excavations from nearly 2000 archaeological sites in eastern England. There was a 44.7 percent

decrease overall (p. 780) in the number of pits yielding potsherds following the Black Death, which may reflect declines in the size of the human population. This approach allows for the mapping of both the occurrence and severity of population decline associated with the epidemic.

28. For example, Sloane, *Black Death in London*; Watts, "Black Death"; James, *Black Death,* and the map in Gummer, *Scouring Angel*, 422 (reproduced as Figure 1, below).

29. Benedictow, *What Disease Was Plague?* 420–84. For modern plague, Bacaër, "Model of Kermack," 410; Kreppel et al., "A Non-Stationary Relationship," 3; Migliani et al., "Epidemiological Trends," 1232; Moore et al., "Seasonal Fluctuations," 2.

30. Benedictow, *Black Death*, 124–42; the figures are on p. 142. The spread of both medieval and modern plague is discussed in Benedictow, *What Disease Was Plague?*, 151–93, albeit with the aim of defending his previously published views.

31. Benedict, *Bubonic Plague,* 108.

32. Horrox, *Black Death*, 44, 81, 107–08; quotation at 83.

33. Hinnebusch, "Bubonic Plague," 646.

34. M. Ziegler, "Black Death," 266; Hinnebusch, "Bubonic Plague," 646; Perry and Fetherston, "*Yersina pestis*," 37, 51–53.

35. Royer, "Blind Men," 108.

36. Drali et al., "A New Clade"; Eisen et al., "Early-phase Transmission"; Houhamdi, et al., "Experimental Model"; Ratovonjato et al., "*Yersinia pestis*"; M. Ziegler, "Case for Louse-Transmitted Plague."

37. Mathematical models reduce complex phenomena to a manageable number of variables and the relationships between them. Infectious disease models can be used to predict the behavior of diseases or can be compared to observations of past diseases in an attempt to understand transmission patterns, mortality rates, or other epidemiological variables and their outcomes. Dean ("Modeling Plague Transmission") used an SIR (Susceptible-Infected-Recovered) model to assess the spread of the Black Death assuming different vectors, including the louse.

38. Royer, "Blind Men," 101.

39. For others who agree on the special value of English sources for estimating mortality, see Benedictow, *Black Death*, 126; Olea and Christakos, "Duration," 296.

40. See below for estimates of the mortality of the well-off.

41. The method depends on the fortunate survival of manorial account rolls or court rolls (the documents where heriots were liable to be recorded), as well as documentation (such as a rental) showing the total number of customary tenants on the manor; see Postan and Titow, "Heriots and Prices," and Titow, *English Rural Society*, 69–70. See also Ohlin, "No Safety," 85–89, for further criticisms of the method.

42. Ecclestone, "Mortality"; Titow, *English Rural Society*, 71.

43. Tithings were part of the frankpledge system. Russell, *British Medieval Population*, 226–27; Poos, *A Rural Society*, 106–07. See also Postles, "Demographic Change."

44. The numbers taxed, moreover, could have changed due to in- or out-migration, or lack of administrative fervor during the plague in enforcing the tax, all of which further reduce the tithing lists' usefulness as an indicator of population losses through plague mortality. Poos, *A Rural Society*, 106–08, recognizes these problems. See also Benedictow, *Black Death*, 369–74, for a detailed critique, although his conclusion (p. 373) that the average of the tithing mortality figures of c. 43–46 percent need to be adjusted upward to 60 percent is based on educated speculation, not firm numbers.

45. Razi, *Life, Marriage and Death*, 106–07; Campbell, "Population Pressure," 96.

46. Arthur, "Black Death"; Benedictow, *Black Death*, 376–77. Benedictow's assumptions about the "super-mortality" of women, are, however, based on mortality rates in modern not medieval plague; see Kowaleski, "Gendering," 185.
47. His Majesty's Stationary Office, "Statistical Abstract," 232–35.
48. Perry and Fetherston, "*Yersina pestis*," 36.
49. Paleomicrobiology is the study of ancient biomolecules such as DNA recovered from human and other animal remains.
50. Bos et al., "A Draft Genome," 509.
51. Wagner et al., "*Yersinia pestis*"; Wiechmann and Grupe, "Detection."
52. Devault et al., "Ancient Pathogen DNA."
53. DeWitte, "Setting the Stage. "
54. Ibid.
55. Gage, "Are Modern Environments Really Bad for Us?" 97.
56. Horrox, *Black Death*, 63, 64.
57. Ibid., 35; Russell, *British Medieval Population*, 216; P. Ziegler, *Black Death*, 227–30; McFarlane, *Nobility*, 169–70.
58. For bishops, see Coulton, *Medieval Panorama*, 496, 749. For monastic clergy, see Russell, *British Medieval Population,* 223 (45 percent based on a small sample of 566 monks in twelve monasteries); he also calculated a 41.5 percent mortality rate for abbots and priors who died in 1349–1350 (p. 225). For an extended discussion of how to interpret the mortality figures for secular clergy based on institutions to benefices, see Benedictow, *Black Death*, 343–59.
59. For the conflicting evidence on sex and plague mortality, see Kowaleski, "Gendering," 185. For recent medieval and early modern opinions, see Curtis and Roosen, "Sex-selective Impact." For bioarchaeological evidence on sex, see below, and for the 1360–1361 plague, see the next section, below.
60. R. Lomas, "Black Death," 130 (based on 155 female tenants of a total of 718 tenants at risk). When women are subtracted from the total, the mortality rates of women and men were virtually the same.
61. Curtis and Roosen, "Sex-selective Impact."
62. Benedictow, *Black Death,* 260–61; id., "Black Death." For a discussion of the overall greater mortality and morbidity in towns, see Kowaleski, "Medieval People," 583–93.
63. Butcher, "English Urban Society," 93–97; Britnell, "Black Death in Durham," 47. The best overview is in Britnell, "Black Death in English Towns."
64. Röhrkasten, "Trends of Mortality," 182.
65. Horrox, *Black Death*, 62–85; only one chronicler of the 1348–1349 plague noted age in claiming that more "young and strong" people died (p. 81). For 1361–1362, see below, note 79.
66. Russell, *British Medieval Population*, 216–18; Russell's figures, however, are highly questionable since he did not allow for "normal" deaths amongst an older population; see Ohlin, "No Safety," 78–80, and Poos, Oeppen, and Smith, "Re-assessing." Razi (*Life, Marriage and Death*, 107–09) presents data showing that male peasants in their 50s in the parish of Halesowen had a higher mortality rate than younger men, but admits how crude his roundabout method of calculation is, and how small the numbers are.
67. Lewis, *Bioarchaeology of Children*, 30–33; Milner, Wood, and Boldsen, "Paleodemography," 573–74.
68. This occurs because these methods are biased toward known-age reference samples (a problem called age-mimicry), which tend to be composed predominantly of young individuals; see Bocquet-Appel and Masset, "Farewell to Paleodemography"; Buikstra and Konigsberg, "Paleodemography"; Müller, Love, and Hoppa, "Semiparametric Method"; Van

Gerven and Armelagos, "Farewell to Paleodemography?"; Milner and Boldsen, "Transition Analysis."

69. Buikstra and Konigsberg, "Paleodemography"; Boldsen et al., "Transition Analysis."

70. Milner, Wood, and Boldsen, "Paleodemography," 563; Wood et al., "Osteological Paradox," 344.

71. In some cases, the distribution of skeletons' ages might, counter-intuitively, be more reflective of fertility than mortality; Sattenspiel and Harpending, "Stable Populations."

72. Wood et al., "Osteological Paradox,"345; Vaupel, Manton, and Stallard, "Impact of Heterogeneity,"439.

73. Ortner, "Theoretical and Methodological Issues," 9–10; Wood et al., "Osteological Paradox," 353–54; DeWitte and Stojanowski, "Osteological Paradox," 407–08.

74. Wood et al., "Osteological Paradox."

75. Grainger and Hawkins, "Excavations at the Royal Mint Site"; Grainger et al., *The Black Death Cemetery*; Hawkins, "Black Death," 637–38. There are a limited number of excavated burial grounds associated with the Black Death or subsequent outbreaks of plague. In addition to East Smithfield, in London there are burials in the St. Mary Graces cemetery attributed to the second outbreak of plague in 1361 (detailed below). There are also known plague burials from the Black Death or afterward from Hereford, England (Gowland and Chamberlain, "Detecting Plague"); Germany (Seifert et al., "Genotyping *Yersinia pestis*"); Barcelona, Spain; Dendermonde, Belgium; France (Castex and Kacki, "Demographic Patterns"); and from Bologna, Italy (Rubini et al., "Mortality Risk Factors").

76. The Museum of London's Department of Greater London Archaeology excavated East Smithfield in the 1980s as part of a planned redevelopment project. The collection is currently curated by the Museum of London's Centre for Human Bioarchaeology.

77. DeWitte, "Age Patterns." DeWitte used hazards analysis to examine age patterns of mortality. With this approach, a model of mortality (such as the Gompertz model used by DeWitte) is fitted to data from skeletal samples to reveal age patterns of mortality. This approach is suited to small samples, as the mortality models smooth the random variation present in the skeletal data but do not impose any particular patterns on the data. See Wood et al., "Mortality Models"; and Gage, "Bio-Mathematical Approaches." DeWitte also used the transition analysis method of age estimation; see Boldsen et al., "Transition Analysis."

78. Only about 8 percent of the East Smithfield sample was below the age of 5, and it is difficult to estimate risks of mortality for children without very large sample sizes. It is not unusual for infants and young children to make up a relatively small proportion of skeletal samples, as mentioned above. Such "infant under-enumeration" is often attributed to the fact that the bones of infants and children are relatively small and thin and thus might be more likely to decay rapidly or be overlooked during excavation compared to the bones of older individuals.

79. DeWitte assessed sex differences by modeling sex as a covariate affecting the Gompertz model of mortality. This approach assumes that sex differences (or lack thereof) in mortality are constant across all ages, which might not be realistic. For example, in many living populations, females face higher risks of death at reproductive ages (because of high maternal mortality resulting from increased susceptibility to some diseases like malaria, complications during childbirth, and other factors) but lower risks at pre- and post-reproductive ages. See DeWitte, "Effect of Sex," for details. Castex and Kacki ("Demographic Patterns," 8) also failed to find evidence that the Black Death disproportionately affected either sex using data from Hereford, England and Dreux, France.

80. DeWitte and colleagues used a hazard model (the Usher model) that allows for the estimation of the difference in risk of death between individuals with and without skeletal lesions and that addresses some of the issues associated with the Osteological Paradox. Usher,

"A Multistate Model." See DeWitte and Hughes-Morey, "Stature and Frailty"; DeWitte and Wood, "Selectivity."

81. Horrox, *Black Death*, 85–86; Sloane, *Black Death in London*, 122–23. See also Shrewsbury, *Bubonic Plague*, 126–32, for further contemporary comments, although Shrewsbury believes it was not bubonic plague.

82. *Calendar of Close Rolls 1361–64*, 181–82, 197–98; Röhrkasten, "Trends of Mortality," 192–94; Sloane, *Black Death in London*, 122–42 (will data at p. 125).

83. Dickens, "Historical Graffiti." 181.

84. Above, note 58.

85. Titow, *English Rural Society*, 70; Watts, "Black Death," 23–25; Razi, *Life, Marriage and Death*, 103, 127.

86. Röhrkasten, "Trends of Mortality," 192.

87. Mullan, "Mortality, Gender and the Plague of 1361–62," 9.

88. For the nobility, see McFarlane, *Nobility*, 169–70. For the data from the inquisitions post mortem of tenants-in-chief, see Russell, *British Medieval Population*, 217–18, 222, though his calculations are considered somewhat flawed (above, n. 65). See also Nash, "Mortality Pattern."

89. Mullan, "Mortality, Gender and the Plague of 1361–62," esp. 22–23.

90. Gilchrist and Sloane, *Requiem*; Grainger and Phillpotts, *Cistercian Abbey*; Sloane, *Black Death in London*, 136–40. It continued until the Reformation in 1538; see Grainger and Hawkins, "Excavations at the Royal Mint Site"; Grainger et al., *Black Death Cemetery.* An associated cemetery was used for burials of the general populations, whereas monks and important lay people were buried within the Abbey's church and chapels; see also Rogers and Waldron, "DISH and the Monastic Way of Life"; Grainger and Phillpotts, *Cistercian Abbey.*

91. These results are based on the Kolmogorov-Smirnov test, a nonparametric test that can be used, as in this case, to determine whether two distributions are significantly different from one another.

92. See the discussion of the documentary evidence for 1361–1362, above.

93. A study of long-term recovered plague patients has found antibodies against *Y. pestis* in people more than ten years following initial infection; see Li et al., "Humoral and Cellular Immune Responses."

94. This analysis used the same Gompertz model (above, n. 78) that DeWitte employed for studies of East Smithfield. Initial analyses of mortality across the lifespan failed to produce good estimates of the immature component of mortality associated with infant and childhood causes of death.

95. DeWitte, "The Effect of Sex."

96. The effect of sex (female) on risk of death is 0.23 (95 percent confidence interval: -0.24, 0.64).

97. DeWitte and Wood, "Selectivity."

98. This analysis was done using the Usher model described above (n. 79, above). For details about periosteal new bone formation, see Larsen, *Bioarchaeology,* 86–88; Ortner, *Identification of Pathological Conditions*, 206; Weston, "Investigating."

99. The effect of having lesions on risk of death is 1.87 (95 percent confidence interval: 1.16, 7.1).

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