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The Anthropology of Plague: Insights from Bioarcheological Analyses of Epidemic Cemeteries

Sharon DeWitte
University of South Carolina, dewittes@mailbox.sc.edu

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Sharon N. DeWitte
University of South Carolina - Columbia, dewittes@mailbox.sc.edu

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PANDEMIC DISEASE IN THE MEDIEVAL WORLD
RETHINKING THE BLACK DEATH

Edited by MONICA H. GREEN

CONTENTS

FEATURED SOURCE

THE ANTHROPOLOGY OF PLAGUE: INSIGHTS FROM BIOARCHEOLOGICAL ANALYSES OF EPIDEMIC CEMETERIES

SHARON N. DEWITTE

THE BLACK DEATH, the first outbreak of medieval plague that swept through Eurasia and Northern Africa in the mid-fourteenth century, was one of the most devastating epidemics in human history.[1] The epidemic killed tens of millions of people in Europe alone within a very short period of time (Benedictow 2004; Cohn 2002; Dols 1977; Wood, Ferrell, and DeWitte-Avina 2003). This disease initiated or enhanced social, demographic, and economic changes throughout Western Eurasia and Northern Africa (see, for example, essays by Borsch 2014, Carmichael 2014, Colet et al. 2014, and Green 2014—all in this issue), and thus has attracted the interest of a variety of researchers for decades. In addition to its importance in shaping events hundreds of years ago, the Black Death continues to be of interest today, in part because the epidemic was caused by the same pathogen that causes modern plague, the bacterium *Yersinia pestis*. Given molecular evidence that the Plague of Justinian (*c.* sixth to eighth centuries CE) was also caused by *Y. pestis* (Harbeck et al. 2013; Wagner et al. 2014; Wiechmann and Grupe 2005), the Black Death was a terrible manifestation of a disease that has affected humans for nearly fifteen hundred years. The continued existence and threat of plague means that it is crucial to understand the extent of the geographic and temporal variation of the disease so that we might best be prepared for its effects on our species in the future.

This paper focuses on the reconstruction of medieval Black Death mortality patterns, particularly those patterns that have been estimated using

1 Here, and throughout this paper, the "Black Death" refers only to the first outbreak of the Second Pandemic of plague during the mid-fourteenth century.

the skeletal remains of victims of the epidemic in the mid-fourteenth century. Such research is essential for several reasons. Though it is now clear from molecular evidence, particularly the results of recent whole genome sequencing studies, that the Black Death was caused by *Y. pestis* (Bos et al. 2011; Drancourt and Raoult 2002; Haensch et al. 2010; Raoult et al. 2000; Schuenemann et al. 2011), this is a relatively recent discovery. The most convincing and least controversial molecular studies, which have yielded sequences specific to *Y. pestis* and used samples clearly dated to the fourteenth-century epidemic (rather than merely suspected victims or victims of more recent historic epidemics), have all been published since 2009 (Bos et al. 2011; Haensch et al. 2010; Schuenemann et al. 2011). Prior to these new molecular insights, the cause of the Black Death was much more hotly debated, and researchers were motivated to clarify in exacting detail how the historic and modern diseases differed epidemiologically. The ultimate goal was determining whether Black Death was, in fact, plague (and if so, whether the strain of *Y. pestis* that caused it was of exceptionally heightened virulence), or whether it was caused by some other disease, such as anthrax or viral hemorrhagic fever (Scott and Duncan 2001; Twigg 1984).

The exciting nature of the molecular findings has made many people forget why the identification of the specific causative agent of medieval plague matters at all. Now that we know (insofar as anything is truly known in scientific research) that it was caused by *Y. pestis*, research can clarify the temporal and geographic variation associated with the disease, to further broaden and improve our understanding of: first, plague virulence (e.g., why was mortality during the Black Death so high?); second, host-pathogen interactions (e.g., how did host population dynamics affect the disease dynamics during medieval plague epidemics?); and third, how this particular disease might manifest in the future (e.g., is it possible that future outbreaks of *Y. pestis* will ever approximate medieval patterns of mortality and transmission?).

Clarifying the human context of the Black Death via bioarcheological research is essential to these goals. Bioarcheology is the study of human skeletal remains excavated from archeological sites with the aim of furthering our understanding of the demography, health, and ways of life of people in past populations. Bioarcheologists examine these remains to determine the ages at which people died, whether they were male or female, what foods they regularly consumed, how healthy or unhealthy they were prior to their deaths (based on the presence or absence of certain markers of physiological stress on the skeleton), whether they experienced interpersonal violence, and other phenomena that leave evi-

dence on or in the skeleton. Through the direct observation and analysis of human skeletal remains, bioarcheological research can provide demographic data that are crucially important for contextualizing the results of the ongoing molecular analyses (e.g., DNA and immunological studies) of both *Y. pestis* and the humans affected by historic plague epidemics. Such context is particularly important given recent evidence that the medieval and modern strains lack significant functional differences in regions of the genome associated with virulence. This suggests that factors other than genetic changes in the causative pathogen are responsible, at least in part, for changes in plague epidemiology (see also Ziegler 2014, in this issue), who argues that research should focus on factors other than a genetic "smoking gun" to further our understanding of plague virulence).

The combination of demographic and molecular data will allow for a disentanglement of the factors that affected medieval health and demography—that is, a determination of whether it was the biological effects of plague or the social and economic consequences of the epidemic that primarily shaped demographic and health patterns in the surviving population. Such interdisciplinary research on the Black Death also has the potential to clarify population dynamics and human biological responses to disease episodes in general, and thus can contribute to our understanding of precisely how and why diseases shape human demography (including age and sex structures, and population density and growth) and human evolution (e.g., acting as forces of natural selection resulting in relatively robust immune responses or genetic resistance to disease). Regarding the latter phenomenon, Crespo and Lawrenz (2014, in this issue) detail how heterogeneity in immune competence during the Black Death might have affected risks and levels of mortality during the epidemic, and how Black Death mortality might have shaped variation in immune function in the surviving population.

In addition to improving our understanding of the dynamics of the Black Death in particular, bioarcheological studies of the epidemic are relevant to current concerns about emerging diseases. Emerging diseases are those that increase in frequency after being introduced into a new, immunologically naïve host population. More than fifty diseases have emerged in the past thirty years, including HIV/AIDS, West Nile Virus, and SARS, and new diseases will emerge in the future (Cleaveland, Haydon, and Taylor 2007; Morens, Folkers, and Fauci 2004; Morse 1995). The Black Death was an emerging disease in the fourteenth century, and the persistence of this ancient emerging disease to the present day provides an ideal opportunity to examine, from an anthropological perspective, long-term trends in emerging disease dynamics, and thereby understand the effects such

diseases have had on human populations and predict how they can affect us in the future. In addition to improving our understanding of plague, bioarcheological research on the Black Death can provide much needed data to evaluate models of the evolution of virulence in emerging pathogens (Ebert and Bull 1999; Frank 1996) and help to resolve questions about whether changes in emerging disease epidemiology are the result of human adaptation, changes in human or non-human host demography or behavior, or evolution of the causative pathogens (Alizon et al. 2009; André and Hochberg 2005; Grenfell et al. 2004).

Furthermore, Black Death bioarcheological research contributes to efforts to understand the phenomena of heterogeneous frailty (an individual's risk of death, relative to other members of the population: Vaupel, Manton, and Stallard 1979) and selective mortality (disproportionate deaths of those with highest frailty), and how they shape population dynamics in living and past populations. Because individuals differ in their susceptibility to disease and death as a result of genetic, biological, environmental, socioeconomic, or other causes, differences in frailty exist among individuals within a population (Aalen 1994; Wood et al. 1992). Mortality is expected to be selective, at least under normal, non-catastrophic conditions; that is, because of differences in frailty, the individuals who die at each age will not be a random sample of all the individuals living at that age, but rather will be disproportionately selected from among those with the highest risk of dying (the highest frailty).

Many researchers are interested in revealing the sources and consequences of heterogeneous frailty in living populations so that health and mortality disparities can potentially be ameliorated or eliminated (Olden and White 2005). It is just as important to understand these processes in the past, particularly in the context of parasitic and infectious disease (which were the most common causes of death for most of human history and continue be the most significant causes of death for many living populations), in order to understand modern human biology. By revealing how biological factors such as age, sex, and history of physiological stress (such as malnutrition or infectious disease) affected risks of mortality during one of the most dramatic disease epidemics in human history, Black Death research has the potential to clarify why and how affected populations have changed in the intervening centuries. The Black Death also offers a case study of how heterogeneous frailty and selective mortality operate during times of crisis mortality, which characterized much of the mortality experience of pre-industrial populations throughout the world (Gage 2005).

Documentary Evidence of Black Death Mortality Patterns

Studies using data from historical documents have confirmed the devastating nature of the Black Death and have shown that mortality during the epidemic was perhaps even higher than previously thought (Benedictow 2004; Cohn 2002; Dols 1977; Scott and Duncan 2001; Wood, Ferrell, and DeWitte-Avina 2003). Using data from court records of payments made by residents, Poos (1991) estimated the annual totals of males ages twelve years and older living on manors in Essex. He found that several manors lost as much as 54% of their male residents during the Black Death. Wood, Ferrell, and DeWitte-Avina (2003) analyzed data on deaths of beneficed priests in the Lincoln diocese in England. In the eighteen months before the Black Death, the mean mortality rate for priests was 38.9 per 1000, but during the twelve-month period beginning with the outbreak of the Black Death in Lincoln, the mean mortality rate increased to 463.6 per 1000. During the Black Death, annual mortality rates for priests were eleven times higher and, more dramatically, *monthly* mortality rates were about thirty-five to forty-five times higher than in the pre-epidemic period. Total mortality from the Black Death was typically 30% to 50% of the total population in affected regions of Europe. Following the Black Death, plague mortality decreased steeply and steadily with subsequent outbreaks (Carpentier 1971; Cohn 2002; Hatcher 1977), which might indicate that the population was generally less frail, that there were changes in susceptibility to plague following the epidemic, or that there were factors involving nonhuman animal hosts that affected plague epidemiological patterns in humans.

It is clear that the mortality caused by the Black Death was extraordinarily high. What has not been as clear is how this excess mortality was distributed by age, sex, socioeconomic status, and other biological and social factors. Several medieval European chroniclers suggest that the Black Death killed people indiscriminately, irrespective of age or sex. For example, Matteo Villani (Florence, d. 1363) described the Black Death as "a pestilence among men of every condition, age and sex" (cited and translated in Cohn 2002: 126). According to the chronicler Michele da Piazza (Sicily, fourteenth century), the mortality from the Black Death was "so heavy that sex and age made no difference, but everyone died alike" (cited and translated in Horrox 1994: 41). Both of these statements suggest indiscriminate mortality, at least with respect to age and sex. Some contemporaries, however, believed that the epidemic was selective, and, for example, killed more women than men, and that in subsequent outbreaks of plague (which are believed to be the same disease) men were

killed in greater numbers than were women (Cohn 2002: 210–12; Horrox 1994: 85). According to Jean de Venette (1307–70), a French Carmelite friar and chronicler, "the young were more likely to die than the elderly" (cited and translated in Horrox 1994: 54). Many chroniclers noted that the age pattern of subsequent outbreaks was quite different from that of the Black Death. For example, the 1361 outbreak was called the "Pestilence of Children," as young people were more severely affected than were adults (Cohn 2002: 212–15; Cohn 2008: 86–87; Holmes 1971: 92).

Reconstruction of age-specific mortality patterns during the Black Death has been attempted using historical data. Based on information from inquisitions post mortem, Russell (1948) produced life-table estimates of age-specific mortality among high-status individuals during the Black Death in England.[2] These were royal inquisitions into the cause of death of tenants-in-chief, people who held land directly from the king. The inquisitions provide a wide sample of the highest rank of landholders. When a tenant died, if the heir was underage, the king assumed guardianship of the property until the heir came of age. Such guardianships were quite lucrative, and it was in the interest of all parties to establish accurately the age of the heir; thus, the age estimates provided by the inquisitions are supported by multiple documents (Russell 1948). Unfortunately, the samples are too small to provide useful life-table estimates, particularly for intervals below the age of twenty; indeed, for some age intervals, the 95% confidence intervals for probability of death include negative values (Wood et al. 2002a). Despite the limitations of the available data, Russell concluded from the life-table estimates that age did have an effect on Black Death mortality. He argued that older men were particularly susceptible (although individuals over the age of sixty apparently fared better than those in their late fifties), and children between the ages of ten and fifteen were at a reduced risk of dying from the disease compared to other age groups. Ohlin (1966), however, has criticized Russell's approach and argued that the data do not support the conclusion that mortality varied by age during the Black Death.

Using manorial court records from the English village of Halesowen (West Midlands), Razi (1980) estimated ages at death among the peas-

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2 Life tables are frequently used in demography to display the numbers of deaths at each age or age interval, age-specific mortality rates, survival rates at various ages, and other related demographic measures for a particular population. Model life tables are constructed using data from living populations and can be applied to small samples obtained from living or past populations to compensate for missing data.

antry during the epidemic. Individuals appear in the manorial court rolls multiple times, and the last appearance of each individual is the death duty (*heriot*) payment made by heirs to the lord of the manor before taking possession of the deceased's property. Razi assumes that each individual appeared for the first time in the court rolls at the age of twenty years and counted forward from this first appearance to the death-duty payment in order to estimate ages at death. His age estimates are therefore imprecise, and by definition no information is available for individuals younger than twenty. Estimating age-specific mortality rates for males during the Black Death, he finds, similar to Russell (1948), that for males between twenty and fifty-nine years of age, mortality rates increased with age, but that age-specific mortality rates for those over sixty years were lower than those of younger men. Such a pattern of mortality is typical under conditions of normal mortality, though a plateau in mortality is usually observed at later adult ages in living populations (Gage 1988). Given the small samples available for Razi's study, the results might not be generalizable to the entire English population.

As limited as the reconstructions of Black Death age-patterns have been using historic documents, such sources are even less forthcoming with respect to the effects of other biological or social factors. For example, the determination of whether one sex was disproportionately affected or both sexes faced equal risks of death during the epidemic is even more problematic than that of age patterns given that there is very little documentary data on age-at-death for women and children at the time of the Black Death (Razi 1980; Russell1948). Though there are data on sex ratios during the medieval period (see, e.g., Kowaleski 2013), these data do not allow reconstruction of sex differences in mortality across the lifespan. There is also no information, as far as I am aware, from any documentary sources about whether pre-existing health conditions had any effect on an individual's risk of death during the epidemic. Historical records, therefore, do not provide a clear and complete picture of Black Death mortality patterns. Without such data, our ability to learn more about the ways in which the Black Death, and other diseases, have the power to shape human demography, biology, and evolution is severely hindered.

Fortunately, skeletal material exists that provides empirical evidence about mortality patterns that is not available in written records. Though human skeletal samples are not without their own limitations, they can, when analyzed properly, reveal much more (and more direct) information about the individuals who died during the Black Death.

Black Death Cemeteries

During the Black Death, existing cemeteries proved inadequate to accommodate the overwhelming numbers of people killed by the epidemic, and providing normal burials for all the victims of the epidemic became difficult (Horrox 1994: 21, 64, 268–71). Though many victims of the disease were buried in existing cemeteries, mass burial grounds were established across Europe during the Black Death to accommodate the excess mortality. One such mass burial ground was the East Smithfield cemetery in London.

The Black Death was apparently introduced to England during the summer of 1348 via the Dorsetshire port of Melcombe Regis, and it reached the suburbs of London as early as September 1348 (the disease was recorded in the suburb of Stepney, two miles east of the city walls, in December 1348). The epidemic ravaged the city throughout 1349 and ended by the spring of 1350 (Gasquet 1977; Hawkins 1990). Aware that the Black Death was spreading towards London, "substantial men of the city" ordered that emergency burial grounds be established prior to its arrival (Grainger et al. 2008; Hawkins 1990). The East Smithfield cemetery was established in late 1348 or early 1349 just outside the city walls in East London, northeast of the Tower of London, on land that had previously been used as a vineyard. The exact dimensions and location of the cemetery are provided by the cartulary of the priory of Holy-Trinity-without-Aldgate, and it was originally called the Churchyard of the Holy Trinity, as the land was acquired from the priory (Grainger et al. 2008; Hawkins 1990).

East Smithfield was partially excavated in the 1980s, as part of the larger Royal Mint Site, by the Museum of London's Department of Greater London Archaeology (now Museum of London Archaeology [MOLA]). The burials in East Smithfield were concentrated in two areas: first, a western area with two mass burial trenches, a mass burial pit, and individual graves arranged in several parallel north-south rows; and second, an eastern area with one mass burial trench and individual graves arranged in several parallel rows (Hawkins 1990). Over six hundred skeletons were excavated from the cemetery, a fraction of the estimated twenty-four hundred individuals originally buried in East Smithfield, and they are now curated by the Museum of London Centre for Human Bioarchaeology. The cemetery could have accommodated many more burials, but the epidemic waned before it was necessary to use all the available space. Excavation of the cemetery revealed that the individuals interred in East Smithfield were buried carefully. Almost without exception, the bodies were bur-

ied in standard medieval Christian fashion: extended on their backs with their heads oriented west and feet oriented east. Some of the skeletons were missing limbs at the time of excavation, indicating that these individuals had been partly disarticulated as a result of putrefaction before burial (Grainger et al. 2008; Hawkins 1990). However, even these bodies were buried in the standard Christian fashion and were treated with apparent care, rather than tossed hastily into a burial trench.

East Smithfield was one of two known emergency burial grounds established in London (Grainger et al. 2008; Hawkins 1990). The other burial ground was at West Smithfield, which later became the site of the London Charterhouse. The West Smithfield site is now beneath Charterhouse Square and thus will not be excavated in its entirety (or even in large part) in the foreseeable future, though a few individuals from the cemetery were recently uncovered during construction of a new London Underground line (Palmer 2013). The East Smithfield site, which yielded several hundred individuals, is thus one of a very few large *excavated* cemeteries in all of Europe with unambiguous documentary and archeological evidence linking it to this outbreak of the medieval disease. At the time of writing, to my knowledge, the only other large, excavated burial grounds that are clearly associated with the Black Death are the Hereford Cathedral cemetery in England (Gowland and Chamberlain 2005) and the Heiligen-Geist Hospital mass burial in Lübeck, Germany (Lütgert 2000). Of these, East Smithfield has been investigated most extensively by various researchers, according to the number of publications recorded in the Wellcome Osteological Database.[3]

The ability to study the skeletal remains of victims of the Black Death and subsequent plagues that have been recovered from East Smithfield and other medieval plague burial grounds yields insights that are simply not supported by existing historical documents. The advantage of bioarcheological research is that the information derived from skeletal remains of victims is not subject to the same biases that are inherent in historical documents, thereby allowing bioarcheologists not only to reveal what is missing from historical data, but also to challenge narratives based on those conventional sources (Perry 2007).

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3 WORD Database (Wellcome Osteological Research Database) 2013: <www.museumoflondon.org.uk/Collections-Research/LAARC/Centre-for-Human-Bioarchaeology/Resources/Medievaldatadownloads.htm> [accessed August 31, 2014].

Previous Bioarcheological Studies of the Black Death

Several researchers have analyzed the East Smithfield Black Death cemetery to discern the mortality patterns of the Black Death (Gowland and Chamberlain 2005; Margerison and Knüsel 2002; Waldron 2001). The aim of some of these inquiries was to determine whether Black Death cemeteries are more representative of the demographics of a living population than is generally true of mortality samples derived from other cemeteries. As discussed above, normal causes of mortality are expected to be selective, targeting the weakest individuals in a population, such as infants, the elderly, and those with immune systems compromised by disease or malnutrition (Milner, Wood, and Boldsen 2008). Because of this selective mortality, skeletal assemblages are inherently biased samples of the once living populations from which they derived. For each age group, the individuals within a cemetery are not a representative sample of all the individuals who were once alive at that age, but rather those individuals who were at the highest risk of dying at that age. This makes it difficult to infer the characteristics of the once-living population in a straightforward manner based on observations of the cemetery assemblage. However, some researchers have suggested that the Black Death might have been so virulent that it killed indiscriminately, rather than selectively, such that mortality samples resulting from the epidemic would contain relatively healthy, robust individuals not usually found in normal cemeteries. If this were the case, Black Death cemeteries might provide a true cross-section of the once-living population not typically available in normal mortality cemeteries (Chamberlain 2006; Margerison and Knüsel 2002).

Waldron (2001) compares the mortality profile from East Smithfield to that of the St. Mary Graces cemetery (*c.* 1350–1538), which was established in the same location as East Smithfield after the Black Death ended, and to the age distribution of the living population of medieval London as predicted from a model life table.[4] He finds no systematic differences between the mortality profiles of the two cemeteries. Furthermore, the profiles of both cemeteries differed from the expected living age structure. Waldron argues that the skeletal evidence does not support the idea that any particular age or sex was at an elevated risk of dying during the Black Death. Waldron also finds that the two cemeteries had generally similar frequencies of skeletal pathologies. He concludes that the East Smithfield

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4 Model life tables are constructed using data from living populations and can be applied to small samples obtained from living or past populations to compensate for missing data.

cemetery is not representative of the once-living population, as one might expect if the Black Death killed people indiscriminately.

However, several researchers have found that the East Smithfield age-at-death distribution more closely resembles a model living population age distribution than the age-at-death distribution of a normal mortality sample. Margerison and Knüsel (2002) compare East Smithfield to the St.-Helen-on-the-Walls cemetery in York, northern England, a normal non-epidemic cemetery in use from the late twelfth century to 1550. The East Smithfield mortality profile differs significantly from that of the St.-Helen-on-the-Walls cemetery, but generally resembles the living age distribution from a model life table assumed to be representative of the age distribution of a living (poor) medieval urban population; in particular, that is, East Smithfield, like a living medieval population, contained many young people and few older individuals. Gowland and Chamberlain (2005) compare East Smithfield to a pre-Black Death sample from Blackgate cemetery in Newcastle, northeastern England. They also find that the East Smithfield distribution differs from the normal mortality sample and more closely resembles a model life table living age distribution, suggesting that all age groups were equally affected by the Black Death.

More recently, I have examined the age and sex patterns associated with the epidemic and the health of the victims, as evidenced by skeletal pathologies (or, more generally, skeletal stress markers, which are visible anomalies of the skeleton that indicate exposure to physiological stress, such as infection or malnutrition at some point in life) (DeWitte 2009; DeWitte and Hughes-Morey 2012; DeWitte and Wood 2008). My research, however, differs in several key ways from the studies described above. With the exception of Gowland and Chamberlain (2005), other studies of East Smithfield use comparison samples that partly or completely post-date the Black Death. This is potentially problematic because the Black Death itself initiated profound demographic changes throughout Europe. Paine (2000) finds, using population modeling, that episodes of catastrophic mortality may have effects on age-at-death distributions that last for several generations. Further, as my work has indicated, if the mortality during the Black Death was selective with respect to preexisting health conditions, it is possible that the epidemic altered patterns of health, as indicated by skeletal stress markers, in the surviving population. It is thus possible that post-Black Death samples, at least those dating to just after the epidemic, more strongly reflect the effects of the Black Death than they do typical medieval mortality patterns. Therefore, in order to determine whether the Black Death killed selectively and how its mortality patterns differ from normal medieval mortality, I have used an exclusively

pre-Black Death comparison sample composed of people who died in the two centuries just before the Black Death arrived.

Another way in which my research has differed from most other investigations of East Smithfield (except for that of Gowland and Chamberlain) is my use of a relatively unbiased method of adult age estimation. Paleodemographic age estimation in adults is based on observations of the morphology of certain parts of the skeleton that change in fairly predictable ways with age (though the timing of such changes can be highly variable). These include changes to parts of the pelvis and to the skull. Studies of known-age-at-death reference samples have yielded various age-estimation methods (which I hereafter refer to as "traditional" methods) which involve assigning a skeleton to one of several stages based on the morphology of a particular skeletal feature. Each stage has a corresponding mean age at death and an age interval, both of which are estimated from known-age individuals in the reference sample who display the morphological features distinctive of each stage. Unfortunately, traditional paleodemographic age-estimation methods have a built-in bias. They tend to lead to underestimations of older adult ages because the age estimates are biased toward known-age reference samples, which are often composed predominantly of young individuals (Bocquet-Appel and Masset 1982; Buikstra and Konigsberg 1985; Milner and Boldsen 2012; Müller, Love, and Hoppa 2002; Van Gerven and Armelagos 1983). For example, the McKern-Stewart Korean War reference sample is made up primarily of young men (Milner, Wood, and Boldsen 2008). This "age mimicry" means that the numerous paleodemographic adult age-at-death distributions that have been estimated for cemetery samples are nearly identical regardless of their temporal or geographic context. Such biased distributions hinder informative comparisons of paleodemographic mortality profiles. Another potential limitation of traditional age-estimation methods is the use of broad terminal age intervals, for example, those fifty years or older, because of the difficulty of estimating age in older adults (Boldsen et al. 2002; Buikstra and Konigsberg 1985). These methods, therefore, are not capable of making distinctions between, for example, a fifty-year-old and a ninety-year-old, two ages for which one would expect very different morbidity and mortality regimes, at least in living populations.

Because of these limitations of traditional age-estimation approaches, I have estimated ages using the method of transition analysis described by Boldsen et al. (2002). Transition analysis resolves the problem of age estimates that are biased toward the known-age reference sample. It also yields point estimates of age and their corresponding 95% confidence intervals (rather than broad and fixed interval estimates), even for older

adults. In transition analysis, data from a known-age reference collection are used to obtain the conditional probability that a skeleton will exhibit a particular age indicator stage, or suite of age indicator stages, given the individual's known age. Using Bayes's theorem and maximum likelihood estimation,[5] this conditional probability is combined with a prior distribution of ages at death to determine the posterior probability that an individual in the cemetery sample died at a particular age given that the skeleton displays particular age indicator stages. In transition analysis, the prior distribution of ages at death can either be an *informative prior* based on documentary data or a *uniform prior*, and I have used an informative prior based on data from seventeenth-century Danish rural parish records. By combining the conditional probability from a known-age reference sample with a prior distribution of ages at death, transition analysis avoids imposing the age distribution of the reference sample on the unknown-age cemetery sample (Boldsen et al. 2002). I have applied transition analysis to East Smithfield data using the Anthropological Database, Odense University (ADBOU) Age Estimation software (Boldsen et al. 2002).

Lastly, my research differs from that of others by incorporating hazard analyses (statistical assessment of risks of death) to examine mortality patterns, rather than using life tables as a standard of comparison. Life tables are typically constructed for living populations using huge datasets that include, among other things, the number of individuals alive at each age and the number who die within a particular interval. They have been used for several decades in paleodemography to examine intra- and inter-population demographic variation (Acsádi and Nemeskéri 1970; Wood et al. 2002b).[6] The construction of paleodemographic life tables is based on the assumption that the age-at-death distribution in a cemetery is equiva-

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5 Bayes's theorem allows for the estimation of the probability of a trait (such as the unknown age of an individual in a cemetery) that cannot be directly observed, but which is associated with another trait that can be observed (such as a skeletal age indicator). In the case of age-estimation, this requires information about the association between age and skeletal indicators of age, which is derived from known-age reference samples in which both the ages of individuals and their skeletal age indicators are observable. The joint distribution of ages at death and the skeletal age indicators for all individuals in a reference sample is used to determine the probability that an individual in a cemetery sample (for whom only the skeletal age indicator can be observed) is a particular age given the skeletal age indicators observed for that individual (Milner, Wood, and Boldsen 2008).

6 Paleodemography is a subfield of bioarcheology that examines demographic patterns, such as mortality and fertility, in past populations using skeletal samples.

lent to the cohort age-at-death column in a life table;[7] however, this is true only under a set of stringent criteria that many past populations do not fulfill (e.g., no migration and no changes in age-specific fertility or mortality rates) (Milner, Wood, and Boldsen 2008). Further, the use of life tables in paleodemography is complicated by small sample sizes and a lack of quantitative information about the original population at risk of death. Life tables are considered by many researchers to be an inefficient way to make use of the data that are available from cemetery samples, as these samples are almost always too small to allow for unbiased estimates of the numerous parameters necessary to construct a life table (Buikstra 1997; Hoppa and Vaupel 2002; Konigsberg and Frankenberg 2002; Milner, Wood, and Boldsen 2008; Wood et al. 2002b). Some paleodemographers, including previous researchers of East Smithfield, have adopted a model life-table approach by applying a theoretical life table that matches or provides a baseline comparison for the age-at-death distribution observed in a cemetery (Weiss 1973; Wood et al. 2002b). Unfortunately, a single paleodemographic age-at-death distribution will often match more than one model life table, and uncertainty exists about which model life tables are most appropriate to use (Gage 1988; Milner, Wood, and Boldsen 2008; Wood et al. 2002b).

In light of these problems, many researchers argue that rather than using life tables, some form of hazard analysis is the most reliable way to derive information from the small samples typical of paleodemography (Buikstra 1997; Gage 1988; Hoppa and Vaupel 2002; Konigsberg and Frankenberg 1992; Konigsberg and Frankenberg 2002; Wood et al. 2002b). Most human age-at-death distributions can be described using five or fewer parameters, and parsimonious parametric models provide a useful alternative to life tables in paleodemographic studies (Holman, Wood, and O'Connor 2002). This is the approach I took in my research and examples of such models are described below.

Hazard Analysis of Black Death Mortality

Hazard analysis of Black Death mortality patterns has, for the most part, involved comparison of the East Smithfield cemetery to a normal (i.e., non-epidemic) mortality sample from the medieval Danish urban parish cemeteries of St. Albani Church, Odense, and St. Mikkel Church, Viborg (*c.* 1100s to mid-1500s), both of which form part of the current ADBOU collection (DeWitte and Hughes-Morey 2012; DeWitte 2010; DeWitte and

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7 A cohort in this context refers to individuals who were all born at the same time.

Wood 2008). The advantages of using the Danish sample as a comparison for East Smithfield include its large size, which allows for estimation of the hazard model parameters; the generally good preservation of the Danish skeletons (which facilitates estimation of age and sex); and the ability to accurately date skeletons and, thus, to select a predominantly (if not exclusively) pre-Black Death sample. The Danish sample contains only individuals who died before the Black Death arrived in Denmark in 1350, and thus provides a baseline of normal, non-epidemic mortality patterns for comparison with East Smithfield. The populations of Denmark and Southern England at this time were similar economically, socially, and demographically (Benedictow 1996; Poulsen 1997; Roesdahl 1999; Sawyer and Sawyer 1993; Widgren 1997). However, the fact that the Black Death and Danish normal mortality samples were drawn from two different geographic regions means that possible population differences have to be considered when interpreting the results of these studies. An ideal comparison sample for East Smithfield should be drawn from England, and therefore, in future research I will use exclusively London samples, some of which have been made available to researchers only recently.[8]

I have previously examined the age patterns of Black Death mortality, and how they compared to those produced by normal mortality conditions, by using age-at-death data from each sample to estimate the parameters of a parsimonious model of human mortality: the Siler model. The Siler model of mortality describes the typical pattern of population-level risk of mortality across the lifespan, which begins relatively high at birth, drops rapidly during childhood before leveling off between late childhood and young adult ages, and then increasing again during later adult ages (Siler 1979). Though the absolute levels of mortality and timing of declines and increases in mortality can vary tremendously across populations, this same general pattern (the so-called "bathtub curve") has been observed in nearly all human populations (and even nonhuman animal populations) for which data are available (Gage 1989).[9]

The results of this research indicate that the Black Death did not kill indiscriminately with respect to age. The results of estimation of the Siler model in the East Smithfield and normal mortality samples indicates that

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8 The Museum of London Centre for Human Bioarchaeology, which provides access to these samples, was established in 2003, and some of the samples that will be used in future research were made available for general research following the publication of the corresponding site report in 2012.

9 Further details about the Siler model can be found in Gage (1988 and 1989) and Wood et al. (2002b).

for both, the risk of death increased with advancing adult age. This means that elderly adults faced higher risks of death compared to their younger peers during times of both catastrophic Black Death mortality and under normal medieval mortality conditions (DeWitte 2010). However, use of the Siler model did not yield convincing estimates of the parameters of the juvenile component for the East Smithfield sample. Typically, in human populations, the juvenile risk of mortality is initially relatively high at birth, but it declines very rapidly with small changes in age for the first few years of life; this change might reflect increases in immune competence with age and thus consequent decreases in risks of infection (Gage 1989). The estimated juvenile risk for the normal mortality sample conforms to such expectations; however, for the East Smithfield sample, the juvenile risk was initially low at birth and increased slowly with age, with no drop in risk during early childhood. At face value, this might suggest that infants faced relatively low risks of mortality during the Black Death; however, the estimated juvenile pattern in East Smithfield might have been the result of the small sample of infants and children available, as they only made up 8% of the East Smithfield sample (which might reflect poor preservation of these bodies or different burial practices for children). Estimation of the juvenile component of mortality is based primarily on individuals in the sample who are between the ages of zero and five years, and it is difficult to estimate accurately the juvenile risk with small sample sizes (Wood et al. 2002b). Such small sample sizes in East Smithfield means that clarification of mortality for the youngest individuals in the population during the Black Death awaits further work with larger samples.

Assessment of whether the Black Death differentially affected one sex has been limited to adults, given the lack of current methods to accurately assess sex in immature individuals. Thus in my previous work, I modeled sex as a covariate affecting the parameters of the Gompertz-Makeham model, which fits the general human pattern of relatively low mortality during the young adult ages and an increasing risk of death with increasing adult age (Wood et al. 2002). The effect of the sex covariate was modeled in such a way that it was free to take on values indicating either an elevated or decreased risk of mortality for females compared to males, or a complete lack of differences in risk between males and females. The results indicate that, under both conditions of the Black Death and normal medieval mortality, males and females did not differ significantly in their risks of death (DeWitte 2009). It should be noted, however, that the way I modeled the covariate effect (i.e., the proportional hazard specification) does not allow for variation in differences in risk between the sexes across adult ages, and thus this approach cannot detect potential cross-

overs in mortality. It is possible that female mortality was higher than that of males during reproductive ages, as some diseases, such as malaria, disproportionately affect pregnant women. However, examination of such variation in sex differentials across adult ages requires further analysis using larger sample sizes.

To determine whether the Black Death disproportionately affected people already in poor health, I used the Usher model, which allows for variation in the risk of death between individuals with and without skeletal stress markers (see DeWitte and Wood 2008 for a description of these markers) (Usher 2000). The Usher model allows researchers to avoid making the conventional assumption that the presence of stress markers always indicates poor health and similar or identical levels of frailty for all individuals in a particular population, an assumption that might not be justified (Milner, Wood, and Boldsen 2008; Ortner 1991; Wood et al. 1992). Most detectable skeletal stress markers take a minimum of weeks to form. Thus, an individual must be minimally healthy to survive a physiological stressor long enough for an associated stress marker to develop. Some individuals in a skeletal sample might lack stress markers despite exposure to physiological stress because they were very frail and died before skeletal stress markers formed. Alternatively, individuals in the sample might lack stress markers because they were fortunate enough to avoid exposure to a particular stressor or had immune systems robust enough to fight off disease before stress markers formed, both of which reflect relatively low frailty. Under these circumstances, individuals who lack a particular stress marker might have had higher or lower levels of frailty compared to individuals who have them. Thus, the presence of skeletal stress markers might actually indicate relatively good health in some cases, but this will not be apparent in a simple dichotomous comparison of stress marker frequencies.

The Usher model allows for an explicit examination of the relationship between skeletal stress markers and risks of mortality, and thus the determination of whether, in a particular context, stress marker presence is indicative of good or poor health. The model has three non-overlapping states: State 1 includes people with no stress markers; State 2 includes people with stress markers; and State 3 is death. Individuals in a cemetery are observed after they have made the transition to State 3, and the presence or absence of visible stress markers indicates which of the two living states they were in just before they died. Transitions between the states are governed by age-specific hazard rates, and data on age and the presence of stress markers allow for the estimation of the model parameters. The model allows for differences in the risks of death associated

with State 1 and State 2. The model thus enables one to estimate whether individuals with stress markers are at an elevated or decreased risk of dying compared to those without them.

I have applied the Usher model to data on a variety of nonspecific skeletal markers of physiological stress within the Black Death cemetery. Some of these stress markers reflect exposure to physiological stressors (such as malnutrition or infectious disease) strictly during childhood, and others can manifest in response to stressors at any point during the lifespan. (See DeWitte and Wood 2008 and DeWitte and Hughes-Morey 2012 for details about these stress markers.) Fitting the Usher model to data on age and the presence of these stress markers in the normal mortality sample reveals that these stress markers are, at least under normal medieval mortality conditions, indicative of frailty or poor health, and that the risk of mortality was higher for people with the markers compared to those without them. The results from East Smithfield indicate that the Black Death disproportionally killed people who had them. That is, people who experienced physiological stressors, and who developed stress markers in response to those stressors, at some point (perhaps even long) before the arrival of the epidemic were subsequently more likely to die during the Black Death compared to their peers who lacked the stress markers (DeWitte and Hughes-Morey 2012; DeWitte and Wood 2008).[10]

Together, the results of analysis of age and frailty reveal that contrary to the common assumption that the Black Death killed indiscriminately, there was variation in risks of dying during the epidemic. This means we should question any historical, molecular, or bioarcheological study that implicitly or explicitly assumes that the Black Death was not a selective killer. Further, the similarities between the East Smithfield and normal mortality cemeteries indicate that we should expect mortality to be selective and for people to differ in their risks of death under all mortality conditions, even in the event of catastrophic mortality. This has implications, not just for our understanding of the effects of disease in the past but also for our expectations of and preparedness for disease outbreaks in living populations.

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10 Initial analysis, using the Usher model, of the effect of physical stature on risk of death found no effect in either the Black Death or the normal mortality sample (DeWitte and Wood 2008). However, the Usher model is an unnecessarily complicated approach to assess stature. Reanalysis using a more appropriate and simpler analytical approach did find an effect of short adult stature on risk of death (DeWitte and Hughes-Morey 2012).

Bioarcheological research on the Black Death has revealed not only the mortality patterns of this particular historical epidemic, but also more generally it demonstrates that bioarcheological research has important contributions to make to lines of inquiry that have traditionally been the purview of historians. The unique contribution of anthropology to medieval plague studies is that it allows the victims themselves (or, more precisely, their skeletal remains) to bear witness to their experiences while they were alive and to the context of their deaths—in this case, how an individual's age and health status elevated or reduced his or her risk of mortality during the Black Death. This is particularly important for our understanding of the experiences of those who are not typically included in the historical record: that is, the vast majority of people who lived in the past.

Future Directions in Black Death Bioarcheology

The results of bioarcheological research on the Black Death raise questions about the demographic and health consequences of the epidemic. The very high levels of Black Death mortality, and the results from hazard analysis which indicate that this mortality was selective and targeted frail people of all ages, means that the epidemic might have exerted a strong selective force on the European population, removing the frailest, unhealthiest individuals on a very large scale. If the post-Black Death population included individuals who were exposed to and survived the Black Death, this episode of selection might, at least in part, explain the very rapid apparent changes in medieval plague epidemiology. These changes include the apparent decline in plague mortality as described in contemporaneous historical documents. As explained by Carmichael (2014, this issue), patterns of human plague deaths might reflect the disease dynamics of nearby animal host populations, which can influence exposure of humans to plague. However, in the case of medieval plague, the lower estimated mortality rates in later outbreaks of plague (Carmichael 1986; Cohn 2008; Russell 1966) might reflect lower average frailty among people who survived the first outbreak, as well as among their descendants. Furthermore, health might have been strongly shaped by the apparent improvements in standards of living that occurred after the Black Death and that resulted primarily from the massive depopulation caused by the epidemic. Such changes in standards of living included improvements in diet for people of all social classes (Dyer 2002; Hatcher 1977; Poos 1991; Rappaport 1989; Stone 2006). Given the effects of nutrition on immune competence, dietary improvements and decreases in social inequities in access to food (which might have strongly benefitted the lower social ech-

elons who made up the bulk of the English populace) might have acted to reduce average levels of frailty within affected populations.

There is some evidence to suggest improvements in survival and thus health following the Black Death. Russell (1966) found—based on comparisons of British and Spanish documentary data (tax records and inquisitions post mortem) to skeletal data from seventy-seven burial grounds primarily from Central Eastern Europe—that the ratio of individuals above the age of sixty relative to those between ages twenty to sixty increased after the Black Death in some areas. However, a comparison of pooled skeletal data from a wide geographic region (mostly Eastern Europe) to documentary data from Western Europe is not ideal. Among other potential problems, each source is subject to different sources of bias (e.g., poor skeletal preservation of children in some cemeteries, biased skeletal age estimates, or underrepresentation of the poorest individuals in some tax records) which complicates comparison. Thus, this line of inquiry needs to be further pursued using large skeletal samples from cemeteries that pre- and postdate the Black Death, preferably from one relatively circumscribed area, and the application of newer, unbiased age-estimation methods and rigorous quantitative approaches such as hazard analysis. Ongoing bioarcheological research is examining temporal trends in survival, risks of mortality, and patterns of skeletal stress markers using samples from London cemeteries that date to the pre-Black Death (1000–1300) and post-Black Death (1350–1540) periods. Thus far, initial results using these samples indicate improvements in survival and health in general following the Black Death (DeWitte 2014).

Not only is there much to learn about the ways in which the Black Death shaped the demographic and health characteristics within surviving populations, there are also questions about the epidemic itself that remain unresolved, and which could benefit from study of other Black Death burial grounds that have been excavated or that will potentially be excavated in the future. As mentioned above, the relatively small proportion of children in the East Smithfield cemetery has prohibited accurate reconstruction of the risk of death for infants and young children during the Black Death. Larger sample sizes of children would allow for the clarification of patterns of mortality for children during the Black Death. This is particularly important given evidence from historical sources that the second outbreak of plague in 1361 disproportionately targeted children. In order to understand precisely how and why mortality patterns might have changed between the first and second outbreak, it is essential to clarify what the risks for children were during the former.

As noted by Varlık (2014, in this issue), there is variation in the manifestation of plague based on local conditions. Thus, it is crucial to repeat and expand the bioarcheological work that has been done with the East Smithfield sample using samples from other areas to reveal how such variation might have produced geographic or temporal differences in the mortality patterns by age, sex, frailty, and other factors during medieval plague epidemics. Given the Eurocentric perspective common to Black Death scholarship highlighted by contributions to this issue of *The Medieval Globe*, it is particularly important to examine plague mortality using cemetery samples from regions outside of Europe if they become available.

Bioarcheological analysis also allows us to better understand the social responses to the Black Death. For example, Colet et al. (2014, in this issue) describe a cemetery containing the victims of violence perpetrated against the Jewish community of the Catalan town of Tàrrega following the epidemic's arrival in the region. This cemetery provides rare insights regarding the specific victims of the violence perpetrated against the Jewish minority (which was blamed for the disease) and how survivors dealt with the victims' burial. In the case of Black Death cemeteries, such as that of East Smithfield, the organization of the burial ground and the positions of the individuals interred therein are informative about how the living treated the victims of the epidemic and how this treatment might have differed from practices discernible during conditions of normal mortality (Grainger et al. 2008; Hawkins 1990; Kacki et al. 2011). In East Smithfield, for example, we have evidence that individuals were afforded a level of care similar to what they would have received during times of normal mortality (Grainger et al. 2008).

Black Death cemeteries, including and in addition to East Smithfield, also provide opportunities for expanding molecular analyses of the medieval plague pathogen. As mentioned above, the integration of bioarcheological research with paleomicrobiological approaches (e.g., ancient DNA analyses) will provide a powerful tool for understanding changes in plague epidemiology, the effects of other circulating diseases on the emergence and epidemiology of plague, and human-pathogen coevolution, thereby addressing questions of great interest to a variety of researchers, including historians, anthropologists, and evolutionary geneticists. Bioarcheology is ideally suited to bridge historical and molecular research on plague (or, indeed, on any important disease in human history). More than any other field, bioarcheology provides the human context that might be missing from other approaches, but which ultimately makes plague research relevant both to the study of human history as well as to the study of biological variation and responses to infectious disease among living people.

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Sharon N. DeWitte (dewittes@mailbox.sc.edu) is a biological anthropologist with interests in bioarcheology, paleodemography, paleoepidemiology, and infectious and epidemic diseases. For over a decade she has been investigating the mortality patterns, demographic context, and consequences of medieval plague epidemics, including the Black Death of 1347–51. By applying hazard analysis to large skeletal samples from Europe, Dr. DeWitte is able to examine population-level phenomena associated with past crisis mortality events, including the effects of biological factors like age and sex on risks of death during epidemics as devastating as the Black Death, how and why those risks change over time, and how epidemics shape demography and health. Her Black Death research is summarized in publications such as "Selectivity of Black Death Mortality with Respect to Pre-Existing Health," co-authored with James Wood in *Proceedings of the National Academy of Sciences USA* 105 (2008): 1436–41; and "Age Patterns of Mortality during the Black Death in London, A.D. 1349–1350," in *Journal of Archaeological Science* 37 (2010): 3394–3400. She is also interested in the molecular biology and evolution of the pathogen that caused the Black Death, *Yersinia pestis*, and is part of a collaborative project that has produced the first complete genome of the fourteenth-century bacterium (Bos et al. 2011). She is currently an associate professor in the Department of Anthropology at the University of South Carolina.

Abstract Most research on historic plague has relied on documentary evidence, but recently researchers have examined the remains of plague victims to produce a deeper understanding of the disease. Bioarcheological analysis allows the skeletal remains of epidemic victims to bear witness to the contexts of their deaths. This is important for our understanding of the experiences of the vast majority of people who lived in the past, who are not typically included in the historical record. This paper summarizes bioarcheological research on plague, primarily investigations of the Black Death in London (1349–50), emphasizing what anthropology uniquely contributes to plague studies.

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