Setting the Stage for Medieval Plague: Pre-Black Death Trends in Survival and Mortality

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ABSTRACT *OBJECTIVES:* The 14th-century Black Death was one of the most devastating epidemics in human history, killing tens of millions of people in a short period of time. It is not clear why mortality rates during the epidemic were so high. One possibility is that the affected human populations were particularly stressed in the 14th century, perhaps as a result of repeated famines in areas such as England. This project examines survival and mortality in two pre-Black Death time periods, 11–12th centuries vs 13th century CE, to determine if demographic conditions were deteriorating before the epidemic occurred.
MATERIALS AND METHODS: This study is done using a sample of individuals from several London cemeteries that have been dated, in whole or in part, either to the 11–12th centuries ($n = 339$) or 13th century ($n = 258$). Temporal trends in survivorship and mortality are assessed via Kaplan–Meier survival analysis and by modeling time period as a covariate affecting the Gompertz hazard of adult mortality.
RESULTS: The age-at-death distributions from the two pre-Black Death time periods are significantly different, with fewer older adults in 13th century. The results of Kaplan–Meier survival analysis indicate reductions in survival before the Black Death, with significantly lower survival in the 13th century (Mantel Cox $p < 0.001$). Last, hazard analysis reveals increases in mortality rates before the Black Death.
CONCLUSIONS: Together, these results suggest that health in general was declining in the 13th century, and this might have led to high mortality during the Black Death. This highlights the importance of considering human context to understand disease in past and living human populations. Am J Phys Anthropol 000:000–000, 2015. © 2015 Wiley Periodicals, Inc.

The 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. The epidemic was so devastating not just because of its extremely high mortality levels, but also because those deaths were concentrated within a short period of time. For example, in the 4 years the Black Death was at its height in Europe (between 1347 and 1351), it is estimated that the epidemic killed tens of millions of people (30–60% of the affected populations) (Green, 2014). Despite decades of research on the Black Death by scholars in history, economics, anthropology, molecular biology, and other fields, there are still questions about why the Black Death emerged when it did and why mortality rates during the epidemic were so extraordinarily high. This study addresses the latter question by examining demography (and, by inference, health) in pre-Black Death London using skeletal samples that provide data not available from existing historical documents. In particular, what has been missing in studies of pre-Black Death populations are data that are informative about the experiences of those not typically included in medieval documents (such as women and the poor) and about demographic trends (survivorship and mortality rates) prior to 1270. Data from a wider spectrum of the pre-Black Death population and with greater temporal depth are necessary both for answering questions particular to the emergence of the Black Death and for understanding the dynamics of emerging diseases in general. The Black Death provides an excellent opportunity to examine the human context of a catastrophic epidemic to improve our understanding of how biological, demographic, economic, and social factors can shape disease epidemiology.

The bubonic plague bacterium, *Yersinia pestis*, has long been suspected as the cause of the Black Death, though this diagnosis has generated considerable debate (e.g., Scott and Duncan, 2001; Twigg, 1984). Conclusive evidence that *Y. pestis* was the cause of the Black Death was only recently produced by molecular analyses that yielded DNA diagnostic of *Y. pestis* from victims of the mid-14th century epidemic (Bos et al., 2011; Haensch et al., 2010; Schuenemann et al., 2011). Modern bubonic plague is a zoonosis, a disease among animals that is transmissible to humans, and is transmitted by an insect vector (Hinnebusch, 1997). *Y. pestis* is an obligate parasite that can survive only within a mammal or insect. The maintenance of bubonic plague is, therefore, dependent upon the cyclic transmission between animal hosts and the ectoparastites that feed on them

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(Green, 2014; Hinnebusch, 1997; Perry and Fetherston, 1997). Humans typically become infected if they come into contact with infected animals (often rodents) and are bitten by the animals' fleas. Case fatality rates (the proportions of infected individuals who die) resulting from different forms of untreated plague are high, 30% and higher (pneumonic plague has been described as "invariably fatal") (Perry and Fetherston, 1997:58). However, modern plague mortality in terms of the *proportion* of the total population killed is rarely more than 2–3% even in the absence of antibiotic treatment (Plague Rearch Commission, 1907), a level of mortality that is substantially lower than that of the Black Death, which killed 30–60% of the population.

The question of why the Black Death emerged in the 14th century might be answered by analysis of climatological data and an increased understanding of nonhuman animal host population dynamics (Carmichael, 2014; Schmid et al., 2015). Both of these are relevant to reconstructing how medieval plague initially (and perhaps repeatedly) made the transition from animals to humans and how it spread as far and as quickly as it did in the 14th century. Regarding the high mortality levels of the Black Death, it is possible that the epidemic was so deadly, and in particular more deadly than any modern (i.e., post-1890s) outbreak of bubonic plague, because it was caused by a particularly virulent strain of *Y. pestis*. The high mortality during the Black Death might have also occurred because the affected populations were already in relatively poor health. Comparisons of the genome of the 14th-century *Y. pestis* (assembled from DNA fragments isolated from Black Death victims from London) and that of a modern reference strain have not yet revealed any significant functional differences between the strains in regions of the genome associated with virulence (Bos et al., 2011). The lack of compelling evidence that there was something unique, genetically, to the 14th-century strain of *Y. pestis* itself that rendered it more deadly than modern stains suggests that the alternative hypothesis, that the Black Death was a syndemic whose outcome was influenced by pre-existing poor health conditions in affected populations, should be tested using skeletal and historical data. This focus on the characteristics of the medieval human population that might have made it vulnerable to an emerging disease may also prove beneficial in terms of understanding disease dynamics in living populations.

Previous bioarchaeological analyses of Black Death victims and of people who died just before and after the epidemic in London have been informative about the mortality patterns associated with the first outbreak of medieval plague and how demographic and health patterns changed in the aftermath of the epidemic, at least in one locale (DeWitte, 2010; DeWitte, 2014a; DeWitte, 2014b; DeWitte and Hughes-Morey, 2012; DeWitte and Wood, 2008). However, this previous work did not explicitly address the question of why the Black Death was so deadly. There were several factors that could have reduced immune competence in the pre-Black Death population and contributed to the extremely high mortality of the epidemic. Most of the skeletal stress markers that were assessed in a previous study of selective mortality in the East Smithfield Black Death cemetery in London (DeWitte and Hughes-Morey, 2012; DeWitte and Wood, 2008) are associated primarily with nutritional stress and infectious disease. The presence of these stress markers among people buried in the Black Death cemetery adds to documentary evidence that pre-Black Death London was particularly stressed by disease (e.g. tuberculosis, smallpox, leprosy, anthrax, and ergotism) and malnutrition (Behbehani, 1983; Donoghue et al., 2005; Lamb, 1995; Lee, 2009; Manchester, 1984; Sternbach, 2003). Such stressors were likely exacerbated by the crowded population of London (Roberts and Cox, 2003).

Further, before the Black Death, people were at risk of starvation because of famines, at least some of which resulted from excessive rainfall associated with climatic changes (Dawson et al., 2007), and famine has previously been hypothesized as a determining factor in the emergence of the Black Death (DeWitte and Slavin, 2013; Russell, 1966). The period under consideration in this study (1000–1250 CE) falls within and at the end of the Medieval Warm Epoch/Period (alternatively called the Medieval Climate Anomaly and the Medieval Solar Maximum given that a warming trend was not observed globally), a shift in global climate patterns that began in the 10th century and ended in the late-13th century (Brooke, 2014). In England, during the Medieval Warm Period, temperatures were highest at the end of the 12th century, and this was followed by a period of rapid cooling, with minimum temperatures reached by the middle of 15th century (Galloway, 1986). From 1000–1200 CE, economic and demographic growth peaked at a time of relative climatic stability, i.e. when, based on tree-ring data, there were relatively few fluctuations in average temperature and precipitation (Büntgen et al., 2011). Cooling temperatures in the 13th and 14th centuries led to widespread famines across parts of Europe (Büntgen et al., 2011). Crop failure was compounded by the loss of livestock as a result of severe winters and deadly epizootics (infectious disease outbreaks among cattle and sheep, often referred to as murrains) (Jordan, 1996; Slavin, 2012). England experienced over 20 famines and several epizootics between 1000 and 1350 (Farr, 1846). There is evidence of famine every 14 years on average in the 11th–13th centuries. With respect to the time period under consideration in this study (i.e., for which skeletal samples are available, as detailed below), there were seven famines between 1000 and 1100 and also seven between 1101 and 1200; between 1201 and 1250 alone, there were five famines (Farr, 1846).

During the 12th–13th centuries, the English economy experienced growth, but in the beginning of the 13th century, there were rapid declines in the country's prosperity (Campbell, 2005; Campbell, 2010). Importantly, for this study, real wages declined from 1200 to 1300 as prices increased (Campbell, 2010; Clark, 2005; Farmer, 1988). For example, real wages for craftsmen dropped by about 25% between 1200 and 1219 (Clark, 2005). Population growth, and simultaneous increases in taxes, rent, and grain prices in the 12th–13th centuries created stark social inequalities, particular with respect to food availability. The poorer social echelons, who were comprising an ever-growing proportion of the English population, were highly vulnerable to food crises (Bailey, 1998; Campbell, 2005; Curtis, 2014; Kitsikopoulos, 2000), and there is evidence that food crises in pre-industrial populations most severely affect the mortality risks of lower socioeconomic status individuals (Hayward et al., 2012). Because such individuals formed the majority of the medieval English population, and given that malnutrition lowers resistance to disease (Ortner, 1998;

Scrimshaw, 1987), famine and epizootics might have increased frailty (or heterogeneity in frailty), at least among lower socioeconomic strata, in the pre-Black Death population. This might have made a large proportion of the population of England particularly vulnerable to disease and death in the context of the emergence of plague in the 14th century.

One way of assessing frailty (an individual's relative risk of dying compared to other people in the same population; (Vaupel et al., 1979)) within a population is by examining demographic phenomena, specifically patterns of survivorship and mortality at the population level, given that death is the ultimate outcome of poor health (DeWitte, 2014b; Gage, 2005). Examination of demographic trends just before the Black Death has previously relied on historical data, most of which is limited to the late 13th and early 14th centuries and is biased toward adults, males, and wealthy individuals (Jonker, 2009; Poos et al., 2012; Russell, 1958; Smith, 2001). This study extends our view of pre-Black Death demographic trends farther back in time by examining paleodemographic data from skeletal samples that date to the 11th–13th centuries. Further, by using skeletal samples that include people of all ages, both sexes, and higher and lower socioeconomic strata, this study provides information about a wider segment of society than is typically represented in historical documents. Using skeletal samples from London, this study tests the hypothesis that survivorship decreased while overall mortality risks increased in the period before the Black Death, from the 11th to 13th centuries CE. By using demographic patterns as a proxy for underlying health, this study ultimately addresses the question of whether health was declining before the Black Death emerged and therefore contributed to elevated mortality during the epidemic itself.

The Black Death was an emerging disease of the 14th century and has been described as "one of the most dramatic examples ever" of such a disease (Wheelis, 2002:971). Emerging diseases are those that are new to human populations or that are increasing rapidly in frequency or geographic distribution (Feldmann et al., 2002). More than 50 diseases have emerged in recent decades, including Ebola, HIV/AIDS, West Nile Virus, and SARS, and new diseases will almost certainly emerge or reemerge in the future as a result of a number of factors, including human encroachment on new habitats, the speed and scale of global transportation, and the overuse of antibiotics (Cleaveland et al., 2007; Morens et al., 2004; Morse, 1995). The current (at the time of writing) devastating outbreak of Ebola (WHO, 2014) has focused the world's attention on the dangers of emerging infectious diseases, but it is important to recognize that emerging diseases also existed in the past. The persistence of some ancient emerging diseases to the present day, as is the case with the Black Death, allows for an ideal opportunity to examine long-term trends in emerging disease dynamics, the effects such diseases have had on human populations, and the factors that favor the emergence of these diseases.

MATERIALS AND METHODS

Skeletal samples

All skeletal samples for this study come from medieval London cemeteries and are curated at the Museum of London Centre for Human Bioarchaeology. Samples were selected from two non-overlapping periods: an early pre-Black Death period that dates from the 11th through the 12th centuries CE, and a late pre-Black Death period that dates to the beginning of the 13th century (from 1200 to 1250 CE). The sample sizes used from each cemetery are shown in Table 1.

TABLE 1. Sample sizes from and dates of each cemetery used in this study. In the case of cemeteries with multiple phases of use, dates are provided only for the samples included in this study.

	Cemetery	Dates (CE)	Sample size
Early pre-Black Death	St. Nicholas Shambles	1000–1150	197
	Guildhall Yard	1050–1150	17
	St. Mary Spital Period 14	1120–1200	125
Late pre-Black Death	St. Mary Spital Period 15	1200–1250	258

Early pre-Black Death period: 11th–12th centuries CE

The early pre-Black Death sample comes from three cemeteries: St. Mary Spital (SRP98), Guildhall Yard (GYE), and St. Nicholas Shambles (SNS). Based on stratigraphic and documentary data and artifacts, St. Nicholas Shambles dates to the 11th–12th centuries and Guildhall Yard dates to the 11th to early 13th centuries (Bowsher et al., 2007; Schofield, 1997; White, 1988). Burials in Guildhall Yard date to two periods: 1050–1150 and 1140–1230; more precise dates within each of those two periods are not available, thus it is not possible to identify individuals from the latter period (1140–1230) who were buried in the 12th *vs* the 13th century. Therefore, individuals from 1140 to 1230 were excluded from the early pre-Black Death sample in order to prevent temporal overlap with the late pre-Black Death sample described below. This study includes 197 individuals from St. Nicholas Shambles and 17 from Guildhall Yard; this sample represents all the individuals in these two cemeteries from the relevant time period that were preserved well enough to provide data on age using the methods described below. The main cemetery associated with the hospital and priory of St. Mary Spital has been divided into four periods using Bayesian radiocarbon dating: Period 14 (c. 1120–1200), Period 15 (c. 1200–1250), Period 16 (c. 1250–1400), and Period 17 (c. 1400–1539), and there are both single and multiple burials in each period (Connell et al., 2012; Sidell et al., 2007). Periods 14 and 15 are believed to predate the use of the cemetery for infirmary burials (Connell et al., 2012). For the early pre-Black Death sample for this study, a random sample of 125 individuals who were preserved well enough to provide data on age was selected from among the single inhumations (burial type A) from Period 14. The samples (combined $n = 339$) from Guildhall Yard, St. Nicholas Shambles, and St. Mary Spital contain both sexes, all age groups, and a combination of lower status lay individuals, high status lay individuals, and members of religious communities.

Late pre-Black Death period: 13th century CE

The late pre-Black Death sample comes from Period 15 (c. 1200–1250) burials in St. Mary Spital cemetery. A

random sample of 258 individuals, who were preserved well enough to provide data on age, was selected for this study.

As is typical of bioarchaeological research, the samples used in this study are subsamples of the total number of individuals originally buried in the cemeteries. Excavation of St. Mary Spital yielded 10,516 individuals, over half of the estimated 18,000 or so individuals originally buried in the cemetery (Connell et al., 2012). Of the individuals excavated, 6950 were 35% complete and had regions of the skeleton suitable for estimating age or sex, of which 5387 were recorded by the Museum of London into WORD and are thus available to researchers (250 type A burials from Period 14 and 291 type A burials from Period 15). Though the primary focus of this study was adult demographic patterns, it was necessary to include subadults in order to estimate the fertility proxy described below. For this reason, a random sample of the available individuals from Period 14 and 15 type-A burials was selected for analysis. Though excavation of the larger site that included the St. Nicholas Shambles church and cemetery was limited by modern streets, nearly all of the cemetery area was excavated and was available for analysis (White, 1988), and archaeologists conducted a full excavation of the Guildhall site (Bowsher et al., 2007). However, this study includes only those individuals from St. Nicholas Shambles and Guildhall Yard dated to 1000–1200 for whom it was possible to estimate age. Readers should note that the samples might be biased to an unknown degree and thus the results presented here should be viewed with the typical caution reserved for bioarchaeological analyses.

Age estimation

Adult ages were estimated using transition analysis (Boldsen et al., 2002), which minimizes the biases associated with traditional methods of age estimation and provides point estimates of age and individual standard errors for those point estimates, even for older adults (i.e., rather than a broad terminal adult age category). In transition analysis, data from a known-age reference collection are used to obtain the conditional probability, $Pr(c_j|a)$, that a skeleton will exhibit a particular age indicator stage or suite of age indicator stages given the individual's known age. This conditional probability is combined, using Bayes' theorem, with a prior distribution of ages at death to determine the posterior probability that a skeleton in the cemetery sample died at a certain age, given that it displays particular age indicator stages. By combining the conditional probability, $Pr(c_j|a)$, 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 target sample (Boldsen et al., 2002). For this study, transition analysis was applied to skeletal age indicators on the pubic symphysis and the iliac auricular surface and to cranial suture closure as described by Boldsen et al. (2002). The Anthropological Database, Odense University (ADBOU) Age Estimation software was used to determine individual ages-at-death. The ADBOU program uses a conditional probability estimated from the Smithsonian Institution's Terry Collection and an informative prior distribution of ages at death based on data from 17th-century Danish rural parish records (the Gompertz-Makeham parameter estimates for this prior are: $\alpha_1 = 0.01273$, $\alpha_2 = 0.00002478$, and $\beta = 0.01618$). For the hazard analyses described below, point estimates of adult age were used without their associated errors to estimate the parameters of the models. Readers should therefore view the estimated values of the covariate effects as indicative of general trends rather than attending to the specific numerical values.

Ages for immature individuals (i.e., those individuals with at least one unfused epiphysis) were estimated based on epiphyseal fusion, and dental development and eruption (Buikstra and Ubelaker, 1994; Gustafson and Koch, 1974; Moorrees et al., 1969; Scheuer et al., 1980; Scheuer and Black, 2000; Smith, 1991). These ages were used, in combination with adult age estimates, to assess temporal differences in age-at-death distributions across the lifespan and to estimate the fertility proxy, as described below. Immature ages, however, were not included in hazard analysis or survival analysis, for reasons outlined below.

Statistical analyses

Hazard model. The differences in risks of mortality between the two pre-Black Death periods are assessed by pooling the adult (15 years of age and above) data from all cemeteries to estimate the Gompertz–Makeham hazard of adult mortality and modeling time period (early pre-Black Death *c.* 11–12th century = 0; late pre-Black Death *c.* 13th century = 1) as a covariate affecting the baseline Gompertz–Makeham hazard. The Gompertz–Makeham hazard is a parsimonious three-parameter model of human mortality with two components of mortality: an age-independent risk, and an exponentially increasing senescent risk. The hazard function including both these components is: $h(a) = \alpha_1 + \alpha_2 e^{\beta a}$. These components of mortality are competing risks, so surviving one does not affect a person's risk of dying during another component (Gage, 1988; Makeham, 1860; Siler, 1979; Wood et al., 2002). The time-period covariate is modeled using a proportional hazards specification: $h_i(t_i|x_i\rho) = h(t_i)e^{(x_i\rho)}$, where $h_i(t_i)$ is the baseline hazard (i.e. Gompertz–Makeham), t_i is the age of the ith person in years, x_i is the time-period covariate, and ρ is the parameter representing the effect of the covariate on the baseline hazard. The proportional hazards specification does not allow for variation in the effect of the covariate with age (the effect of the covariate is assumed to be constant across age). The advantage of using a hazard model is that it requires the estimation of just a few parameters and thus makes efficient use of the available skeletal data. The model can be applied to relatively small samples, as it smoothes random variation in mortality data without imposing any particular age pattern on the data (Gage, 1988). Two versions of the model were estimated: the full model, including all three Gompertz–Makeham parameters and the covariate parameter, and a reduced model with the age-independent component of the Gompertz–Makeham model set to 0 (reducing the model to the Gompertz model), as previous research has shown that this component often cannot be estimated from paleodemographic data (Gage, 1988; Herrmann and Konigsberg, 2002; Nagaoka et al., 2006).

It is possible to assess the risk of mortality across all ages using the five-parameter Siler model (Siler, 1979), which includes an exponentially decreasing immature risk in addition to the age-independent and senescent

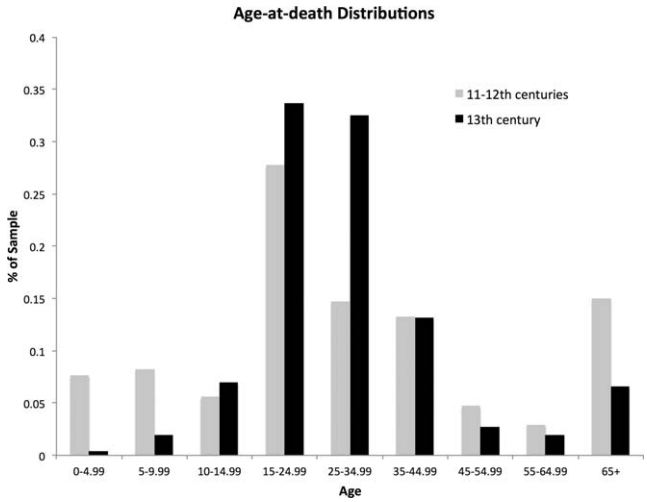

Fig. 1. Age-at-death distributions from the early and late pre-Black Death assemblages.

components of the Gompertz–Makeham model. However, preliminary analyses using the Siler model and including individuals below the age of 15 did not yield good estimates of the parameters of the immature component (the standard errors were large), likely because the available samples sizes for infants and children were too small. Though individuals below the age of 15 were excluded from hazard and survival analyses, they were included in construction of the age-at-death distributions and estimation of the fertility proxy, as described below.

Parameters are estimated using maximum likelihood analysis with the program *mle* (Holman, 2005). A positive estimate for the parameter representing the effect of the time-period covariate would suggest that those in the late pre-Black Death time period were at an elevated risk of death compared to people in the earlier time period. The fit of the full model with the time period covariate compared to a reduced model, in which the value of the parameter representing the time period covariate was set equal to 0 was assessed using a likelihood ratio test (LRT). The LRT tests the null hypothesis that time period was not associated with elevated or decreased risks of death. The LRT was computed as follows: $\mathrm{LRT} = -2[\ln(L_{\mathrm{reduced}}) - \ln(L_{\mathrm{full}})]$, where LRT approximates a χ^2 distribution with $df = 1$.

Kaplan–Meier survival analysis. The effect of time period on survival was assessed using Kaplan–Meier survival analysis with a log-rank test and using pooled data on age from both time periods. Analysis was performed using SPSS version 22. For consistency with the hazard analysis, Kaplan–Meier survival analysis was performed only on adults 15 years of age and above.

Fertility proxy. Given the relatively long period of time across which trends in survival and mortality are examined for this study and because changes in fertility can alter age-at-death distributions even if age-specific mortality does not change (Milner et al., 1989; Paine, 1989; Sattenspiel and Harpending, 1983), this study controls for potential changes in fertility. To control for fertility, this study examines the number of the individuals above the age of 30 divided by the number of individuals above the age of 5 years, i.e., D_{30+}/D_{5+}. Buikstra et al. (1986) found that there is a strong (negative) relationship between D_{30+}/D_{5+} and birth rate, and the 95% comparison intervals reveal whether birth rates differ significantly across samples.

TABLE 2. Maximum likelihood estimates (with 95% confidence intervals in parentheses) of the Gompertz parameters and the effect of the time period covariate (0 = early pre-Black Death and 1 = late pre-Black Death) on mortality (with likelihood ratio test).

Parameter	Estimate (95% CI)	−2LLR
α	0.0095 (0.008, 0.011)	
β	0.032 (0.028, 0.035)	
Time period covariate	0.42 (0.21, 0.62)	20.9 ($p < 0.001$)

RESULTS

Age-at-death distributions

The early and late pre-Black Death age-at-death distributions, for all ages, are shown in Fig. 1; the two distributions are significantly different (Chi-square $p < 0.001$). As can be seen in Fig. 1, there are fewer infants and children below the age of 10 and fewer older adults ages 65 and above in the late pre-Black Death period compared to the earlier period. The late period has more adults between the ages of 15 and 34.99. Analysis of the adult age-at-death distributions (only individuals 15 years of age and above) also reveals a significant difference between the two time periods (Chi-square $p < 0.001$). In particular, there are substantially more adults between ages 25–34.99 and fewer older adults (65+) in the later period.

Hazard model

The results of the hazard analysis are shown in Table 2. These results are for the model with the age-independent component (α_1) of the hazard model set to 0 (i.e., the Gompertz model), as the full model (i.e. the Gompertz–Makeham model) did not yield good estimates of the α_1 parameter. The estimated value of the parameter representing the effect of time period of burial is positive, the corresponding 95% confidence interval includes only positive values, and the results of the likelihood ratio test indicate that inclusion of the covariate improves the fit of the model. These results indicate elevated risks of mortality for adults in the late pre-Black Death period compared to the early period.

Kaplan-Meier survival analysis

The Kaplan–Meier survival curves are shown in Fig. 2, and the mean survival times and corresponding 95% confidence intervals are shown in Table 3. The survival functions reveal lower survivorship among those within the late pre-Black Death period compared to the early pre-Black Death period (Mantel-Cox $p < 0.001$).

Fertility proxy

The fertility proxies, i.e., the D_{30+}/D_{5+} values and their 95% comparison intervals, are shown in Table 4. The D_{30+}/D_{5+} value for the late pre-Black Death sample is lower than that of the early pre-Black Death sample,

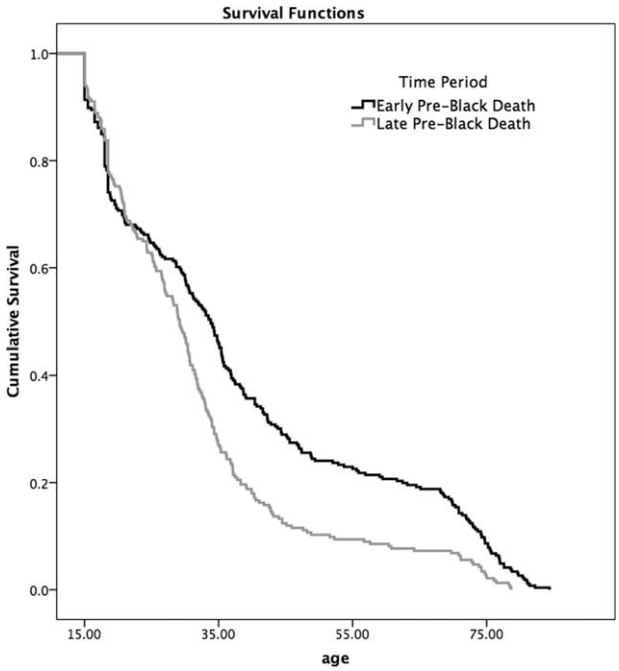

Fig. 2. Kaplan–Meier survivorship curves from the early and late pre-Black Death assemblages.

which might indicate that birth rates increased during the 13th century compared to the earlier period. However, as seen in Table 4, the comparison intervals for the two periods overlap, which indicates a lack of a significant difference in birth rates between them (Buikstra et al., 1986).

DISCUSSION

The results suggest that there were reductions in survivorship and increases in adult mortality in the 13th century compared to the 11th–12th centuries, and that these demographic changes were not an artifact of changes in birth rates over this same time period. Death is the ultimate outcome of poor health, and life expectancy and mortality levels are commonly used to evaluate the general health of living populations (Gage, 2005). Therefore, the demographic trends observed in the pre-Black Death skeletal samples suggest that there were declines in general levels of health in the 13th century compared to the 11th–12th centuries. These declines might have contributed to the extraordinarily high mortality during the Black Death, i.e., making the epidemic more deadly than it might have been had it struck a more robust or resilient population.

This study expands our understanding of the circumstances of the emergence of the Black Death and the timing of demographic changes in England because the bioarchaeological data used here allow for an examination of pre-Black Death demographic trends farther back in time and for a wider sample of the population (in terms of age, sex, and social status) than is possible with existing historical evidence. However, the latest upper limit of the estimated dates (with 95% probability using Bayesian radiometric dating) for Period 15 burials in St. Mary Spital is 1260 CE (this does not include later estimated dates for burials that according to Sidell et al. (2007) cannot be excluded from other periods based on stratigraphic evidence). Thus, there is a lag of several decades between the end date of the late pre-Black Death sample and the emergence of the Black Death in London in 1348. This lag between the samples used in this study and the beginning of the Black Death raises the question of whether the observed downward trend in survivorship and upward trend in adult mortality risk were sustained in the interval and thus whether poor underlying health (as reflected by these demographic measures) was a mechanism that favored extreme excess mortality during the Black Death. With these bioarchaeological data alone, I cannot rule out the possibility that demographic patterns, and by inference, health conditions might have improved between 1250 and 1348. However, there are historical and climatological data that are inconsistent with the suggestion that there might have been demographic improvements in the late-13th and early 14th centuries in England on the eve of the Black Death.

TABLE 3. Kaplan–Meier survival analysis results.

Sample	Mean survival time (years)	95% CI	Mantel-Cox χ^2	p-value
Early Pre-Black Death	38.19	35.7–40.7	18.7	<0.0001
Late Pre-Black Death	31.63	29.7–31.6		

TABLE 4. D_{30+}/D_{5+} values and their 95% confidence intervals for the early and late pre-Black Death samples.

	D_{30+}/D_{5+}	95% CI
Early Pre-Black Death	0.50	0.41–0.59
Late Pre-Black Death	0.43	0.33–0.53

There are several documentary sources that allow for an examination of demographic trends in England before the Black Death. Specifically, these documents provide data for the reconstruction of temporal trends in population sizes from the late 11th century onward and for estimation of life expectancies and mortality rates from the middle of the 13th century onward (Jonker, 2003; Poos and Smith, 1984; Postan, 1950; Razi, 1980; Russell, 1948; Russell, 1958). Historians have long been interested in the timing of medieval demographic decline relative to the Black Death—specifically whether the Black Death was the primary or sole factor that ended population growth and initiated decline or whether the epidemic caused a particularly precipitous drop in population in the context of a preexisting decline (Curtis, 2014; Galloway, 1986). Aggregate population data from sources such as the Domesday Book (*c*. 1086 CE), Hundred Rolls (*c*. 1279), Lay Subsidy Roll (*c*. 1327), and the Poll Tax Returns (*c*. 1377) reveal population growth from the 11th to the mid-13th century followed by decline, at least in some locations in England (Curtis, 2014). According to Smith (2012:49), the "best local evidence" (the earliest of which dates to the 1270s) reveals the beginning of a substantial demographic decline (among males above the age of 12) approximately two generations before the Black Death. In Cambridgeshire, for example, most locations experienced demographic decline beginning in the late 13th century to the early 14th century, though the magnitude of that decline

varied (Curtis, 2014). Hallam's (1988) data on temporal trends in tenant or holdings numbers in England between 1086 and 1317 (and Broadberry et al.'s reassessment thereof) indicate that populations across several regions increased relatively rapidly between 1086 and 1262, but then stagnated afterward through the early 14th century (Broadberry et al., 2011; Hallam, 1988). On some English manors and estates, decline in tenant numbers began even earlier, at the beginning of the 13th century, i.e., during the late pre-Black Death period examined in this study (Langdon and Masschaele, 2006). In general, then, there is historical evidence from several locations in England indicating that populations were declining decades before and continuing to the time of the Black Death. The patterns observed in this study suggest that declines might have occurred even earlier in London than previously thought.

Reductions in population growth can occur because of declines in fertility. The time period under consideration predates (by several centuries) the declines in birth rates associated with the demographic transition in England (Thompson, 1929). However, in the pre-Black Death population, fertility might have dropped during periods of famine (see below) because of amenorrhea induced by poor nutritional status, reduced nuptiality, and other factors (Galloway, 1986). Combined with evidence of declines in life expectancy (described below), this suggests that the downward trend in population before the Black Death should *not* be interpreted as indicating improved conditions in the population from the late 13th century until the Black Death.

According to Russell (1958), there is more material for the study of expectation of life during the medieval period (beginning in the latter half of the 13th century) in England than in any other country. The relevant data come from documents such as the inquisitions *post mortem* (royal inquisitions into the cause of death of people who held land directly from the king that provide a nation-wide sample of the highest rank of landholders) and manorial court records (Poos and Smith, 1984; Razi, 1980; Russell, 1948). These documents are often limited in their representation to adult males and, in the case of inquisitions *post mortem*, higher status individuals (Jonker, 2009; Poos et al., 2012). Based on inquisitions data, Russell (1948) found a general decline in male life expectancy in England for cohorts born between 1276 and 1348 (i.e., 1276–1300, 1301–1325, and 1326–1348) compared to those born before 1276. Jonker (2003, 2009) similarly found that in England, life expectancy at age 25 for those born between 1280 and 1300 was higher than that of individuals born between 1310 and 1323 (though the latter were probably affected by the Black Death). These late-13th–early-14th century patterns suggest that the declines in survivorship between 1000 and 1250 observed in this study did not reverse in subsequent decades.

This study and previous analyses of historical data reveal demographic trends that accompanied and were likely precipitated by changing climatic conditions and consequent factors, such as famine, that were detrimental to health during the medieval period (Campbell, 2011). As mentioned above, the skeletal samples for this study date to a period that falls within and at the end of the Medieval Warm Epoch (Brooke, 2014). In England, temperatures were highest at the end of the 12th century before they cooled rapidly, reaching a minimum by the middle of 15th century (Galloway, 1986). Economic and demographic growth occurred from 1000 to 1200 CE when the climate was relatively stability (Büntgen et al., 2011). Thereafter, however, famines were widespread across parts of Europe as a result of cooling temperatures in the 13th and 14th centuries (Büntgen et al., 2011). This includes one of the best known in human history, the agrarian crisis *c*. 1315–1321 that was caused by back-to-back harvest failure (Campbell, 2010). Decreases in temperature negatively affected agricultural yields by reducing the elevation at which crops could be grown and shortening the growing season; declines in agricultural yields in England and other regions corresponded to decreases in temperature at the turn of the 14th century and continuing until the end of the 15th century (Galloway, 1986). Changing climate conditions also produced excessive summer rainfalls that resulted in crop failures (Dawson et al., 2007), which were compounded by the loss of livestock as a result of deadly epizootics and severe winters (Jordan, 1996; Lamb, 1995). England experienced over 20 famines and several epizootics between 1000 and 1350 (Farr, 1846), including major occurrences of both in 1258 in the aftermath of a massive volcanic eruption (Stothers, 2000), the Great Famine in 1315–1317, and the Great Bovine Pestilence, which killed 62% of bovines in England and Wales between 1319 and 1320 and led to long-term dairy depravations (DeWitte and Slavin, 2013; Jordan, 1996; Slavin, 2012). High humidity during the extraordinarily wet summers also favored fungal diseases that affected not only crop yields but also human health directly (e.g., ergotism caused by ingestion of rye affected by ergot blight) (Dark and Gent, 2001; Lamb, 1995). Famines in the medieval period would have affected human health directly via starvation and indirectly by increasing susceptibility to infectious disease; famines might also have affected health by leading to increased migration, which might have led to exposure of individuals to novel infectious diseases (Galloway, 1986). Heavy taxation and disruption of trade associated with war likely exacerbated the problems of food availability produced by famine (Briggs, 2005; Campbell, 2010).

In summary, the available historical data provide evidence of declining demographic conditions (particularly increased mortality and declines in population growth and size) in England during the 13th and 14th centuries, though the available data are limited primarily to adult males. Further, both historical and climatological sources reveal dramatic climate shifts that affected food availability in the 13th–14th centuries. As mentioned above, what has been missing are data that are informative about the experiences of those not generally included in historical documents (i.e., women, children, and the poor) and about trends in survivorship and mortality prior to the 13th century. Data from a more widely representative sample of the pre-Black Death population and which dates back farther are necessary both for answering questions about long-term trends that might have favored high mortality during the Black Death and for understanding the dynamics of emerging diseases in general. Though this study provides a deeper temporal perspective and includes data on those typically missing from historical documents, it is not directly informative about demography and health in London during the period 1250–1348. However, the skeletal samples used in this study include (though they are not limited to) the individuals best represented in historical documents (adult males). Thus, even though the skeletal data

(*c*. 1000–1250) and the historical data (*c*. 1270–1348) come from two nonoverlapping periods of time, there is partial correspondence in the demographic composition of each dataset. Given the inclusion of adult males in both datasets and the demographic evidence from those historical documents that there was demographic decline and reduced survivorship just before the Black Death, it is parsimonious to conclude that deterioration of demographic conditions, and by inference declines in health, began earlier than 1270 and continued to the time of the Black Death.

The declines in survivorship and increases in mortality observed in this study, from 1000 to 1250, might have been the result of reductions in food availability or in dietary quality, or the result of widespread infection with various diseases or syndemic interactions thereof, or a combination of these or other factors. Teasing these possible causes apart is important for improving our understanding of the biological, social, and economic factors that shape the outcome of disease. However, disentangling the effects of several plausible influential factors in the pre-Black Death population awaits further study of diet and disease at the individual and population levels in these and other samples.

As mentioned above, there is historical evidence of population growth in England from the 11th to the mid-13th century; however, the estimates of the fertility proxies for this study indicate there were no significant differences in birth rates between 1000 and 1200 *vs* 1200 and 1250, as might be expected under conditions of population growth. If fertility was higher in 1200–1250 than it was in 1000–1200, then the results of this study might be an artifact of more young people being introduced into the 1200–1250 cemetery sample with each birth cohort rather than real increases in mortality and reductions in survivorship. However, there are some issues with the available historical data on demographic trends in the medieval period, one of which is the problem of determining the timing and evaluating the consistency of population growth. Inferences about population growth in medieval England are most often based on sources that are widely separated in time, such as the Domesday Book (1086) and 1377 poll tax records. The population in 1377 was higher than it was in 1086, which indicates population growth in the interim. In the absence of documentary data, many historians have assumed that populations peaked in 1300 before being reduced as a result of the mid-century Black Death (Langdon and Masschaele, 2006). However, there is a debate about temporal trends in population growth and about when population sizes actually peaked (Langdon and Masschaele, 2006). In the case of London, its population is thought to have peaked in 1300 (Keene, 2000; Keene, 1989). However, even if the population of London did peak in 1300, this does not necessarily mean that there was uniform, constant growth up to that time or that growth was fueled by fertility. In fact, population growth in London (and in towns in general) during the medieval period was, according to several scholars, the result of migration into the city rather than intrinsic growth (Connell et al., 2012; Kowaleski, 1988). If this is true, then the results from this study indicating a lack of significant changes in birth rates across the time period under consideration are not at odds with evidence of population growth.

Further, there is evidence that patterns of population growth were not uniform across England (Broadberry et al., 2011; Hallam, 1988; Langdon and Masschaele, 2006). Though there is evidence (with good temporal control) of sustained population increases in many locales in England between 1086 and the late 13th century, on some English manors and estates, there was stagnation or decline in tenant numbers in the early 13th century (Langdon and Masschaele, 2006). In the East Midlands, for example, though the population grew from 1149 to 1230, there was stagnation between 1230 and 1262 and decline from 1262 to 1292. On Glastonbury manors, there was stagnation and population decline from 1189 through the mid-13th century (Hallam, 1988). According to Langdon and Masschaele (2006:62) "trends were geographically specific" and "rates of growth could vary quite substantially even between manors on the same estate". Thus, generalizations about population growth in the period before the Black Death might mask important local conditions. Perhaps in London, there was stagnation in population growth or even population decline in the first half of 13th century because of increases in mortality even if fertility remained the same. Because all of the estimates reported in this article are derived from the same skeletal samples, I have emphasized the birth rates estimated from the skeletal samples over population growth estimates from historical documents for the purposes of contextualizing the findings of this study. However, changes in birth rates cannot be ruled out as a confounding factor with complete certainty. Future research on skeletal stress markers can potentially provide additional insights into temporal trends in health during the time period considered here.

In addition to improving our understanding of the characteristics of the pre-Black Death population, at least in London, one larger goal of this study is to emphasize the importance of focusing on the human context of disease, i.e., examining large-scale interactions of biological, social, economic or political factors that influence human health (Armelagos, 1998; Goodman and Leatherman, 1998; Singer and Clair, 2003). The human context of disease includes more than the effect of a disease on the human body and morbidity and mortality patterns during disease outbreaks. It also includes the characteristics of human populations that might affect the emergence, transmission, severity, and persistence of diseases. The current (at the time of writing) outbreak of Ebola, which began in December 2013, highlights the crucial role of human behavior in shaping the effects of disease epidemics. Previous outbreaks of Ebola were devastating to local communities because of their high case-fatality rates. However, the current outbreak has had an unprecedented number of victims and a wide geographic distribution (Agusto et al., 2015), with cases and deaths totaling nearly ten times those from all other outbreaks since 1976 combined (Tomori, 2015). According to the World Health Organization, the current outbreak does not differ from previous ones in terms of the clinical course of the disease, transmissibility, case fatality rates, and other epidemiological variables (WHO, 2014). Genomic analysis of the variant of the virus responsible for the outbreak confirms that it does not differ from other variants in ways that would enhance virulence or transmissibility (Azarian et al., 2015; Olabode et al., 2015). That is, the disease itself has not changed; instead the current epidemic is so devastating and widespread because of factors such as easy travel between rural and densely populated urban areas, failed

surveillance systems, delayed and insufficient control measures, an inadequate health care delivery system, and traditional beliefs regarding treatment of the dead (Abramowitz et al., 2015; Agusto et al., 2015; Tomori, 2015; WHO, 2014). Human behavior has clearly played an important role in spreading the disease (Agusto et al., 2015). Ebola viruses are highly transmissible by direct contact with virus-infected body secretions, fluids or tissues from infected individuals (Bausch et al., 2007), and thus traditional West African funeral practices that include washing, touching, and kissing the deceased have promoted transmission of the disease (in both the current and previous outbreaks) (Hewlett and Amola, 2003; Muyembe-Tamfum et al., 1999; Pandey et al., 2014). Efforts to contain outbreaks of Ebola have also been thwarted by healthy people refusing to be quarantined for fear of becoming deliberately infected while under quarantine, people with symptoms failing to seek medical treatment because of fears of not seeing family following admission to the hospital, family members hiding infected loved ones from health-care workers because of fears that normal burial rituals will not be carried out, and family members caring for sick relatives without adequate personal protective equipment (Abramowitz et al., 2015; Agusto et al., 2015; Hewlett and Amola, 2003).

Importantly, the scale of the current epidemic was not inevitable. Ebola has caused over 11,000 deaths in Guinea, Liberia, and Sierra Leone, but it was also introduced into large cities in Nigeria, where it only caused 8 deaths (Centers for Disease Control and Prevention, 2015). The WHO highlights the swift implementation of rigorous control measures in Nigeria to explain this discrepancy (Shuaib et al., 2014; WHO, 2014), and Tomori (2015: 2) describes "fortuitous circumstances" that were unique to the introduction of the disease to Lagos (i.e., the index case was a diplomat who was symptomatic when he arrived in Lagos, and he was immediately admitted to private hospital). It is clearly important to understand how the human context of modern diseases like Ebola affects disease outcomes so that proper preventative measures can be taken in living populations. It is also important to examine these phenomena in past populations, such as in the case of the Black Death, to more fully understand the range of variation in interactions between human factors (human biology and economic, social, and political conditions) and disease throughout human history.

CONCLUSION

The results of this study indicate that survivorship declined and risks of mortality increased, at least for adults, in London in the 13th century compared to the 11th–12th centuries. These demographic changes occurred at a time of dramatic climate change, and might reflect general deterioration in health as a consequence of periodic, severe food shortages that resulted from changing climate conditions. The demographic changes might also reflect the effects of numerous diseases that were circulating in the population at the time, or the interaction of famine and disease. Whatever the precise cause(s) of the apparent declines in demography and, by inference, health in the 13th century, the end result might have been a population that was extremely vulnerable to the effects of the Black Death. In addition to improving our understanding of the circumstances that might have contributed to extraordinarily high mortality during the Black Death, this study highlights the importance of considering the human context of disease. The Black Death, like Ebola, is a dramatic example of an emerging infectious disease, and thus the particulars of its emergence and epidemiology might not be directly applicable to responding to diseases in living populations. However, the evidence at hand thus far indicates that human factors might strongly influence the trajectory of epidemics even in the case of catastrophic epidemic diseases. Improved understanding of the components of human populations that can influence disease outcomes, and how those have done so throughout human history, might prove beneficial to living populations.

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