Health in Post-Black Death London (1350–1538):
Age Patterns of Periosteal New Bone Formation in a
Post-Epidemic Population

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ABSTRACT Previous research has shown that the Black Death targeted older adults and individuals who had been previously exposed to physiological stressors. This project investigates whether this selectivity of the Black Death, combined with post-epidemic rising standards of living, led to significant improvements in patterns of skeletal stress markers, and by inference in health, among survivors and their descendants. Patterns of periosteal lesions (which have been previously shown, using hazard analysis, to be associated with elevated risks of mortality in medieval London) are compared between samples from pre-Black Death (c. 1,000–1,300, n = 464) and post-Black Death (c. 1,350–1,538, n = 133) London cemeteries. To avoid the assumptions that stress markers alone provide a direct measure of health and that a change in frequencies of the stress marker by itself indicates changes in health, this study assesses age-patterns of the stress marker to obtain a more nuanced understanding of the population-level effects of an epidemic disease. Age-at-death in these samples is estimated using transition analysis, which provides point estimates of age even for the oldest adults in these samples and thus allows for an examination of physiological stress across the lifespan. The frequency of lesions is significantly higher in the post-Black Death sample, which, at face value, might indicate a general decline in health. However, a significant positive association between age and periosteal lesions, as well as a significantly higher number of older adults in the post-Black Death sample more likely suggests improvements in health following the epidemic. Am J Phys Anthropol 155:260–267, 2014. © 2014 Wiley Periodicals, Inc.

The Black Death, the first outbreak of medieval plague that swept through Europe between 1347 and 1351, was one of the most devastating epidemics in human history. The epidemic killed an estimated 30–50% of populations in affected regions, which amounted to tens of millions of people (Poos, 1991; Cohn, 2002; Wood et al., 2003). This single, massive, extremely rapid depopulation event initiated or enhanced social, demographic, and economic changes throughout the continent, and thus has attracted the interest of a variety of researchers for decades (Twigg, 1984; Cohn, 2002; Hinde, 2003).

My previous research examined the selectivity of Black Death mortality, i.e., whether the medieval epidemic targeted particular individuals or whether, as is often assumed given its very high mortality levels, it killed indiscriminately. This previous research was done using individuals excavated from the East Smithfield Black Death cemetery in London. East Smithfield was established specifically for use during the Black Death and was only used while the epidemic lasted in London, from 1349 to 1350 (Hawkins, 1990). East Smithfield is one of a very small number of large, excavated cemeteries that has both excellent documentary and archaeological evidence linking it clearly to the Black Death (Grainger et al., 2008). Using a sample from East Smithfield, I examined sex and age patterns of Black Death mortality and whether the Black Death disproportionately targeted people who had been exposed to physiological stressors before the epidemic (and thus, by inference, whether the Black Death was selective with respect to frailty). The results of this research indicated that the Black Death was not an indiscriminate killer, but rather that people varied in their risks of dying during the epidemic (DeWitte and Wood, 2008; DeWitte, 2010; DeWitte and Hughes-Morey, 2012). In particular, older adults and people of all ages who had been exposed to physiological stressors (and had developed skeletal lesions or exhibited short adult stature as a result of responses to those stressors) before the Black Death were more likely to die during the epidemic than their younger peers or their age-peers who lacked skeletal stress markers. Given that the skeletal stress markers that I selected for this previous work were shown by my own research and that of others (e.g., Usher, 2000) to be associated with elevated risks of mortality, it is safe to say that the stress markers are, at least under the circumstances examined in these studies, good indicators of health (or frailty) more generally, and thus that the Black Death targeted people in poor health.

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The extraordinarily high and selective nature of Black Death mortality raises questions about how it might have shaped patterns of health and demography in the surviving population.

By targeting frail individuals of all ages, the Black Death might have exerted a strong selective force on the European population, selecting out the weakest individuals in huge numbers. Historical documents indicate that plague mortality declined steeply between the initial outbreak in 1347–1351 and the second outbreak in 1361 and that mortality levels remained lower in subsequent plague outbreaks throughout the medieval and early modern periods (Russell, 1966; Cohn, 2008). Such changes in mortality patterns following the initial Black Death might mean that people who survived the first outbreak and their descendants were generally less frail and less likely to die from a variety of causes compared to the pre-epidemic population. Survivors of the Black Death might have had characteristics (e.g., strong immune systems or beneficial genetic traits) that conferred resistance to plague and other diseases. If this was the case, it is possible that medieval plague had positive (though perhaps short-lived) effects on population-level patterns of health similar to those observed following modern plague epidemics; e.g., in the early 20th century in India, all-cause mortality dropped in the years following plague outbreaks with high mortality (Office, 1915).

Decreases in plague mortality levels after the Black Death might mean that the pathogen responsible for the epidemic, and subsequent plague outbreaks, evolved in ways that made it less virulent. Researchers have long suspected, based primarily on similarities of some symptoms between the medieval and modern diseases, that the Black Death was caused by the same pathogen responsible for modern bubonic plague, the bacterium *Yersinia pestis*. Over the last few years, these suspicions have been confirmed by ancient DNA studies that have found molecular evidence of *Y. pestis* in the skeletal remains of documented or putative victims of medieval plague (Drancourt et al., 1998; Raoult et al., 2000; Bos et al., 2011; Kacki et al., 2011; Ny Tran et al., 2011; Schuenemann et al., 2011). Such molecular evidence has the potential to provide insights into how *Y. pestis* epidemiology has changed over time. However, a recently published mortality study of 14th-century *Y. pestis* revealed no unique derived positions in the ancient compared to the modern reference strain in regions of the genome associated with virulence (Bos et al., 2011). Given that pathogen virulence is the result of host-pathogen interaction and not simply a characteristic of the pathogen itself (Casadevall and Pirofski, 2001; André and Hochberg, 2005), these genetic results suggest that factors other than, or in addition to, evolution of the pathogen itself were at play in the changing epidemiology of plague.

Furthermore, there is evidence from historical documents that standards of living improved after the epidemic, at least in some areas of Europe such as England, resulting in large part from the massive depopulation caused by the Black Death. Following the Black Death, there was a severe shortage of laborers and consequently wages improved dramatically and prices for food, goods, and housing fell (Bailey, 1996). The post-Black Death period saw the decline of serfdom, a major redistribution of wealth (with real wages rising to levels that were not exceeded until the 19th century), and improvements in housing and diet for people of all social status levels (Postan, 1950; Hatcher, 1977; Rappaport, 1989; Poos, 1991; Dyer, 2002; Stone, 2006). Grain prices in England dropped steeply after 1375 and generally remained low for almost a century and a half thereafter (Dyer, 2005). In England it took several years for real wages to rise, and in fact they may have dropped in the immediate aftermath of the epidemic, but by the late 14th–century real wages had risen sharply to their medieval peak (Murno, 2004); in the late 15th century, real wages were at least three times higher than they had been at the beginning of the 14th century (Dyer, 2005).

To deal with a pool of workers that was not only smaller than that which existed before the epidemic but which had more opportunities for both mobility and new employment if existing conditions were unsatisfactory, employers also increased payments in kind (such as extra food and clothing) after the epidemic to attract workers (Bailey, 1996).

Given the effects of nutrition on immune competence (Scrimshaw, 2003), dietary improvements (and decreases in social inequities in access to food that might have strongly benefitted the lower social echelons who made up the bulk of the English populace), in particular might have acted to reduce average levels of frailty within England. Following the Black Death, there were increases in the amount of money spent per capita on food, and people ate higher quantities of relatively high quality wheat bread and meat and fish, much of which was consumed fresh rather than salted as had been common prior to the epidemic (Dyer, 2005). Such changes probably improved the nutritional quality of the diet (Dyer, 2005), and given that the diet of lower classes became more similar to that of high status individuals, this meant that following the Black Death, a greater proportion of the English population was consuming a nutritious diet than had been true before the epidemic.

This study examines whether the selectivity of the Black Death, combined with rising standards of living after the epidemic, resulted in a healthier post-epidemic population. In particular, it examines the temporal changes, from the pre- to the post-Black Death periods, in the frequencies and age patterns of periosteal lesions. It is important, particularly in the context of this issue, to emphasize that I am being careful to avoid relatively simplistic interpretations of skeletal stress markers and that dealing consistent with the principles advocated by Wood et al. (1992). In their seminal paper over 20 years ago, Wood et al. (1992) challenged paleodemographers to more carefully consider the effects of heterogeneous frailty and selective mortality on skeletal assemblages by using models that account for within-population variation in risks of death. One facet of their argument—first articulated by Ortner (Ortner and Auferheide, 1991)—is that skeletal lesions might not, under all circumstances, reflect poor health. This is based on the fact that the skeletal stress markers commonly used by bioarchaeologists to evaluate health take a long time to form to the extent that they are visible to the naked eye. Thus, an individual who exhibits a particular stress marker would have been at least sufficiently healthy enough to survive long enough to develop the marker; a very frail individual, on the other hand, might have succumbed to the stressor before developing a stress marker in response. In this scenario, the presence of a stress marker might actually indicate relatively good health, whereas the absence of a stress marker might indicate both very low frailty (e.g., someone who was not exposed to a particular stressor or who
fought off infection before formation of a lesion occurred) or someone with very high frailty (i.e., who died relatively quickly following exposure to the stressor).

Given the uncertainty typically inherent in using skeletal stress markers, for this study, I do not assume that stress markers by themselves indicate poor health. Rather, I am incorporating into my analyses a stress marker that I previously established, using samples from both medieval London and medieval urban centers in Denmark, to be associated with elevated risks of mortality (DeWitte and Wood, 2008). Because death is clearly a poor health outcome, periosteal lesions in this population do indicate relatively poor health. If the Black Death affected general levels of health, there should be significant differences in the patterns of periosteal lesions between the pre- and post-Black Death populations.

In this study, I also avoid the assumption that temporal differences in raw frequencies of periosteal lesions alone are indicative of changes in general patterns of health. Research in fields such as demography, gerontology, and public health has addressed health as a multidimensional phenomenon with physical, mental, and social domains, and including factors such as physical functioning, emotional problems, social functioning, pain, energy, and fatigue (Molla et al., 2003). A change in the frequency of a stress marker might indicate changes in the proportion of individuals in a past population that were free from disease, which is how health is frequently defined in medicine (Gage and DeWitte, 2009). However, importantly, such an interpretation does not account for the fact that not all disease states leave skeletal evidence. Furthermore, an increase or decrease in frequencies of skeletal stress markers is not necessarily directly informative about other aspects of health, such as an individual’s ability to perform the activities of daily life or risk of dying (death being the ultimate unhealthy state). Moreover, research in gerontology has shown that in living populations, improvements in mortality and longevity (both of which are used as markers of general improvements in health) are not always associated with being free of disease or debilitating conditions (Crimmins et al., 1994; Molla et al., 2003; Crimmins, 2004; Ailshire et al., 2011). Bioarchaeologists are increasingly recognizing the need to go beyond simple counts of pathologies (e.g., see Klaus in this issue regarding the importance of considering age-at-death distributions in bioarchaeological investigations of pathologies in general), as using frequencies of stress markers as an isolated measure of health ignores the multidimensionality of health and the potential for a complex relationship between morbidity and mortality. Therefore, for this study, in an effort to examine health in a more nuanced manner than is possible with an examination of raw frequencies of lesion alone, I analyze stress markers in the context of age-at-death distributions to assess how both were shaped by the Black Death and how changes in the age-structure of the population might have affected population-level patterns of stress markers.

**MATERIALS AND METHODS**

**Skeletal samples**

The skeletal samples for this study are all from medieval London cemeteries. Because the pre- and post-Black Death samples are drawn from a single population, I avoid the potential confounder of population differences, and thus observed differences between the two time periods can be attributed to the effects of the Black Death.

**Pre-Black Death cemeteries: St. Mary Spital, Guildhall Yard, and St. Nicholas Shambles.** The pre-Black Death sample comes from three cemeteries, St. Mary Spital, Guildhall Yard, and St. Nicholas Shambles. Guildhall Yard and St. Nicholas Shambles date to the 11–12th centuries, based on stratigraphic and documentary data and artifacts (White, 1988). 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). I selected a random sample of 200 individuals from among the single inhumations (burial type A) from Periods 14 and 15, both of which are believed to predate the use of the cemetery for infirmary burials (Connell et al., 2012). This study uses a combined sample of 464 individuals from these three cemeteries; this sample represents all of the individuals in the St. Nicholas Shambles and Guildhall Yard cemeteries that were preserved well enough to provide data on age using the methods described below, as well as the random sample from St. Mary Spital (who were similarly well preserved). A smaller subset of individuals (n = 222) from the combined sample provided data on tibial periosteal lesion presence/absence.

**Post-Black Death cemetery: St. Mary Graces.** The post-Black Death sample comes from the cemetery associated with the Cistercian Abbey of St. Mary Graces, which was established in London in 1350, soon after the Black Death ended, and it was in use until the Reformation in 1538 (Grainger and Hawkins, 1988; Grainger et al., 2008). This study uses a sample of 133 individuals from St. Mary Graces who were preserved well enough to provide data on age using the methods described below. A smaller subset of individuals (n = 81) from the combined sample provided data on tibial periosteal lesion presence/absence.

**Age estimation**

Adult ages (generally 20 years and older) were estimated using the method of transition analysis described by Boldsen et al. (2002). The advantages of transition analysis include the avoidance of age estimates that are biased toward the known-age-reference sample, as occurs with traditional methods of age estimation, and the estimation of point estimates of age and individual standard errors for those point estimates, even for older adults (i.e., rather than the broad terminal adult age categories typical of traditional methods). It therefore allows for an examination of patterns of skeletal stress markers even at advanced adult ages.

In transition analysis, data from a known-age reference collection (in this case, the Smithsonian Institution’s Terry 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’ theorem, this conditional probability is combined with a prior distribution.
of biological stress. As mentioned above, my previous formation, supports the use of these lesions as markers reveals the highly complex nature of periosteal lesion pathophysiology of periosteal lesions, which, though it often traumatize the periosteum and as a result of local or systemic infection or inflammation associated with a variety of factors (Larsen, 1997; Ortner, 2003; Weston, 2008). Periosteal lesions are estimated based on epiphyseal fusion, and dental development and eruption (Moorrees et al., 1969; Gustafson and Koch, 1974; Scheuer et al., 1980; Smith, 1991; Buikstra and Ubelaker, 1994; Scheuer and Black, 2000).

**Periosteal lesions**

Periosteal new bone formation (a periosteal lesion) occurs in response to stimuli that tear, stretch, or otherwise traumatize the periosteum and as a result of local or systemic infection or inflammation associated with a variety of factors (Larsen, 1997; Walton and Rothwell, 1983; Ortner, 2003; Weston, 2008). Periosteal lesions are often identified macroscopically and scored as present if the there was at least one distinct patch, of any size, of woven or sclerotic bone (or a combination of the two) laid down on the surface of the diaphysis. The periosteal lesions were scored as woven if the patch of bone appeared porous with sharp, unremodeled edges; lesions were scored as sclerotic if the patch of bone had rounded, remodeled edges (Weston, 2008). Individuals with a combination of women and sclerotic new bone formation were given a score of “mixed”. For this study, the woven/sclerotic/mixed scores were pooled into a score of “present” to maximize the sample size for analysis. Only tibiae with anterior diaphyseal surfaces that were free of both periosteal new bone growth and postmortem damage were scored as lacking periosteal lesions; tibiae with no visible lesions but with postmortem damage that prevented visual assessment of the anterior surface were given a score of “unobservable” with respect to periosteal lesions and thus excluded from analyses. Both right and left tibiae, if present, were scored for periosteal lesions during data collection; however, only data from the left tibia were used in the analyses described here, as this maximized the sample size for analysis.

**Statistical analyses**

Differences in the frequencies of periosteal lesion presence were assessed using a Chi-square test. The relationship between age and the presence of periosteal lesions in the medieval population of London is assessed using binary logistic regression. To estimate odds ratios using binary logistic regression, data on age (point estimates) and periosteal lesion presence were pooled from the pre- and post-Black Death samples. The effect of periosteal lesions on survival was also assessed using Kaplan-Meier survival analysis with a log rank test and using pooled data (point estimate of age and periosteal lesion presence) from both time periods. Differences in the age-at-death distributions between the pre- and post-Black Death samples are assessed using a Kolmogorov-Smirnov test. All statistical analyses were done using IBM SPSS Statistics version 20.

**RESULTS**

The overall frequencies of periosteal lesions in the pre- and post-Black Death samples are 54.5% and 67.9%, respectively. The frequency of periosteal lesions is

![Fig. 1. Kaplan-Meier survival functions for individuals with and without periosteal lesions.](image)
The higher frequency of periosteal lesions in the post-Black Death sample indicates, at face value, that health deteriorated after the Black Death. However, interpretation of temporal changes in periosteal lesion frequencies in medieval London requires consideration of the relationship between age and periosteal lesions and the age structure of the pre- and post-Black Death samples given that positive associations between periosteal lesions and age have been observed in other bioarchaeological investigations. For example, Rose and Hartnady (1991) found an increase of tibial periosteal lesions with age in adults in a historic Afro-American cemetery from Arkansas, which they interpreted as suggesting diminishing disease resistance in the population. In a medieval sample from northern England, Grauer (1993) found that the frequency of periosteal lesions increases with older adult age, which she suggests indicate accumulation of nonlethal conditions with age. Grauer also suggests that the age-associated increase in the frequency of such lesions might represent a selection process whereby individuals capable of withstanding certain physiological stressors survive to adulthood with skeletal evidence of the past stressors. More generally, Klaus, in this issue, argues for the consideration of age-at-death distributions when analyzing skeletal pathologies, as a population with a high proportion of older adults is one in which people would have collectively had more time to accumulate certain skeletal pathologies.

The results of binary logistic regression using the pooled data from the pre- and post-Black Death samples reveal that periosteal lesions were positively associated with age in medieval London, and the Kolmogorov-Smirnov test indicates that the proportion of older adults is significantly higher in the post-Black Death sample. The latter result is consistent with Russell’s (1966) finding, based on cemetery data primarily from central eastern Europe, that the ratio of individuals above the age of 60 relative to those between ages 20–60 increased after the Black Death. Together, these results indicate, perhaps unintuitively, that the increased frequency of periosteal lesions in the post-Black Death sample reflects a healthier population in general if we define health in terms of longevity and not simply in terms of the absence of a particular disease (or in this case, several diseases or other physiological stressors that can cause periosteal lesions).

Improvements in survival and, by inference, health following the Black Death might have occurred because of improvements in diet or lower average frailty within the population resulting from the strong selective effects of the epidemic. However, such positive changes in longevity, with more people surviving to later adult ages, resulted in a larger portion of the population exhibiting periosteal lesions, which, as observed here and in

### Table 1. Joint probability distributions of age and periosteal lesions

<table>
<thead>
<tr>
<th>Age at Death</th>
<th>Pre-Black Death</th>
<th>Post-Black Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>No lesion</td>
<td>Lesion</td>
<td>No lesion</td>
</tr>
<tr>
<td>0–14.99</td>
<td>0.109</td>
<td>0.109</td>
</tr>
<tr>
<td>15–29.99</td>
<td>0.159</td>
<td>0.159</td>
</tr>
<tr>
<td>30–44.99</td>
<td>0.109</td>
<td>0.194</td>
</tr>
<tr>
<td>45+</td>
<td>0.08</td>
<td>0.095</td>
</tr>
</tbody>
</table>

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other studies, are (at least in some circumstances) positively associated with age. Perhaps lower average frailty in the post-Black Death population meant that fewer people died from certain physiological stressors that lead to periosteal lesions, and thus individuals were more likely to survive to later adult ages with those lesions than was true of the pre-Black Death population. In the pre-Black Death population, people might have been frailer on average and more likely to die from those stressors, potentially entering the skeletal sample at relatively young ages with or without the associated skeletal lesions.

Enhanced longevity might also have led to increasing frequencies of periosteal lesions at older ages because, as a consequence of living longer, individuals spent more years accumulating the effects of nonfatal physiological stressors that lead to such lesions. The positive relationship between age and periosteal lesions might reflect the chronic inflammatory status of individuals in the medieval samples, as aging is associated with systemic low-grade inflammatory activity in living populations (Brittensgaard et al., 2003). This phenomenon has been suggested to explain the age pattern of periodontal disease, which, as is often the case with periosteal lesions, has an inflammatory etiology. According to Benatti et al. (2009), periodontal disease (and other age-associated diseases) increase with age because of an underlying chronic inflammatory status; in the case of periodontal disease, as people age, the accumulated effects of years of exposure to oral pathogens and their byproducts lead to elevated levels of pro-inflammatory cytokines (which are associated with periodontal tissue destruction) and the reduced production of anti-inflammatory cytokines in periodontal tissues. Pro-inflammatory cytokines also play a role in the proliferation of bone, at least in response to stimuli such as trauma (Frost et al., 1997; Lange et al., 2010). If inflammatory status changed with age in medieval London, and inflammatory status is associated with periosteal lesions, enhanced survival in the post-Black Death population might have increased the proportion of individuals with chronic inflammation and thus a high probability of developing periosteal lesions.

This possible scenario—enhanced survival, but consequentially relatively poor skeletal health at late adult ages—is similar to trends that have been observed in some living populations. Until recently, researchers assumed that improvements in longevity overall and in mortality for the elderly reflected improvements in health at late ages; however, numerous studies have revealed that improvements in mortality and longevity are actually often associated with declines in health at older adult ages (Crimmins et al., 1994; Molla et al., 2003; Crimmins, 2004). For example, mortality in the US decreased between 1970 and 1990, but the prevalence of conditions such as heart disease, cancer, and diabetes increased among older adults during that same period. According to Crimmins et al. (1994), in general, improvements in mortality that occur without concomitant improvements in morbidity can lead to higher proportions of nonfunctioning (i.e., unhealthy) individuals in living populations. For example, with respect to heart disease, a leading cause of death in many living populations, there has been a “tradeoff of mortality for morbidity” (Bonneux et al., 1994: p 20). Even when the incidence of heart disease decreases, the prevalence among older adults increases as people survive longer in a morbid state because of improvements in medical care (Crimmins, 2004). Some studies in living populations have found that increasing longevity is associated with increased health at the latest adult ages (Doblhammer and Kyri, 2001; Ailshire et al., 2011), so a decline in health among the elderly is not an inevitable consequence of improvements in survival and mortality. Nonetheless, perhaps in the population of London that lived just before and after the Black Death the prevalence of conditions that led to periosteal lesions remained unchanged, and thus the frequency of such lesions increased as people survived to later adult ages following the epidemic.

CONCLUSIONS

Improvements in longevity after the Black Death indicate improvements in population health overall in medieval London. However, these population-wide patterns mask variation in health that might have existed by age; i.e., improvements in health were not necessarily uniform across all ages. The results of this study indicate that people were living longer after the Black Death, but as a consequence were likely to experience declines in health, as reflected by periosteal lesions, at the latest adult ages.

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LITERATURE CITED


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