

Frailty and Famine: Patterns of Mortality and Physiological Stress Among Victims of Famine in Medieval London

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ABSTRACT

Objectives: Famine can be defined as a shortage of foodstuffs that instigates widespread excess mortality due to starvation, infectious disease, and social disruption. Like other causes of catastrophic mortality, famine has the potential to be selective. This study examines how famines in medieval London were selective with respect to previous stress, age, and sex.

Methods: This study compares famine burials to nonfamine (attritional) burials from the St Mary Spital cemetery, London (c. 1120–1540 AD). We evaluate the associations between age, sex, and skeletal stress indicators [cribra orbitalia, linear enamel hypoplasia (LEH), and periosteal lesions] using hierarchical log-linear analysis. Additionally, sex is modeled as a covariate affecting the Gompertz hazard of mortality.

Results: Significant associations exist between famine burials and LEH and between attritional burials and periosteal lesions, independent of age or sex. Sex did not significantly affect risk of mortality in the 12th–13th centuries. However, males interred in attritional burials c. 1400–1539 AD faced a lower risk of mortality compared to females.

Discussion: The LEH results suggest that early exposure to stressors increased frailty in the context of famine. The periosteal lesion results suggest that individuals were more likely to survive stressors and thus form these lesions under nonfamine conditions. Hazard analysis suggests that a cultural or biological transformation during this period affected sex differences in mortality. Possible causes include the selective mortality during the Black Death, which might have influenced risks of mortality among survivors, or unequal distribution of improvements in standards of living after the epidemic. *Am J Phys Anthropol* 160:272–283, 2016. © 2016 Wiley Periodicals, Inc.

For years, historians, economists, anthropologists, and others have been interested in periods of exceptionally high mortality, i.e., crisis or catastrophic mortality (e.g., Torry, 1984; Boyle and Ó Gráda, 1986; Campbell, 1992; DeWitte and Wood, 2008; Sawchuk et al., 2013). The causes of crisis mortality include war, famine, epidemics, and natural disasters (Margerison and Knüsel, 2002; Chamberlain, 2006). Previous paleodemographic research has compared catastrophic burials to attritional mortality assemblages, which are accumulated over long periods of time (i.e., years, rather than days or weeks) (e.g., Margerison and Knüsel, 2002; Gowland and Chamberlain, 2005; DeWitte and Wood, 2008). Though it is often assumed that catastrophic mortality kills more indiscriminately than normal mortality, even extraordinarily catastrophic events have been shown to be selective. For example, although the Black Death (c. 1348–1351 AD) in London was highly virulent, research has suggested that it was selective with respect to frailty; individuals in poor health faced higher risks of death compared to their healthier peers (DeWitte and Wood, 2008; DeWitte and Hughes-Morey, 2012). This study examines possible famine burials from medieval London to broaden our understanding of selective mortality under conditions of medieval crises.

FAMINE: THE COMPLEXITY OF CAUSES AND EFFECTS

Famine can be caused by natural events (e.g., droughts, floods, and blights), but also by anthropogenic

phenomena (e.g., war, inadequate communication or transportation systems, market panic, and lack of political or social power) (Parrack, 1978; Sen, 1981; Torry, 1984; Pottier, 1986; Scrimshaw, 1987; Ó Gráda, 2007; Morgan, 2013). Though many scholars equate famine mortality with death from starvation, the majority of deaths during famines are often caused by infectious diseases because of the negative effects of malnourishment on immune competence (Scrimshaw, 1987; Walter and Schofield, 1989; Ó Gráda, 2007).

Famine mortality has historically been worsened by market panic, issues with food transport, the deterioration of sanitation and medical services, social and political dearth of entitlements, lack of physically able labor, and political upheavals (Ravallion, 1997; Ó Gráda,

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2007). These indirect causes exacerbate the already elevated rates of mortality that result from starvation and disease. For example, Sen (1981: 76) notes that farmers and grain merchants panicking about a *possible* famine can convert a “moderate short-fall in production...into an exceptional short-fall in market release.” This can occur, for example, when people hoard food resources in anticipation of a short-fall, thereby reducing availability for others.

In general, biologically and culturally determined heterogeneity in frailty (an individual’s relative risk of dying compared to other people in the same population; Vaupel et al., 1979) can lead to selective mortality. Such selectivity has been observed during famines within the last 150 years. For example, there is evidence for lower female mortality during recent historical famines, and this might result from female subcutaneous fat content, which makes them biologically less susceptible to starvation than men (Dyson and Ó Gráda, 2002; Ó Gráda, 2007; Morgan, 2013). Historically, famine has also been strongly selective with respect to age. Some scholars characterize the mortality curve as an exaggerated attritional curve with mortality peaking at the youngest and oldest ages (e.g., Morgan, 2013). Others emphasize that the greatest proportional increase in mortality is seen in age groups that are at lowest risk during “normal” periods (Maharatna, 1996; Ó Gráda, 2007). For example, Watkins and Menken (1985) cite several modern examples of famine mortality in which some of the greatest proportional increases in mortality occurred among children between the ages of 5–9 years and people over the age of 45. Exceptions to this pattern appear to be cases in which cultural biases may have favored the allocation of food to those under the age of 40 (cf. Ashton et al., 1984).

In sum, the effects of famine on mortality in recent history are complex. Very often, men, the young (particularly those between the ages of 1 and 10), and the old (particularly those over the age of 45) have exhibited elevated mortality. However, cultural factors and regional variance undoubtedly affect mortality distributions within a population, and thus studies of famine need to take societal and cultural context into account.

FAMINE: MEDIEVAL LONDON

The majority of the medieval British population lived in rural settlements and practiced mixed agriculture (Poos, 1998; Rigby, 2010; Benedictow, 2012). Crisis mortality could substantially impact a small settlement, instigating population movement (Dyer, 1998). However, for medieval London, economic deprivation was the most likely motivator for rural-urban migrants. Rural areas were overpopulated, making the city an attractive option for men and women looking for alternative sources of work (Dyer, 2002). Largely a result of such migration, London was one of the largest urban centers in Europe by the 12th century. By 1200, the population was about 40,000, and by 1300 it is estimated to have grown to between 80,000 and 100,000 (Keene, 1984).

Like other urban areas, London relied on surrounding rural areas for food. However, unlike smaller cities (which drew from approximately 10–12 km away), London drew its primary food source (grain) from 10 counties, some of which were nearly 80 kilometers away (Dyer, 2002). Urban crowding and a reliance on foodstuffs from elsewhere made medieval London

particularly susceptible to starvation and infectious disease—the hallmarks of famine. According to Farr (1846), there were famines every 14 years on average in the 11th through 13th centuries, with a slight decrease in the following three centuries; overall, there were approximately 7 famines per century, or 10 years of famine per century.

Some of the most severe famines in medieval London involved back-to-back crop failures (e.g., the famine of 1257–1258 AD and the Great Famine of 1315–1317 AD; see Farr, 1846; Lucas, 1930). Records from England note that many people died of hunger and disease during the 1257–1258 famine, particularly the urban poor, many of whom had migrated to London when their farms failed (Farr, 1846; Stothers, 2000). In 1259, a pestilence—likely influenza—aggravated the situation further, killing many of the surviving inhabitants of the city (Stothers, 2000). Interestingly, Farr (1846) reports that grain was available during the famine, but was too expensive for most urban dwellers. Consequently, much of this famine—initially instigated by weather conditions and crop failures—was blamed upon political and economic factors, rather than scarcity. This suggests that the entitlements and social disruption described by Sen (1981) in modern-day famine were also at play in the past.

In summary, London during the 12th through 16th centuries experienced both growth and crisis mortality. Famines were common, and, occasionally, quite severe. However, patterns of selectivity during medieval famines (and mortality patterns more generally) are difficult to discern from historical evidence because most of the available documentary evidence from the medieval period is biased towards adult males (Kowaleski, 2013), and this period mostly predates the introduction of the London Bills of Mortality in 1592. There is a dearth of information on the mortality of women and children. In fact, much of the historical research into sex differentials in mortality during Europe’s medieval period is considered speculative (Kowaleski, 2013). Fortunately, skeletal samples from this period can potentially provide information about patterns of mortality in medieval London that are currently unavailable from surviving historical documents. Few studies utilize the available skeletal evidence to understand famines during this period, resulting in a loss of meaningful information on these populations, their heterogeneity, and the selectivity of famines in general. Thus, one goal of this study is to integrate these two sources of data to understand famine in medieval London populations.

This study will further our understanding of how episodes of catastrophic mortality might differ (or not) from normal mortality, i.e., whether individuals in relatively poor health are consistently at higher risk of mortality, regardless of cause of death (DeWitte and Wood, 2008; DeWitte and Hughes-Morey, 2012). To examine whether medieval famine mortality was selective with respect to frailty, we compare patterns of stress indicators between famine and attritional samples from the medieval London cemetery of St Mary Spital. We also use hazard analysis to examine the relationship between sex and famine mortality. Like many of England’s hospitals in the medieval period, St Mary Spital was intended to care for the sick by providing regular meals and shelter, or, in the case of dying individuals, spiritual care and comfort. Although London was relatively clean compared to similarly sized cities at the time, its rapid growth and expansion during the 11th through 14th centuries led to

disease outbreaks typical of dense, crowded urban centers. Living conditions further complicated matters, as many homes—often constructed of timber, wattle, and daub—lacked proper ventilation for cooking fires or oil lamps, as well as clean, dry floors. In terms of work, London boasted a variety of industries (e.g., fisheries, brick and tile makers, bakers, and meat and produce sellers, etc.), but this also exposed workers to potentially dangerous or unhealthy conditions, such as dangerous construction work or noxious fumes and air pollution (Connell et al., 2012). Consequently, even during non-famine periods, St Mary Spital hospital and the accompanying cemetery had a regular influx of migrants and city inhabitants suffering from crowd diseases and illnesses or infections brought on by dangerous or unsanitary living environments.

The stress markers we examine have been shown previously to be associated with elevated risks of mortality in medieval London (DeWitte and Wood, 2008). Previous analyses of St Mary Spital cemetery found differences between famine and attritional burials in the frequencies of some stress markers (Jones, 2012); however, those analyses did not control simultaneously for age and sex, so it remains unclear whether their results truly reflect differences in attritional vs. famine mortality or rather were artifacts of differences in age or sex distributions between the two burial types. For this study, we use an approach that controls for age and sex and thus yields results that are unconfounded by these factors. Jones and Walker (2012) also found significantly higher proportions of males in attritional burials but no differences in numbers of males and females in famine burials. Their analysis, however, relied on χ^2 tests, which are not ideally suited to paleodemographic analysis of mortality risks, given that they are influenced by missing data. The hazard-analysis approach we take here accommodates missing data and allows for an efficient comparison of sex differences in risk that controls for age and thus allows for clearer insights into patterns of mortality.

MATERIALS AND METHODS

St Mary Spital Cemetery sample

The sample for this study comes from the St Mary Spital cemetery (SRP98), located outside of the City of London, near the Borough of Tower Hamlets. The cemetery was established by the medieval priory and hospital of St Mary Spital, which was founded in 1197 AD by a group of wealthy London merchants to serve the poor, pilgrims, wayfarers, and women in childbirth (Connell et al., 2012). The larger part of the cemetery ($\sim 40,000$ m²) was excavated between 1998 and 2001 by Museum of London Archaeology (MoLA) (Connell et al., 2012). Based on the context of the site and Bayesian radiometric dating of its stratigraphic layers, the burials were assigned to four phases: Period 14 (1120–1200 AD), Period 15 (1200–1250 AD); Period 16 (1250–1400 AD); and Period 17 (1400–1539 AD) (Sidell et al., 2007; Connell et al., 2012). A total of 10,516 individuals of an estimated 18,000 individuals originally buried in the cemetery were excavated (Connell, 2012). The protocol used during data collection for the Museum of London Wellcome Osteological Research Database (WORD) stipulated that only individuals who were 35% complete and had regions of the skeleton suitable for estimating age or sex were included in the database ($n = 5,387$).

There is no archaeological evidence to suggest that males and females were buried in different parts of the cemetery, though there are status differences in burial location within the cemetery. Overall, the cemetery is interpreted as being a secular one, drawing on communities in the City and surrounding areas (Connell et al., 2012), and our sample is not limited to hospital inmates.

Different burial types in each period are believed to correlate with periods of attritional mortality and crisis mortality (see diagrams in Connell et al., 2012: 13). About half of the burials are single interments (Type A burials) of hospital inmates, officials, and benefactors (Thomas et al., 1997). Type B burials consist of single horizontal layers of two to seven bodies. Type C burials consist of bodies stacked directly on top of one another and are typically 2–11 bodies deep. Type B and C burials may represent individuals who died on the same day and were therefore buried as a group. Lastly, Type D burials consist of multiple layers of horizontally arranged bodies stacked on top of each other within a single grave cut; known as “the catastrophic group,” these burials typically contain 8–45 bodies. The manner in which they were dug suggests that they were constructed to accommodate large numbers of deaths within a short time period (Connell et al., 2012; Jones, 2012). According to Jones (2012), individuals in the Type D burials do not exhibit higher levels of interpersonal violence compared to the attritional (A, B, and C) burials, suggesting that Type D burials are not associated with warfare or local incidences of violence. Instead, they note that the size and timing of the mass burials corresponds with historically documented famines (Farr, 1846). For example, mass burials in Period 14 are radiometrically dated to the middle of the 12th century, during which there was a documented famine in 1162. Connell et al. (2012) therefore argue that these burials are associated with famine-related catastrophic mortality.

The well-established chronology and burial types of St Mary Spital can be used to better understand the impact and selectivity of famines in the periods before and after the Black Death, which lasted from 1349 to 1350 AD in London; Periods 14 and 15 pre-date and Period 17 post-dates the epidemic. Because we are not investigating the crisis mortality caused by the Black Death, Period 16 (1250–1400 AD) has been omitted from this study. It is possible that the Black Death obscured patterns that were normally produced by famines in that period. Some of the mass burials from Period 16 may be associated with famines that are unrelated to the plague, but it is not yet feasible to distinguish between famine and Black Death burials in St Mary Spital. For this study, attritional burials (Type A burials, $n = 713$) are compared to the famine burials (Type D, $n = 814$) from Periods 14, 15, and 17. We take a conservative approach of comparing only Type A and D burials to minimize the chances of including nonattritional burials in our attritional samples. All data for this study were previously collected by researchers at MoLA and were obtained from WORD. The samples used in this study are subsamples of the total number of individuals originally buried in SRP98, excavation of which was incomplete. Thus, our samples might be biased to an unknown degree, and the results of this study should be viewed with the same caution that must be applied when interpreting most bioarchaeological analyses.

Age estimation

Ages were previously estimated by researchers at MoLA via traditional methods (Powers, 2012). Particularly, morphological changes to the pubic symphysis (Brooks and Suchey, 1990), iliac auricular surface (Lovejoy et al., 1985), and costochondral junction (Işcan et al., 1984, 1985), and dental wear (Brothwell, 1981) were evaluated. These features allowed adults to be assigned to one of four age intervals: 18–25, 26–35, 36–45, and ≥ 46 years.

Sex determination

Sex determinations were made on the basis of sexually dimorphic features of the skull and pelvis using the standards described in Phenice (1969), Ferembach et al. (1980), Brothwell (1981), and Bass (1995). Features were scored on a five-point scale and individuals were assigned a sex of male, probable male, intermediate, probable female, or female. In some cases, poor preservation, truncation, or missing skeletal features resulted in a sex classification of “undetermined.” Data for males and probable males and for females and probable females were pooled for this study.

Skeletal indicators of stress

To assess the effect of physiological stress on famine mortality, trends in the presence of the following skeletal stress markers were examined: cribra orbitalia, linear enamel hypoplasia, and periosteal new bone formation (periosteal lesions). Previous studies have shown these to be associated with episodes of malnutrition or disease and with elevated risks of mortality (Roberts and Manchester, 2007; DeWitte and Wood, 2008).

Cribra orbitalia is characterized by porotic lesions on the orbital roofs that result from expansion of the diploë and resorption of the outer table of the skull in response to bone marrow hypertrophy (Stuart-Macadam, 1992; Ortner, 2003; Walker et al., 2009). Paleopathologists commonly attribute these lesions to anemia (particularly iron-deficiency anemia). However, histological studies of cribra orbitalia have shown evidence of causes such as inflammation, osteoporosis, and rickets (Wapler et al., 2004). Walker et al. (2009) argue that iron-deficiency anemia is not a likely cause based on evidence that iron deficiency inhibits marrow hypertrophy rather than causing the marrow expansion (however, see Oxenham and Cavill, 2010 and McIlvaine, 2013). We do not attempt to determine the specific etiology of cribra orbitalia in our sample, but instead use it as an indicator of previous exposure to stressors, regardless of etiology. Cribra orbitalia was scored as absent or as present, using the grading system of Stuart-Macadam (1991); Scores 1–5 were collapsed into a single “presence” score for analysis.

Linear enamel hypoplasias (LEH) consist of horizontal lines or pits on tooth enamel. They are caused by the disruption of enamel formation in response to infection or malnutrition (Goodman et al., 1980; Larsen, 1997; White et al., 2011). Because permanent teeth are formed during childhood, defects in enamel formation, such as LEH, remain as a lasting record of early life, even in later adult life (unless the affected teeth are excessively worn or lost antemortem). Only those LEH that were identified macroscopically on the mandibular canines were analyzed. These canines were selected for analysis

because they provide reliable information on an individual's exposure to stress given their relatively long developmental time span and high sensitivity to physiological stress (Goodman et al., 1980). LEH were scored as present if one or more defects on the surface of the tooth were visible to the naked eye under good lighting.

Periosteal lesions occur in response to trauma to the periosteum or inflammation associated with a variety of factors (Weston, 2008; White et al., 2011). As with the other skeletal stress indicators examined, we do not attempt to diagnose individuals with a specific disease on the basis of periosteal lesions, but rather use them as a general indicator of exposure to stressors. Because we did not use differential diagnosis to exclude individuals with specific conditions (e.g., leprosy or treponemal infection), it is possible that our sample does include people who suffered from such diseases; however, we argue that these diseases would have caused physiological stress and thus their potential inclusion does not undermine the inferences we make about frailty. We analyze data from individuals possessing at least two-thirds of the right tibia. The right tibia was selected for two reasons: 1) in general, the tibia is more likely than other skeletal elements to present with periosteal lesions indicative of stress—it is the body's least vascularized skeletal element, it has little soft tissue between it and the external environment, it is subject to recurrent injury and susceptible to bacterial infection, it has a particularly slow immune response, and it has an elevated osteogenic potential (Gallay et al., 1994; Roberts and Manchester, 2007; Klaus, 2014); 2) the right tibia was more frequently preserved in the St Mary Spital skeletal sample and therefore provided a large sample size for statistical analysis.

Hierarchical log-linear analysis

The associations among age, sex, stress markers, and famine were evaluated using hierarchical log-linear analysis with SPSS version 22. This approach is appropriate because our data include both categorical (i.e., age-interval) and binary (i.e., presence or absence of stress indicators) variables. Hierarchical log-linear analysis was used to test the significance of the four-way interactions among age, skeletal stress marker, burial type (famine or attritional), and sex, as well as all other lower order interactions (e.g., the three-way interaction between skeletal stress marker, age, and burial type, and the two-way interaction between stress marker and sex). Backward elimination was used to remove non-significant interactions among variables with a statistical significance criterion of 0.05, unless otherwise noted. Hierarchical log-linear analysis allows for the evaluation of the interactions among more than two variables, so that it is possible to determine whether there are significant associations between burial type and skeletal stress markers that exists in the absence of either age or sex (DeWitte and Bekvalac, 2011). Further, if a significant association is observed between the two conditions, this approach is informative about whether the relationship is consistent across all age groups or if it is dependent on age (Green, 1988).

Although we report statistical significance (for the hierarchical log-linear analyses and hazard analyses described below), we recognize that *P*-values must be used with caution and are in fact replaced by many medical, psychological, and epidemiological researchers with

TABLE 1. Stress indicator frequencies by burial type

	Attritional			Famine		
	No stressor (n)	Stressor (n)	% with stressor	No stressor (n)	Stressor (n)	% with stressor
Linear enamel hypoplasia (n = 1,049)	331	143	30.2	367	208	36.2
Cribra orbitalia (n = 1,031)	309	153	33.1	361	208	36.6
Periosteal new bone (n = 1,154)	348	164	32.0	479	163	25.4

TABLE 2. Stress indicator frequencies by age and sex

	Age	Sex	No stressor (n)	Stressor (n)	% with stressor	
Linear enamel hypoplasia (n = 1,049)	18–25	Female	72	61	45.9	
		Male	76	57	42.9	
	26–35	Female	129	66	33.8	
		Male	126	64	33.7	
	36–45	Female	75	39	34.2	
		Male	127	44	25.7	
	46+	Female	49	8	14.0	
		Male	44	12	21.4	
Cribra orbitalia (n = 1,031)	18–25	Female	73	49	40.2	
		Male	81	43	34.7	
	26–35	Female	122	67	35.4	
		Male	122	65	34.8	
	36–45	Female	63	49	43.8	
		Male	116	64	35.6	
	46+	Female	43	13	23.2	
		Male	50	11	18.0	
	Periosteal new bone (n = 1,154)	18–25	Female	100	25	20.0
			Male	97	37	27.6
		26–35	Female	167	45	21.2
			Male	137	80	36.9
36–45		Female	97	29	23.0	
		Male	116	85	42.3	
46+		Female	59	6	9.2	
		Male	54	20	27.0	

TABLE 3. Ages at death for males and females in the attritional and famine burials (all periods)

Age	Sex	Attritional (Type A burials) (n = 713)	Famine (Type D burials) (n = 814)
18–25	Female	78	97
	Male	89	98
26–35	Female	116	161
	Male	140	141
36–45	Female	72	96
	Male	136	138
46+	Female	32	48
	Male	50	35

other methods (Lang et al., 1998; Rothman, 1998; Goodman, 1999; Cohen, 2011; Trafimow and Marks, 2015). For the analyses presented here, *P*-values of less than 0.10 are considered to be significant, or suggestive of a real effect.

Gompertz model

We use the Gompertz model to estimate sex differences in adult mortality. The Gompertz model is a biomathematical hazard model of mortality and thus incorporates biological principles, such as the physiologi-

cal processes that influence mortality, into the analysis of mortality patterns (e.g., Gage, 1988, 1989; Wood et al., 2002). Importantly, hazard models can be fruitfully applied to small skeletal samples, which are the norm in bioarchaeological studies (Milner et al., 2008).

The two-parameter Gompertz age-dependent mortality function (Gompertz, 1825) fits the general human pattern of relatively low risks of mortality at younger adult ages and an increasing risk of death with senescence (Wood et al., 2002). Although a third parameter, Makeham's (1860) age-independent component, can be added to the Gompertz model (yielding the three-parameter Gompertz-Makeham model), it is often difficult to estimate with paleodemographic samples (Gage, 1988; Herrmann and Konigsberg, 2002; Nagaoka et al., 2006). To determine whether sex affected risks of mortality among the famine (Type D) and attritional (Type A) burials, sex was modeled as a covariate affecting the Gompertz model using a proportional hazard specification:

$$h_i(t_i|x_i\rho) = h(t_i)e^{(x_i\rho)}$$

where the baseline Gompertz hazard $h(t_i) = \alpha e^{\beta t}$, t_i is the age of the i th skeleton in years, x_i is the sex covariate, and ρ is the parameter representing the effect of the covariate on the baseline hazard. We coded females as 0, and males as 1. A significant positive estimate for the

TABLE 4. Results of the four-way hierarchical log-linear analyses

Variables	P
Burial type × age × sex × LEH	0.886
Burial type × age × sex × CO	0.996
Burial type × age × sex × PNB	0.031
Burial type: famine or attritional. Age groups: 18–25, 26–35, 36–45, and 46+.	
LEH, linear enamel hypoplasia; CO, cribra orbitalia; PNB, periosteal new bone.	

TABLE 5. Results of the three-way hierarchical log-linear analyses

	Variables	P
Linear enamel hypoplasia	Burial type × age × LEH	0.479
	Burial type × sex × LEH	0.145
	Age × sex × LEH	0.511
	Age × LEH	0.000
	Sex × LEH	0.534
	Burial type × age	0.977
Cribra orbitalia	Burial type × LEH	0.047
	Burial type × age × CO	0.968
	Burial type × sex × CO	0.487
	Age × sex × CO	0.782
	Age × CO	0.003
	Sex × CO	0.160
	Burial type × age	0.979
	Burial type × CO	0.300
Periosteal new bone	Burial type × age × PNB	0.675
	Burial type × sex × PNB	0.939
	Age × sex × PNB	0.455
	Age × PNB	0.002
	Sex × PNB	0.000
	Burial type × age	0.476
	Burial type × PNB	0.028

Burial type: famine or attritional. Age groups: 18–25, 26–35, 36–45, and 46+.
LEH, linear enamel hypoplasia; CO, cribra orbitalia; PNB, periosteal new bone.

parameter representing the effect of the covariate would suggest males were at an increased risk of death compared to females, while a negative estimate would suggest males were at a decreased risk of death.

Using individual age and sex estimates, parameters were estimated separately for the famine and attritional burials using maximum likelihood analysis with the program *mle* (Holman, 2002). A likelihood ratio test (LRT) was used to assess the fit of the full model compared to the baseline model, which does not include sex as a covariate. The LRT tests the null hypothesis that sex had no effect on risk of mortality (H_0 : effect of sex covariate = 0). The LRT was computed as follows: $LRT = -2[\ln(L_{sex}) - \ln(L_{baseline})]$, where LRT approximates a χ^2 distribution with $df = 1$.

RESULTS

Hierarchical log-linear analysis

The frequencies of stress indicators (LEH, cribra orbitalia, and periosteal lesions) by burial type and by sex and age in St Mary Spital are shown in Tables 1 and 2, respectively (as crude prevalence rates). The age-distributions by sex in the two burial types are shown in Table 3. The results of the hierarchical log-linear analysis for the three stress indicators are presented in Tables 4 and 5. For two of the

TABLE 6. Numbers of adult males and females in each burial type and period

Period	Burial type	Males		Females	
		(n)	%	(n)	%
14/15 (n = 1,115)	Attritional	217	55.8	172	44.2
	Famine	363	50.0	363	50.0
17 (n = 412)	Attritional	198	61.1	126	38.9
	Famine	49	55.7	39	44.3

The proportions are reported with respect to burial type.

stress indicators there is no significant association among all four variables (i.e., age, sex, burial type, and stress indicator). However, a significant four-way association was found between burial type, sex, age, and periosteal lesions ($P = 0.031$); therefore, in order to expose other significant three- and two-way relationships, the significance criterion for backwards elimination was reset to 0.01. For every three-way analysis, no association was found among the three variables analyzed (with the significance criterion set at 0.05 for cribra orbitalia and LEH, and at 0.01 for periosteal lesions). Because our primary goal is to investigate whether a relationship between burial type and stress indicator exists independent of age or sex (i.e., how previous exposure to stress impacted an individual's inclusion in famine or attritional burials, regardless of age or sex), some three-way analyses (e.g., Burial type × age × sex) were omitted from Table 4 for the sake of simplicity.

There is a significantly higher frequency of LEH in famine burials, but a higher frequency of periosteal lesions in attritional burials. However, there is no significant difference between famine and attritional burials with respect to cribra orbitalia. Other significant two-way relationships include: age and LEH (highest frequency in those aged 18–25 years), age and cribra orbitalia (highest frequency in ages 36–45 years), age and periosteal lesions (highest frequency in people ages 26–45 years), and sex and periosteal lesions (higher frequency in males).

Gompertz model

Table 6 summarizes the numbers of adult males and females in the famine and attritional burial types within each period. The sex distributions (for all time periods pooled) differ significantly between the two burial types ($\chi^2 P = 0.003$). There are more males than females in attritional burials; within famine burials, males (slightly) outnumber females only in Period 17. Analysis of the effect of sex on risk of death (Table 7) reveals a significant effect only in Period 17 attritional burials (1400–1539 AD). In that period, the estimated value of the parameter representing the effect of sex is negative for the attritional burials, the corresponding 95% confidence interval includes only negative values, and the results of the likelihood ratio test indicate that inclusion of the covariate improves the fit of the model. These results indicate that males were at reduced risks of death compared to females in the attritional burials. However, there is no significant effect of sex in the famine burials from Period 17.

DISCUSSION

Patterns of physiological stress markers

The results of this study reveal significant differences in the presence of some stress indicators between the

TABLE 7. Maximum likelihood estimates of the effect of the sex covariate (with the 95% confidence interval in parentheses) and likelihood ratio tests (LRT) of H_0 : effect of sex covariate = 0

	Famine			Attritional		
	Sex (95% CI)	LRT	P	Sex (95% CI)	LRT	P
Period 14/15	-0.12 (-0.34, 0.09)	1.35	0.24	-0.06 (-0.23, 0.10)	0.68	0.41
Period 17	0.20 (-0.29, 0.62)	0.87	0.35	-0.23 (-0.46, -0.01)	3.87	0.05

famine and attritional burials. Specifically, there is a higher frequency of periosteal lesions in attritional burials, and a higher frequency of LEH in famine burials, but no association between burial type and cribra orbitalia. The results also indicate that there are significant associations between age and stress indicator presence and between sex and stress indicator presence.

Explanations for burial type differences in stress indicator presence

Given that periosteal lesions occur only if an individual survives the causative stressor, the higher frequency of periosteal lesions among attritional burials might indicate that under conditions of normal, nonfamine mortality, individuals were more likely to survive nonlethal physiological stressors that can cause periosteal lesions. In contrast, periods of repeated crop failure, migration to an urban environment, the disruption of social norms and communities, malnutrition, and high rates of infectious disease experienced by individuals in the famine burials might have increased individual levels of frailty and thus decreased their chances of survival in the face of physiological stressors, independent of age or sex. The significant association between periosteal lesions and attritional burials found here and in another study (Jones, 2012) may actually reflect relatively low underlying frailty (Ortner, 1991; Wood et al., 1992). In other words, individuals in attritional burials with observable lesions may have been able to survive physiological stressors long enough to develop lesions, while people in the famine burials without observable lesions were relatively frail and perished before lesions could develop. It may be worthwhile to explore whether famine burials are primarily associated with active lesions (high frailty), while attritional burials are associated with healing or healed lesions (low frailty) as previous research has revealed significant differences in survivorship between individuals with active vs. healed lesions (DeWitte, 2014a). The existing data do not generally include lesion activity, and thus such an analysis is beyond the scope of the current study.

LEH was the only stress indicator that was found at significantly higher frequencies in the famine pits compared to the attritional burials, both in the hierarchical analyses presented here and in Jones's (2012) study comparing raw lesion frequencies between burial types. Early exposure to physiological stressors, as indicated by LEH, could have impacted these individuals' overall frailty, and thus their morbidity and mortality later in life. It is possible that stress in fetal development led to maladaptive epigenetic changes (e.g., see Heijmans et al., 2008), or compromised immune function (e.g., see Sullivan et al., 1993). Likewise, insults in childhood could also have resulted in a body that was underprepared for additional physiological insults during famine periods. Further, given the high number of migrants and

impoverished individuals that would have utilized St Mary Spital, it is possible that many of the people included in the skeletal sample were marginalized people who were frequently exposed to environmental stressors and thus more likely to perish during famine events than London's wealthier inhabitants. Recent evidence of delayed completion of maturation in low status London residents compared to other contexts suggests high levels of stressors within the city (Lewis et al., 2015), though that study was not specific to famine victims. We should note that enamel defects can also be caused by trauma to the jaw (Andreasen and Ravin, 1973), and thus the higher frequency of LEH in the famine burials might indicate higher rates of trauma among individuals who succumbed to famine mortality. However, as mentioned in the introduction, previous analysis of St Mary Spital did not reveal higher rates of evidence of interpersonal violence in the famine burials compared to attritional burials and, thus, we do not think that a conclusion of higher rates of trauma for famine victims based on patterns of LEH is justified.

Unlike periosteal lesions and LEH, there was no significant association between burial type and cribra orbitalia, independent of age and sex. The results of this study do not suggest that individuals who suffered from the stressors associated with cribra orbitalia were any more likely to die during periods of famine than they would have been under normal mortality conditions. Perhaps cribra orbitalia, though clearly indicative of exposure to stressors, is not as strongly indicative of frailty as LEH.

Explanations for age differences in stress indicator presence

The results of this study reveal a significant association between age and LEH, with the highest frequency in those aged 18–25 years. Following Armelagos et al. (2009), Goodman and Armelagos (1988), and Duray (1996), the association between LEH and earlier age at death could suggest that 1) individuals with LEH are socially or culturally marginalized, resulting in increased exposure to environmental stressors that could both generate stress-induced growth disruption and earlier mortality; 2) early exposures to stressors could result in an individual being less likely to successfully cope with later insults; 3) individuals genetically or biologically prone to being influenced by stress events are likely to experience lifelong illness, which would result in both LEH and early mortality; and 4) individuals with LEH represent people who survived during resource scarcities, meaning that both the LEH and the early mortality are results of the resource scarcity.

With respect to the first possibility, clearly defined social classes in medieval London could have caused poor individuals to be more frequently exposed to environmental stressors that could have disrupted their

childhood growth and led to their earlier mortality. However, although St Mary Spital broadly served migrants and the poor, they also served the sick, as well as hospital officials and benefactors (Thomas et al., 1997). As a result, differentiating between potential underlying factors like socioeconomic status is difficult, yet cannot be dismissed when attempting to explain patterns in age-at-death. It is indeed possible that many of the individuals interred at St Mary Spital were urban paupers who, in comparison to their more wealthy counterparts, were more likely to experience lifelong stressors that impact canine enamel development and age at death, regardless of famine or sex.

The final three explanations are not mutually exclusive (Duray, 1996) and can reasonably be encompassed by the idea that early exposure to physiological stressors impacts later frailty, and thus morbidity and mortality. This idea is not new, but recent research has made anthropologists more aware of the potential mechanisms connecting early life environment and later life morbidity and mortality (alternatively described as the fetal origins, fetal programming, Barker, or developmental origins of health and disease hypotheses), and there has been an increase in bioarchaeological research that explicitly addresses ways to assess this phenomenon when investigating health in the past (Klaus, 2014; Temple, 2014; Gowland, 2015). With respect to famine, for example, individuals prenatally exposed to famine during the Dutch Hunger Winter in 1944–1945, 60 years later had DNA methylation patterns that differed from those of their unexposed siblings (Heijmans et al., 2008; Tobi et al., 2012). Changes in methylation, a type of epigenetic marker, are one way that early environmental cues and stressors can lead to increased lifetime disease risk and early mortality. In other words, experiences in fetal development or early childhood could have persistent effects on later morbidity and mortality. Stressors documented in the canines could reflect the early instigation of lifelong frailty, which ultimately impacted earlier mortality, as reflected in the early ages at death of individuals with LEH.

Periosteal lesions show an entirely different association with age compared to LEH. Periosteal lesions were most common in individuals between the ages of 26 and 45. This pattern is not uncommon in bioarchaeological studies. For example, in Grauer's (1993) study of a medieval sample from northern England, periosteal lesions were found to increase with age. Grauer interprets this pattern as an accumulation of nonlethal conditions over time. Similarly, in her study of pre- and post-Black Death skeletal samples from London, DeWitte (2014b) found that enhanced longevity led to increasing frequencies of periosteal lesions at older ages, which might indicate that living longer resulted in an accumulation of nonlethal physiological stressors (e.g., chronic inflammation associated with aging) that caused the observed lesions. Thus, the positive association between periosteal lesions and age observed here may actually reflect relatively low underlying frailty, as proposed by Ortner (1991) and Wood et al. (1992); that is, individuals with periosteal lesions may have been less frail and thus more likely to survive certain physiological stressors than their peers, allowing them to develop lesions. In contrast, individuals without observable lesions (e.g., individuals 18–25 and 46+ years) may have been relatively frail and unable to survive physiological stressors long enough to develop periosteal lesions. Interestingly,

it has been proposed that the type of periosteal lesion (active vs. healed) may be more informative about frailty than presence alone. Though it was not explored in this study because the WORD database does not include "activity," previous research in medieval London suggests that active periosteal lesions may represent individuals of high frailty, while healed or healing lesions are a marker of relatively low frailty (DeWitte, 2014a). Consequently, it is important to keep in mind that the association between age and periosteal lesion presence may not be representative of all of the heterogeneous subgroups (or their relative frailty) that made up the population of medieval London.

The presence of cribra orbitalia was most common in those below the age of 46. Like LEH, the peak of cribra orbitalia at younger age groups could reflect experiences in early life that had persistent effects on later morbidity and mortality. Physiological stressors encountered in infancy or childhood may have had the twofold effect of producing cribra in the orbits and increasing frailty, which ultimately led to earlier mortality for many in the St Mary Spital population. However, given the findings by DeWitte (2014a) mentioned above, it may be of interest to evaluate the degree of healing seen in cribra orbitalia lesions, as this study may have overlooked some important association between healing, age, and underlying frailty. However, given the available data, this is a topic that should be pursued in the future pending further data collection.

Explanations for sex differences in stress indicator presence

Periosteal lesions were significantly more frequent in males than females, independent of age or burial type. In contrast, LEH and cribra orbitalia were not significantly associated with either sex. Although the patterns of LEH and cribra orbitalia suggest that each sex was exposed to similar environments and stressors, the significant association between sex and periosteal lesions suggests that some aspects of life were different for the two sexes, resulting in different exposures to traumas, infections, or other stressors. For example, periosteal lesions are known to result from inflammation to the periosteum, which may be a consequence of repeated minor traumas to the tibia (Weston, 2008). At St Mary Spital, differences in tibial periosteal lesions may reflect cultural differences in activity and environmental exposure. Males may have engaged in labor-intensive occupations that exposed them to repeated minor tibial traumas, such as building construction (Dyer, 2002). Further, these different work environments may have meant that exposure to pathogens differed between the sexes as well, putting males at greater risk of developing infection-related periosteal lesions. However, biological causes could be implicated as well. Numerous studies demonstrate that estrogen generally promotes stronger immune responses to infectious disease in females (e.g., Klein, 2000; Klein et al., 2011; Zuk and Stoehr, 2010; Fischer et al., 2015); this might have allowed females to fight infections before skeletal involvement leading to periosteal lesions occurred.

Ultimately, it is important to keep in mind that the host's immune status, the infecting organism or pathogen's virulence, and environmental conditions (e.g., population density) will all interact in complex ways that might not always be visible in the skeleton. Further,

these conditions will also interact with cultural differences between the sexes (e.g., activity differences or food consumption practices) in ways that could influence how an individual's skeleton reacts to infection, inflammation, and nutrient deficiency. Any combination of these factors could be implicated when considering why males in medieval London exhibit significantly higher frequencies of periosteal lesions but not cribra orbitalia or LEH.

Hazard analysis of sex differences in mortality

As detailed in the Introduction, during famines in the 19th and 20th centuries, males often experienced higher mortality than females (Dyson and Ó Gráda, 2002; Ó Gráda, 2007; Morgan, 2013). This evidence from historical famines might suggest that males experience an increased risk of mortality during famines when compared with females in general, perhaps for biological reasons (e.g., higher subcutaneous fat content and estrogen-enhanced humoral immunity in females) (Klein, 2000; Dyson and Ó Gráda, 2002; Morgan, 2013); research using animal models has revealed the mechanisms underlying the protective effects of estrogen during periods of starvation (e.g., Lebeck et al., 2012), though similar evidence for humans is currently lacking. However, the results of hazard analysis (Table 7) suggest that the same may not have been true for adults during the medieval period in London. In Periods 14 and 15 (1120–1250 AD), estimates of the parameter representing the effect of the sex covariate suggest that sex did not significantly affect risk of death in either the famine or attritional samples. In Period 17 (1400–1539 AD), however, males in the attritional burials faced a lower risk of mortality compared to females; the risk of death for males and females during famine in Period 17 (1400–1539 AD) does not appear to have differed significantly.

The change over time in sex differentials in attritional mortality risk apparent in the results suggests that a cultural or biological transformation took place during Period 16 (1250–1400 AD) or Period 17 (1400–1539 AD). The most likely source of such a change was the medieval Black Death (1347–1351 AD). Previous research on medieval London cemeteries suggests that there was enhanced survival and improvements in mortality in the 200 years following the Black Death (DeWitte, 2014b, c). The causes could have been the selectivity of the epidemic itself (i.e., frail individuals were disproportionately killed, so the surviving population would have been less frail in general), or subsequent improvements in diet and standards of living. However, the current study may suggest that these health benefits were distributed unequally among the population. Improvements following the Black Death may have benefitted males more than females in the nonfamine years of Period 17.

One possible explanation for males' comparatively lower mortality risk in the Period 17 attritional burials is the improvements in diet that followed the Black Death. The availability of meat increased in the latter half of the 14th century, supplementing the grain-dominated diets of Londoners (Hanawalt, 1993; Woolgar, 2006). In medieval society, females were undervalued (Kowaleski, 2013), which could have stemmed from the fact that only sons could retain heritable properties when married (Bennett, 1987; Wiesner, 2000). This preference for male heirs could have resulted in sex-based differences in food access, which would have influenced nutrition and health. If so, the sex receiving preferential

treatment (males) would have experienced a reduced risk of mortality during attritional periods, while the undervalued sex (females) would have died in greater numbers. However, the sex differential in favor of males under conditions of normal mortality disappears during famines. It is possible that the male advantage in terms of food access was matched by the female biological advantage that has been recorded during historical famines (Dyson and Ó Gráda, 2002; Ó Gráda, 2007; Morgan, 2013). Estrogen enhances the immune competence of females, which may defend them from many of the infectious diseases that claim lives during famines (Klein, 2000). Further, their greater subcutaneous fat content may make females relatively less likely to succumb to starvation during food shortages (Dyson and Ó Gráda, 2002; Morgan, 2013). We note that preferential treatment of male heirs might not explain the observed pattern in our study, given that our samples likely include many lower status individuals. Lower status female workers would presumably have had the ability to purchase nutritious foods just as well as their male counterparts, and thus disproportionate access to such resource might not have been true for many of the people in the St Mary Spital population.

An alternative explanation for the lower risk of mortality for males in attritional burials from Period 17 (1400–1539 AD) is the selectivity of the Black Death itself. DeWitte (2010) found that excess mortality associated with skeletal indicators of frailty was higher in males than females in the East Smithfield Black Death cemetery in London. This might indicate that males were frailer than females prior to the Black Death, and, as a result, more males died during the epidemic. Such results would be consistent with findings that females are biologically less susceptible to a variety of diseases because of the effects of estrogen (e.g., Klein and Roberts, 2010). Excess mortality of frail males during the plague might have produced lower frailty among surviving males compared to females following the Black Death. This might have had a multi-generational effect if frailty was at least in part determined by genetic variation that was shaped by the selective sweep of the Black Death.

It should be noted that one potential reason for the overall lack of differences in risk of mortality between males and females is the way in which we modeled sex as a covariate affecting the Gompertz model. In this study, the effect of the sex covariate was modeled as proportional to the baseline hazard, independent of age. Thus, it is possible that there was variation in sex differences in mortality across age that is not detected using this approach. If famine mortality in medieval London was selective against females in early adulthood and males later in life (or vice versa), the way in which sex was modeled here affecting the Gompertz model may not reveal a significant difference.

Other potential sources of error include preservation bias and sex estimation bias. In an analysis of post-medieval crypt burials from St Bride's Church in London, it was found that elderly women were disproportionately relegated to the "undetermined" category during sex estimation, because the features of the pelvis necessary for sex estimation were often poorly preserved in this group (Walker, 1995). In addition to sex differences in preservation, sex estimation can be biased, particularly if we rely on features of the cranium. In postmenopausal females, the supraorbital ridge is

considerably more pronounced than in younger females, making it more likely that the older female is placed in the “male” or “undetermined” categories (Walker, 1995). Both preservation bias and biases in sex estimation could produce an excess of males in the analyzed sample, as females in the “undetermined” category would not have been included in the hazard model analysis.

The possibility also remains that the general excess of males may actually reflect a real disparity in the numbers of males and females living in London in the 12th through 16th centuries. It is possible that males disproportionately migrated to London seeking work, especially when agricultural occupations were limited in rural areas (Sloane, 1999). However, such a statement begs caution, as female migrants were also able to access employment in the city and may have outnumbered their male counterparts yet gone unrecorded in historical documents (Leyser, 1995; Dyer, 2002; McIntosh, 2005; Kowaleski, 2013, 2014).

CONCLUSION

Comparisons of the presence of skeletal indicators of stress between famine and attritional burials from medieval London suggest that famine during this period was selective with respect to frailty. Individuals who experienced early life stressors (indicated by LEH) were likely more frail than their peers, and thus were more likely to die during famines. In contrast, periosteal lesions may be indicative of relatively low frailty and the ability to survive during nonfamine periods long enough to develop lesions. *Cribra orbitalia* was not significantly associated with famine or attritional burials, and therefore may not be as sensitive an indicator of frailty as periosteal lesions and LEH, at least in the context of medieval London.

This study also examined stress indicator differences across age groups, finding that *cribra orbitalia* was more common in people below the age of 45, LEH was most common in those under the age of 25, and periosteal lesions peaked between the ages of 26 and 45. Further, periosteal lesions are significantly more common in males. Collectively, these findings may reflect culturally and biologically induced differences in frailty among age and sex groups within the larger population of medieval London.

Finally, the results of this study suggest that adult males and females in London did not experience significantly different risks of mortality during famine and nonfamine periods prior to the Black Death (1348–1351 AD). After the Black Death, however, the results indicate that males faced a significantly lower risk of mortality during nonfamine periods. It is possible that improvements in diet and standard of living in London following the Black Death primarily benefitted males, allowing them to survive many causes of death during nonfamine periods. Another explanation is that the Black Death discriminated more strongly against frail males than frail females, causing the surviving males to be healthier and less frail relative to the surviving females. As a result, the surviving males would have experienced reduced mortality in the nonfamine years of the 14th and 15th centuries.

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LITERATURE CITED

- Andreasen JO, Ravin JJ. 1973. Enamel changes in permanent teeth after trauma to their primary predecessors. *Scand J Dent Res* 81:203–209.
- Armstrong GJ, Goodman AH, Harper KN, Blakey ML. 2009. Enamel hypoplasia and early mortality: bioarchaeological support for the Barker hypothesis. *Evol Anth* 18:261–271.
- Ashton B, Hill K, Piazza A, Zeitz R. 1984. Famine in China, 1958–61. *Popul Dev Rev* 10:613–645.
- Bass WM. 1995. *Human osteology: a laboratory and field manual*. Springfield, MO: Missouri Archaeological Society.
- Benedictow O. 2012. New perspectives in medieval demography: the medieval demographic system. In: Bailey M, Rigby S, editors. *Town and countryside in the age of the black death: essays in honour of John Hatcher*. Turnhout: Brepols Publishers. p 3–42.
- Bennett JM. 1987. *Women in the Medieval English Countryside: gender and household in Brigstock before the plague*, Vol.15. New York: Oxford University Press.
- Boyle PP, Ó Gráda C. 1986. Fertility trends, excess mortality, and the Great Irish Famine. *Demography* 23:543–562.
- Brooks ST, Suchey JM. 1990. Skeletal age determination based on the os pubis: a comparison of the Ascádi-Nemeskéri and Suchey-Brooks methods. *J Hum Evol* 5:227–238.
- Brothwell DR. 1981. *Digging up bones: the excavation, treatment and study of human skeletal remains*, 3rd ed. Ithaca, New York: Cornell University Press.
- Buikstra JE, Ubelaker DH, editors. 1994. *Standards for data collection from human skeletal remains*. Arkansas Archaeological Survey Research Series 44. Fayetteville, Arkansas.
- Campbell BMS, ed. 1992. *Before the Black Death: Studies in the “crisis” of the early fourteenth century*. Manchester: Manchester University Press.
- Chamberlain A. 2006. *Demography in archaeology*. Cambridge: Cambridge University Press.
- Cohen HW. 2011. P values: use and misuse in medical literature. *Am J Hypertens* 24:18–23.
- Connell B. 2012. Materials and methods. In: Connell B, Jones AG, Redfern R, Walker D, editors. *A bioarchaeological study of medieval burials on the site of St Mary Spital*. London: Lavenham Press. p 19–24.
- Connell B, Redfern R, Thomas C. 2012. Introduction. In: Connell B, Jones AG, Redfern R, Walker D, editors. *A bioarchaeological study of medieval burials on the site of St Mary Spital*. London: Lavenham Press. p 1–18.
- DeWitte SN. 2010. Sex differentials in frailty in medieval England. *Am J Phys Anthropol* 143:285–297.
- DeWitte SN. 2014a. Differential survival among individuals with active and healed periosteal new bone formation. *Int. J. Paleopathol.* 7:38–44.
- DeWitte SN. 2014b. Health in post-Black Death London (1350–1538): age patterns of periosteal new bone formation in a post-epidemic population. *Am J Phys Anthropol* 155:260–267.
- DeWitte SN. 2014c. Mortality risk and survival in the aftermath of the medieval Black Death. *PLoS ONE* 9:e96513.
- DeWitte SN, Bekvalac J. 2011. The association between periodontal disease and periosteal lesions in the St. Mary Graces cemetery, London, England AD 1350–1538. *Am J Phys Anthropol* 146:609–618.
- DeWitte SN, Hughes-Morey G. 2012. Stature and frailty during the Black Death: the effect of stature on risks of epidemic mortality in London, A. D. 1348–1350. *J. of Archaeological Sci.* 39:1412–1419.

- DeWitte SN, Wood JW. 2008. Selectivity of Black Death mortality with respect to preexisting health. 105:1436–1441. PNAS
- Duray SM. 1996. Dental indicators of stress and reduced age at death in prehistoric Native Americans. *Am J Phys Anthropol* 99:275–286.
- Dyer C. 1998. Standards of living in the later Middle Ages: social change in England c 1200–1500, revised edition. Cambridge: Cambridge University Press.
- Dyer C. 2002. Making a living in the middle ages: the people of Britain 850–1520. New Haven, CT: Yale University Press.
- Dyson T, Ó Gráda C, eds. 2002. Famine demography: perspectives from the past and present. Oxford: Oxford University Press.
- Farr W. 1846. The influence of scarcities and of high prices of wheat on the mortality of the people of England. *J. Stat Soc. Lond* 9:158–171.
- Ferembach D, Schwidetzky I, Stoukal M. 1980. Recommendations for age and sex diagnoses of skeletons. *J Hum Evol* 9: 517–549.
- Fischer J, Jung N, Robinson N, Lehmann C. 2015. Sex differences in immune responses to infectious diseases. *Infection* 43: 399–403.
- Gage TB. 1988. Mathematical hazard models of mortality: an alternative to model life tables. *Am J Phys Anthropol* 76:429–441.
- Gage TB. 1989. Bio-mathematical approaches to the study of human variation in mortality. *Yearb. Phys Anthropol* 32:185–214.
- Gallay SH, Miura Y, Commisso CN, Fitzsimmons JS, O'Driscoll SW. 1994. Relationship of donor site to chondrogenic potential of periosteum in vitro. *J Orthop Res* 12:515–525.
- Gompertz B. 1825. On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. *Philos Trans R. Soc. London, Ser A* 115:513–585.
- Goodman AH, Armelagos GJ. 1988. Clinical stress and decreased longevity in a prehistoric population. *Am Anthropol* 90:936–944.
- Goodman AH, Armelagos GJ, Rose JC. 1980. Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Hum Biol* 52:515–528.
- Goodman SN. 1999. Toward evidence-based medical statistics. 1: The P-value fallacy. *Ann Intern Med* 130:995–1004.
- Gowland RL. 2015. Entangled lives: implications of the developmental origins of health and disease hypothesis for bioarchaeology and the life course. *Am J Phys Anthropol* DOI: 10.1002/ajpa.22820.
- Gowland RL, Chamberlain AT. 2005. Detecting plague: palaeodemographic characterization of a catastrophic death assemblage. *Antiquity* 79:146–157.
- Grauer AL. 1993. Patterns of anemia and infection from medieval York, England. *Am J Phys Anthropol* 91:203–213.
- Green JA. 1988. Log-linear analysis of cross-classified ordinal data: applications in developmental research. *Child Dev* 59:1–25.
- Hanawalt BA. 1993. Growing up in medieval London. Oxford: Oxford University Press.
- Heijmans BT, Tobi EW, Stein AD, Putter H, Blauw GJ, Susser ES, Slagboom PE, Lumey LH. 2008. Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proc Natl Acad Sci USA* 105:17046–17049.
- Herrmann NP, Konigsberg LW. 2002. A re-examination of the age-at-death distribution of Indian Knoll. In: Hoppa RD, Vaupeel JW, eds. *Paleodemography: age distribution from skeletal samples*. Cambridge: Cambridge University Press. p 243–257.
- Işcan MY, Loth SR, Wright RK. 1984. Age estimation from the rib by phase analysis: white males. *J Forensic Sci* 29:1094–1104.
- Işcan MY, Loth SR, Wright RK. 1985. Age estimation from the rib by phase analysis: white females. *J Forensic Sci* 30:853–863.
- Jones AG. 2012. Defining catastrophe: mass burial at St Mary Spital. In: Connell B, Jones AG, Redfern R, Walker D, eds. *A bioarchaeological study of medieval burials on the site of St Mary Spital*. London: Lavenham Press. p 217–231.
- Jones AG, Walker D. 2012. Demography. In: Connell B, Jones AG, Redfern R, Walker D, eds. *A bioarchaeological study of medieval burials on the site of St Mary Spital*. London: Lavenham Press. p 25–35.
- Keene DJ. 1984. A new study of London before the great fire. In: *Urban History Yearbook*. London: Leicester University Press. p 11–21.
- Klaus HD. 2014. Frontiers in the bioarchaeology of stress and disease: cross-disciplinary perspectives from pathophysiology, human biology, and epidemiology. *Am J Phys Anthropol* 155: 294–308.
- Klein SL. 2000. The effects of hormones on sex differences in infection: from genes to behavior. *Neurosci Biobehav Rev* 24: 627–638.
- Klein SL, Hodgson A, Robinson DP. 2011. Mechanisms of sex disparities in influenza pathogenesis. *J Leukoc Biol* 92:67–73.
- Klein SL, and Roberts C, editors. 2010. *Sex hormones and immunity to infection*. Heidelberg: Springer.
- Kowaleski M. 2013. Gendering demographic change in the Middle Ages. In: Bennett JM, Karras RM, editors. *The Oxford handbook of women and gender in medieval Europe*. Oxford: Oxford University Press. p 181–196.
- Kowaleski M. 2014. Medieval people in town and country: new perspectives from demography and bioarchaeology. *Speculum* 89:573–600.
- Lang JM, Rothman KJ, Cann CI. 1998. That confounded P-value. *Epidemiology* 9:7–8.
- Larsen CS. 1997. *Bioarchaeology*. Cambridge: Cambridge University Press.
- Lebeck J, Gena P, O'Neill H, Skowronski MT, Lund S, Calamita G, Praetorius J. 2012. Estrogen prevents increased hepatic aquaporin-9 expression and glycerol uptake during starvation. *Am J Physiol Gastrointest Liver Physiol* 302:G365–374.
- Leyser H. 1995. *Medieval women: a social history of women in England 450–1500*. New York: St. Martin's Press.
- Lovejoy CO, Meindl RS, Pryzbeck TR, Mensforth RP. 1985. Chronological metamorphosis of the auricular surface of the ilium: a new method for the determination of adult skeletal age at death. *Am J Phys Anthropol* 68:15–28.
- Lucas HS. 1930. The great European famine of 1315, 1316, and 1317. *Speculum* 5:343–377.
- Maharatna A. 1996. *The demography of famines: an Indian historical perspective*. Delhi: Oxford University Press.
- Margerison BJ, Knüsel CJ. 2002. Paleodemographic comparison of a catastrophic and an attritional death assemblage. *Am J Phys Anthropol* 119:134–143.
- McIlvaine BK. 2013. Implications of reappraising the iron-deficiency anemia hypothesis. *Int J Osteoarchaeol* DOI: 10.1002/oa.2383.
- McIntosh MK. 2005. *Working women in English society, 1300–1620*. Cambridge: Cambridge University Press.
- Milner GR, Wood JW, Boldsen JL. 2008. Advances in paleodemography. In: Katzenberg MA, Saunders SR, editors. *Biological anthropology of the human skeleton*, 2nd ed. New York: Wiley-Liss. p 561–600.
- Morgan J. 2013. The invisible hunger: is famine identifiable from the archaeological record? *Antrocom Online J Anthropol* 9:115–129.
- Nagaoka T, Hirata K, Yokota E, Matsu'ura S. 2006. Paleodemography of a medieval population in Japan: analysis of human skeletal remains from the Yuigahama-minami site. *Am J Phys Anthropol* 131:1–14.
- Ó Gráda C. 2007. Making famine history. *J. Econ. Lit.* 45:5–38.
- Ortner DJ. 1991. Theoretical and methodological issues in paleopathology. In: Ortner DJ, Aufderheide AC, eds. *Human paleopathology: current syntheses and future options*. Washington, DC: Smithsonian Institution Press. p 5–11.
- Ortner DJ. 2003. *Identification of pathological conditions in human skeletal remains*, 2nd ed. Waltham, MA: Academic Press.
- Oxenham MF, Cavill I. 2010. Porotic hyperostosis and cribra orbitalia: the erythropoietic response to iron-deficiency anaemia. *Anthropol Sci* 118:199–200.

- Parrack DW. 1978. Ecosystems and famine. *Ecol Food Nutr* 7: 17–21.
- Phenice TW. 1969. A newly developed visual method of sexing the os pubis. *Am J Phys Anthropol* 30:297–302.
- Poos LR. 1998. Population and demography. In: Szarmach PE, Tavormina MT, Rosenthal JT, eds. *Medieval England: an encyclopedia*. New York: Garland. p 605–607.
- Pottier JP. 1986. The politics of famine prevention: ecology, regional production and food complementarity in Western Rwanda. *Afr Aff (Lond)* 85:339–
- Powers N. 2012. Human osteology method statement. Available online at <http://archive.museumoflondon.org.uk/NR/rdonlyres/3A7B0C25-FD36-4D43-863E-B2FDC5A86FB7/0/OsteologyMethodStatementrevised2012.pdf>.
- Ravallion M. 1997. Famines and economics. *J Econ Lit* 35: 1205–1242.
- Rigby SH. 2010. Urban population in late Medieval England: the evidence of the lay subsidies. *Econ Hist Rev* 63:393–417.
- Roberts C, Manchester K. 2007. *The archaeology of disease*, 3rd ed. Ithaca, NY: Cornell University Press.
- Rothman KJ. 1998. Writing for epidemiology. *Epidemiology* 9: 333–337.
- Sawchuk LA, Tripp L, Damouras S, DeBono M. 2013. Situating mortality: quantifying crisis points and periods of stability. *Am J Phys Anthropol* 152:459–470.
- Scrimshaw NS. 1987. The phenomenon of famine. *Annu Rev Nutr* 7:1–21.
- Sen A. 1981. Ingredients of famine analysis: availability and entitlements. *Q J Econ* 96:433–464.
- Sidell J, Thomas C, Bayliss A. 2007. Validating and improving archaeological phasing at St. Mary Spital, London. *Radiocarbon* 49:593–610.
- Sloane B. 1999. Reversing the dissolution: reconstructing London's medieval monasteries. *Trans London Middlesex Archaeol Soc* 50:67–77.
- Stothers RB. 2000. Climatic and demographic consequences of the massive volcanic eruption of 1258. *Clim Changes* 45:361–374.
- Stuart-Macadam PL. 1991. Anemia in Roman Britain: Poundbury Camp. In: Bush H, Zvelebil M, editors. *Health in past societies: biocultural interpretations of human skeletal remains in archaeological contexts*. Oxford: British Archaeological Research International Series. p 101–113.
- Stuart-Macadam PL. 1992. Anemia reevaluated: a look to the future. In: Stuart-Macadam PL, Kent S, editors. *Diet, demography, and disease: changing perspectives on anemia*. New York: Transaction Publishers. p 261–268.
- Sullivan DA, Vaerman JP, Soo C. 1993. Influence of severe protein malnutrition on rat lacrimal, salivary and gastrointestinal immune expression during development, adulthood and aging. *Immunology* 78:308–317.
- Temple DH. 2014. Plasticity and constraint in response to early-life stressors among late/final Jomon period foragers from Japan: evidence for life history trade-offs from incremental microstructures of enamel. *Am J Phys Anthropol* 155:537–545.
- Thomas C, Sloane B, Phillpotts C, editors. 1997. *Excavations at the priory and hospital of St Mary Spital, London*. Museum of London Archaeology Monograph Series 1. London: Lavenham Press.
- Tobi EW, Slagboom PE, van Dongen J, Kremer D, Stein AD, Putter H, Heijmans BT, Lumey LH. 2012. Prenatal famine and genetic variation are independently and additively associated with DNA methylation at regulatory loci within IGF2/H19. *PLoS ONE* 7:e37933
- Torry WI. 1984. Social science research on famine: a critical evaluation. *Hum Ecol* 12:227–252.
- Trafimow D, Marks M. 2015. Editorial. *Basic Appl Soc Psych* 37:1–2.
- Vaupel JW, Manton KG, Stallard E. 1979. The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16:439–454.
- Walker PL. 1995. Problems of preservation and sexism in sexing: some lessons from historical collections for palaeodemographers. In: Saunders SR, Herring A, editors. *Grave reflections: portraying the past through cemetery studies*. Toronto: Canadian Scholars' Press. p 31–47.
- Walker PL, Bathurst RR, Richman R, Gjerdrum T, Andrushko VA. 2009. The causes of porotic hyperostosis and cribra orbitalia: a reappraisal of the iron-deficiency-anemia hypothesis. *Am J Phys Anthropol* 139:109–125.
- Walter J, Schofield R, eds. 1989. *Famine, disease, and the social order in early modern society*. Cambridge: Cambridge University Press.
- Wapler U, Crubézy E, Schultz M. 2004. Is cribra orbitalia synonymous with anemia? Analysis and interpretation of cranial pathology in Sudan. *Am J Phys Anthropol* 123:333–339.
- Watkins SC, Menken J. 1985. Famines in historical perspective. *Popul Dev Rev* 11:647–675.
- Weston DA. 2008. Investigating the specificity of periosteal reactions in pathology museum specimens. *Am J Phys Anthropol* 137:48–59.
- White TD, Black MT, Folkens PA. 2011. *Human osteology*. Walnut, MA: Academic Press.
- Wiesner ME. 2000. *Women and gender in early Modern Europe*. New York: Cambridge University Press.
- Wood JW, Milner GR, Harpending HC, Weiss KM. 1992. The osteological paradox: problems of inferring prehistoric health from skeletal samples. *Curr Anthropol* 33:343–370.
- Wood JW, Holman DJ, O'Connor KA, Ferrell RJ. 2002. Mortality models for paleodemography. In: Hoppa R, Vaupel J, editors. *Paleodemography: age distributions from skeletal samples*. Cambridge: Cambridge University Press. p 129–168.
- Woolgar CM. 2006. Meat and dairy products in late medieval England. In: Woolgar CM, Serjeantson D, Waldron T, eds. *Food in Medieval England: diet and nutrition*. Oxford: Oxford University Press. p 88–101.
- Zuk M, and Stoehr AM. 2010. Sex differences in susceptibility to infection: an evolutionary perspective. In: Klein SL, and Roberts C, editors. *Sex hormones and immunity to infection*. Heidelberg: Springer. p 1–17.