The Pennsylvania State University

The Graduate School

College of the Liberal Arts

THE PALEODEMOGRAPHY OF THE BLACK DEATH 1347-1351

A Thesis in

Anthropology

by

Sharon N. DeWitte

Submitted in Partial Fulfillment
of the Requirements
for the Degree of

Doctor of Philosophy

December 2006

The thesis of Sharon N. DeWitte was reviewed and approved* by the following:

James W. Wood
Professor of Anthropology and Demography
Thesis Advisor
Chair of Committee

Kenneth M. Weiss
Evan Pugh Professor of Anthropology and Genetics

Jeffrey Kurland
Associate Professor of Anthropology

Ottar Bjørnstad
Associate Professor of Entomology

Jesper Boldsen
Associate Professor, Institute of Forensic Medicine
University of Southern Denmark
Special Member

Nina Jablonksi
Professor of Anthropology
Head of the Department of Anthropology

*Signatures are on file in the Graduate School

ABSTRACT

The Black Death of 1347-1351 has long been considered one of the most devastating epidemics in human history; it killed an estimated 30-50 percent of the European population and initiated profound social, economic, and demographic changes throughout the continent. Among other things, the Black Death has been credited with ending the medieval feudal system and exacerbating social conflict between the wealthy and poor. Because the Black Death had important consequences both culturally and demographically, it has fascinated researchers for decades, yet there are still important questions about the medieval epidemic that have remained unanswered.

By comparing a Black Death cemetery to a pre-Black Death, normal mortality cemetery, this project seeks to determine how Black Death mortality was distributed by age and sex and whether the disease was selective with respect to frailty. This project incorporates a newly developed method of adult age-at-death estimation and a multistate model of morbidity and mortality. The results indicate that the Black Death differentially affected individuals with pre-existing health conditions. The Black Death, however, was not as strongly selective as was normal mortality. The epidemic was highly virulent and therefore killed otherwise healthy individuals who would have been at low risk of death under normal circumstances. However, the Black Death did not, as many have assumed, killed people indiscriminately.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACKNOWLEDGEMENTS

I have been fortunate enough in my graduate school career to have worked with and been instructed and advised by so many of the wonderful faculty at Penn State. I am very grateful to my advisor, Jim Wood, for his guidance, instruction, seemingly infinite patience, and great friendship during the last seven years of my graduate career (and, hopefully, for the years to come). I am also thankful for his generosity in loaning novels to me, which helped in no small way to keep me sane during my months of research in Denmark. I am thankful to the other members of my dissertation committee – Kenneth Weiss, Jeffrey Kurland, Ottar Bjørnstad, and Jesper Boldsen – who have generously given their time to help me improve this dissertation. Kenneth Weiss has also provided invaluable career advice, and I appreciate his strong encouragement to pursue whichever career path will make me happiest. It was a joy to be a teaching assistant for so many years for Jeffrey Kurland; he is an excellent teacher and a wonderful friend (and a great musician), and I strive to live up to his example. I would like to thank Mark Shriver, who generously supported me during my "pre-Black Death" phase. I would like to thank Pat Johnson for welcoming me into her home so many times, for her friendship, and for advising me on matters both professional and personal.

This dissertation would not have been possible without the generous help of people at the Anthropological Database at Odsense University, Denmark and the Museum of London. At ADBOU, Jepser Boldsen and Ulla Freund gave me unlimited access to the Danish skeletons included in my sample. Bill White and and his team at the Museum of London Centre for Human Bioarchaeology helped me with the sometimes

arduous task of retrieving East Smithfield skeletons from storage and truly made me feel like a member of their team.

I am grateful to Bethany Usher, whose model I use and who generously gave me examples of her own programs. I would like to thank Darryl Holman for patiently answering my many questions about *mle* and for helping me at each step of my analyses.

I would like to thank my Shippensburg University colleagues, Christine Loveland and Karl Lorenz, for their encouragement, advice, and friendship.

I have made so many friends at Penn State who have made the tough times bearable and the rest of the time (which was most of the time) so much fun. I would like to thank Jason DeLeon, Abby Bigham, Holly Dunsworth, Corey Sparks, Maria Inclan, Geoff Vasile, Erick and Sarah Rochette, Sam Sholtis and Felicity for making me laugh and for making grad school so much more than an academic pursuit. In addition to being a great friend, Corey Sparks also helped me immeasurably with the execution of my analyses. I am so happy to have Eric Jones in my life - my best friend, he has believed that I could finish this, even when I doubted it myself, and encourages me to relax once in a while.

Lastly, I want to thank my parents, Gordon and Dorothy DeWitte, and my sister, Jennifer DeWitte for supporting me and believing in me. I am so lucky to have such a great family, and I could not have finished without their love and encouragement ("I can BO it!").

This research was funded by the American-Scandinavian Foundation, the National Science Foundation (#0406252), the Wenner-Gren Foundation, a grant from the

Penn State Research and Graduate Studies Office, and a Hill Fellowship from the Penn State Anthropology Department.

Chapter 1

INTRODUCTION AND BACKGROUND

Introduction

The Black Death of 1347-50 has fascinated both researchers and lay people for over six hundred years[1]. The medieval epidemic had profound consequences both culturally and demographically and it did much to shape human history. There are references to the Black Death in popular films and novels. For example, the main character of Ingmar Bergman's film The Seventh Seal is pursued by Death during the medieval epidemic. The company Giant Microbes™ has even made a plush toy representing the purported cause of the Black Death: the plague bacterium *Yersinia pestis*.

The Black Death has been studied extensively for decades by historians, demographers, anthropologists, epidemiologists, molecular biologists, and others. Yet,

---

[1] The term "Black Death" was not actually used at the time of the fourteenth-century epidemic. Contemporaries referred to the epidemic with such terms as *pestis*, *pestilentia*, *epidemia*, and *magna mortalitas*. The first known uses of the term "Black Death" appear in sixteenth-century Danish and Swedish chronicles (*den sorte död*, *swarta dödhen*) (Simpson and Weiner 1989), and the term did not appear in English writings until the nineteeth century (e.g. Penrose 1859).

despite such a long history of study, there are several important questions about the Black Death that either remain unanswered, or have not yet been answered completely. The Black Death was obviously extremely virulent, as it killed between 30 and 50 percent of the European population. But it is not clear what the true pattern of Black Death mortality was. We do not know, for example, how Black Death mortality was distributed by age and sex. Were the very young and the elderly at higher risk of death than other age groups, or were all ages at equal risk of dying during the epidemic? Was either sex at higher risk, or were men and women at equal risk? We also do not know if the Black Death was selective with respect to health, or if it killed people indiscriminately. Were people who were already in poor condition before the Black Death at a higher risk of death during the epidemic than healthier individuals? The Black Death was new to medieval Europe, and it was clearly quite virulent, so it is tempting to think that all people were at equal risk of dying during the epidemic. But is this true? I have attempted to answer these questions about the epidemiology of the Black Death using large skeletal collections from Denmark and England and new paleodemographic methods: the Rostock protocol for adult age-at-death estimation (Hoppa and Vaupel, 2002) and the Usher multistate model of morbidity and mortality (Usher, 2000).

The Black Death can be viewed as one of the most important emerging diseases in human history. Emerging diseases are those that are increasing in frequency after being introduced into a new host population, and much research attention is currently focused on understanding modern emerging diseases such as HIV/AIDS, West Nile Virus, Ebola, and SARS. Understanding how emerging diseases have behaved in the past may help us

to understand, predict or even ameliorate the effects of those emerging today and in the future.

Outline of Thesis

In Chapter 1, I provide a general description of the Black Death, including some of the purported demographic and cultural effects of the epidemic. I describe the epidemiological patterns of the medieval disease as reconstructed from historical documents. Chapter 1 summarizes previous studies of the mortality patterns of the Black Death using historical documents and cemetery samples, stressing the potential limitations of those previous studies.

Chapter 2 describes the cemetery samples and methods I used to evaluate the mortality patterns and selectivity of the Black Death. I compared the East Smithfield Black Death cemetery from London (n = 490) to a pre-Black Death, normal mortality cemetery sample from medieval Denmark (n = 291). I scored all skeletons for age, sex (in adults), presence of non-specific skeletal lesions, and state of preservation. For adults in each sample, I estimated age-at-death using a new method of estimation, the Rostock protocol, that is designed to eliminate the biases associated with traditional age estimation methods. I analyzed the age pattern of mortality using the Siler mortality model, and I analyzed the selectivity of the Black Death with respect to frailty and sex using the Usher (2000) model of morbidity and mortality. Chapter 2 also describes a measurement error study evaluating my consistency in scoring skeletons for age, sex, and presence of skeletal lesions.

The results are presented in Chapter 3, and Chapter 4 provides a discussion of these results, their relevance to understanding the mortality patterns of the Black Death and other disease epidemics, and possible studies I might undertake in the future using the same dataset. The appendices provide the scoring protocols for sex and age, estimates of individuals sex and age, parameter estimates, the Usher model likelihood equations, results of the measurement error studies and of the simulation studies to test the capabilities of the estimation program *mle*, and an example of a computer program written for this study.

The Black Death

Consequences of the Black Death

The Black Death has long been considered the most devastating epidemic in human history; it killed an estimated 30-50 percent of the European population and initiated or hastened profound social, economic, and demographic changes throughout the continent (Bowsky, 1971; Gottfried, 1983; Horrox, 1994; Herlihy, 1997; Cohn, 2002; Hinde, 2003). Between 1346 and 1353, the Black Death spread throughout western Asia, the Middle East, North Africa, and Europe. The spread of the Black Death in Asia and Africa is not documented as well as the spread in the West, and the exact geographic origin of the Black Death is not certain; the general consensus is that the epidemic began somewhere in Asia and traveled west along trade routes to the Black Sea (Horrox, 1994). Gottfried (1983) argues that the Golden Horde (the Mongols who ruled over the steppes

of southern Russia) were ultimately responsible for spreading the disease. In 1345-46, the Khan of the Golden Horde besieged a Genoese colony in the Black Sea port of Kaffa (the present-day Crimean port of Theodosia) and allegedly infected the inhabitants (Horrox, 1994; Herlihy, 1997). Italian merchant ships then brought the Black Death from Kaffa to Constantinople and other ports along the coast of the Mediterranean Sea in 1347; from these ports, the Black Death swept throughout Asia Minor, the Middle East, North Africa, and Europe (Horrox, 1994; Benedictow, 2004).

The Black Death has been credited with ending the feudal system, hastening the emergence of a capitalist market society, exacerbating social conflict, motivating technological innovations, and changing people's perceptions of and attitudes about death. Most of the social and economic changes wrought by the Black Death are related to the massive depopulation caused by the epidemic. There is some debate regarding whether the Black Death hastened an existing trend of population decline. Some researchers argue that on the eve of the Black Death, population growth in Europe had been curtailed by the natural limits of the prevailing agricultural system and some areas experienced declining populations (Poos, 1991; Herlihy, 1997; Hinde, 2003). For example, according to Poos (1991), the three decades before the Black Death were characterized by persistent decreases in population in the English county of Essex. Similar trends of slowly declining populations had begun even earlier in other regions of England (Hinde, 2003). Regardless of whether the European population was already declining before the Black Death, there is no doubt that the epidemic had a significant effect upon the population of Europe and was the primary reason for the *steep* decline in population in the mid-fourteenth century (Hatcher, 1977; Hinde, 2003).

In addition to killing so many people, the Black Death caused a dramatic displacement of local populations; many people fled to uninfected areas, as they realized at the time of the Black Death that flight was the only effective method of avoiding the disease – an option that was of course available only to those with sufficient means (Horrox, 1994). Some areas in Europe were entirely depopulated by death and abandonment (Horrox, 1994).

Following the Black Death, the European population was slow to recover, despite apparent increases in standards of living; this slow recovery was likely the result of repeated outbreaks of plague (Hatcher, 1977; Poos, 1991; Horrox, 1994; Hinde, 2003). In England, for example, repeated outbreaks of plague caused a period of demographic stagnation that lasted until the beginning of the sixteenth century (Hinde, 2003); according to Smith (2002), it was not until the eighteenth century that the English population finally returned to pre-Black Death levels.

By killing so many people, the Black Death created a scarcity of labor that ultimately may have led to the end of the feudal system. There is a debate regarding the importance of the epidemic in the demise of feudalism (Horrox,1994). Some researchers argue that the Black Death alone changed the European economic system. Others argue that the epidemic was just one of many factors influencing economic change; the Black Death may have simply hastened changes that were inevitable. Regardless of one's perspective on the priority of the Black Death, there is little doubt that the epidemic had economic consequences. The medieval feudal system was dependent upon an abundance of labor and a shortage of land (Hinde, 2003). Peasants paid rent to lords of the manor for arable land and for homes and gardens within manor villages; some peasants were

also obligated to work on the demesne (manorial land reserved for the use of the lord), the products of which were kept by the lord (Gottfried, 1983). Because of the scarcity of arable land, peasants had little ability to negotiate wages, labor obligations, or rents. These conditions changed in many regions after the Black Death. The epidemic created a shortage of workers and in general the supply of land exceeded demand, and many peasants thereby gained the bargaining power to reduce their labor obligations or rents or to increase their wages (Poos, 1991; Horrox, 1994). Peasants were able to leave their lords' manors and acquire employment or land elsewhere (Dyer, 2002). The power of lords was diminished (Horrox, 1994), and many of the nobility and clergy who had previously depended upon the rents paid by peasants were ruined. According to Benedictow (2004), the Hundred Years War, which began in 1337, lasted as long as it did because it provided a way to make money for nobility whose incomes had been reduced as a result of the Black Death.

The Black Death provided new employment opportunities for some people; for example, labor obligations of peasants were forgotten or cancelled in many cases and people were therefore free to find new lines of work or establish new kinds of businesses (Thompson, 1971; Horrox, 1994). Wages for surviving paid laborers increased following the epidemic; according to Hatcher (1977), in late fourteenth and fifteenth centuries, the real wages of craftsmen and laborers in England apparently reached levels that were not surpassed until the late nineteenth century. In Essex, for example, wages rose by approximately one-third or more in the decades following the Black Death (Poos, 1991). With rising wages and decreasing rents, the standard of living for many people increased following the Black Death (Poos, 1991; Horrox, 1994; Hinde, 2003).

According to some researchers, the Black Death created conditions that allowed for a more diversified economy (Horrox, 1994; Herlihy, 1997; Smith, 2002). In the feudal system, the basic mode of production, the small peasant farm, was worked with technology that had remained unchanged for many years before the Black Death. The only way to increase production within the feudal system was to expand areas under grain cultivation, but arable land was, of course, limited, and production was subject to the law of diminishing returns. By reducing population sizes, the Black Death made it possible for people to use the land in different ways (Poos, 1991). In general, land was not simply abandoned, but rather was withdrawn from agricultural use and put to other, less intensive uses; for example, much agricultural land was converted to pasture (Smith, 2002). Mills that had previously been used exclusively for grinding grain could be used for cloth production and sawing wood. In Essex, the rural cloth industry expanded after the Black Death, as former agricultural workers were free to pursue alternative employment (Poos, 1991). According to some researchers, the tremendous loss of life and the resulting high cost of labor spurred the development of labor-saving devices, and such technological innovations perhaps paved the way for the Industrial Revolution (Herlihy, 1997; Benedictow, 2004).

The Black Death may have exacerbated social conflict between peasants and nobility in the countryside and in cities. Governments attempted to restrict rising wages and to curtail the decreases in rents following the epidemic. In England, for example, King Edward III issued the Ordinance of Labourers in 1349 and thereby ordered that all individuals able to work who were offered employment were "obliged to accept the employment offered, and they should be paid only the fees, liveries, payments or salaries

which were usually paid in the part of the country where they are working in the twentieth year of our reign [1346]" (Luders et al., 1994: 288); thus the government attempted to freeze wages at their pre-Black Death levels. Any worker who demanded higher wages was ordered to be imprisoned until he or she agreed to work for pre-Black Death wages. Employers who paid high wages were to be fined. The English Parliament later attempted to reinforce the ordinance with the Statute of Labourers in 1351 (Poos, 1991).

These attempts to cap wages may have motivated social uprisings. According to Poos (1991), the attempts of lords to enforce labor obligations and the governments' attempts to restrict wages created an anti-authoritarian sentiment among the English peasantry that ultimately led to the peasant revolt of 1381. Renouard (1971) credits the Black Death with directly leading to such rural peasant uprisings as the Jacquerie (1358) and the Tuchins (1381-82) in France and the Laborers (1381) in England, and such urban working class insurrections as the Weavers (1379) and the Maillotins (1382) in France.

The Black Death was viewed by some as a punishment from God for their sins (Horrox, 1994). This belief motivated penitential movements, such as the Flagellant movement, as efforts to appease God and prevent further epidemics. The Flagellants traveled from town to town and scourged themselves in public to make amends for the sins of the world (Zeigler, 1971; Horrox, 1994). Many new churches were built throughout Europe and charitable donations increased in an attempt to curtail the wrath of God and attain salvation (Gottfried, 1983; Horrox, 1994; Benedictow, 2004). Some people responded to the epidemic by punishing those they believed to be at fault for the disease, including foreigners, the poor, travelers, and, most commonly, Jews (Horrox,

1994). The Black Death incited persecution of the Jews, as they were accused of poisoning wells and rivers and corrupting the air and thereby causing the epidemic (Horrox, 1994). The fact that Jews seemed to die of the disease as readily as Christians was apparently ignored, and they were massacred in many parts of Europe (Kosminskii, 1971; Horrox, 1994; Benedictow, 2004).

Epidemiological Patterns of the Black Death

Historical documents provide some information about the epidemiology of the Black Death. For example, many contemporary chroniclers described the symptoms of the disease. According to Herlihy, (1997), the "most common sign" of medieval plague was *lenticulae* (the Latin term for freckles), pustules or subcutaneous hemorrhages (petechiae and ecchymoses) covering large areas of the body. The Black Death was also characterized by the development of huge swellings, or buboes, in the armpit, groin, or neck. Giovanni Boccaccio describes the "plague-boils" of the Black Death in *The Decameron*: "certain swellings, either in the groin or under the armpits, whereof some waxed of the bigness of a common apple, others like unto an egg, some more and some less" (in Bowksy, 1971:7). Many contemporary descriptions of Black Death mention the awful stench produced by sufferers of the disease; according to the chronicler Papon, the "sweat, excrement, spittle, breath" of Black Death victims was "so foetid as to be overpowering" (quoted in Ziegler, 1969:20). Other symptoms included fever, bloody sputum, and chills. Victims died within about five days of the appearance of such symptoms (Scott and Duncan, 2001).

Modern epidemiological studies of historical documents have confirmed the devastating nature of the Black Death and have shown that mortality was perhaps even higher than previously thought (Wood et al., n.d.; Wood et al., 2001; Scott and Duncan, 2001; Cohn, 2002). Using data from court records of payments made by residents, Poos (1991) estimated the annual totals of males ages twelve years and older living on manors in Essex; he found that several manors lost as much as 54 percent of their male residents during the Black Death. Wood et al. (2002) analyzed data on deaths of beneficed priests in the Lincoln diocese; in the eighteen months before the Black Death, the mean mortality rate for priests was 38.9 per 1000; during the twelve-month period beginning with the outbreak of the Black Death in Lincoln, the mean mortality rate increased to 463.6 per 1000. During the Black Death, annual mortality rates for priests were eleven times higher, and, more dramatically, *monthly* mortality rates were about thirty-five to forty-five times higher than in the pre-epidemic period. Total mortality from the Black Death was typically 30-50 percent of the total population in affected regions. Following the Black Death, plague mortality decreased steeply and steadily with subsequent outbreaks, which strongly suggests that Europeans adapted to the presence of the disease by acquiring immunity (Carpentier, 1971; Hatcher, 1977; Cohn, 2002).

According to the majority of accounts, the Black Death was spread very readily from person to person. Some contemporaries were aware that the Black Death was spread from person to person; for example, people observed that if one individual in a household was infected, other members of the household soon thereafter became infected (Cohn, 2002). Boccaccio wrote about the Black Death that “this pestilence was so powerful that it was communicated to the healthy by contact with the sick, the way a fire

close to dry or oily things will set them aflame" (Musa and Bondanella, 1977: 4). According to Louis Heyligen, a medieval chronicler, "when one person dies everyone who saw him during his illness, visited him, had any dealings with him, or carried him to burial, immediately follows him, without any remedy" (Horrox, 1994: 42). In late medieval Italy, during the epidemics following the Black Death, the overwhelming majority of families (96 percent of those for whom we have records) experienced multiple cases of the disease (Scott and Duncan, 2001).

Though there was little debate that the disease spread from person to person, contemporaries differed in their beliefs regarding how exactly the disease spread from person to person, whether it was by breath, touch, sharing food, sweat, the odor from feces or decaying bodies or some other mechanism (Cohn, 2002). Boccaccio wrote that "not only did it infect healthy persons who conversed or had any dealings with the sick … but it also seemed to transfer the sickness to anyone touching the clothes or other objects which had been handled or used by its victims" (1994: 28). Many contemporaries believed the Black Death was caused by corruption of the air, and people were therefore advised to surround themselves with "pleasant smells" and thereby "create a barrier of aromatic vapours through which the bad air could not penetrate" in order to prevent infection (Horrox, 1994: 100). Doctors tending to victims of the epidemic were advised to stand near an open window or hold a sponge soaked with vinegar or something aromatic to their noses (Michon, 1994). During outbreaks of plague subsequent to the Black Death, physicians wore stylized masks filled with herbs and spices when they treated patients suffering from the disease (Figure **1-1).** The herbs and spices were

thought to ward off infection, and the use of these masks further suggests that contemporaries believed the disease was spread from person to person.

In an effort to stem the spread of the medieval disease, forty-day quarantines were established beginning in the late fourteenth century; such measures were believed effective and could only have been so if the disease was spread from person to person.

Figure 1-1: Physician during the 17th-century wearing clothing thought to ward off infection. Etching by Paulus Furst of Nuremberg, Germany, 1656. Illustration from http://bcm.bc.edu/issues/winter_2005/ll_plague.html

Wood et al. (2003) examined bishops' registers from England to investigate the timing of the spread of the Black Death and found that the epidemic lasted only about four to six months in each parish and spread very quickly between parishes. Parishes

included several communities, so the timing of the disease would have been even more rapid at the individual community level. The Black Death spread quickly almost certainly because it was spread from person to person.

Using detailed historical documents from English villages, Scott and Duncan (2001) estimated the epidemiological parameters of epidemics of plague subsequent to the Black Death; the authors argue that these subsequent outbreaks were caused by the same disease that caused the Black Death. Scott and Duncan estimated that these plagues were characterized by a latent period (the period following exposure to the disease during which the patient is asymptomatic) of 10 to 12 days, an infectious period before symptoms lasting 20 to 22 days, and a period of symptoms lasting 5 days. Thus, during the medieval epidemic, infected individuals were capable of infecting others for 25 to 27 days, and the total time from point of infection to death was as long as 37 days. Because of the time course of the medieval disease, people could travel long distances and infect many others before being aware of any symptoms. According to Scott and Duncan, plague was brought to the village of Penrith in 1598 by a visitor who was infected elsewhere and arrived in the village without any symptoms; the death of the visitor was followed by an interval of 22 days before a villager succumbed to the same disease. Individuals during the Black Death would have been infectious and yet still healthy enough to travel long distances and thereby spread the disease from one village to another.

Despite the wealth of information available in historical documents, there is still much that is unknown about the epidemiology of the Black Death, and there remains a need to study the epidemic using appropriate biological materials.

The East Smithfield Black Death Cemetery

During the Black Death, existing cemeteries proved inadequate to accommodate the huge numbers of people killed by the disease. Several chroniclers commented on the near impossibility of providing normal burials for all the victims of the epidemic. Mass burial grounds were therefore established across Europe during the epidemic. As one chronicler, Agnolo di Tura de Grasso, wrote, "in many places … great pits were dug and piled deep with the multitude of dead" (quoted in Bowsky, 1971). One such mass burial ground was the East Smithfield cemetery in London. The East Smithfield cemetery is the only known, excavated cemetery that has indisputable evidence linking it to the Black Death, and it therefore provides an ideal sample for investigating the epidemic.

The Black Death was apparently introduced to England during the summer of 1348 via the Dorsetshire port of Melcombe Regis, and it reached the suburbs of London as early as September 1348 (the disease was recorded in the suburb of Stepney, two miles east of the city walls, in December 1348); the epidemic ravaged the city throughout 1349 and ended by the spring of 1350 (Gasquet, 1893; Hawkins, 1990). Aware that the Black Death was spreading towards London, "substantial men of the city" ordered that emergency burial grounds be established prior to its arrival (Hawkins, 1990.). The East Smithfield cemetery was established in late 1348 or early 1349 on land just outside the city walls that had previously been used as a vineyard. The East Smithfield cemetery was originally called the Churchyard of the Holy Trinity, as the land was acquired from the prior of Holy Trinity without Aldgate. The Cartulary of Holy Trinity provides the exact dimensions and location of the cemetery (Hawkins, 1990).

For years, researchers believed that part or all of the East Smithfield cemetery had been destroyed, but trial excavations in 1984 revealed intact burials (Hawkins, 1990). The cemetery was fully excavated in the 1980s as part of the larger Royal Mint Site by the Museum of London's Department of Greater London Archaeology (one of the precursors to the current Museum of London Archaeological Service). The Royal Mint site is located in East London, northeast of the Tower of London. **Figure 1-2** shows the East Smithfield cemetery during the excavation. The burials in East Smithfield were concentrated in two areas: 1) a western area with two mass burial trenches, a mass burial pit, and individual graves arranged in several parallel north-south rows, and 2) an eastern area with one mass burial trench and individual graves arranged in several parallel rows (Hawkins, 1990). **Figure 1-2** shows part of the western area of the cemetery, and numerous individual graves and part of a mass burial trench with several skeletons still in situ are visible. Approximately 600 skeletons were excavated from the cemetery, a fraction of the estimated 2400 individuals originally buried in East Smithfield. The cemetery could have accommodated many more burials, but the epidemic apparently waned before it was necessary to use all the available space.

Excavation of the cemetery revealed that the individuals interred in East Smithfield were buried carefully. Almost without exception, the bodies were buried in standard medieval Christian fashion: extended on their back with their heads oriented west and feet oriented east. **Figure 1-3** shows a portion of one the mass burial trenches in the western area of the cemetery; all six adults in the photo are in the standard position

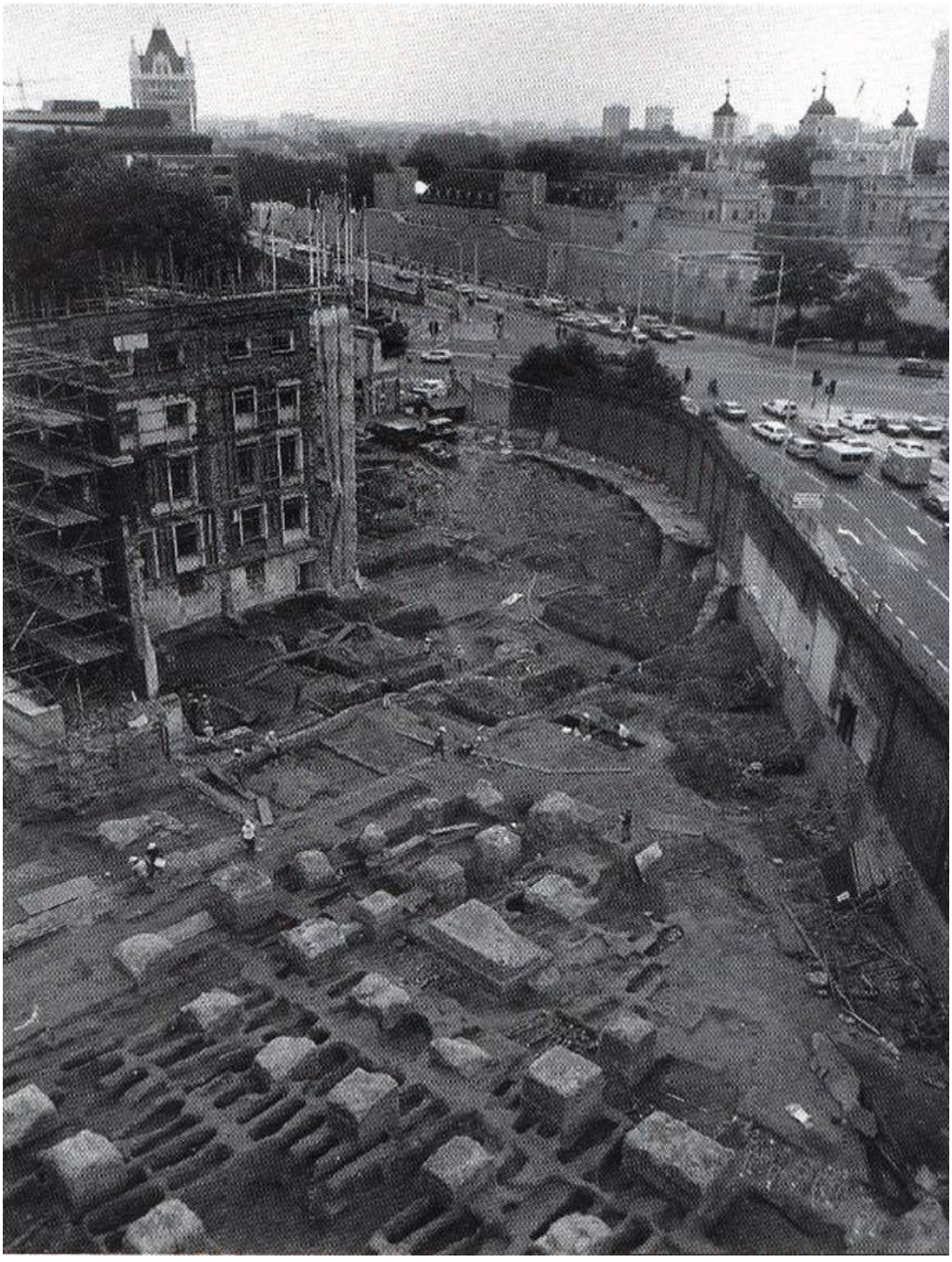

Figure 1-2: Photo of Royal Mint site during excavation. Individuals graves and sections of mass burial trenches that were part of the East Smithfield Black Death cemetery are visible in the lower half of the photo. Note the Tower of London and part of the Tower Bridge in the background. From Platt (1996: 6).

(note the arrow indicating north at the feet of the first skeleton in the trench). Some of the skeletons were missing limbs at the time of excavation, indicating that these individuals had been partly disarticulated as a result of putrefaction (Hawkins, 1990); even these bodies were buried in the standard Christain fashion and were treated with apparent care rather than tossed hastily into a burial trench.

The shape of the one of the trenches in the western area provides more evidence about burial practices in East Smithfield. This trench has a stepped base and is much deeper at the southern end, which would allow for access within the trench during burial; the trench was apparently filled from the southern end to the northern end with stacks of bodies, rather than from the bottom to the top of the trench with layers of bodies (Hawkins, 1990). In this manner, the bodies were interred carefully, rather than merely tossed into the trenches, and those responsible for burial also avoided stepping on previously buried, rotting bodies.

Typical medieval Christian burials do not include grave goods, but, perhaps because of the relative haste with which people were buried during the Black Death, some individuals in East Smithfield were interred with coins and other objects. The ages of these objects provide further evidence that the cemetery dates to the time of the Black Death. Several individuals in the East Smithfield cemetery were interred with coins whose minting dates provide evidence of the age of the cemetery. For example, one adult male was found with two distinct groups of coins, one to the right of his chest (likely from a pouch worn around the neck or over the shoulder) and the other within his pelvis (likely from a waist pouch); these two caches contained 181 coins, 75 of which were

Figure **1-3**: Photo of partially excavated mass burial trench within the East Smithfield cemetery. Six adult skeletons are visible *in situ*. From www.museumoflondon.org.uk.

preserved well enough for identification. Of the coins found within the cemetery, ten date from sometime within the interval 1344-51; all the rest predate this interval by a few years or a few decades. Thus all the coin evidence is consistent with the first outbreak of the Black Death in 1349-50. Archaeologists also found pottery in the East Smithfield cemetery, most of which was likely deposited before the cemetery was established (Grainger et al., nd.). Some of the pottery can be dated, with confidence, to the period just before the Black Death, thereby establishing the starting point for the cemetery. For example, excavation yielded sherds from Saintonge jugs; such jugs, which likely contained wine from Gascony, represent the predominant type of imported pottery from France into London between 1270 and 1350 (Grainger et al., nd.). Also found were sherds of Andalucian lusterware from Spain, which first appeared in London during the late thirteenth or early fourteenth century (Grainger et al., nd).

East Smithfield was one of two known emergency burial grounds established in London (Grainger et al., n.d.; Hawkins, 1990). The other burial ground was at West Smithfield, which later became the site of the London Charterhouse. The West Smithfield site is now beneath Charterhouse Square and thus will not be excavated in the foreseeable future. The East Smithfield site in London is currently the only large *excavated* cemetery in all of Europe with unambiguous documentary and archaeological evidence linking it to this first outbreak of the medieval disease.

The only other *possible* Black Death cemetery to have been excavated is that of Heiligen Geist hospital in Lübeck, Germany (Lütgert, 2000). Excavators revealed a mass grave and two potentially contemporary smaller burial pits that they estimate contained a combined total of over 800 individuals. The skeletons are not buried

according to standard medieval Christian burial practices with their heads oriented to the east; rather, the bodies were piled into the mass graves, in stacks up to five to six bodies deep with little soil between the bodies; the majority of the bodies had their heads oriented to the west; some skeletons were even upside-down or sitting up, with their legs bent to better fit them into the graves, indicating that they were buried in haste during some mortality crisis. Archaeological evidence, including radiocarbon dating and ceramic sherds, dates this cemetery to the late thirteenth to mid-fourteenth centuries, and there are two possible explanations for the mass burials: the Great Famine of 1315-22 and the first outbreak of the Black Death. It is unlikely that the Great Famine would have necessitated mass burials, as the increased mortality from the famine was spread out over several years, and survivors would probably have been able to maintain normal burial practices. The Heiligen Geist site is most likely associated with the Black Death; however, unlike East Smithfield, there are no documents linking the Heiligen Geist mass graves specifically to the Black Death.

East Smithfield provides an opportunity to address several important questions about the Black Death that cannot be answered with existing historical documents. I studied the East Smithfield skeletons at the Museum of London to investigate the following questions: 1) How high was the excess mortality associated with the Black Death, and how was this excess mortality distributed by age and sex? Were infants, juveniles, and the elderly at a higher risk than other age groups, or were all ages at equal risk of death? Was either sex at higher risk of dying? Or were males and females at equal risk? 2) Was the Black Death selective with respect to pre-existing health conditions (i.e. frailty)? That is, were people in poor health at a higher risk of dying from

the Black Death, or did the disease kill people indiscriminately, as is often assumed? As described below, some of these questions have been addressed by other researchers. But I believe that the Black Death cemetery at East Smithfield, when compared to an appropriate pre-epidemic sample (described below), and new paleodemographic methods enabled me to address these questions more thoroughly and with a higher degree of confidence than has been possible to date.

Age Patterns of Black Death Mortality: Estimates from Historical Documents

Several chroniclers claimed that the Black Death killed people indiscriminately, irrespective of age or sex. For example, Matteo Villani, a Florentine chronicler, described the Black Death as "a pestilence among men of every condition, age and sex" (quoted in Cohn, 2002). According to the chronicler da Piazza, the mortality from the Black Death was "so heavy that sex and age made no difference, but everyone died alike" (da Piazza, 1994: 41). Some contemporaries, however, believed that the epidemic was selective, and, for example, killed more women than men, and that in subsequent outbreaks of medieval plague (which are believed to be the same disease) men were killed at a higher rate than were women (Cohn, 2002). According to Jean de Venette, a Carmelite friar and chronicler, during the Black Death "the young were more likely to die than the elderly" (1994: 55). Many chroniclers note that the age pattern of subsequent outbreaks was quite different from that of the Black Death; for example, the 1361

outbreak was called the "Pestilence of Children", as young people were more severely affected than were adults (Holmes, 1971); several chroniclers noted that this outbreak differentially affected young men and children (Hatcher, 1977). Such a change in the age-pattern of mortality suggests that survivors of the Black Death acquired immunity to the disease.

Several researchers have attempted to infer the age pattern of Black Death mortality from documentary evidence; unfortunately, it is much more difficult to analyze the sex pattern of mortality from such documents given that most provide information only about males (Russell, 1948; Razi, 1980).

Based on information from the *inquisitions post mortem*, Russell (1948) produced life-table estimates of age-specific mortality among high-status individuals during the Black Death in England. The *inquisitions* were royal inquisitions into the cause of death of tenants-in-chief, people who held land directly from the king. The *inquisitions* provide a nation-wide sample of the highest rank of landholders. When a tenant died, if the heir was under age, the government assumed guardianship of the property until the heir came of age. Such guardianships were quite lucrative, and it was in the interest of all parties to establish accurately the age of the heir; thus the age estimates provided by the *inquisitions* are quite good and are supported by multiple documents (Russell, 1948). Unfortunately, the samples are too small to provide useful life-table estimates, particularly for intervals below the age of twenty; indeed, for some age intervals, the 95 percent confidence intervals for probability of death include negative values (Wood et al., 2002). Despite the limitations of the available data, Russell concluded from the life-table estimates that age did have an effect on Black Death mortality; he argued that older men

were particularly susceptible (although individuals over the age of 60 apparently fared better than those in their late fifties), and children between the ages of ten and fifteen were at a lower risk of dying from the disease than other age groups.

Using manorial court records from the English village of Halesowen, Razi (1980) estimated ages at death among the peasantry during the epidemic. Individuals appear in the manorial court rolls multiple times; the last appearance of each individual is the death duty (*heriot*) payment made by his heirs to the lord of the manor before they could take possession of the deceased's property. Razi assumed each individual appears for the first time in the court rolls at the age of 20 years and counted forward from first appearance to the death-duty payment to estimate ages at death; his age estimates are therefore quite poor, and by definition no information is available for individuals younger than 20. Razi estimated age-specific mortality rates for males during the Black Death, and he found, similar to Russell (1948), that for males between 20 and 59, mortality rates increased with age, and that rates decreased with age after 60. Razi cautioned that his observations are not necessarily valid given the small sample sizes.

Very little about the age-pattern of Black Death mortality can be inferred from the life table estimates derived from historical documents. The basic problem with life tables, when applied to historical demographic or paleodemographic data, is that small samples are further subdivided into several age-intervals; parameters must be estimated for *each* interval, and this is an inefficient use of the data. In an attempt to use these *inquisitions* and Halesowen data more effectively, Wood et al. (2002) fit a Gompertz-Makeham function by maximum likelihood methods; this model has been shown to provide a satisfactory fit to most adult human age-patterns of mortality (Gage, 1988). By

combining all the data (except for individuals under the age of twenty), we needed to estimate just three parameters. We found that age *did* have an effect on risk of death during the epidemic such that elderly people were differentially affected by Black Death mortality. As the Gompertz-Makeham function is a monotonically increasing function of age, it cannot capture the decrease in mortality after age 60 that was found in other studies. Unfortunately, because of the paucity of information on individuals under the age of 20, we were unable to determine whether infants and juveniles were also at a disproportionate risk of death during the Black Death.

In general, the existing historical records do not provide very good age estimates, and there is very little documentary information about women and children at the time of the Black Death. There is also little information about whether pre-existing health conditions had any Effect on an individual's risk of death during the epidemic. Historical records, therefore, do not provide a clear, complete picture of Black Death mortality patterns. Fortunately, skeletal material exists that potentially provides information about mortality patterns that is not available in written records.

Age Patterns of Black Death Mortality: Estimates from a Black Death Cemetery

In principle, skeletal samples can provide information on the age and sex pattern of mortality that can complement surviving historical documents. For example, in a recent study of eighteenth-century documents and the remains of victims of the 1722 outbreak of plague in southern France, Signoli et al. (2002) have shown that the analysis

of skeletal remains is a valid method for investigating mortality dynamics in the past. They estimated the age distribution of the populations in several localities in Provence during the early eighteenth century, and compared those distributions to the age-at-death distributions estimated from two mass burials associated with the 1722 epidemic. They found that the mortality profile of the cemeteries was very similar to the age distribution of the living populations; they conclude, therefore, that victims of plague provide a representative sample of the age structure of the pre-epidemic population.

Several studies of the fourteenth-century Black Death have been done using the East Smithfield cemetery (results summarized in Table **1-1**). Waldron (2001) compared the mortality profile of the East Smithfield cemetery to that of the overlying cemetery of St Mary Graces to determine: 1) whether the age distribution of the mass burial resembles that of a living population more closely than does a normal cemetery, as would be expected if the Black Death killed indiscriminately, and 2) whether the mortality patterns of the medieval epidemic were like that of modern plague; according to some sources, modern bubonic plague differentially affects males and young children (Ell, 1984; Butler, 1989).

Table 1-1: Summary of Previous Studies of East Smithfield

Author	Comparison Sample			Results
	Site	Size	Date	
Conheeney (1999)	Living population	---	---	East Smithfield similar to living population
Margerison and Knüsel (2002)	St. Helen On-the-Walls	636	1100s-1550	East Smithfield similar to living population
Waldron (2001)	St. Mary Graces	236	1350-1538	East Smithfield similar to normal mortality cemetery

The Abbey of St. Mary Graces was established in 1350 and was in use until 1538; the associated cemetery was formed over a long period of time by the gradual attrition of the population (a so-called "attritional" cemetery assemblage). Monks and important lay people were buried within the Abbey's church and chapels, but the cemetery was used for the general population (Rogers and Waldron, 2001). Excavation of St. Mary Graces yielded 133 skeletons within the church and chapels and 301 skeletons within the larger cemetery (Rogers and Waldron, 2001).

Waldron compared the 600 Black Death skeletons excavated from East Smithfield to a sample of 236 skeletons from the St. Mary Graces cemetery. All individuals were assigned to one of six age-at-death intervals: 0 – 4, 5 – 14, 15 – 24, 25 – 34, 35 – 44, and 45+. Many of the adult skeletons in each sample were too poorly preserved to allow for age and/or sex estimation; furthermore, sex differences in skeletal morphology generally do not appear until after puberty, so sex is typically not estimated for juveniles in paleodemographic studies. Of the East Smithfield sample, Waldron was unable to

determine sex for 37.2 percent of the entire sample and age for 29.7 percent of the adult skeletons; for the St. Mary Graces sample, he was unable to determine sex for 27.5 percent of the entire sample and age for 34.3 percent of the adult skeletons.

Because "the presence of so many skeletons of unknown age or sex seriously distorts the age/sex distributions", Waldron assigned sex and/or age to juvenile and poorly preserved adult skeletons by making two assumptions. First, Waldron assumed that the sex ratio of children was the same as that of the adults in each assemblage, and thereby assigned sex to juveniles. Such an assumption is not necessarily realistic. Innumerable studies have shown that there are sex-based differences in age-specific morbidity and mortality rates, such that sex ratios within populations change with age. In humans, males are at a higher risk of mortality from puberty onward and often at prepubertal ages as well (Wells, 2000; Owens, 2002). Additionally, differences in the sex ratios of adults and juvenile within a population may arise because of secular trends in sex ratios (Allan et al., 1997). Therefore, the sex ratios of adults in the East Smithfield and St. Mary Graces may very well have differed from those of the children.

Waldron also assumed that the adults of unknown age were distributed evenly among each of the adult age categories; such an assumption requires that there was no differential preservation of the skeletons by age in either cemetery; according to Waldron, "there seems to be no reason to suppose that the skeletons of any one sex or any particular age group are less likely to preserve well than another" (2001: 107). This assumption also requires that the survival function was flat, and this does not make sense biologically. Stojanowski et. al. (2002) studied skeletal preservation at an 8000 year-old site in Florida and found that there was no significant relationship between bone

preservation and age or sex. However, others have found that in poorly preserved assemblages, age at death does have a significant effect on preservation. Gordon and Buikstra (1981) found in Late Woodland burials (*circa* A.D. 830 – 1200) that soil pH and preservation of juvenile bones are significantly correlated and that preservation declines more rapidly with decreasing pH in juveniles than in adult skeletons. Walker et al. (1988) studied an early nineteenth-century cemetery with poor preservation as a result of sandy soils that allow water to saturate the burials; burial records show that most of the people interred in the cemetery were infants and elderly individuals; however, the skeletal assemblage comprised mostly young adults. Given the poor preservation of parts of the East Smithfield cemetery, as described below, it is perhaps not safe to assume that skeletons of all age groups were preserved equally well.

Waldron (2001) compared the mortality profiles of the East Smithfield and St. Mary Graces cemeteries to the age distribution of a living population of medieval London predicted from a model life table (**Figure 1-4**). Contrary to expectations, there seem to be relatively few infants in the normal mortality cemetery; infant mortality is usually quite high and then decreases with increasing age, so normal mortality cemeteries are expected to have more infants than older children. The apparently low number of infants in St. Mary Graces might be the result of poor preservation of infants in that cemetery or an artifact of the of very broad age intervals Waldron used in the study. Compared to St. Mary Graces, there was a significant deficit of males between the ages of 15-24 and females over the age of 45, and a significant excess of females between the ages of 25-34 in East Smithfield. Waldron also found that both East Smithfield and St. Mary Graces have more post-infant juveniles than might be expected from a model life table.

However, he found that there were no systematic differences between the mortality profiles of the two cemeteries, and that there was no systematic over-representation of young children or males in the Black Death cemetery as *might* be expected from a modern bubonic plague death assemblage (Grainger et al., n.d).

According to Waldron, in both skeletal assemblages, there is an apparent overestimation of the number of individuals in the younger age groups and an underestimation of individuals in the oldest age group (over 45 years) compared to a model life table; this might be the result of age mimicry of the known age-at-death reference sample (see discussion of age estimation below). Waldron concluded that the skeletal evidence does not support the idea that any particular age or sex was at an elevated risk of dying during the Black Death, and that the East Smithfield cemetery probably does not provide a representative sample of the population of London.

Conheeney (1999) used Waldron's age and sex estimates and compared the East Smithfield age-at-death distribution to living age distribution of a modern relatively poor society without access to modern medicine (**Figure 1-5**). He found that the two distributions appear similar, the only notable deviation being a deficit of individuals between the ages of 15-25 and an excess of individuals older than 25 in the East Smithfield cemetery. Conheeney suggests that there are fewer 15-25 year-olds than expected because this age group would be the most mobile and have the fewest ties to the community and would therefore have had the ability to flee the city and escape the epidemic.

Margerison and Knüsel (2002) compared the age-at-death distribution of the East Smithfield burials to that of St Helen-on-the-Walls in York, a parish cemetery in use

from the late twelfth century to 1550. Although the St Helens-on-the-Walls cemetery was in use during the Black Death, and therefore might contain some victims of the epidemic, Margerison and Knüsel consider it a normal attritional cemetery and an appropriate comparison for the epidemic cemetery. Both distributions were compared to a model life table that the researchers assumed was representative of the age distribution of a living poor medieval urban population (**Figure 1-6**). Margerison and Knüsel used Waldron's (2000) age and sex estimates for the East Smithfield cemetery, but they eliminated the adult individuals who could not be assigned an age and/or sex. They estimated sex and age for the St. Helen-on-the-Walls cemetery using the same methods used by Waldron. The St Helen-on-the-Walls profile was similar to that of the model life table, the only major difference being a high number of juveniles in the former. The East Smithfield mortality profile was significantly different from that of the non-epidemic cemetery. There were fewer infants and more juveniles in East Smithfield than expected from the life table, but in general the epidemic age-at-death distribution resembled the age distribution of a living population as might be expected of a catastrophic cemetery; that is, East Smithfield, like a living medieval population, contained many young people and few older individuals.

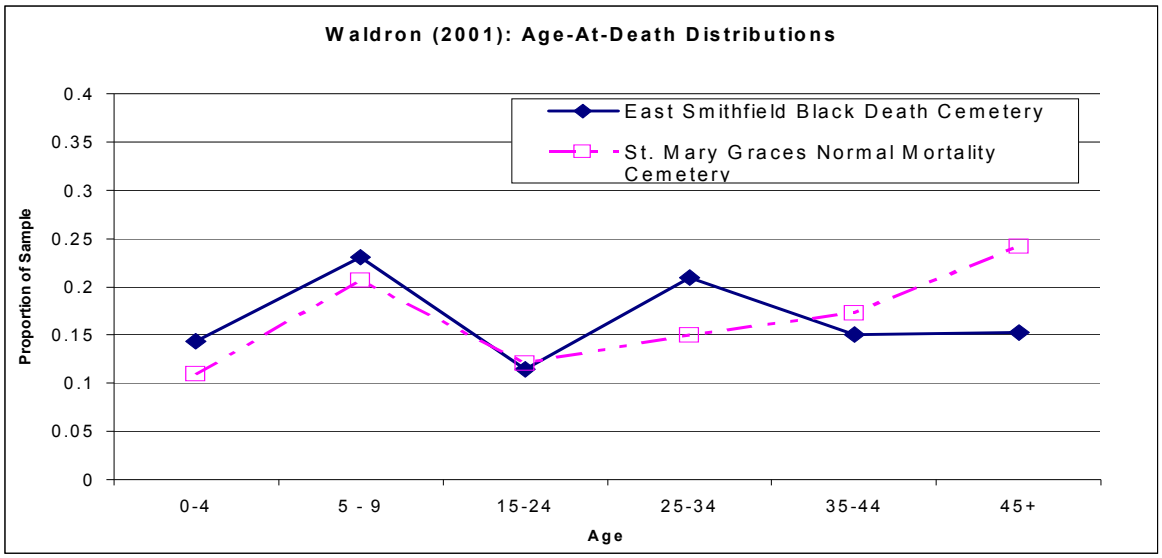

Figure 1-4: Comparison of age-at-death distributions from the St. Mary Graces normal mortality cemetery and the East Smithfield Black Death cemetery. See text for a description of the age intervals used in the study. Adapted from Waldron (2001).

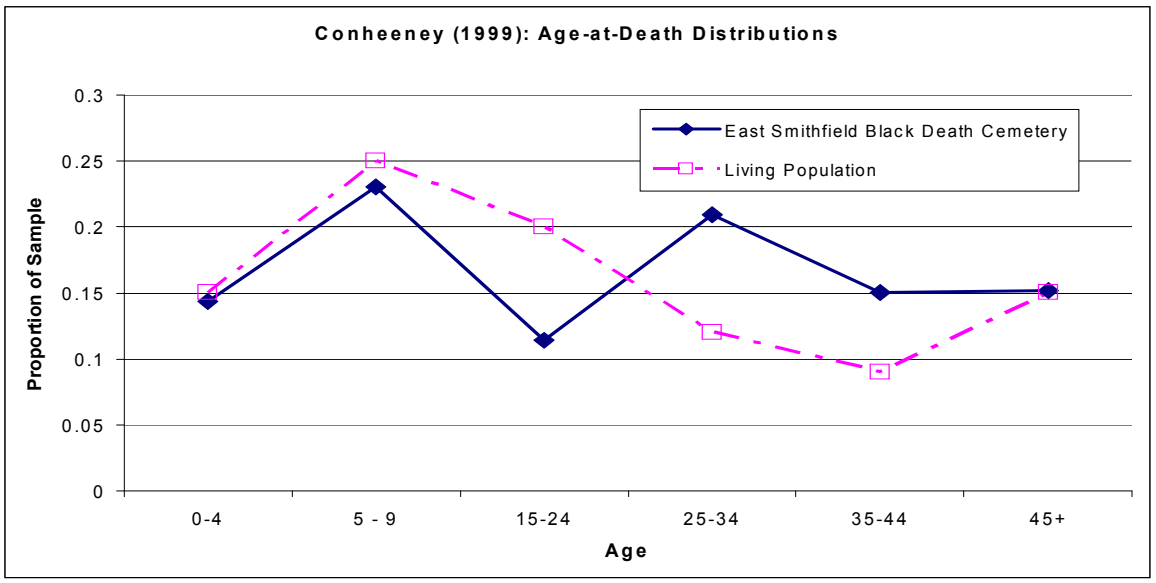

Figure 1-5: Comparison of the age distribution of a living population to the age-at-death distribution from the East Smithfield Black Death cemetery. See text for a description of the age intervals used in the study. Adapted from Conheeney (1999).

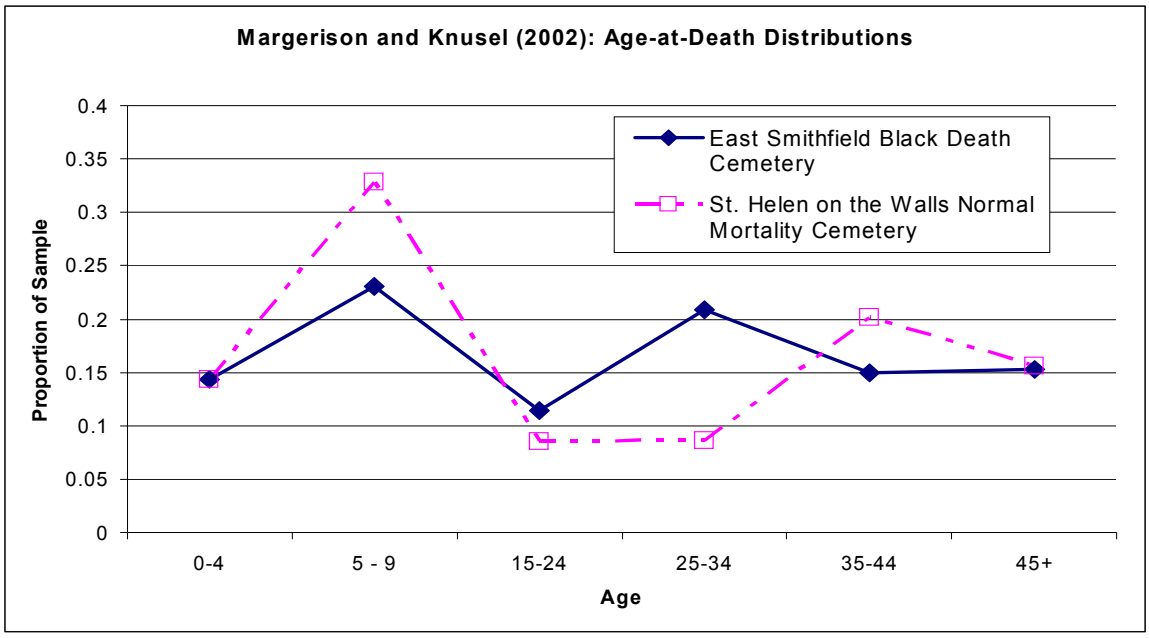

Figure 1-6: Comparison of age-at-death distributions from the St. Helen-on-the-Walls normal mortality cemetery and the East Smithfield Black Death cemetery. See text for a description of the age intervals used in the study. Adapted from Margerison and Knüsel (2002).

Problems with Previous Studies

There are several problems with these studies of Black Death skeletons, most importantly their choice of comparison samples. To make sound inferences about the epidemiology of the Black Death, one needs to know what the affected population would have looked like if the epidemic had never happened. Since this is impossible, I had to

find a control cemetery that approximates conditions in the affected population just *before* the epidemic. No post-Black Death burials will meet this need, for the Black Death itself initiated profound demographic changes throughout Europe (Bowsky, 1971; Gottfried, 1983; Herlihy, 1997; Cohn, 2002); moreover, any selective mortality associated with the epidemic would immediately render the surviving population unrepresentative of pre-Black Death conditions. Paine (2000) found, using population modeling, that episodes of catastrophic mortality may have lasting affects on age-at-death distributions; even at the end of a 100-year population projection, an episode of catastrophic mortality continued to have noticeable effects on the simulated age-at-death distribution.

For an investigation of the mortality patterns of the Black Death, the proper control cemetery would be made up of individuals from an urban community as similar as possible to lower-class London *circa* 1345, that is, just before the epidemic. Ideally the only difference between the East Smithfield cemetery and the control cemetery would be that the Black Death affected the former but not the latter. The further one strays from this ideal, the less confident one can be about any inferences drawn about the epidemiology of the Black Death. Previous studies of East Smithfield have all used comparison samples that include large numbers of individuals who died well after the Black Death. In addition, the burials at the Abbey of St. Mary Graces are unlikely to be representative of the larger city because they probably include a disproportionate number of upper-class individuals and monks. Although the perfect control sample does not exist, I have tried to identify a comparison sample that should more closely approximate the ideal than did those in previous studies (see below).

In some of the previous analyses (Waldron,2001; Margerison and Knüsel, 2002), model life tables were used as standards of comparison for gauging the age pattern of excess mortality. I have decided not to adopt this approach because of the inherent uncertainty about which model life table to use (Wood et al., 2002). Use of an appropriate empirical comparison sample, such as the pre-Black Death Danish sample I use in this project, is a better approach (see below).

The other problems with previous epidemiological studies of the East Smithfield burials are mainly statistical in nature. For example, the previous studies all used life-table analysis, which is now considered an inefficient way to deal with paleodemographic age-at-death data (Buikstra, 1997; Konigsberg et al., 1997; Konigsberg and Frankenberg, 1992; Hoppa and Vaupel, 2002; Müller et al., 2002; Wood et al., 2002). Life tables require the estimation of one parameter (the central mortality rate) for every age interval used, and there is no way to do this reliably without huge samples and knowledge of the original population at risk (Wood et al., 2002). According to Holman et al. (2002), most human age-at-death distributions can be described using five or fewer parameters, and parsimonious parametric models provide a useful alternative to life tables in paleodemographic studies.

Further, the fixed age intervals in the life table imply that the age estimates of each skeleton in each age interval are known with the same degree of error, and this is simply not true. For example, age estimates of young children that are based on the relatively invariant process of tooth formation and eruption are much more precise and reliable than age estimates of adults that are based on highly variable changes in the pelvis. The life table approach is also problematic because it assumes the population

from which the skeletal collection is derived was stationary: closed to migration, with an intrinsic rate of growth equal to zero, and age-specific schedules of fertility and mortality that were unchanging over time and which induce an equilibrium age distribution; such an assumption is not necessarily realistic. It is now widely accepted (Konigsberg, 1992; Buikstra, 1997; Konigsberg et al., 1997; Hoppa and Vaupel, 2002; Müller et al., 2002; Wood et al., 2002) that some form of parametric or semi-parametric hazards analysis is the most powerful way to derive information from the small samples typical of paleodemography – and that is the approach I adopted here.

Another problem with previous studies of East Smithfield is the use of traditional osteological aging methods, which are known to be badly biased for adult skeletons (Bocquet-Appel and Masset, 1982; Konigsberg, 1992; Konigsberg et al., 1997; Hoppa and Vaupel, 2002). I chose to use the so-called "Rostock protocol" for age estimation, which was designed to correct this bias (Hoppa and Vaupel, 2002; Müller et al., 2002); even with biased reference samples, the Rostock protocol produces good age estimates (see details about the Rostock protocol in Chapter 2). A further advantage of the Rostock protocol is that it provides both a point estimate of age and the complete error distribution of the estimate, rather than the crude and arbitrary interval estimates provided by traditional methods. The Rostock protocol, which has been validated against several simulated and real known-age data sets represents a significant improvement over older aging techniques (Boldsen et al., 2002; Holman et al., 2002; Hoppa and Vaupel, 2002; Müller et al., 2002).

Selectivity of the Black Death

A second important question about the epidemiology of the Black Death is whether mortality associated with the disease was selective with respect to pre-existing health conditions – or what Vaupel et al. (1979) call *frailty*, which is an individual's age-adjusted relative risk of death. In this study, frailty means an individual's relative risk of death *before* the Black Death (i.e. during normal, non-epidemic times). Did the Black Death kill people indiscriminately, regardless of frailty, or did Black Death mortality behave like normal, non-epidemic mortality in that those individuals with highest frailty were at highest risk of death?

The Black Death was clearly new to Europe in the mid-fourteenth century, at least as far as the contemporary population was concerned, and it was exceptionally virulent. It has been tempting to believe, therefore, that all people, regardless of health status, were at equal risk of dying from the epidemic, all being equally immunologically naïve. If this were true, it would mean that Black Death cemeteries could be used to obtain unbiased estimates of the prevalence of health-related osteological lesions in the once-living populations from which they were drawn (Grainger et al., n.d.; Waldron, 1992; Waldron, 2001; Margerison and Knüsel, 2002). If the Black Death was not selective, and instead killed people indiscriminately, then everyone in the original population would have been at equal risk of dying regardless of health status; Black Death cemeteries, such as the East Smithfield cemetery, should therefore have the same proportion of individuals with high and low frailty, and thus the same proportion of individuals with and without lesions associated with frailty, as did the original living population.

Normal mortality, however, is almost always selective, preferentially affecting those individuals with the highest frailty (the weakest members of the population), such as very young children, the elderly, and severely malnourished and diseased individuals. Individuals with low frailty do occasionally make their way into normal mortality cemeteries as the result of homicide, suicide, accidental death, and sheer bad luck; but such individuals usually represent a minority of deaths (Wood et al., 1992). Normal mortality cemeteries should therefore look nothing like the original living population; rather, they should be comprised mostly of very young and old people and individuals with skeletal lesions associated with high frailty.

Thus, were catastrophic mortality not selective, catastrophic burials would provide insights into the original population that are relatively uncomplicated by the selectivity biases associated with normal mortality burials (Wood et al., 1992). That is, catastrophic cemeteries would allow for much more straightforward investigations of past populations than is possible with normal cemeteries. But is any catastrophe ever truly non-selective? It appears that numerous individuals were exposed to the Black Death pathogen but did not come down with the disease, and a fair fraction of those who were infected managed to survive. Was this differential susceptibility real or a matter of chance?

Recently it has been suggested that the Black Death was selective with respect to the CCR5-△32 deletion, which occurs in western Europeans (Fukushima, 1994). The CCR5-△32 deletion is a thirty-two basepair deletion in the human chemokine receptor CCR5 gene, and it has an average frequency of ten percent in modern European populations. Kremeyer et al. (2005) hypothesize that the deletion arose through a single

mutation event, and the frequency of the allele increased to its current level through the strong selective pressure of the Black Death. The allele is believed to confer resistance to HIV, and might also have conferred resistance to the causative agent of the Black Death, although this matter is still being investigated and is currently debated (Elvin et al., 2004; Sabeti et al., 2005). But whatever the outcome of this debate, it is at least as important to determine if the Black Death was selective in regard to overall health or frailty, which is more likely to reflect differences in social and economic environment than in genetics.

Waldron (2001) attempted to address the question of selectivity by comparing the frequencies of skeletal lesions in the East Smithfield cemetery to those in the overlying cemetery of St Mary Graces. Waldron examined skeletons for cribra orbitalia, dental disease (e.g. dental caries and abscesses), osteoarthritis and other degenerative diseases, broken bones, and lesions characteristic of tuberculosis, rickets, and diffuse idiopathic skeletal hyperostosis (DISH, a condition characterized by excessive bone growth and associated with obesity and advanced age). Waldron found that the two cemeteries differed with respect to the frequencies of degenerative lesions and dental disease, and most of the lesions were at a lower frequency in East Smithfield compared to the normal mortality cemetery. However, none of the observed differences was significant. Finding that the two cemeteries had generally similar frequencies of skeletal lesions, he concluded that the East Smithfield cemetery is not representative of the once-living population, as one might expect if the Black Death killed people indiscriminately. I believe, however, that this conclusion is premature given that his comparison sample was made up entirely of individuals who died *after* the epidemic.

The Study Populations and Samples

To investigate selectivity and the age and sex pattern of excess mortality, I compared a sample from the East Smithfield cemetery to an appropriate pre-Black Death sample from medieval Denmark. Little is known about the catchment area of the East Smithfield cemetery, so finding a perfectly matched sample is impossible. A previous analysis of the East Smithfield skeletons revealed that the males might have been, on average, slightly shorter than the medieval average, so, according to Grainger et al. (n.d.) the individuals in the East Smithfield cemetery appear to be of lower social status. Several studies have found that stature can be used as an indicator of social status. Steckel (1995:1911) argues that "income is a potent determinant of stature that operates through diet, disease, and work intensity." In nineteenth-century England, upper class teenage boys at the Sandhurst military academy were ten to fifteen centimeters taller than their contemporaries from the slums of London (Steckel, 1995). Komlos and Kriwy (2002) found striking differences in adult stature among social classes in Germany. For example, in former West Germany between 1920 and 1980, upper-class men were 3.9 centimeters taller and middle-class men were 1.7 centimeters taller than lower-class men. Hatch and Willey (1974) analyzed stature of high and low status skeletons from the Late Mississippian "Dallas" society of Tennessee and Georgia and found that larger overall stature was associated with high status males, but that there was no significant relationship between stature and status for females.

The ideal comparison sample – a large, lower socioeconomic status parish cemetery from London dating from just before the Black Death – unfortunately does not exist. Nor indeed do such samples exist anywhere in England (Barney Sloane, pers. comm.). I therefore identified a comparable sample from the Anthropological Database, Odense University (ADBOU), in Denmark, a skeletal collection well known for its size and the quality of its chronological control (Boldsen, 1998).

In general, Denmark and southern England were quite similar socially, economically, and demographically during the Middle Ages up to the time of the Black Death – more similar, for example, than were southern and northern England (Benedictow, 1993; Sawyer and Sawyer, 1993; Roesdahl, 1999). According to Benedictow (1993), England and southern Scandinavia had much in common culturally and climatically. Sufficient historical records that would allow for an exhaustive comparison of the demographic patterns of medieval Denmark and southern England do not exist, but the evidence that does exist reveals similarities between the two regions. For example, estimates for mean life expectancy at birth, based on historical documents and skeletal data, are the same for England and Scandinavia circa 1300 (22-28 years) (Benedictow, 1993).

During the middle ages, English and Danish societies were based primarily on lords and peasants (Poulsen, 1997); in both countries, the peasantry comprised the majority of agricultural labor, though there were also large numbers of freeholders and semi-free tenants. In Denmark and southern England there existed wide river valleys and level ground which facilitated crop cultivation; peasants in those regions cultivated extensive fields of cereal crops. In contrast, northern England is an upland environment

that even today is ill suited for intensive cultivation; the peasants in northern England were therefore much more pastoral (Dyer, 2002). In Denmark and much of southern England, the open field system dominated (Poulsen, 1997; Widgren, 1997); in the open-field system, villages were surrounded by several open fields that were divided into strips, and each peasant was allocated a certain number of strips to farm; to maintain and improve the productivity of arable land, each year the peasants would plant crops in one or more fields and let other fields remain fallow (Benedictow, 1993; Sawyer and Sawyer, 1993). Given the similarities between Denmark and southern England, and the ability to date medieval Danish burials (see details in the *Materials and Methods* discussion in Chapter 2), Danish cemeteries can provide good comparison samples for a study of East Smithfield.

My Danish comparison sample comprises nearly 300 skeletons from the urban parish cemeteries of Albani Church and St Mikkel Church, both of which form part of the ADBOU collection. Individual graves in each cemetery can be reliably dated on the basis of arm position (see description of arm position in Chapter 2), so I was able to select a sample of individuals who mostly died before the Black Death struck Denmark in 1349-1350. I also selected the Albani and St Mikkel cemeteries because they represent lower socioeconomic urban communities (Kristensen, 1987; Christensen, 1988). Thus, my comparison sample is as close an approximation to the ideal control group for East Smithfield as is currently possible. I do not argue, of course, that the Albani and St. Mikkel skeletons are *the* ideal comparison samples, merely that they are a much more appropriate comparison than those used in previous studies of East Smithfield.

For the analytical framework for my epidemiological studies, I used a multistate

hazards model of morbidity and mortality developed for paleodemography by Usher (2000). Usher's model is estimated using the maximum likelihood estimation program *mle* written by Holman (2000). The model allowed me to estimate the excess mortality associated with the Black Death and determine if there was differential mortality with respect to sex or osteological indicators of frailty.

The paleodemographic analyses described here represent an improvement over previous studies in several respects. Extensive research on the dating of burial arm positions has been done in Denmark (Kieffer-Olsen, 1993; Jantzen et al., 1994), enabling me to select a predominantly pre-Black Death comparison sample. Unlike previous studies, I neither used a sample that includes only people who died after the Black Death nor one that likely includes many individuals who died during the epidemic. Thus, the patterns observed in the Danish comparison sample are unlikely to reflect either Black Death selectivity or demographic changes induced by the Black Death itself. Because the once-living samples from which the East Smithfield and Danish comparison cemetery samples derived were broadly similar (urban, lower socioeconomic status, etc.), any differences I observed with respect to excess mortality, age and sex patterns of mortality, and frailty can be attributed to the Black Death with some confidence.

I also used powerful new methods of age estimation that provide unbiased point estimates of age as well as the complete error distribution of the estimates (Hoppa and Vaupel, 2002; Müller et al., 2002). The age-at-death distributions that I estimated for both cemeteries are therefore more reliable and informative than those estimated in previous studies. Usher's multistate model, combined with Holman's *mle* estimation program, provides efficient and unbiased estimates of excess mortality and selectivity.

These new methods have enabled me to provide new and important details about the epidemiology of the Black Death. Not only are these results exciting in terms of furthering our understanding of the Black Death in particular, but they also are informative about selective mortality and emerging infectious diseases in general. These results also help demonstrate, in general, the utility of new paleodemographic methods in the study of infectious diseases that have played an important role in shaping human history.

Chapter Summary

- The Black Death was one of the most important emerging diseases in human history and it had profound demographic, economic, and social consequences.
- There is still much that is unknown about the epidemiology of the Black Death.
- This project investigates the age and sex patterns and the selectivity of the Black Death in an effort to contribute to our understanding of emerging diseases in general.
- This project is an improvement over previous studies of the East Smithfield Black Death cemetery in terms of the comparison sample and the application of new paleodemographic methods.

Chapter 2

MATERIALS AND METHODS

Samples

Medieval Danish Cemeteries: St. Mikkel and Albani Church

As argued in Chapter 1, in order to make valid inferences about the age and sex patterns of Black Death excess mortality and about selectivity of the disease with respect to frailty, I need to compare the East Smithfield skeletal sample to one that pre-dates the epidemic. I have selected such a sample from the Anthropological Database at Odense University, Denmark. My Danish pre-Black Death comparison sample is a combined sample of 291 skeletons from the urban parish cemeteries of Albani Church and St Mikkel Church, both of which form part of the current ADBOU collection.

The Church of St. Mikkel in Viborg is assumed to date before 1129, and it was first mentioned in mid-twelfth century written sources; there is evidence that burials on the site predate the construction of the church. St. Mikkel was located just south of the walled city of Viborg, similar to other early churches in Denmark. The city of Viborg converted to Lutheran Protestantism in 1529, and by April of that year, all churches associated with the city were demolished, including St. Mikkel. According to Boldsen (personal communication), St. Mikkel was a suburban parish with a congregation comprising mostly lower socioeconomic people.

The St. Albani church in Odense was founded in the early eleventh century, and is first mentioned in written records in 1086 (Boldsen and Mollerup, 2006). St. Albani was used until 1529 and the church was demolished in 1542. According to Boldsen and Mollerup (2006), St. Albani served an urban parish. The St. Albani cemetery covered approximately 4500 square meters. Excavation of the site revealed 398 skeletons, and most were excavated from north and south of the church; some skeletons were excavated from the interior of the church itself.

Though the Albani and St Mikkel cemeteries were both in use from the 1100s until the early 1500s (Kristensen, 1987; Christensen, 1988), individual graves can be dated with reasonable accuracy on the basis of arm positions (Kieffer-Olsen, 1993; Jantzen et al., 1994). Examination of medieval and early modern Danish cemeteries has revealed a series of rapid changes in the predominant arm position of interred individuals**.** Jacobzon (1982) described the following arm positions: A) arms fully extended alongside the body, B) hands clasped over pelvis, C) hands clasped over stomach, and D) arms crossed over chest (Figure **2-1**).

Kieffer-Olsen (1993) analyzed eight cemeteries in Denmark and found that in medieval Danish cemeteries, arm position provides a better dating method than does radiocarbon dating, as the margin of error associated with arm position is much narrower (Figure **2-2)**. The Hocker position, used infrequently in the early eleventh century, is the traditional pre-Christian Viking burial form (body on side with legs drawn up). According to Kieffer-Olsen (1993), arm position A was the predominant position used between AD 1000 and 1250, and the transition to arm position B was quite rapid. The

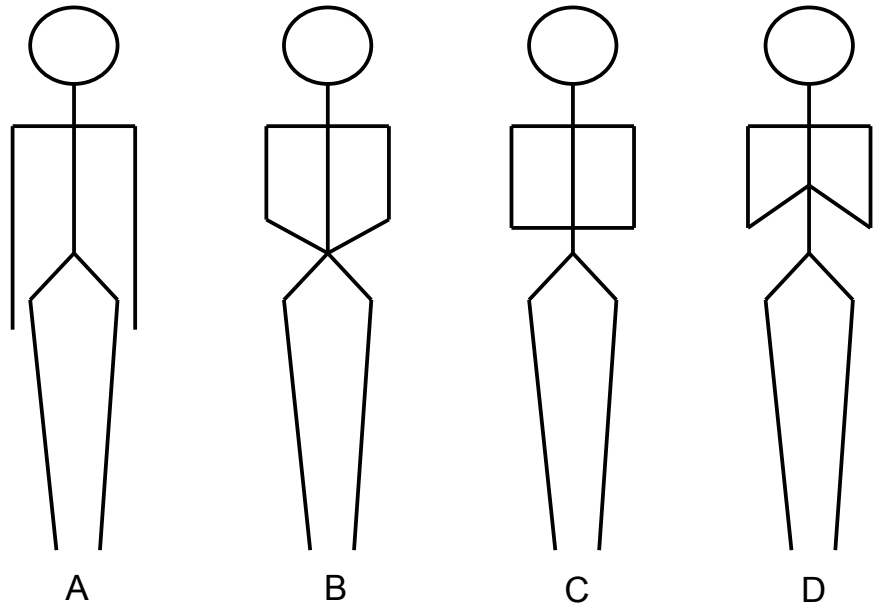

Figure 2-1: Arm positions in medieval Danish cemeteries

somewhat slower transition from arm position B to C occurred in the mid-fourteenth century; arm position B was used until about 1375, although rarely after 1350. The transition from position C to D occurred in the mid-1400s. After the Protestant Reformation in the early sixteenth century, several different arm positions were used at any given time. To increase the likelihood that my comparison sample predates the Black Death, I sampled only skeletons with arm positions A and B from cemeteries that were not used after the Reformation. Based on the distribution of arm positions in Denmark (Kieffer-Olsen, 1993), approximately 97 percent of randomly-selected burials with arm positions A or B are likely to have died before the Black Death hit Denmark in

1349. The error associated with the dates shown in Figure 2-2 may slightly lower this probability.

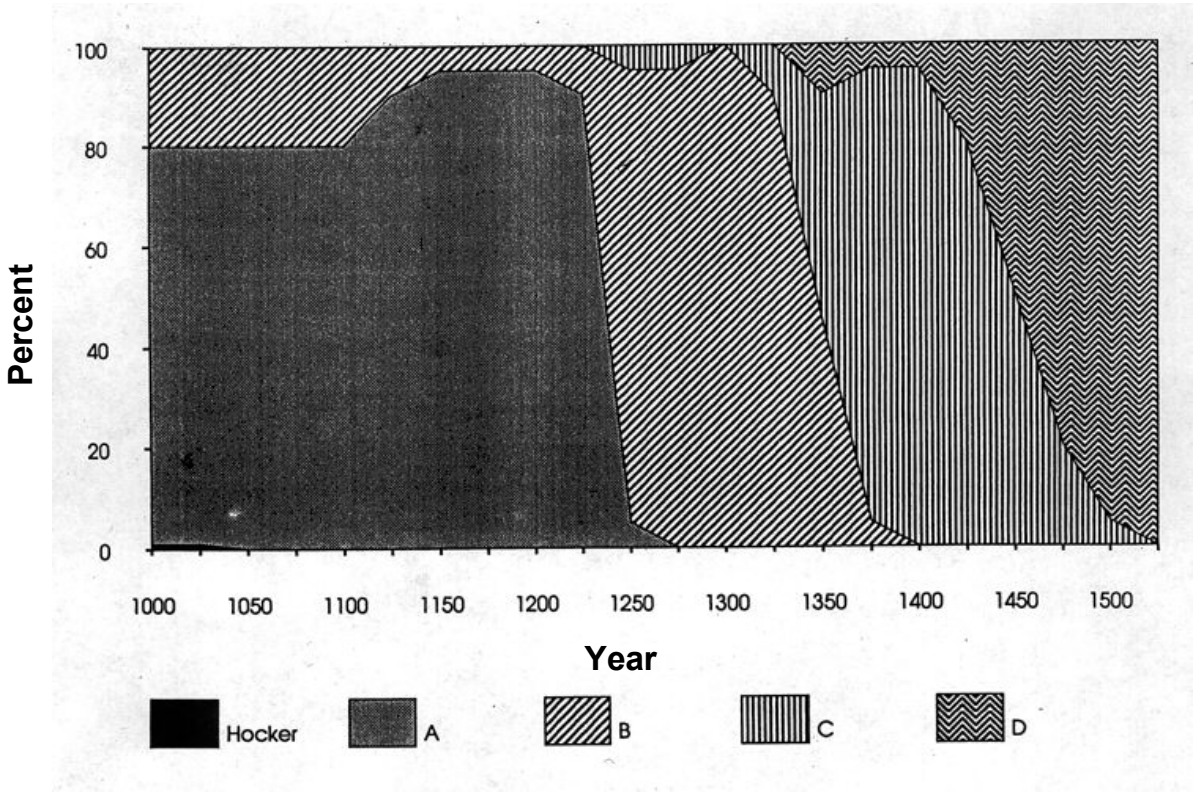

Figure 2-2: The chronology of changes in arm position in medieval Danish cemeteries.

According to Jantzen et al. (1994), the transitions between arm positions occurred about fifty years later than shown in Figure 2-2, and the fraction of individuals with arm position B who died during the Black Death may be larger than Kieffer-Olsen's (1993) earlier work suggested. However, Jantzen et al. (1994) do not provide a revised version of Figure 2-2, and with the available data, I cannot calculate the probability that a randomly-selected individual buried with arm position B died before the Black Death. It is possible that some of the individuals that I included in my Danish sample died during or after the Black Death, and the sample therefore might not perfectly represent normal mortality conditions.

There is some controversy regarding whether the sequence of arm positions described by Kieffer-Olsen (1993) can be generalized to all of Denmark and other parts of Scandinavia; however, with respect to the areas of medieval Denmark from which my samples come, the consensus among Danish archaeologists is that the arm positions are consistent and provide reliable dates (Boldsen, personal communication; Arneborg et al., 1999). Of the skeletons with arms positions A and B, I only selected those that were preserved well enough for me to score them for age, sex, and the presence of skeletal lesions (n = 291).

For this study, *I assumed that all of the individuals in my Danish sample died before the Black Death,* and thus that the Danish sample includes no victims of the epidemic; this might not be true, as work done by Kieffer-Olsen (1993) and Jantzen et al. (1994) indicates that, although not common, arm position B was still used during and for a short time after the Black Death in Denmark. I cannot determine the likelihood that any particular individual in my Danish sample died before the Black Death, and it is

possible that the Danish sample includes victims of the Black Death. Such individuals are likely to be a small minority of the normal mortality sample; nonetheless, the potential inclusion of Black Death victims in the Danish sample would decrease apparent differences between the Danish sample and East Smithfield. In Chapter 4, I discuss how my results and the interpretation of those results might be affected if the assumption that all individuals in the Danish sample died before the Black Death is wrong.

East Smithfield Cemetery

The East Smithfield cemetery in London (Figure **2-3)** was established in late 1348 or early 1349 expressly to accommodate the overwhelming number of people killed during the Black Death; in 1348, there were likely over one hundred burial grounds in the city of London and the surrounding suburbs, yet the East Smithfield cemetery was necessary to supplement or perhaps temporarily replace existing cemeteries. The purpose, location, and size of the East Smithfield cemetery are carefully noted in contemporary records. Further evidence of the site's date is provided by a group of coins

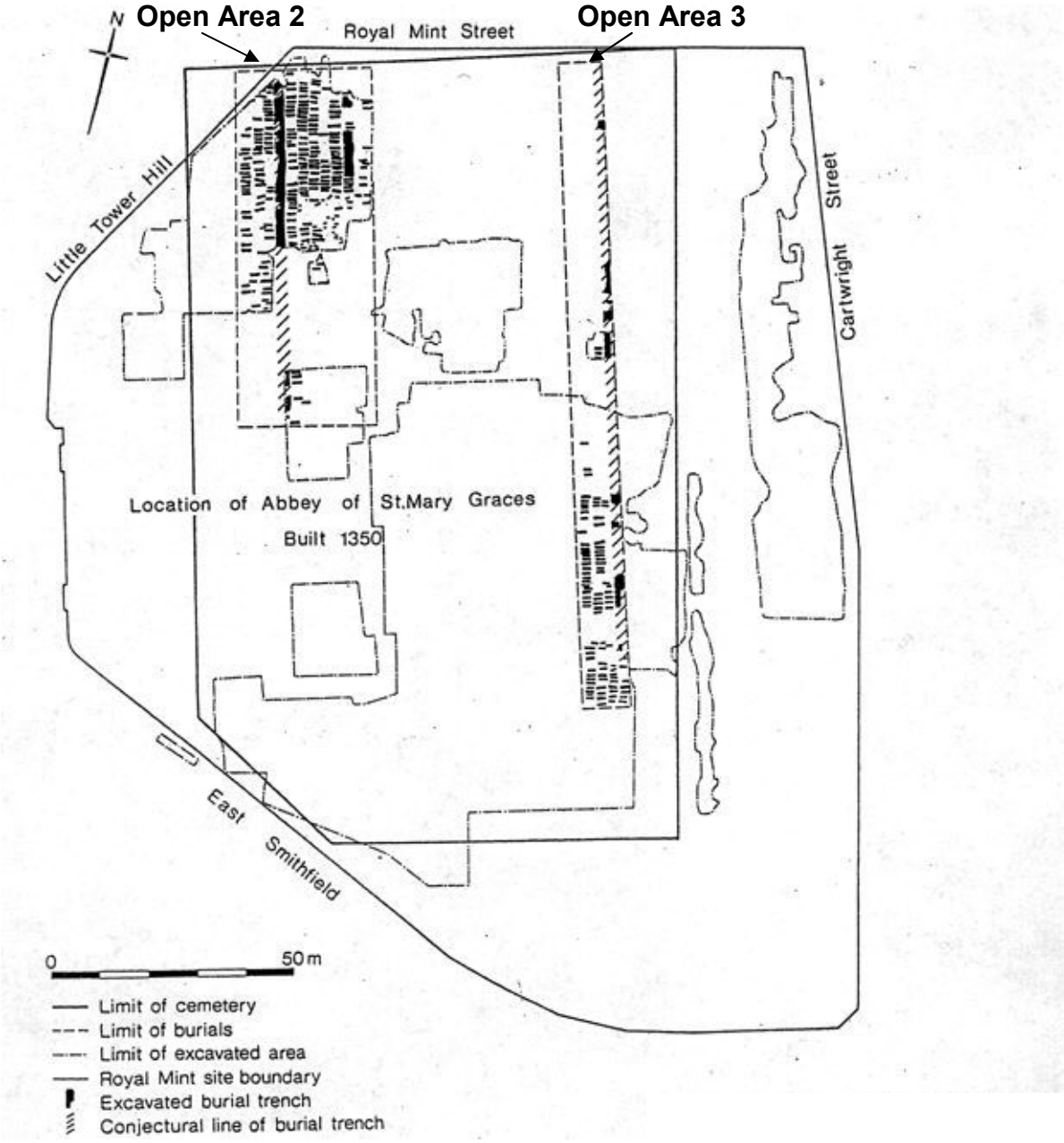

Figure 2-3: **The East Smithfield Black Death cemetery.** The Royal Mint site is the larger area enclosed by a solid line and is bounded by modern roads; the East Smithfield Black Death cemetery is circumscribed by a solid line within the larger Royal Mint site. The excavated Black Death burials were concentrated in Open Areas 2 and 3. (Adapted from Hawkins, 1990).

found with one of the skeletons interred in the cemetery, the latest of which can be securely dated to sometime in the interval 1344-51. The cemetery was situated on the eastern edge of the city of London in an area that was previously undeveloped agricultural land (Grainger et al., n.d.).

The East Smithfield cemetery was excavated as part of a larger archaeological investigation of the Royal Mint site in eastern London. The Museum of London Department of Greater London Archaeology began full excavation of the Royal Mint site in 1986 and the project lasted about two years. Excavations revealed the Black Death cemetery, the Cistercian Abbey of St. Mary Graces (in use between 1350 and 1538), and a Royal naval victualling yard (in use between 1560 and 1785). The Abbey of St. Mary Graces was built a short time after the Black Death, and there is a small but distinct cemetery associated with the abbey. However, there is no evidence that the larger Black Death cemetery was used for burials after the fourteenth-century epidemic (Hawkins, 1990). Approximately 80 percent of the larger Royal Mint site was excavated or found to be archaeologically sterile (Hawkins, 1990). The site was not completely excavated, and only about a quarter of the estimated total number of Black Death burials was ultimately recovered. It is possible that the available skeletons are not representative of the entire original East Smithfield cemetery.

The Black Death burials are concentrated in two distinct parts of the East Smithfield cemetery: Open Area 2 in the north-west corner of the site and Open Area 3 in the eastern part of the site (**Figure 2-3**). Approximately fifty to sixty percent of Open Area 2 was fully excavated, revealing two mass burial trenches, a mass burial pit, and 262 individual graves arranged in eleven parallel rows. The forty to fifty percent of Open

Area 2 that remains unexcavated is preserved *in situ* under the courtyard of the Royal Mint Court. No evidence of boundary markers of Open Area 2 was found, nor were grave markers or graveyard structures found; the edges of the area were defined archaeologically by the absence of burials and by documentary sources. All burial features in Open Area 2 are stratigraphically contemporaneous, and there is no evidence of any inter-cutting of the graves or trenches or of re-deposition of bodies. In both burial trenches, corpses were carefully placed in dense stacks up to five bodies deep, and infants and small children were often used to fill in the spaces between adults. Approximately fifty percent of the larger trench survived and was excavated, yielding 243 individuals. The smaller burial trench was completely excavated and contained fifty individuals. Thirty-eight individuals, representing both sexes and all age ranges, were buried in coffins within the trenches; coffins were identified by the presence of nails and/or traces of wood. The mass burial pit held eight individuals, all carefully placed in the grave; some individuals were partially disarticulated prior to burial, indicating some delay in burial as putrefaction had likely set in before the bodies were interred. Of the individual burials, 128 contained evidence of coffins. All but two excavated individuals were extended and supine with their heads oriented toward the west; the two exceptions are from the mass burial trenches in Open Area 2: one individual was prone and one was in a crouched position. According to Grainger et al. (n.d.), the individual graves would probably be indistinguishable from other, non-epidemic burials from the same period.

Open Area 3 was unfortunately disturbed and severely truncated by the construction of plant rooms for the Royal Mint during the nineteenth century. The surviving features of Open Area 3 are individual graves and one mass burial trench

arranged in four rows. The long burial trench contained 102 skeletons, which might represent a relatively small fraction of the original number of individuals buried in the trench, the remainder being destroyed or covered by the construction of the Royal Mint. Within the long burial trench, there was clear evidence of thirteen coffins. Excavation revealed 97 individual graves, fifty of which held coffins. Many of the skeletons revealed by excavation in Open Area 3 were contaminated by chemicals used in the minting process, resulting in extremely poor preservation. Some skeletons were so highly degraded by chemicals that archaeologists were unable to remove them from the site; of the 97 surviving individual graves, seven were empty because chemicals had completely dissolved the skeletons, leaving only a stain in the soil.

According to Hawkins (1990), the presence of both mass burial trenches and individual graves in Open Areas 2 and 3 might be evidence that there were fluctuations in the numbers of people dying during the epidemic in London. Presumably, during times of relatively fewer deaths, there was more time to construct coffins and dig individual graves; when the death toll increased, the survivors were overwhelmed, and the mass burial trenches and pits, being more expedient than individual graves, were necessary. However, archaeologists were unable to determine the relative chronology of the digging or filling of the graves and burial trenches "as there was no stratigraphic relationship between the graves and trenches" (Hawkins, 1990: 640).

During excavation of East Smithfield, 762 skeletons were recorded in the ground; however, many skeletons were too fragile to remove from the site (Waldron 1992). In total, approximately 600 skeletons were recovered from East Smithfield, a fraction of the total number individuals originally buried at the site. According to the archaeologists

who excavated East Smithfield, up to 2400 people were buried in the cemetery during the epidemic in London; this estimate is based on the dimensions of Open Areas 2 and 3 and the density of surviving burials within those areas. Of the 600 excavated skeletons, I only included in my sample those that were adequately preserved to allow scoring of age, sex, and presence of skeletal lesions (n = 490).

For this study, *I assumed that all of the individuals in my East Smithfield sample were victims of the Black Death*. This is not an unreasonable assumption, given that Black Death mortality swamped normal mortality during the epidemic. However, it is possible that some individuals in the East Smithfield cemetery died from causes other than the Black Death. In Chapter 4, I discuss how the possible inclusion of non-Black Death victims in my East Smithfield sample could affect my results and interpretations.

Preservation

According to Walker et al. (1988:183), "inaccuracies introduced through biases in preservation are a major source of error in paleodemographic reconstructions". Archaeological samples are not representative of the populations from which they are derived, partly because of differences in preservation. For example, the bones of infants are small, soft, and fragile, and therefore do not preserve as well as those of adults. Elderly individuals with severe osteoporosis might not preserve as well as younger adults. Variation in soil pH and mineral and water content within a cemetery can cause differential preservation of skeletons. Additionally, differences in preservation between geographic locations complicate comparisons of cemeteries from different populations. Because

variation in preservation can cause biases in skeletal samples, it is important to score skeletons for degree of preservation and take any differences into account when making any inferences about the once-living population from the skeletal sample.

I scored all the skeletons in my samples for degree of preservation using categories based on those described by Gordon and Buikstra (1981). I divided the skeleton into four regions: head, torso (vertebral column, ribs, sternum, scapula, and clavicle), arms (humerus, radius, ulna, carpals, metacarpals, phalanges), and legs (innominate, femur, tibia, fibula, tarsals, metatarsals, phalanges). I focused primarily on those bones within each region that were important in terms of estimating age or sex and determining the presence or absence of the skeletal lesions used in my analyses. I scored each region separately as good, fair, poor, or missing. I assigned a score of "missing" if the region was not present. A score of "good" was given to a region if the relevant bones were whole (or fragmented but could be easily and completely reassembled) and relatively undamaged (i.e. most or all of the surface of the bone is visible, unbroken, and free of abrasion or other damage). I was able to score the bones in regions with "good" preservation for all the desired characteristics (i.e. all of the age features, sex features, and skeletal lesions). A score of "fair" was given to a region if the relevant bones had superficial damage such that fewer than half the features I wanted to score were obscured. I assigned a score of "poor" to regions in which the relevant bones were damaged or missing to the extent that more than half of the features I wanted to score were obscured or missing. I did not include in my samples any skeleton that was missing all of the desired information.

Age Estimation

Adult Age-at-Death Estimation

As mentioned above, one of the potential problems with previous studies of East Smithfield is that they were based on traditional methods of paleodemographic age estimation, which have been shown to be biased for adults. With traditional methods, one estimates an individual skeleton's age at death based on some osteological age indicator stage, and population-level estimates are determined by combining the estimates for all individuals in the sample. Bocquet-Appel and Masset (1982) showed that paleodemographic age estimates using traditional methods are inherently biased towards the age composition of the modern known-age-at-death reference sample. Each stage of an osteological indicator of age corresponds to a mean age in the reference sample, and these mean ages depend primarily upon the age structure of the reference sample. For example, in a reference sample composed mostly of older individuals, by chance some old individuals may have pubic symphyses with Todd (1921) stage 1 morphology (the most youthful pubic symphysis morphology); therefore the mean age for Todd stage 1 determined from this reference sample will be older than that from a reference sample which includes many young individuals.

This bias towards the reference sample might not present a problem if the reference sample was completely representative of the original population (or in the unlikely event that the age-at-death distributions of the target and reference samples were identical), but unfortunately, most reference samples have unusual age-at-death

distributions quite unlike the age distribution of the overall population. For example, McKern and Stewart (1957) studied age-related changes using a reference sample comprising young men killed in the Korean War, so age estimates based on this reference sample might underestimate true ages in the target sample.

By regressing an osteological trait (Y) on age (X), one can obtain unbiased estimates of osteological age indicator given age from a reference sample; Bocquet-Appel and Masset (1982) showed that the linear regressions of osteological trait on age from two reference samples with very different mean ages at death were very similar. However, in paleodemography, the goal is to determine the probability that a skeleton is a certain age (X) given the appearance of an osteological trait (Y). Simply regressing X on Y does not produce the same line as regressing Y on X (i.e. they are inverse functions). When regressing age on osteological trait, two samples with different age compositions produce two different regression lines; the result is age-mimicry of the reference sample, as estimation of age is highly sensitive to the age distribution of the reference sample. Bocquet-Appel and Masset (1982) viewed age-mimicry as an insurmountable problem, and they concluded that, "save unforseen developments - unpredictable in the near future at least," paleodemographic age estimation and any demographic research or inferences based on such estimates are fruitless (1982:329). To indicate how serious they regarded this problem, their 1982 paper was entitled "Farewell to Paleodemography."

The Rostock Protocol

Recently, researchers have developed statistical methods to solve the problem of age mimicry and provide more accurate and informative estimates of age-at-death

(Konigsberg and Frankenberg, 1992 and 1994; Boldsen et al., 2002; Holman et al., 2002; Hoppa and Vaupel, 2002; Müller et al., 2002). The recently-developed Rostock protocol, which I used to estimate age-at-death, uses maximum likelihood estimation and Bayes' Theorem to produce unbiased estimates (the protocol is thus named because it was developed at a series of meetings held at the Max Planck Institute for Demographic Research in Rostock, Germany). Additionally, the Rostock protocol provides not only a point estimate of individual age-at-death, but also the error distribution of that estimate; thus, the Rostock protocol provides more accurate and informative age-at-death estimates than are possible with traditional methods.

With traditional methods, one estimates individual ages-at-death for a cemetery sample, and then combines all of those individual estimates to create an age-at-death distribution for the entire sample. However, with the Rostock protocol, one first estimates the cemetery sample age-at-death distribution (Müller et al., 2002), and *then* estimates individual age-at-death using Bayesian inversion (Konigsberg and Frankenberg, 1992 and 1994).

The first step in the Rostock protocol is to use nonparametric methods to estimate from a known-age-at-death reference sample the conditional probability that a bone is classified into a specific age-indicator stage given age at death (the "weight function"). Müller et al. (2002) assumed weight functions are invariant; they do not depend on the populations under consideration as aging of the human skeleton is assumed to be invariant across time and space, so the weight functions for the target and reference samples are the same. That is, the weight functions are unbiased estimates of skeletal age indicators given age, and are thus not subject to age mimicry. This assumption of

invariance may or may not be biologically correct, but without it, paleodemographic age estimation is impossible.[2]

The next step of the Rostock protocol is to select a parametric distribution to represent the age-at-death distribution of the target sample; one useful distribution for this purpose is the Gompertz-Makeham distribution. The Gompertz-Makeham model describes adult mortality, and was developed to fit the general human pattern of relatively low mortality during the young adult ages and an increasing risk of death with age. The Gompertz-Makeham model has two components, one independent of age, the other increasing with age, and the hazard function is:

$$\mu(a) = \alpha_1 + \alpha_2 e^{\beta a} \qquad \textbf{2.1}$$

where a is age, α_1 is the constant, age-independent risk of death, and $\alpha_2 e^{\beta a}$ is the exponentially increasing senescent risk of death (the Gompertz function). The α_2 parameter describes the absolute level of mortality associated with older age, and the β parameter describes the rate at which this risk increases with age. The Gompertz-Makeham model has been shown to fit well to human mortality data for the ages between 30 and 85 years (Wood et al., 2002).

Having selected a parametric distribution, one then combines that distribution with the weight functions and the observed frequencies of osteological age categories

---

[2] Some traits are more variant than others. Paleodemographers should try to work with the less variable ones.

from the target sample and uses maximum likelihood estimation to find parameters for the age-at-death distribution that maximize the likelihood of the observed age-indicator frequencies in the target sample. The likelihood function takes the following form (Eq. **2.2):**

$$L = \prod_{i=1}^{n} \Pr(c_i) = \prod_{i=1}^{n} \int_0^{\infty} \Pr^*(c_i|a) \Pr(a) \, da \qquad \textbf{2.2}$$

where a is age, c_i is some vector of age-indicator stages, Pr* is the weight function estimated from a reference sample, and Pr(a) is the age-at-death distribution from the target sample that one wants to estimate (Wood et al., 2002). At this point, one has estimated the age-at-death distribution for the target sample. This is essentially the reverse of the traditional method; with traditional methods, one estimates individual ages at death from skeletal age-indicators, and then combines all the individual age estimates to construct the target sample age-at-death distribution. But with the Rostock protocol, the target age-at-death distribution is estimated *before* one estimates individual ages at death.

Müller et al. (2002) conducted a simulation study to verify that: 1) the target sample age-at-death distribution estimated using the Rostock protocol is not biased toward the age distribution of the living sample, and 2) the Rostock protocol is capable of recovering accurate estimates of the parameters of the target sample age-at-death distribution. They simulated a target population distribution using the Gompertz model and generated 300 ages at death; thus they knew the true age-at-death distribution of the

target sample. They then attempted to recover the parameters of this distribution using the Rostock protocol. Weight functions for the simulation study were obtained from the Suchey-Brooks reference sample (Brooks and Suchey, 1990). They ran simulations to obtain observations of skeletal age indicator frequencies in the target sample and then used the observed frequencies to estimate the parameters of the Gompertz function using maximum likelihood estimation. They compared the estimated target age-at-death distribution to both the known reference sample and true target sample distributions. The estimated target distribution was very similar to the true target distribution, and both differed from that of the reference sample; specifically, the reference sample distribution was bi-modal with a major mode at age 25 and a second mode at age 50, whereas both the estimated and true target age-at-death distributions had a single mode at around age 78. Müller et al. (2002) demonstrated that the Rostock protocol does indeed produce accurate estimates of the parameters of the age-at-death distribution that are not subject to age mimicry (i.e. the estimated target distribution was unlike that of the reference sample distribution).

Once the age-at-death distribution of the target sample is determined, individual ages can be determined by using Bayes' Theorem:

$$\Pr\left(a|c_i\right) = \frac{\Pr^*\left(c_i|a\right)\Pr\left(a\right)}{\int_0^{\omega}\left[\Pr^*\left(c_i|a\right)\Pr\left(a\right)\right]\mathrm{d}a} \qquad \textbf{2.3}$$

where a is age, c_i is some vector of age-indicator stages, Pr* is the weight function estimated from a reference sample, Pr(a) is the age-at-death distribution from the target sample, and ω is the maximum attainable age in the target population.

Using the Rostock protocol, Müller et al. (2002) estimated individual ages at death for the simulated target sample described above and compared the results to those obtained using traditional methods. They found that that the estimates obtained using the traditional method were very different from the true ages in the simulated target sample and were biased toward the age distribution of the reference sample. However, encouragingly, the estimated ages using the Rostock protocol were very close to the true ages in the simulated target sample and were not biased.

To summarize, the steps of the Rostock protocol are as follows: 1) estimate the probabilities of certain skeletal age-indicators given age from a reference sample, 2) observe the frequencies of skeletal indicators of age in the target sample, 3) calculate the maximum likelihood estimates for the parameters of a parametric age-at-death distribution that maximize the likelihood of the observed age-indicator frequencies in the target sample, and 4) use Bayes' Theorem to determine individual ages at death for the target sample.

For this project, I used a modified version of the Rostock protocol called transition analysis that was developed by Boldsen et al. (1998, 2002). In transition analysis, the age-at-death distribution is assumed uniform or is based on documentary information independent of the target sample, whereas in the Rostock protocol, the entire age-at-death distribution of the target sample is estimated from the observed age-indicator frequencies in the target sample. Additionally, the transition analysis approach assumes the

age indicators are independent of one another once they have been conditioned on age. Boldsen et al. (2002) argue that transition analysis is more applicable to the small sample sizes typical to paleodemography than is the original Rostock protocol. For this project, I used the *ADBOU Age Estimation* software developed by Boldsen et al. (2002) to determine individual ages-at-death. The *ADBOU* program uses an age-at-death distribution from 17th-century Danish rural parish records to provide estimates for the age-at-death distribution in the target sample (Boldsen et al., 2002).

The first step of the Rostock protocol, estimating the probabilities of certain age indicators given age from a known age-at-death reference sample (the weight functions), was completed by George Milner and Jesper Boldsen prior to the current study (see Boldsen et al., 2002). In this analysis, I used the weight functions estimated by Milner and Boldsen using the Terry reference collection. The Terry collection, which is curated by the National Museum of Natural History of the Smithsonian Institution, comprises 1728 skeletons of known age, sex, ethnicity, cause of death, and pathological conditions. The individuals in the Terry collection were either donated or unclaimed bodies primarily from the St. Louis, Missouri, area *circa* 1920s – 1960s; age-at-death in the Terry Collection ranges from 16 to 102 years. The Terry collection is a valuable resource for anthropologists, and it has been used, among other things, to determine age-related changes in the adult skeleton. Boldsen et al. (2002) used weight functions derived from the Terry Collection to obtain age estimates using transition analysis. Boldsen et al. found that the age estimates obtained using transition analysis were unbiased.

I scored all adults in my samples for the skeletal indicators of age described by Boldsen et al. (2002); these nineteen skeletal indicators include cranial suture closure and

features of the pubic symphysis and iliac auricular surface (pictures of typical age-related changes in these regions of the skeleton are provided in Appendix A). I scored the age-indicators using the protocol described in Boldsen et al. (2002; see Appendix A).

Juvenile Age-at-Death Estimation

To estimate age of juvenile skeletons, I examined epiphyseal closure and dental development and eruption; these processes are relatively invariant and orderly, so estimation of juvenile age is not as problematic as is estimating adult age. I scored epiphyseal closure according to the standards provided by Buikstra and Ubelaker (1992). According to White (2000), dental development is more closely associated with chronological age than is development of most other parts of the skeleton. Dental ages are considered very reliable because typically dental development is not as susceptible to environmental conditions such as nutrition and disease as are other skeletal growth and developmental processes. I assigned dental ages based on the standards produced by Moorrees et al. (1963a,1963b) and Thoma and Goldman (1960).

Age Pattern of Mortality: Siler Model

To determine the complete age pattern of Black Death mortality across all ages, adult and juvenile, I estimated the parameters of the Siler mortality model (Eq. **2.4**)

within each cemetery sample using the individual age point estimates for the adults and the midpoint of the estimated age range for juveniles:

$$\mu(a) = \alpha_1 e^{-\beta_1 a} + \alpha_2 + \alpha_3 e^{\beta_3 a} \qquad \textbf{2.4}$$

The Siler model has three components. The first component, $\alpha_1 e^{-\beta_1 a}$, describes the juvenile risk that declines exponentially with age, where α_1 is the risk of death at birth caused by immaturity and β_1 is the rate at which this risk decreases with age. The second component of the Siler model, α_2, is the constant age-independent or "baseline" risk that everyone within the population faces. The third component of the model, $\alpha_3 e^{\beta_3 a}$, is the exponentially increasing senescent risk, where α_3 is the risk of death associated with senescence at the moment of birth and β_3 is the rate at which this risk increases with age. The second and third components of the Siler model are identical to the two components of the Gompertz-Makeham model. The three components of the model are independent. The Siler model is a parsimonious parametric model of mortality; it fits a wide variety of human mortality patterns as well as or better than most other models (Gage, 1991; Wood et al., 2001). The Siler model does have some limitations in terms of fitting the fine details of human mortality. For example, the juvenile component is difficult to estimate. Juvenile mortality decreases very rapidly with age, and it is hard to capture the details of this decline, particularly with the very small juvenile sample sizes common in paleodemography (Wood et al., 2002). I

estimated the parameters of the Siler model using maximum likelihood estimation with the program *mle* (Holman, 2002).

Sex Estimation

I only estimated sex in adult individuals, that is the skeletons in which the long bone epiphyses and sphenooccipital sychondrosis were fused. I did not attempt to determine the sex of subadult individuals, as sexually dimorphic features do not develop until puberty, and thus there are no reliable indicators of sex in the skeletal remains of juvenile individuals (Powell, 1988; Buikstra and Ubelaker, 1994). I scored features of the cranium and pelvis using the sex determination standards provided by Phenice (1969) and Buikstra and Ubelaker (1994). With respect to the pelvis, I scored the ventral arc, subpubic concavity, ischiopubic ramus ridge, and the greater sciatic notch; on the skull, I scored the nuchal crest (external occipital protruberance), mastoid process, supra-orbital margin, supra-orbital ridge (glabella), mental eminence, and gonial angle (diagrams of these features are provided in Appendix A). According to Buikstra and Ubelaker (1994), sex differences in the pelvis are more reliable than those in the skull; therefore, for cases in which individuals had ambiguous skull morphology or skull morphology consistent with one sex and pelvic morphology consistent with the opposite sex, I determined sex based on the pelvic morphology. Fortunately, in both the East Smithfield and Danish cemeteries, sex determination for adults was relatively easy, with few individuals having ambiguous features.

Usher Multistate Model of Morbidity and Mortality

Analytical Problems in Paleodemography

To analyze the pattern of excess mortality associated with the Black Death and the selectivity of the disease, I used Usher's (2000) multistate model of morbidity and mortality. Usher's full, four-state model has the potential to solve several fundamental problems inherent in paleodemographic analysis, including hidden heterogeneity in frailty, selective mortality, and demographic non-stationarity (Wood et al., 1992; Usher, 2000; Wright and Yoder, 2003). These fundamental problems complicate our study of demographic patterns and disease in past populations, in particular preventing us from making inferences about health and mortality *directly* from such data as mean age at death or prevalence of skeletal lesions in skeletal samples.

Traditionally, paleodemographers have viewed skeletal samples as essentially representative samples of the once-living population and have therefore made rather straightforward conclusions about health and demography in past populations based on skeletal data. For example, Angel (1971) estimated the age at death distribution for the ancient Aegean population of Lerna using skeletal data, and based on a modal age at death of thirty years, he concluded that the population faced a high risk of mortality. Angel also analyzed the frequency of porotic hyperostosis, cranial lesions he believed are indicative of thalessemia, a form of anemia that is protective against malaria. Angel

found a high frequency of porotic hyperostosis in the skeletal samples, and assuming that the frequency of the lesion in the skeletal sample directly reflected the prevalence of malaria in the once living population, he concluded that malaria was highly prevalent in the population. Angel assumed the severity of porotic hyperostosis reflects an individual's genotype for the thalassemia locus, such that individuals homozygous for the thalassemia allele had severe porotic hyperostosis whereas heterozygotes had only slight lesions; based on this assumption, he estimated the frequency of the thalassemia allele within the population based on the moderate and severe lesion frequencies in the skeletal sample. He further concluded that primitive farming methods would have created conditions that promoted malaria, and that the invention of farming was therefore associated with an increase in disease, a protein-poor diet, and a decline in health in past populations.

Many researchers (e.g. Cohen and Armelagos, 1984; Goodman et al., 1988; Cohen, 1989) have followed in Angel's footsteps, in terms of making direct interpretations of the patterns observed in skeletal samples and focusing on the health consequences of the transition from hunting and gathering to agricultural subsistence strategies. According to these researchers, the transition from hunting and gathering to agriculture resulted in a general deterioration of average health; because of this transition, diet generally declined in quality, infectious diseases increased in importance because of a greater degree of sedentism, higher population density, and larger population size, and economic inequality increased, so that a larger segment of the population was at very high risk of death. Such conclusions are based primarily on a general decrease in mean age at death and an increase in the frequency of certain skeletal lesions in skeletal

samples from agricultural populations compared to those from hunting and gathering populations (Wood et al., 1994; Wood et al., 2000).

However, other researchers have suggested that a decrease in mean age at death is actually more reflective of increases in fertility than mortality (see discussion of demographic non-stationarity below; Coale, 1957; Sattenspiel and Harpending, 1983), and thus a decrease in mean age at death might indicate improvements in diet and health. Furthermore, skeletal lesions might sometimes indicate a relatively healthy individual (see discussion of the "osteological paradox" below), and thus increases in the frequency of certain lesions might also indicate general improvements in health.

More recently, several researchers have questioned the assumption that skeletal samples are representative of once-living populations, and have argued that heterogeneous frailty, selective mortality, and demographic nonstationarity would actually tend to make such samples *un*representative of living populations (e.g. Ortner 1991; Wood et al., 1992; Milner et al., 2000; Wright and Yoder, 2003).

Heterogenenous Frailty

One important reason why cemetery samples are unlikely to be representative of living populations is that every individual in a population is *not* at the same risk of dying. On the contrary, all populations are heterogeneous for frailty (the age-standardized relative risk of death); that is, all individuals in a population vary in terms of their relative risk of death compared to others in their birth cohort (Vaupel et al., 1979). Heterogeneous frailty exists because of differences in susceptibility to disease and death

that may have genetic, environmental, or socioeconomic causes. In paleodemography, what we observe are aggregate patterns, such as the age pattern of mortality estimated from skeletal samples. This aggregate pattern of mortality is determined by individual variation in frailty and the relationship between frailty and risk of death, and Vaupel and Yashin (1985) argue that heterogeneity in terms of frailty can have significant effects on aggregate patterns.

Vaupel and Yashin (1985) and Wood et al. (1992) demonstrate that infinitely many combinations of subpopulation age-specific mortality rates can give rise to the observed aggregate mortality rate. For example, the aggregate mortality pattern for human populations is characterized as a "bathtub curve"; this aggregate pattern suggests that mortality is high immediately following birth, decreases during infancy and is eventually constant for a while, and then increases with age. But this does not necessarily mean that any particular individual's risk decreases, increases, or remains constant at different times during his/her life. Vaupel and Yashin show that the aggregate pattern could be produced by two subgroups, each of which has its own specific hazard rate; the hazard rate for one subgroup is high and constant, while the hazard rate for the other subgroup increases gradually with age. The initial decline in mortality observed in the aggregate pattern reflects the fact that over time, the individuals in the subgroup with the high hazard are selected out of the population. The hazard for the aggregate can only decline as low as the hazard of the second subgroup, and as the hazard of that second group increases with age, the aggregate hazard increases as well. The same aggregate bathtub curve could also be produced by three, four, ten, or one hundred different subgroups, each with its own age-specific hazards. Wood et al. (1992) write that, in fact,

each individual in the population could have his/her own unique hazard, the combination of which produces the observed aggregate pattern.

Wood et al. (1992) describe "hidden" heterogeneity, wherein the variation in frailty cannot be measured. If not controlled for statistically, heterogeneous frailty can make it difficult to infer an individual's level of health or risk of dying from aggregate measures such as age-specific mortality rates (Vaupel and Yashin, 1985; Wood et al., 1992).

Selective Mortality

Selective mortality acts upon heterogeneous frailty (Vaupel and Yashin, 1985; Wood et al., 1992). Selective mortality refers to the fact that individuals who die at a given age are unlikely to be representative of the entire *living* population at risk of death at that age; individuals with the highest frailty are most likely to die and thus be selected out of the population. Once individuals are selected out of the population, the surviving population is unlike the original population.

Because of selective mortality, one cannot go directly from the prevalence of diagnostic lesions in a skeletal sample to the prevalence of the associated disease(s) in the once-living population. If the skeletal lesions are caused by conditions that increased an individual's risk of death, by using prevalence of lesions as a measure of prevalence of the associated condition in the population, we would tend to overestimate the number of cases in the living population As an analogy, to estimate the prevalence of HIV in the United States, one would not sample individuals seeking care from an AIDS clinic, as

one would grossly overestimate the prevalence of HIV in the overall population from such a biased sample (Milner, lecture, Method and Theory in Archaeology, 10/99). Even skeletons of the victims of violence cannot be assumed to be a representative sample of all people who were once alive in a particular community; Milner et al. (1991) found that many adults in a skeletal sample from late prehistoric Illinois were killed by enemies and showed signs of debilitating conditions that would have hampered their ability to protect themselves or flee from danger. Estimates from this sample of the prevalence of these debilitating conditions in the once-living population would likely be too high.

Demographic Non-stationarity

Until recently, it was often assumed in paleodemography that the population under consideration was stationary: closed to migration and having constant age-specific fertility and mortality rates (i.e. the rates do not change from one generation to the next), a stable age distribution, and a growth rate of zero. Under these assumptions, given enough time, a population will attain a stationary age distribution, such that the absolute number of individuals within each age group is constant over time – which also means that the *proportion* of people at each age is constant. In addition, the age distribution is an equilibrium, so if it is perturbed, for example by a catastrophic but transitory epidemic, it will eventually return to the stable form.

This assumption of stationarity is useful for paleodemography because it allows one to estimate mortality rates for the once-living population from skeletal sample age-at-death distributions. However, if a population is not stationary – if, for example, it has a

non-zero growth rate – the age-at-death distribution can be profoundly distorted. If demographic non-stationarity is not recognized, biased estimates of the age distribution and mortality rates for a once-living population are likely to be obtained (Sattenspiel and Harpending, 1983; Johansson and Horowitz, 1986).

Researchers have shown, perhaps counter-intuitively, that the age-at-death distributions of non-stationary populations are more strongly affected by changes in fertility rates than by changes in mortality rates (Coale, 1957). For example, if a population experiences growth because of increased fertility rates (i.e. non-stationarity), there will be an increase in the proportion of young individuals in the population, and the average age at death in the population will decrease. If population growth is not accounted for, one might conclude that the population experienced increased mortality.

One way to deal with demographic non-stationarity is to assume the population under consideration was stable (but not necessarily stationary) and estimate the parameters of an age-at-death distribution that allows for population growth. A stable population is one that is closed to migration, and has constant age-specific fertility and mortality rates and a stable age distribution (i.e. all the assumptions of the stationary population *except* for zero population growth) (Lotka, 1907 and 1922).

Researchers need not abandon all paleodemographic studies because of the complications caused by heterogeneity, selective mortality and demographic nonstationarity, but we must acknowledge that such problems exist and use appropriate statistical methods, such as the Usher model, to deal with them.

The Full Usher Model

In the full Usher model (Figure **2-4**), individuals are in one of four "states" at any one time: State 1 includes those individuals with no skeletal lesion, State 2 includes those with active lesions, State 3 includes those with healed lesions, and the fourth State is death. Of course, all individuals in cemetery samples are dead, but there are three possibilities (States 1 – 3) for the living state each individual was in at the time of death.

As with all multi-state models, in the Usher model, individuals can only be in one state at a time, and all individuals are in one of the states at a given time. Movement from one state to another is governed by transition rates. The model is governed by the Markovian assumptions that the states are independent of one another and movement from one state to another is only dependent upon the state an individual is in at a given time (i.e. movement is not affected by the states an individual has previously passed through). For states with two or more exits, the risks of leaving in each direction are independent of one another. Individuals enter State 1 (no lesions) through birth, and the birth cohort is assigned a frailty distribution $g_1(z)$. Transitions between states *i* and *j* (e.g. from State 1 to State 2 or from State 2 to State 3), occur at age-specific hazards rates $\mu_{ij}(a|z)$, where *a* is age in years and *z* is individual frailty; these hazard rates are therefore dependent upon an individual's age and level of frailty.

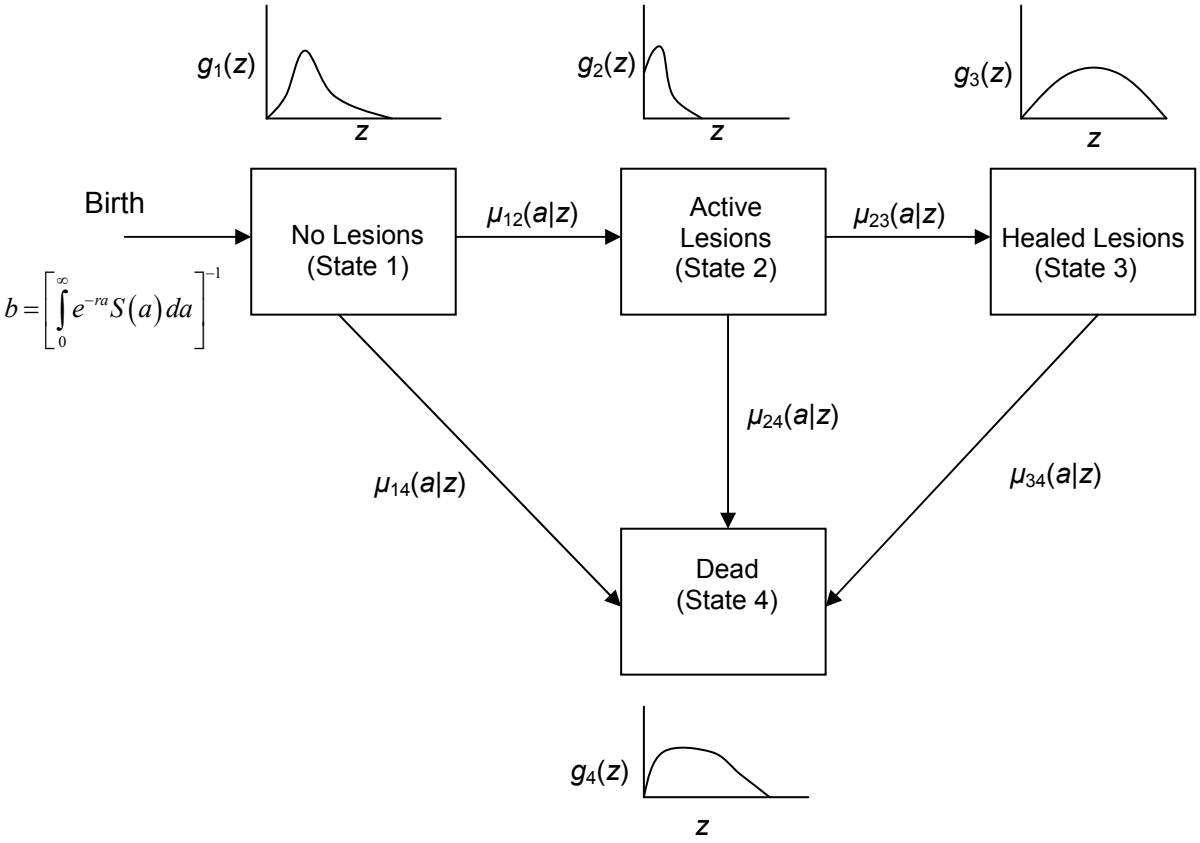

Figure 2-4: Full multi-state model of morbidity and mortality (from Usher, 2000). In this model, a is age in years, b is the crude birth rate of the stable population, $S(a)$ is the aggregate survival function, r is the population growth rate, z is individual frailty, $g_i(z)$ is the frailty distribution in state i, and $\mu_{ij}(a|z)$ is the hazard of making a transition between states i and j at age a given frailty z..

The frailty distributions $g_i(z)$ associated with the different living states differ because all the transitions within the model are selective with respect to frailty. For example, the mean frailty for the State 2 frailty distribution is higher than that of State 1

because individuals with higher frailty are more likely to make the transition from having no lesions to having lesions. A simpler, but also potentially informative approach is to assume that, instead of frailty varying continuously within the population, there are two subpopulations, one of which includes those individuals at high risk of both developing lesions and of dying, the other individuals at a relatively low risk of developing lesions and dying. Both approaches, using a continuous distribution of frailty or using a model with just two distinct risk groups, provide a way to examine the effects of hidden heterogeneity in frailty.

The Usher model also enables one to investigate selective mortality by allowing for differences in the risk of death associated with each of the living states. The model allows one to determine the force of mortality associated with different living states with respect to a given lesion; if the force of mortality is stronger for any one state, the model can help resolve one aspect of the "osteological paradox" (Wood et al., 1992) – that is whether an individual with a certain lesion is more or less healthy than an individual without that lesion.

The traditional view in paleodemography is that a skeletal lesion indicates an individual who was not as healthy as a similarly aged individual without such a lesion, and skeletal lesions would therefore indicate a high risk of death. However, some researchers (e.g. Ortner, 1991; Wood et al., 1992) suggest that skeletal lesions might sometimes actually indicate a relatively healthy individual. This suggestion is based on the fact that visible skeletal lesions take some time to form; they do not form immediately in response to trauma or disease, but rather take weeks or months to become detectable. Individuals with skeletal lesions might therefore have been healthier than their peers

without lesions, given that they were able to survive malnutrition, trauma, or disease long enough for the skeletal lesions to form. Absence of a certain skeletal lesion might indicate relatively poor health, as individuals without lesions were in such poor health that they succumbed to illness, trauma, or malnutrition and died before lesions ever formed.

Wood et al. (1992) do not argue that skeletal lesions are necessarily or even typically associated with better health, but rather that paleodemographers cannot ignore that possibility; paleodemographers should therefore exercise caution when they make inferences about health in past populations based on skeletal lesions (Usher, 2000; Thomas, 2002). The Usher model makes no assumptions about what effect lesions have on risk of death; instead, it provides a means for examining the relationship between skeletal lesions, health, and mortality.

The full Usher model allows for demographic nonstationarity because it includes a birth model (i.e. all individuals enter State 1 through birth):

$$b = \left[\int_0^\infty e^{-ra} S(a)\, da \right]^{-1} \qquad \textbf{2.5}$$

where b is the crude birth rate, r is the population growth rate, a is age, and $S(a)$ is the aggregate survival function (the probability that death has not occurred by time a). When r is not constrained to 0, the model allows for population growth or decline (nonstationarity). The value of r can theoretically be estimated from the data observed in the skeletal sample.

Three-State Usher Model

I was interested in determining: 1) the level of excess mortality associated with the Black Death and how mortality was distributed by age and sex, and 2) the selectivity of the epidemic with respect to frailty. To investigate the level of Black Death excess mortality, I used the model to compare the risk of mortality for those who died during the epidemic compared to those who died during times of normal mortality. To examine the effect of sex on risk of death, I used the model to compare the risk of death for men to that of women during the Black Death.

To investigate the selectivity of the Black Death, I analyzed the risk of death for individuals with lesions compared to the risk for those without skeletal lesions within the East Smithfield Black Death cemetery. I then compared the risk of death associated with lesions in East Smithfield to the risk associated with lesions within the Danish, non-Black Death samples. Because I was interested in just the presence or absence of certain skeletal lesions and not in the activity of those lesions (i.e. active or healed at the time of death), dividing individuals with lesions into two distinct states (active lesions versus healed lesions) was not necessary for this study. Therefore, use of all three living states in the full Usher model is unnecessary for my current research.

Furthermore, I analyzed skeletal lesions only because of the information they might provide about risk of death during the Black Death. I did not attempt to estimate

lesion frequencies in the once-living population based on lesion frequencies observed in the skeletal samples. If I were attempting to estimate lesion frequencies in the once-living population, I would have to examine the effects of hidden heterogeneity in frailty on mortality. However, as this was not my goal, I need not estimate the frailty distributions associated with the different living states. Because my analytical goals were relatively modest, I used a reduced, three-state version of the model, as shown in Figure **2-5,** rather than the full four-state Usher model. This three-state model includes all the necessary parameters that allowed me to determine the excess mortality of the Black Death and the selectivity associated with sex and various skeletal lesions.

In the reduced version, there are three states: State 1 includes those individuals with no skeletal lesions, State 2 includes those with active or healed lesions, and State 3 is death (Figure 3). By allowing variation in the transition rates between each of the two living states and death, the model can be used to estimate the differential risk of death associated with the living states; the model therefore allows one to investigate selective mortality with respect to skeletal lesions. As in the full Usher model, individuals are born into State 1, and transitions between states i and j occur at age-specific hazard rates $\mu_{ij}(a)$, where a is age in years; transitions between the states are determined by an individual's age and level of frailty.

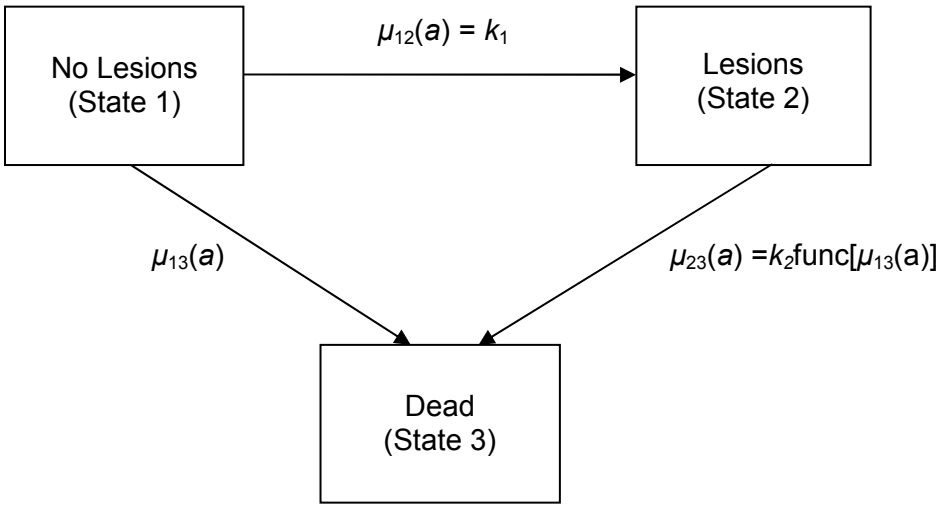

Figure 2-5: Three-state version of the Usher model (from Usher 2000).

The baseline risk of death from State 1, $\mu_{13}(a)$, is specified as a Siler mortality model (Eq. 2-4). The hazard of developing lesions, $\mu_{12}(a)$, in the Usher model is estimated as a constant k_1, as the age of onset of conditions resulting in lesions is generally unknown in paleoepidemiological studies. That is, for most skeletal lesions, one does not know the age at which an individual became ill or suffered some other physiological stress, nor does one know the timing of the development of the lesion. For simplicity, in this study, the age of onset of lesions is an exponential random variable. Thus, for this study, *I assumed that all individuals faced a constant risk of developing a lesion.* However, this assumption might not be true; some individuals might not have

been at any risk of developing lesions, or the risk of developing some of the lesions used in this study might have been lower for adults compared to children.

The age-specific mortality rate from State 2, $\mu_{23}(a)$, is specified as some function of $\mu_{13}(a)$, e.g. a proportional hazard function, an additive function, an accelerated failure-time function, or a function operating on each of the three age components of the Siler model separately. Usher originally modeled the hazard of dying from State 2 as proportional to the baseline age-specific risk of dying from State 1, and this is the approach I took. Under this specification, k_2, the constant of proportionality, indicates the proportional difference in risk of death between individuals with and without lesions. When k_2 is significantly larger than one, individuals with lesions faced an elevated risk of dying compared to similarly aged individuals without lesions. When k_2 is significantly lower than one, individuals with lesions faces a decreased risk of death compared to their peers without lesions. And if k_2 is equal to one, individuals with and without lesions were at the same risk of death. It is tempting to view the k_2 parameter as indicating the relative risk of death for individuals with lesions; however, given that I do not know the true age pattern of lesion formation and death among those with lesions, I will treat k_2 as a general, qualitative measure of excess mortality associated with lesions. That is, for this project, the results I obtain for the k_2 parameter associated with various lesions are more important in terms of the patterns they reveal than their actual numerical values. Note that if the proportional hazards assumption made here is correct, then k_2 *would* be constant and would therefore indicate relative risk of death.

By using the proportional hazards model in this study, *I assumed that the k_2 parameter is constant across age*. That is, I assumed that the differential risk of death

associated with lesions compared to the risk for those without lesions is proportional across age. Such an assumption of proportionality might not be correct; for example, the excess mortality associated with certain lesions might be higher for children than it is for adults. In Chapter 4, I discuss the implications if this assumption is wrong.

States 1 and 2 among the living are latent states; that is, they are not directly observable as one only observes dead individuals. But observations on age at death and the age-specific distribution of lesions among the dead provide the data needed to estimate the entire model by maximum likelihood. With the three-state model, I estimated the baseline risk of death for everyone in the population (the baseline risk of death from State 1), and then I determined the deviations from the baseline risk associated with certain skeletal lesions. Appendix E provides the hazard and likelihood functions used in the analyses.

I estimated the likelihoods of dying from State 1 (Equation E-15) and from State 2 (Equation E-22) using maximum likelihood estimation by writing lesion-specific programs for *mle* and using the simplex method of maximization; I also ran several preliminary analyses using the simulated annealing method and found that it yielded results very similar to those produced using the simplex method. I used the open trapezoidal method of numerical integration; tests of the Romberg method of integration yield results very similar to those obtained with the open trapezoidal method. An example of an *mle* program is provided in Appendix F.

Applications of the Usher Model

Level of Excess Mortality

I applied the Usher model in two distinct ways. In one application, I estimated the level of excess mortality associated with the Black Death; in the second, I determined the risk of death associated with sex and skeletal lesions.

To estimate the level of excess Black Death mortality, I combined the East Smithfield and Danish samples and treated them as a single cemetery sharing the same baseline mortality (specified as a Siler model). I applied the Usher model to the East Smithfield and Danish "sub-samples" as shown in Figure **2-6** . "Risk factor" in this version of the analysis is analogous to "lesion" in Figure 2-5, and refers specifically to dying during the Black Death. Individuals from East Smithfield and Denmark provide the data necessary to estimate the parameters of the Siler model (indicated by $S(\theta)$ in Figure). In this application, *within* the Danish sub-sample, the hazards of moving from State 1 and State 2 to death are the same: $k_{2DK}S(\theta)$; specifically the hazard is the same as that of dying from State 2 in the 3-state model described above. Similarly, the hazards of moving from State 1 and State 2 to death are the same *within* the East Smithfield sub-sample: $k_{2ES}S(\theta)$.

.

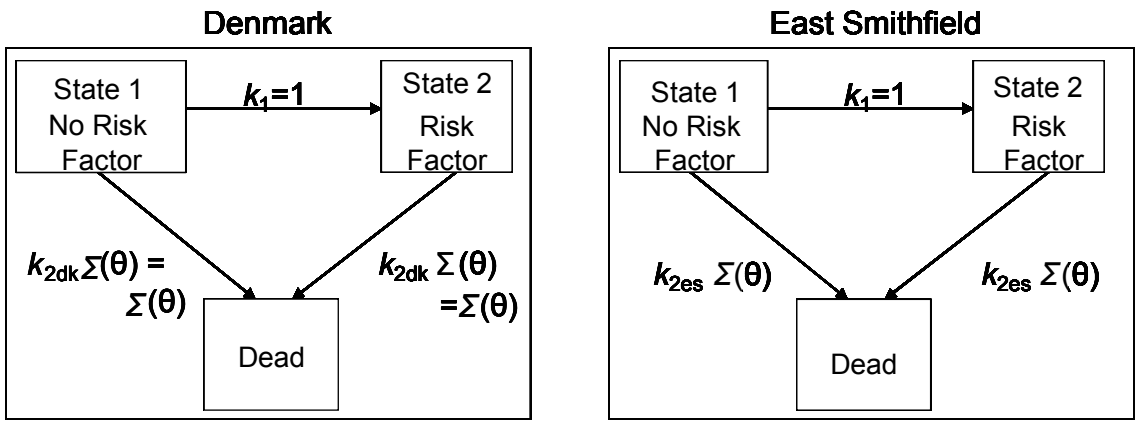

Figure 2-6: Application of Usher model to estimate excess mortality. $\Sigma(\theta)$ is the Siler model with parameters θ, k_{2dk} is the excess mortality within the Danish sample (set to one), and k_{2es} is the excess mortality within East Smithfield (i.e. for individuals who died during the Black Death).

Within each sub-sample, I allow for the theoretical possibility of making the transition from State 1 to State 2. This transition is irrelevant because all individuals within each sub-sample are assumed to face the same risk of dying, so I set the value of k_1 to one. The intuitive approach would be to set k_1 equal to zero, thus eliminating the possibility of transition from State 1 to State 2. However, I could not set k_1 equal to zero, as doing so reduces the likelihood of dying with a lesion to zero (see Equation 22 in Appendix E). To test the effect of the assumption that $k_1 = 1$, I conducted sensitivity analyses using various values of the k_1 parameter.

The hazards for the East Smithfield and Danish sub-samples differ with respect to the k_2 parameter. For the Danish sub-sample, I set the value of k_{2DK} equal to 1, so that the likelihood of dying reduced to the Siler hazard; individuals in the Danish sample are assumed to pre-date the Black Death and thus never to have been exposed to the disease,

so the risk of death for all Danish individuals is simply the baseline risk of death. However, individuals in the East Smithfield sub-sample died during the Black Death, and so I estimated their risk of death relative to the baseline risk of death. Under Usher's constant proportional hazards model, the estimate of k_{2ES} provides a measure of Black Death excess mortality.

This analysis assumes that the Danish sample contains no victims of the Black Death and that the East Smithfield cemetery includes *only* victims of the Black Death. While there may be some individuals in the East Smithfield cemetery who died from causes other than the Black Death, they are likely to be a small minority, as deaths from the Black Death were known to have swamped normal mortality (Wood et al., 2003). Although I tried to select an exclusively pre-Black Death Danish sample based on arm-position data, there might be some victims of the Black Death in the Danish sample. As discussed above, my Danish sample includes individuals buried with arm position B, and Kieffer-Olsen (1993) and Jantzen et al. (1994) found that a small proportion of individuals who died after 1350 were buried with arm position B. If Black Death victims were included in my Danish sample, they are likely to be a minority. However, if the above assumption is untrue, then the excess mortality associated with the Black Death will be under-estimated.

Given estimates of Black Death mortality from historical records, I expected the value of k_{2ES} associated with the Black Death to be substantially greater than 1.

Sex-pattern of Mortality

To determine if there was differential risk of death associated with sex during the Black Death (as is observed with some diseases), I applied the Usher model separately to the East Smithfield and Danish samples. Within each cemetery sample, I applied the model to sex as shown in **Figure 2-7**. Both males and females provide the data necessary to estimate the parameters of the Siler model (indicated by $S(\theta)$ in Figure 2-7). For each sex, the hazards of moving from State 1 and State 2 to death are the same, and I allowed for the theoretical possibility of making the transition from State 1 to State 2. Because men and women during the Middle Ages were presumably not at any risk of becoming the opposite sex, the value of the k_1 parameter is set to one for this analysis. To test the effect of the assumption that $k_1 = 1$, I conducted sensitivity analyses using various values of the k_1 parameter.

I arbitrarily designated maleness as a risk factor to determine if either males or females were at an increased risk of death compared to the opposite sex during the epidemic. For females, I set the value of k_{2f} equal to 1, so that their likelihood of dying reduced to the Siler hazard, and I estimated the value of k_{2m} associated with males.

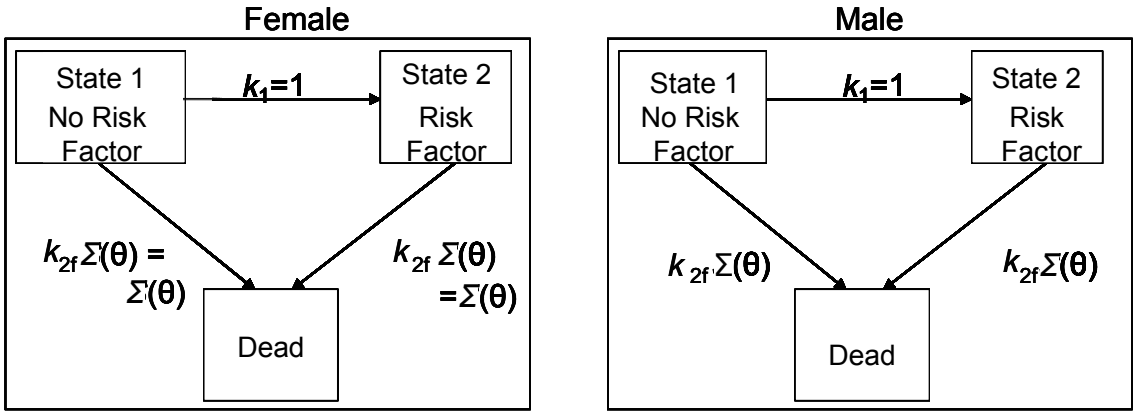

Figure 2-7: Application of Usher model to sex. $\Sigma(\theta)$ is the Siler mode with parameters θ, k_{2f} is the risk for females (set to one), and k_{2m} is the risk for males.

If males and females were at the same risk of death during the Black Death, the value of k_{2m} should be close to or equal to one; however, if males were at an increased or reduced risk of death compared to females during the epidemic, the value of k_{2m} should be less than or greater than one, respectively.

Selectivity with Respect to Frailty

To determine if the Black Death was selective for frailty, I applied the Usher model separately to the East Smithfield and Danish samples. In these analyses, I estimated the excess mortality associated with various osteological lesions or stress markers *within* each sample. I have chosen well-standardized markers that have been shown to be good, non-specific indicators of frailty (Goodman and Rose, 1990; Stuart-

Macadam, 1991; Buikstra and Ubelaker, 1994; Roberts and Manchester, 1995; Usher, 2000; Palubeckaite et al., 2002). For example, Usher (2000) applied the model to observations on some of these stress markers in skeletons from the medieval Danish village of Tirup. She found that enamel hypoplasias on the first maxillary molar, cribra orbitalia, shorter than average femur length, and proliferative and destructive lesions on the femur were associated with significant increases in the risk of death.

By fitting Usher's (2000) model to data from the East Smithfield and Danish skeletons, I tested whether the presence of such markers is associated with significant excess mortality within each sample. Given the results obtained by Usher, I expected lesions to be associated with excess mortality within the Danish samples. By comparing the estimates of excess mortality associated with stress markers obtained from East Smithfield to those from the Danish cemeteries, I determined whether the Black Death was selective with respect to frailty. Given the virulence of the Black Death, I expected little if any excess mortality associated with stress markers in East Smithfield compared to what I found in the Danish samples.

Usher conducted simulation studies to determine if *mle* is able to recover known parameters from a simulated dataset. She did Monte Carlo simulations using the dynamic modeling program Stella, and entered probabilistic hazard rates at the beginning of the simulations. She began with 1000 individuals in State 1 and ran the model until each individual was dead; at the end of the simulation, she knew the age-at-death distribution for the simulated population, and the state each individual was in before death. The dataset, a simulated cemetery sample, was entered into *mle*, and *mle* was always able to recover the k_1 (the constant risk of developing a lesion, i.e. making the transition from

State 1 to State 2) and k_2 (excess mortality) parameters (see Appendix C). The program performed less well at recovering the parameters of the Siler model (the baseline risk of death from State 1), but the recovered estimates were close to the known parameters; such a result is not surprising given that the parameters of the Siler model are correlated with one another, and many different combinations create the same age-at-death distribution. Importantly, *for the analyses of selectivity with respect to frailty*, I was interested in the estimate of excess mortality associated with lesions rather than the values of the Siler parameters.

Usher also conducted sensitivity analyses of sample size and found that *mle* was able to recover reasonably precise and unbiased estimates of the *k* values, which are of primary interest in the present study, with samples of 100 to 200 individuals. The results of these sensitivity analyses are summarized in Appendix C.

In using the Usher model to compare East Smithfield to the Danish cemeteries, I in effect assumed that immediately before the Black Death, East Smithfield had the same population growth rate and age-specific mortality rates as did the communities in Denmark. This is not an unreasonable assumption given the similarities in socio-economic and demographic conditions between southern England and Denmark during the Middle Ages (see Chapter 1). The cemeteries samples used in this study were both drawn from the homogeneous population of medieval Denmark (*circa* 1200 – 1350) and from London (*circa* 1348-1350). Thus, demographic non-stationarity should not pose a problem for this study, as I did *not* likely compare across populations with potentially different growth rates. However, this assumption might not be correct, and in Chapter 4 I discuss the implications if it is wrong.

For this study, *I assumed that the Danish and East Smithfield populations were stable*, i.e. closed to migration, with constant age-specific fertility and mortality rates and stable age distributions. This assumption is reasonable, in general, in paleodemographic studies given that most populations "still closely approximate a stable age distribution" even in the face of migration and changing mortality and fertility rates (Milner et al., 2000: 480). However, because the catastrophic mortality levels of the Black Death would perturb a population's age distribution away from its stable form, assuming the East Smithfield population was stable is problematic *if* the Black Death did not run its course very rapidly. If the Black Death epidemic lasted relatively long in any given area, mortality would be acting upon a very different age distribution at the end of the epidemic compared to that existing at the initial outbreak of disease. However, estimates of time course of the Black Death show that the time course of the disease was very rapid (Wood et al. 2003), such that the population affected at the beginning of the epidemic would not have differed much from that affected at the end of the epidemic. In Chapter 4, I discuss the implications if the stability assumption is wrong.

Osteological Indicators of Frailty Used in This Study

Tibial Lesions

Periostitis and osteomyelitis are conditions that cause macroscopic changes to the surface of bones. Periostitis and osteomyelitis produce nonspecific skeletal lesions as

part of an inflammatory response to trauma or infection. Non-specificity refers to the fact that the lesion resulting from infection with one particular pathogen is indistinguishable from that caused by a different pathogen (Roberts and Manchester, 1995). Very few infectious diseases (e.g. treponemal disease and leprosy) cause specific skeletal lesions, and most pathogens, if they lead to any bony response, cause non-specific lesions. Inflammation is one of the body's responses to invading pathogens, and inflammatory bony lesions typically reflect chronic rather than transitory infection (Roberts and Manchester, 1995). In order for bony lesions to form, an infection or trauma must be severe enough to stimulate such a response, yet the victim must survive long enough for the lesion to form (Ortner, 1992).

Periostitis and osteomyelitis reflect the same general inflammatory response induced by infection or trauma, but the two are often distinguished on the basis of their degree of severity and their location. Periostitis produces less severe lesions, and is usually the result of an elevation of the periosteum (the fibrous membrane covering the surface of bones) and subsequent proliferation of bone caused by the stimulation of osteoblasts (bone-producing cells) within the inner-most layer of the periosteum (Mensforth et al., 1978; Larsen, 1997). Periostitis can be caused by trauma, hemorrhage, the direct spread from an adjacent soft tissue infection (e.g. skin ulcers), or it can be the manifestation of a generalized infection (Ortner, 2003). Often, periostitis causes the formation of woven bone on the external surface of a bone; woven bone has a porous appearance and may eventually be remodeled into lamellar bone.

Osteomyelitis, which produces more severe lesions, is a bacterial infection involving both the periosteum and the endosteum (the layer of cells lining the medullary

cavity). It is therefore characterized by an inflammation of bone and bone marrow. Osteomyelitis is primarily caused by pyogenic (pus-producing) bacteria, and at least 90% of cases are caused by *Staphylococcus aureus;* however viral, parasitic, and fungal infections can also cause osteomyelitis (Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003). Osteomyelitis is characterized by simultaneous bone proliferation and destruction, and pus formation. The affected bone is usually enlarged, and there is a restriction of the medullary cavity as osteoblasts of the endosteum are stimulated. Abscesses containing pus may form within the bone, and sinuses for the drainage of the abscesses may form on the surface of the bone (Roberts and Manchester, 1995; Larsen, 1997). Infection of blood vessels can restrict blood flow to the infected bone and eventually cause necrosis and eventually the formation of a sequestrum – a segment of necrotic bone surrounded by a proliferation of new bone growth; the new bone surrounding the sequestrum is called an involucrum (Aufderheide and Rodriguez-Martin, 1998).

Osteomyelitis can be caused by direct contamination of traumatic or surgical wounds, spread from an adjacent soft tissue infection, or by hematogenous spread from a septic focus somewhere else in the body (Ortner, 2003). Infection resulting in periostitis is not typically fatal, as it tends to be localized to one area on a single bone, but osteomyelitis is caused by a more severe infection that can cause death if it spreads to vital organs (Larsen, 1997).

I chose to score proliferative lesions associated with periostitis and osteomyelitis on the tibia because such lesions are relatively common in archaeological samples, and because the tibia is a robust bone that is therefore often well-preserved.

Paleopathological studies have repeatedly demonstrated that the tibia is the bone most commonly affected by periostitis and osteomyelitis (Eisenberg, 1991; Milner, 1991; Roberts and Manchester, 1995; Larsen, 1997). The tibia may be particularly vulnerable to bacterial colonization and infection anterior and medial surfaces of the diaphysis of the tibia for because circulation is generally slower in the lower legs, the anterior and medial surfaces of the bone are the "most vascularly and physiologically inactive" of the skeleton and are cooler than other parts of the body as they are not surrounded by large amounts of soft tissue, and the bone is not well protected from trauma by soft tissue (Larsen, 1997).

I examined the proliferative lesions (the posterior surface of the tibia is often covered by extensive muscle markings that can interfere with lesion identification). An individual received a score of "present" if there was at least one distinct patch of woven or sclerotic bone (or a combination of the two) laid down on the surface of the diaphysis (see Figure **2-8).**

Figure 2-8: Proliferative tibial lesions on the medial surface of an adult tibia from Denmark.

I assigned a score of "no lesion" only to those tibiae that were complete and well-preserved and lacked the appropriate lesions. I assigned a score of "no information" if the tibia was missing or poorly preserved. I did not attempt to distinguish between lesions caused by periostitis and osteomyelitis, as I was interested in estimating frailty in general and not diagnosing specific causes of skeletal lesions. In the analyses of excess mortality associated with tibial lesions, I only included individuals who were well preserved.

Porotic Hyperostosis

In the normal physiological state, there is a balance between bone formation and bone destruction by osteoblasts and osteoclasts, respectively; physiological stress may upset the balance between these two processes, leading to abnormal proliferation or destruction of bone. Porotic hyperostosis refers to skeletal lesions characterized by a porous appearance of the outer table of the affected bone often associated with expansion of the underlying diploic bone (Roberts and Manchester, 1995). Porotic hyperostosis occurs on the cranial vault bones and there is a great deal of variation in the size and severity of the lesions. Porotic hyperostosis can be caused by any chronic disease that leads to an increase in the volume of hemopoietic (i.e. blood-cell forming) marrow, which, in turn causes an expansion of diploic bone containing the hemopoietic marrow (Powell, 1988; Ascenzi et al., 1991).

Porotic hyperostosisis is associated with *childhood* episodes of physiological stress which cause expansion of the bone marrow; adults in clinical studies subjected to the conditions that cause porotic hyperostosis in children (e.g. iron-deficiency anemia) display no evidence of similar bony changes (Stuart-Macadam, 1991). Half of the bone marrow of an adult is adipose (fat tissue), whereas in children all the long bone marrow is hemopoietic; an adult, therefore, can double his/her volume of hemopoietic marrow without any resulting bony change, but in a child, even a small increase in hemopoietic marrow can lead to an expansion of the diploic bone (Ascenzi et al., 1991). Although porotic hyperostosis tends to occur more frequently in children, the lesions can be retained into adulthood, providing a lasting records of childhood illness.

Diseases including congenital hemolytic anemias (e.g. sickle-cell disease, thalassemia), iron-deficiency anemia, and cyanotic congenital heart disease can all cause porotic hyperostosis (Mensforth et al., 1978). Anemia is characterized by a reduction in the concentration of hemoglobin (oxygen-binding protein in red blood cells) or numbers of red blood cells below normal levels. Iron is a component of hemoglobin (and other proteins) and it functions in oxygen transport from the lungs to the rest of the body's tissues via the bloodstream (Ryan, 1997). Any reduction in the concentration of iron or of red blood cells reduces the body's ability to transport oxygen efficiently and will, thus, stimulate an increase in hemopoietic marrow to increase production of blood cells. This increased production may, in turn, cause an expansion of the diploë and lead to porotic hyperostosis.

In the 1970s, it was believed that skeletal populations with relatively high frequencies of iron-deficiency anemia, as indicated by porotic hyperostosis, were less successful in adapting to their environments than those populations with no porotic hyperostosis. This view is now being challenged by data suggesting that iron-deficiency may strengthen the body's defense against infection, as a low-iron environment inhibits growth of bacteria and other pathogens (Stuart-Macadam, 1991; Ryan, 1997). Iron-deficiency anemia may reflect, among other things, a diet low in iron and/or an 'iron-sequestering' adaptive response to invading pathogens; presence of porotic hyperostosis may, thus, provide information regarding both nutritional levels and exposure to pathogens.

In addition to anemias and heart disease, porotic hyperostosis can be caused by infection of the scalp, periostitis, osteomyelitis, trauma, scurvy, or, more rarely, by

rickets; under these circumstances, there tends to be remodeling of the outer table of the cranial vault creating a porous appearance without expansion of the underlying diploë (Ortner, 2003). According to Martin et. al. (1985), it is very difficult to determine the specific cause of porotic hyperostosis.

I scored the frontal, parietal, and occipital bones for porotic hyperostosis. I looked for lesions ranging from small areas of porosity to severe expansion of the diploë on the frontal and parietal bosses, and on the squamous portion of the occipital bone along the coronal, sagittal, and lambdoidal sutures. I scored a lesion as "present" if there was at least one square centimeter of porosity with the appearance of densely clustered pin-pricks as shown in Figures **2-9** and **2-10.** I assigned a score of "no lesion" only to those bones that were complete and well-preserved and lacked the appropriate lesions. In the analyses of excess mortality associated with porotic hyperostosis, I only included individuals who were well preserved.

Figure 2-9: Porotic hyperostosis on the parietal bones of an adult individual from Denmark (posterior view).

Figure 2-10: Close-up view of porotic hyperostosis on cranial vault of an adult individual from Denmark.

Cribra Orbitalia

Like porotic hyperostotis, cribra orbitalia is a lesion characterized by a porous appearance of the outer table of the affected bone often associated with expansion of the underlying diploic bone, as shown in Figure **2-11**. Cribra orbitalia affects the orbital roof, particularly the anterolateral portion. Cribra orbitalia and porotic hyperostosis are similar skeletal lesions, and are, in fact, considered by some researchers to represent the same pathological processes. Cribra orbitalia is caused by many of the same factors that cause porotic hyperostosis. Additionally, cribra orbitalia can be caused by inflammation of the frontal, maxillary, or ethmoidal sinuses or by inflammation of the lacrimal gland

(Ortner, 2003). Cribra orbitalia occurs more frequently in children than adults, but these lesions can be retained into adulthood.

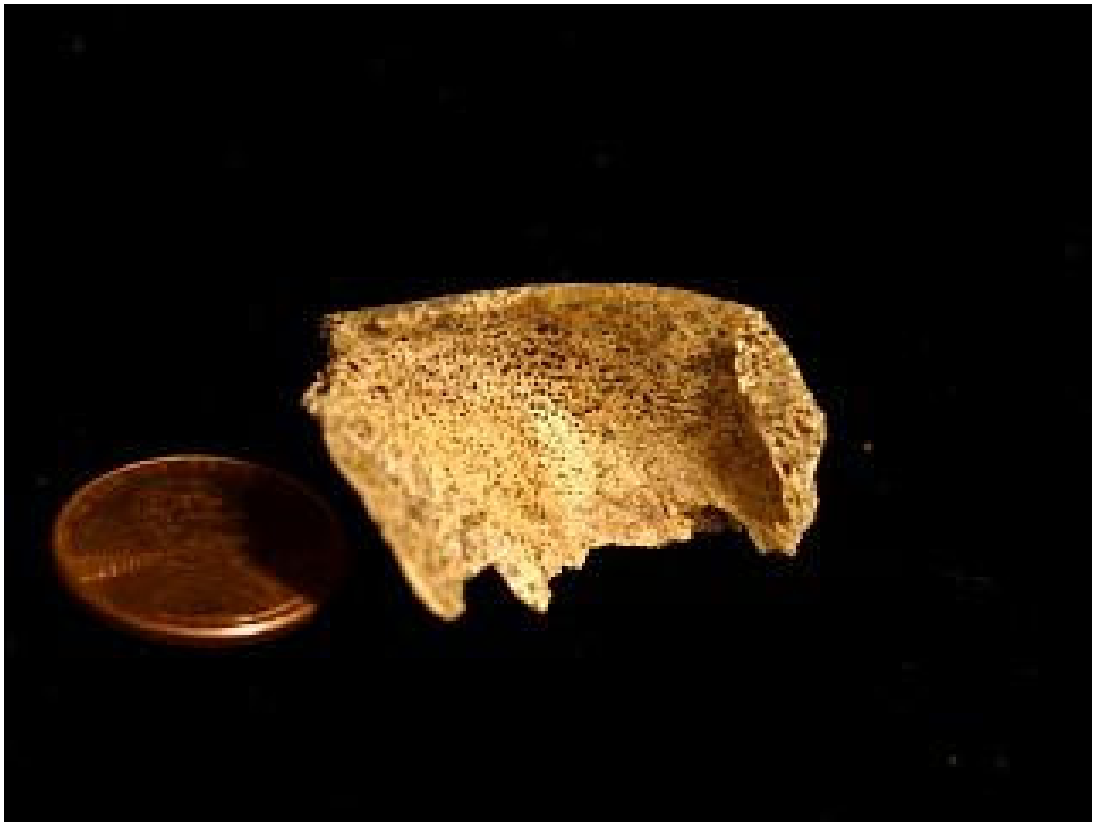

Figure 2-11: Cribra orbitalia on the roof of a left juvenile orbit (from Denmark).

I scored the orbital roofs of the frontal bone for cribra orbitalia. I assigned a score of "present" if there was at least one square centimeter of porosity similar to that shown in Figure 2-8. I assigned a score of "no lesion" only if a bone was complete, well-

preserved, and lacked the porous lesions. In the analyses of excess mortality associated with cribra orbitalia, I only included individuals who were well preserved.

Enamel hypoplasia

Enamel hypoplasia is a macroscopic tooth enamel defect that is caused by disruptions in the metabolism of ameloblasts (enamel forming cells); such disruptions can be caused by both specific and non-specific infection or by nutritional deficiencies, circumstances in which the body is forced to divert energy from enamel formation to routine maintenance. Enamel is calcified in two stages, the first of which is matrix formation; if ameloblasts are disrupted during matrix formation, there is a reduction in enamel thickness (enamel hypoplasia) (Huss-Ashmore et al., 1982). Enamel hypoplasias appear as a ring or circle of pits and adjacent defects on the enamel (Dahlberg, 1991). Enamel hypoplasias occur only while the teeth are developing and are not subject to remodeling, and, thus, they remain as a "permanent record into adulthood" of nutritional deficiency or illness experienced during childhood (Roberts and Manchester, 1995: 58).

According to Goodman (1991), enamel hypoplasia has been found to be inversely correlated with longevity in several archaeological samples, and he suggests at least three possible explanations that may account for such a relationship: 1) the observed data reflect differential life-long patterns of susceptibility to physiological stress (i.e. if an individual is highly susceptible to stress, he/she is more likely to both have enamel hypoplasias and die young); 2) individuals who are exposed to and survive a prolonged

episode of stress during childhood are consequently less able to respond to future stress (i.e. the initial episode of stress weakens the individual's ability to survive future stress); or 3) the data reflect differential life-long patterns of behaviorally and culturally mediated exposure to stress (e.g. socioeconomic status). Goodman argues that the third is the most plausible explanation of the relationship between longevity and enamel hypoplasia.

Often researchers examine only one tooth, the mandibular canine, as it has a relatively long developmental time-span (approximately 10 years) and is highly sensitive to physiological stress (Goodman et al., 1980; Huss-Ashmore et al., 1982). Others examine both the maxillary central incisors and mandibular canines, claiming that these teeth are typically more 'hypoplastic' than others (Santos and Coimbra, 1999). Enamel hypoplasia can be detected either by direct visual examination of the surface of the tooth, as they are easily visible to the naked eye, or by microscopic examination of thin sections of enamel (Thomas, 2002)..

I scored the maxillary central incisor, mandibular and maxillary canines, and the maxillary first and second molars for linear enamel hypoplasias (distinct horizontal lines of decreased enamel thickness). I scored permanent dentition only and I only scored teeth with very little or no wear. I scored only the buccal surfaces of the teeth. I assigned a score of "present" if one or more enamel hypoplasias were visible to the naked eye under good lighting and could be felt as slight depressions by running a fingernail or thin soft wood stick over the surface of the tooth (see Figure **2-12**). I assigned a score of "no lesion" if no enamel hypoplasias were visible or palpable.

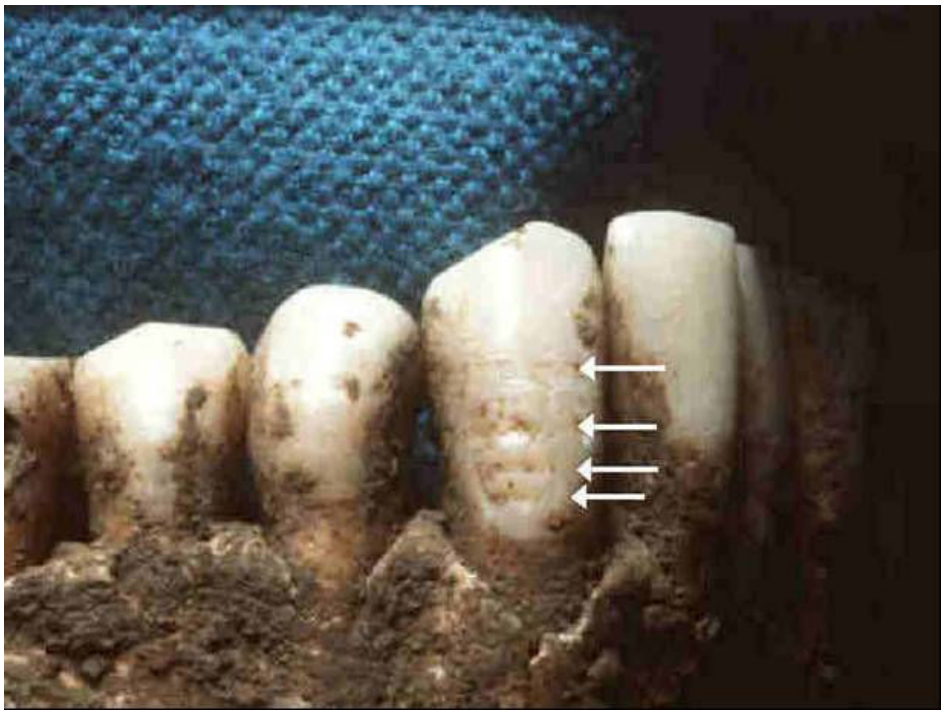

Figure 2-12: Linear Enamel Hypoplasias on an adult mandibular canine (indicated by arrows). From web.utk.edu.

Estimates can be made regarding the age at which enamel hypoplasias formed based on location of the lesion on the tooth, and by determining the timing and frequency of enamel hypoplasias much can be learned about the patterns of stress to which the individual was exposed (Power, 1985-86). Following Usher (2000) and Palubeckaité et al (2002), I determined age at formation using the standards provided by Massler et al. (1941). Reid and Dean (2000) have provided new, potentially more reliable standards forestimating enamel hypoplasia formation time. However, they provide enamel formation sequences only for the anterior teeth (incisors and canines); furthermore

Palubeckaité et al (2002) found that the possible corrections provided by the Reid and Dean (2000) formation times did not substantially affect the results of their study of medieval and early modern Danish and Lithuanian samples compared to using the more conventional method of Massler et al (1941).

For my dissertation, I was concerned with whether individuals experienced one or more episodes of stress during tooth development and not necessarily the exact age at which those events occurred. I therefore used the Massler et al. method so that I could include the maxillary molars and thereby maximize the information I obtained from each individual. These data can be used in future work to determine the variation in risk of death associated with number of enamel hypoplasias (i.e. whether individuals who suffered from multiple episodes of stress were at a higher risk of death compared to those who suffered from just one episode). These data can also be used to investigate whether risk of death associated with enamel hypoplasia varies at all with respect to age at formation.

Stature

Adult stature reflects, among other things, exposure to chronic stress during development (Haviland, 1967; Powell, 1988; Roberts and Manchester, 1995). Short adult stature, relative to other individuals within the population, may indicate poor health and poor nutrition during the developmental years, at least if everyone's genetic composition with respect to stature is similar. Children who are malnourished or fighting infection and disease must expend precious energy resources in basic tissue maintenance and the

immune response, diverting energy from growth and development to these most essential metabolic functions. According to Steckel (1995:1903) stature is "a net measure that captures not only the supply of inputs to health but demands on those inputs." In general, stature is positively correlated with health condition throughout an individual's life (Komlos and Baur, 2003).

A possible complication arises when one attempts to reconstruct health patterns based on adult stature. Adult stature is controlled not only by environmental and cultural factors, such as disease and nutrition, but also by genes; two individuals may be of greatly different stature, not because of differential exposure to physiological stress during childhood, but because of the genetic component of stature (Huss-Ashmore et al., 1982). I hopefully avoided this potential confounder, as I did not compare long bone length to a standard derived from a modern population nor from an archaeological sample that potentially differs from my target samples with respect to genetic composition and/or exposure to environmental and dietary factors that affect stature. As described below, I only compared long bone length *within* cemetery samples, and not across potentially different populations.

I measured the maximum length of the femur adult skeletons; for children with unfused epiphyses, I measured the maximum diaphyseal length of the femur (Buikstra and Ubelaker, 1994). I measured only those long bones that were complete. I measured the long bones using an osteometric board according to the standard methods described in Bass (1995) and Buikstra and Ubelaker (1994).

When using stature as a measure of frailty in paleodemographic investigations, researchers often estimate overall stature for each individual from long bones using a

regression function derived from a reference sample. The stature-estimation formulae produced by Trotter and Gleser (1952, 1958, 1970) have been used frequently by paleodemographers, archaeologists, and forensic anthropologists over the past several decades (e.g. Hershkovitz et al., 1993; Reale et al., 1998; Robb et al., 2001; Papathanasiou, 2005). According to Bass (1995), the most reliable stature estimates are the Trotter and Gleser estimates for white Americans and African-Americans. However, stature estimation is complicated by geographic and racial differences in stature; the ideal reference sample would be one that was very similar to the target sample in terms of genetic composition, diet, and exposure to other factors that affect adult height. Because I was not interested in height *per se*, but rather am using it as a measure of relative frailty, I avoided the potential problems associated with estimating stature. I compared long bone length within my samples directly and thereby eliminated the unnecessary and error-prone intermediate step of calculating stature. Following Usher (2000), I determined mean femur length separately for adult males and females within the East Smithfield and Danish samples separately; I treated femur lengths that were one standard deviation below the mean for the corresponding sex as lesions in my analyses; those individuals with femur lengths that were normal (within one or more standard deviations of the mean for their sex) or above average are considered to be in State 1 (no lesions) with respect to stature.

Relationship Between Osteological Lesions and Frailty

Previous studies of osteological lesions and stress markers similar or identical to those described above have suggested that they really do tell us something about frailty. In most cases, for which we have data, the presence of lesions is associated with increased frailty, but this is not always the case. For example, Usher (2000) found that enamel hypoplasia on the first molar, cribra orbitalia, shorter than average femur length, and proliferative and destructive lesions on the femur were all associated with increased risks of death (with k_2 values of 2.16, 4.97, 10, and 10 respectively). However, Usher also found that enamel hypoplasia on the incisor was associated with a *reduced* risk of death ($k_2 = 0.15$). Given the results of other studies, I expected to find that most of the lesions included in the study would be associated with high excess mortality within the Danish normal mortality cemetery at least. That is, I expected individuals in Denmark with skeletal lesions (i.e. who appear unhealthy) to have higher frailty than their peers without such lesions (the excess mortality associated with lesions in East Smithfield depends upon the selectivity of the Black Death).

Measurement Error Study

Interobserver Error

I collected all the data used in my dissertation myself; nevertheless, I conducted an inter-observer error study of the age and sex estimation methods I used in order to

determine how consistent my scores are with those of other researchers. Using a subset of 63 adult skeletons from the St. Mikkel cemetery, I compared my scores for the nineteen skeletal age indicator traits in a blind test to the corresponding scores of two highly experienced osteologists. (One of the "control" osteologists, Ulla Fruend, is Danish and the other, Corey Sparks, is American; both were trained by leading osteologists in their respective home countries and both have many years of research experience.) To analyze the degree of correlation between our measures, I calculated Spearman correlations separately for each pair-wise combination of observers (summarized in Appendix B Tables B-1 – 3). Figures B-1 – 3 in Appendix B show examples of regressions between my scores and those of Sparks and Freund.

There is a significant correlation between my scores and those of Freund for fifteen of the nineteen age indicators; 60% of these correlations are strong or very strong ($0.6 \leq r \leq 1$), and 40 % were moderate ($0.4 \leq r < 0.6$). There is a significant correlation between my scores and those of Sparks for sixteen of the age indicators; 56 % of the correlations are strong, 31 % are moderate, and 13 % are weak ($0.3 \leq r < 0.4$). There is a significant correlation between the scores of Sparks and Freund for fifteen age indicators; 47% of the correlations are strong, 47% are moderate, and 7 % are weak. In general, the weaker correlations were associated with certain features of the auricular surface (e.g. inferior surface topography, apical morphology) that have yet to be standardized properly.

Using the same sample of 63 adult skeletons from St. Mikkel, I compared my scores for sex to those assigned by the Freund and Sparks. All three of us gave the 63 individuals the same scores for sex.

Intraobserver Error

To evaluate the reliability of my own age, sex, and lesion scores, I conducted an intraobserver error study using a sample of 32 individuals from the cemeteries of St. Mikkel and Albani. I used the test-retest method, and I scored each individual in my error study twice; the intervals between first and second measurements ranged from four to ten weeks. I scored each individual for the nineteen skeletal age indicator traits, sex (in adults), cranial and tibial lesions, and I measured their long bones. To analyze the consistency of my scores for sex, age traits and cranial and tibial lesions, I calculated Spearman correlations separately for each pair-wise combination of repeated measurements (summarized in Appendix B).

All of the correlations were significant ($p < 0.05$), and strong to very strong ($0.6 \leq r \leq 1$). Statistica© can not calculate the significance of correlation coefficients equal to one. Therefore, for each trait with no variation between the initial and repeat measures (marked with an asterisk in Tables B-6 – 8 Appendix B), I calculated the significance of a correlation coefficient of 0.9999 with respect to the corresponding sample size; given that a correlation coefficient of one is very close to 0.9999, the true value of the significance of the correlation coefficients of one must be at least as good as that associated with a coefficient of 0.9999.

To analyze the error associated with my femur measurements, I regressed the second measurement for each femur on the corresponding initial measurement; the results of this regression are shown in Appendix B. In Figure B-1 in Appendix B, the true regression line is compared to an ideal regression line

that reflects absolutely no error in the repeated measurements (i.e. Y = X with an intercept of zero and a slope equal to one). The initial and repeat femur measurements were significantly and very highly correlated; the standard error associated with such measurements was only 0.7 millimeters. I used the Student's t-test to test the difference between the slopes of the true regression line between original and repeated measurements and the "ideal" regression; the results of this test ($t = 0.1943$, $df = 52$) indicate that the slope of the true regression is not significantly different from zero, and thus, the repeated measurements were very similar to the original measurements.

Summary of Measurement Error Study

The results of the interobserver error study suggest that I am capable of scoring skeletons for sex and age-indicators as well as two highly trained osteologists. My scores were significantly correlated with those of each osteologist for most age-indicators, and the vast majority of those correlations were moderate to strong. As mentioned above, the weaker correlations were associated with features of the auricular surface that are not yet properly standardized and therefore given less weight in age-estimation. Importantly, my age-indicator scores were as similar to those of each osteologist as their scores were to each others. There was no variation at all among the three of us with respect to our sex scores.

The results of the intraobserver error study suggest that I am highly consistent in terms of scoring age-indicators, sex, cranial and tibial lesions, and measuring the femur.

All of my repeated measurements were significantly and strongly correlated with the original measurements. The result of these measurement error studies cannot reveal how accurately I actually estimate age-at-death or sex (as the ages and sex of the individuals in the cemetery samples are unknown), but at the very least I am confident in my ability to assign sex and age-indicator scores and identify skeletal lesions.

Chapter Summary

- To investigate the mortality patterns of the Black Death, I compared a sample of 490 skeletons from the East Smithfield Black Death cemetery in London to a combined sample of 291 skeletons from the medieval Danish cemeteries of St. Mikkel and Albani Church.
- For adult age estimation, I used transition analysis to produce unbiased estimates.
- To evaluate the age pattern of Black Death mortality, I estimated the parameters of the Siler mortality model.
- For analyses of the excess mortality and selectivity with respect to frailty and sex, I used the Usher multistate model of morbidity and mortality.
- Meaurement error studies indicate that I was consistent in my scoring of skeletal age indicators, sex, and the presence of osteological indicators of frailty.

Chapter 3

RESULTS

Age Pattern of Black Death Mortality

To evaluate the age pattern of Black Death mortality, I estimated the parameters of the Siler mortality model separately for the East Smithfield and Danish samples using individual age-at-death estimates. I estimated point estimates of adult age-at-death using transition analysis, as described in Chapter 2. I estimated juvenile ages at death based on epiphyseal closure and dental development and eruption; for point estimates of juvenile age, I used the midpoint of the age range provided by these methods. For the sake of comparison with previous studies of East Smithfield, I also produced crude age-at-death distributions for East Smithfield and Denmark using the same age intervals used by Waldron (2001); *these crude age-at-death distributions, however, do not form the basis of any conclusions I ultimately make* about the mortality patterns of the Black Death.

Age-At-Death Distributions: Comparison with Previous Studies

In the previous studies of East Smithfield described in Chapter 1, to determine the age pattern of Black Death mortality, the researchers produced age-at-death distributions using traditional methods of age-at-death estimation (Waldron, 2001; Margerison and Knüsel, 2002). In these studies, individuals were assigned to one of six age-at-death intervals: 0 – 4, 5 – 14, 15 – 24, 25 – 34, 35 – 44, 45+. Figures **3-1** and **3-2** show age-at-death distributions adapted from Waldron (2001) and Margerison and Knüsel (2002). For the sake of comparison with these previous studies, I produced similar age-at-death distributions by allocating my individual age estimates for adults and juveniles into the same age intervals used in those studies (all age estimates are provided in Appendix D). I should emphasize that this approach is *not* one that I advocate, nor is it the focus of this study; I did it merely to make my distributions comparable to those of previous studies.

The age-at-death distributions produced from my data are shown in Figure **3-3**; this crude comparison of East Smithfield and Denmark reveals that the two age-at-death distributions are slightly different. I compared the distributions from the East Smithfield and Danish cemeteries using a likelihood ratio test (the G-test) (Sokal and Rohlf, 1995). The results of the G-test indicate that the two age-at-death distributions are significantly different ($p < 0.001$). Compared to Denmark, the East Smithfield cemetery has fewer infants and young children (ages zero to five years) and adults above the age of 25, and a higher proportion of older children, adolescents and younger adults (ages 5 to 25). Figure 3-4 compares the age-at-death distribution from East Smithfield from this study to that of Waldron (2001); I found a much higher proportion of individuals between the ages of 15 and 25 than Waldron.

A comparison of the age-at-death distributions from all three studies reveals that each study found fewer older adults (from about age 25 to 35) in East Smithfield than in the corresponding normal mortality cemetery. But this is where the general similarities among the three studies end; the East Smithfield distribution produced from my data differs from that of the other studies (both of which used the same age estimates for East Smithfield), and the normal mortality distributions from the three studies are also different. The direction in which the East Smithfield distribution differs from the normal mortality distribution at different ages, and the scale of that difference, varies from one study to the next. Furthermore, nothing much can be said, from this crude comparison, about the significance of any apparent differences between the Black Death and normal mortality cemeteries.

At best, I can conclude from a comparison of the age-at-death distributions, using these broad age-intervals, that the East Smithfield and Denmark distributions appear to differ and that the Danish distribution more closely conforms to expectations of a normal mortality cemetery (i.e. a relatively high proportion of young children and a lower proportion of older children and young adults). However, this crude comparison of the age-at-death distributions is not conclusive. A potentially more informative use of the available data, and the one I emphasize in this study, is to estimate the parameters of a hazard model. According to a comparison of East Smithfield and Denmark samples using that approach, there is no clear evidence that the Black Death was selective with respect to age among adults.

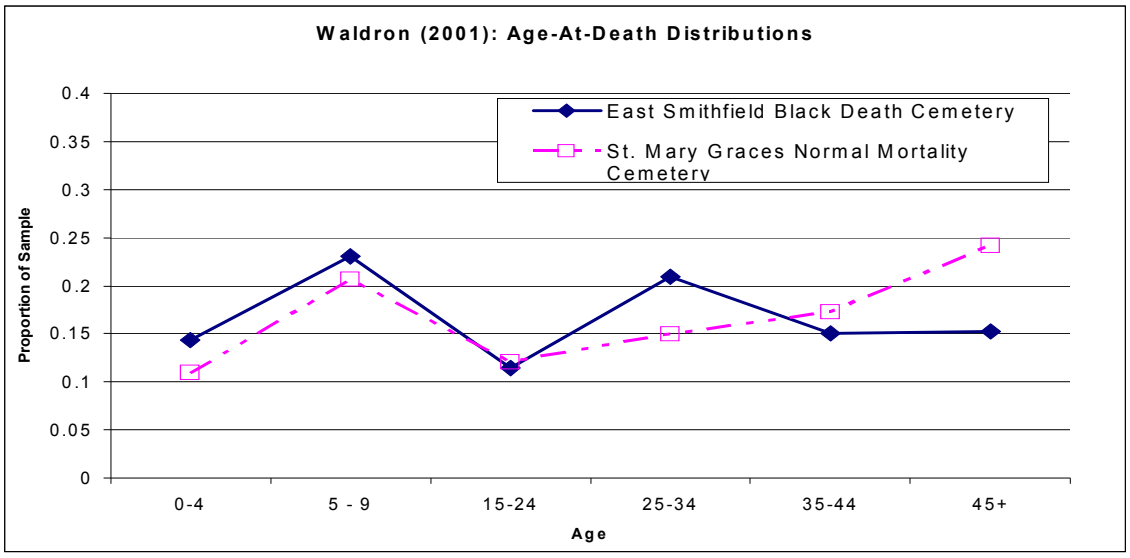

Figure **3-1**: Comparison of age-at-death distributions estimated by Waldron (2001) for the St. Mary Graces normal mortality cemetery and the East Smithfield Black Death cemetery. See text for a description of the age intervals used in the study. Adapted from Waldron (2001)

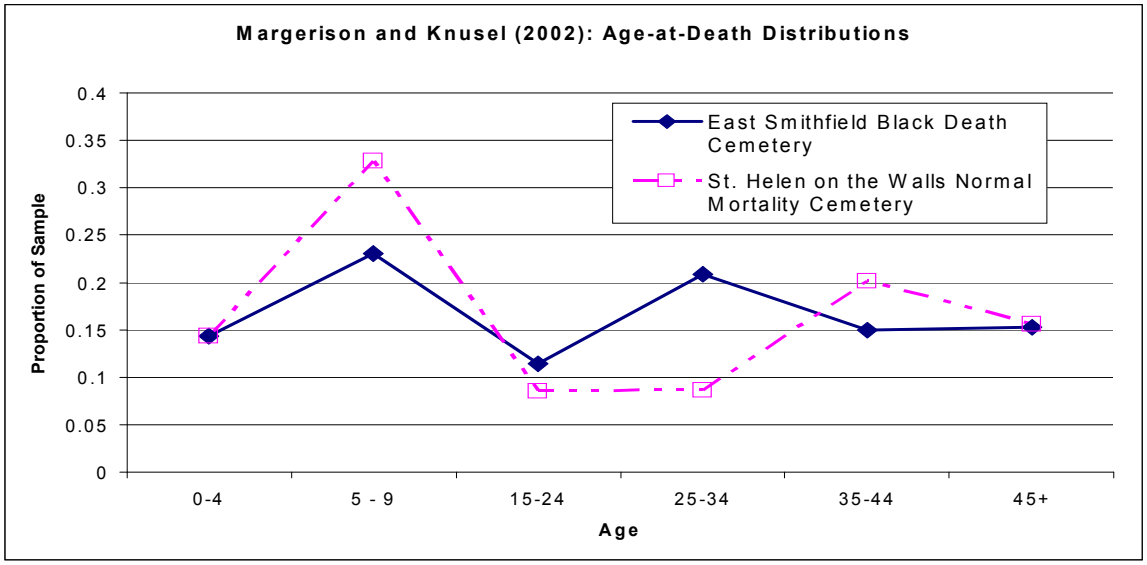

Figure **3-2**: Comparison of age-at-death distributions estimated by Margerison and Knüsel (2002) for a normal mortality cemetery and the East Smithfield Black Death cemetery. Adapted from Margerison and Knüsel (2002).

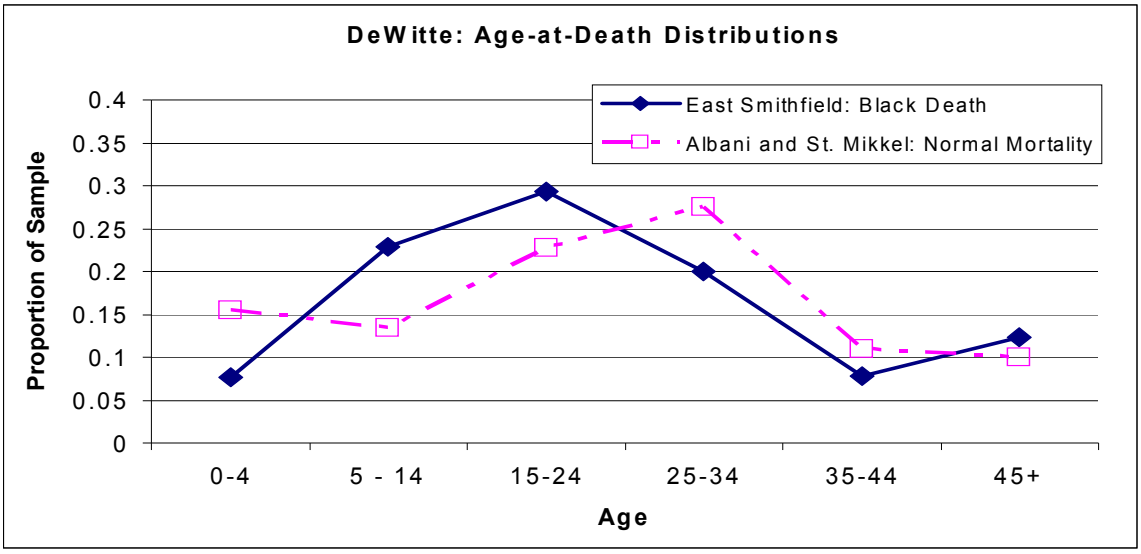

Figure 3-3: Comparison of age-at-death distributions using individual ages estimated in the current study for the Danish normal mortality cemetery and the East Smithfield cemetery.

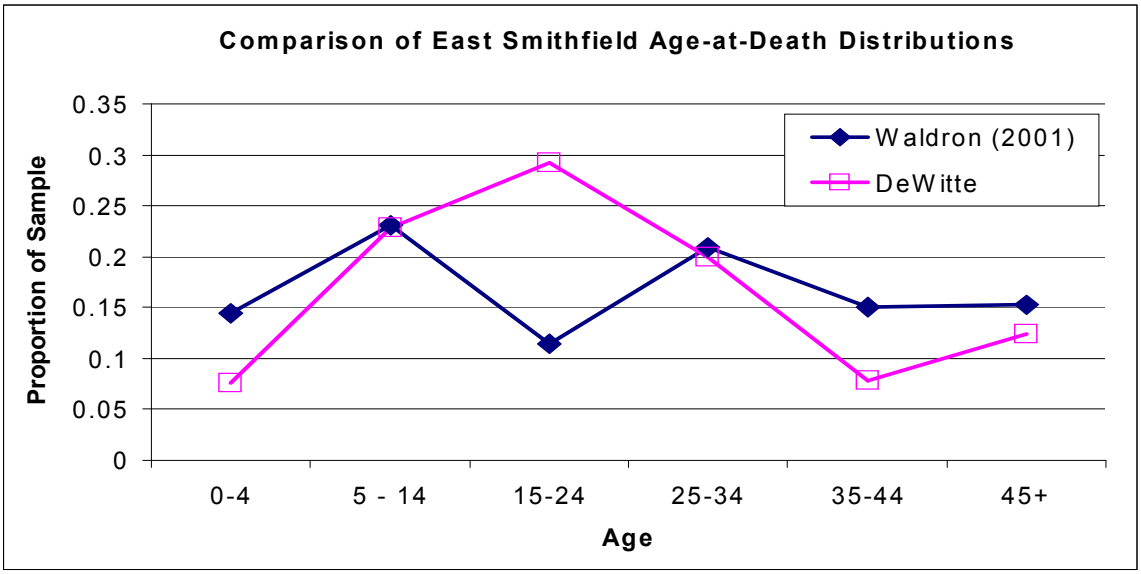

Figure 3-4: Comparison of Waldron's (2001) and DeWitte's age-at-death distributions from the East Smithfield Black Death cemetery

Age Pattern of Mortality: Siler Model

To understand the age pattern of Black Death mortality, I estimated the parameters of the Siler model within the East Smithfield and Danish samples using *mle*. Because I examined different skeletal age indicators in children and adults, I could not estimate Siler parameters for *all ages* directly from the observed frequencies of age indicators using the Rostock Protocol. Instead, I used transition analysis to obtain point estimates for adults, and for point estimates of juvenile age, I used the midpoint of the age range provided by epiphyseal closure and dental development. I then combined all these point estimates to estimate the parameters of the Siler model. By using point estimates for all adults and juveniles, I ommitted the error associated with the adult age estimates.

The Siler parameter estimates for East Smithfield and Denmark are shown in Tables **3-1** and **3-2.** For the East Smithfield cemetery, the β_1 estimate is significantly different from zero ($p < 0.001$); none of the other estimates from either cemetery are significantly different from zero. To determine whether the fitted age-at-death distributions were significantly different, I first treated the two samples as a single cemetery and estimated the parameters of the Siler model using the age estimates for all individuals from both East Smithfield and Denmark. I then computed a likelihood ratio test using the log likelihoods provided by *mle* for the two cemetery samples (when treated separately) and the combined East Smithfield and Danish sample:

$$LLR = -2\{\log(L_{combined}) - [\log(L_{East\ Smithfield}) + \log(L_{Denmark})]\} \quad \textbf{3.1}$$

which has a chi-square distribution with two degrees of freedom. The result of this test indicates that the East Smithfield and Danish functions are *not* significantly different.

These results are quite different from the comparison of the distributions based on individual age estimates described above. That is, the fitted age-at-death distributions from the two cemetery samples are not significantly different, but the distributions based on individual age estimates are significantly different. The reason for this discrepancy is that the distribution using individual ages omits the error associated with the individual estimates, but the fitted age-at-death distribution accounts for that error. When the error associated with paleodemographic age estimation is included, the differences apparent in the individual-age comparison are blurred or even obliterated by that error. Traditional methods of paleodemographic age estimation provide no information about the error distribution of individual age estimates. The results obtained using such methods, therefore, appear "neater", but only *because* they omit what might be a considerable amount of error. One of the strength of the Rostock protocol is that it provides the error associated with age estimates, so we can actually see how accurate (or inaccurate) each particular estimate is. Ideally, we would only include in our analyses those age estimates with the least amount of associated error, but such an approach is not feasible given the already small sample sizes typical of paleodemographic studies. We must therefore be comfortable with the inevitable error associated with age estimation. To learn anything

substantive from skeletal samples, we should not ignore that error, but rather make it as explicit as possible.

Table 3-1: Siler parameter estimates for East Smithfield.

Parameter	Estimate	Standard Error
α_1	0.00	0.02
β_1	1.2	0.00
α_2	0.00	0.1
α_3	0.03	0.1
β_3	0.02	0.03

Table 3-2: Siler parameter estimates for Denmark

Parameter	Estimate	Standard Error
α_1	0.005	0.02
β_1	0.3	1.7
α_2	0.00	0.1
α_3	0.03	0.09
β_3	0.02	0.04

Figure 3-5 shows the graph of the hazard function for each sample. The results of this analysis suggest that age did have an effect on risk of death during the Black Death, such that older individuals were at an increased risk of dying during the epidemic. In both the East Smithfield and Danish cemeteries, the relative risk increases steadily, suggesting that elderly individuals were at much higher risk of dying than younger individuals under both normal mortality conditions and during the Black Death. The graph of the function for Denmark is higher than that for East Smithfield with respect to ages thirty and above, and the difference between the two functions increases with increasing age. This suggests that during the Black Death, just as in normal mortality situations, older individuals were at an increased risk of death compared to adolescents and young adults, but their relative risk of death was not as high as it was during times of normal mortality.

To further explore the age pattern of Black Death mortality, I used the Siler parameter estimates from Denmark to generate a survival function to show the living age distribution in a normal population; by assuming that the East Smithfield and Danish populations were similar before the Black Death, this survival function can be viewed as representing the population at risk in East Smithfield. I then generated an age-at-death distribution using the East Smithfield Siler parameter estimates. I divided the East Smithfield age-at-death distribution by the "living age distribution" for ages zero to eighty years, as shown in **Figure 3-6**. These results also show an apparent increase in the risk of death with age during the Black Death. These results are not conclusive, however, given that I omitted the error associated with adult ages at death.

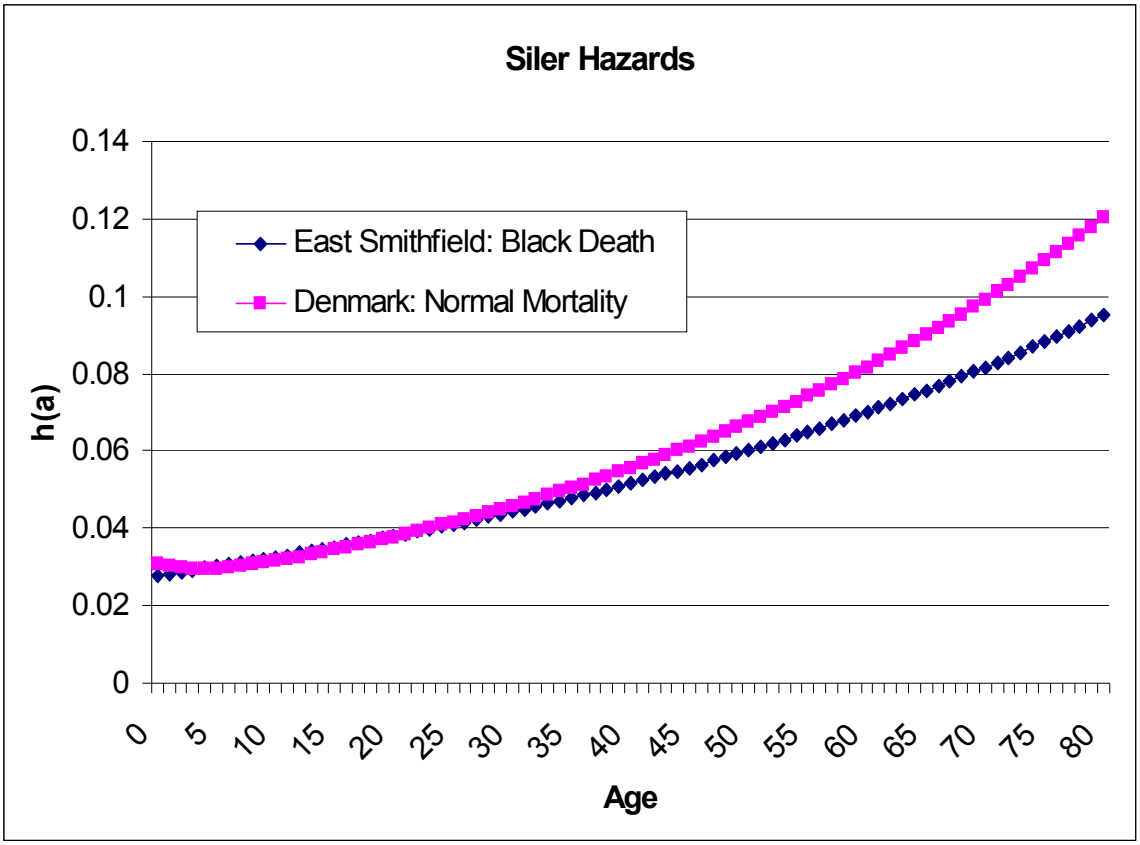

Figure 3-5: Siler hazard functions.

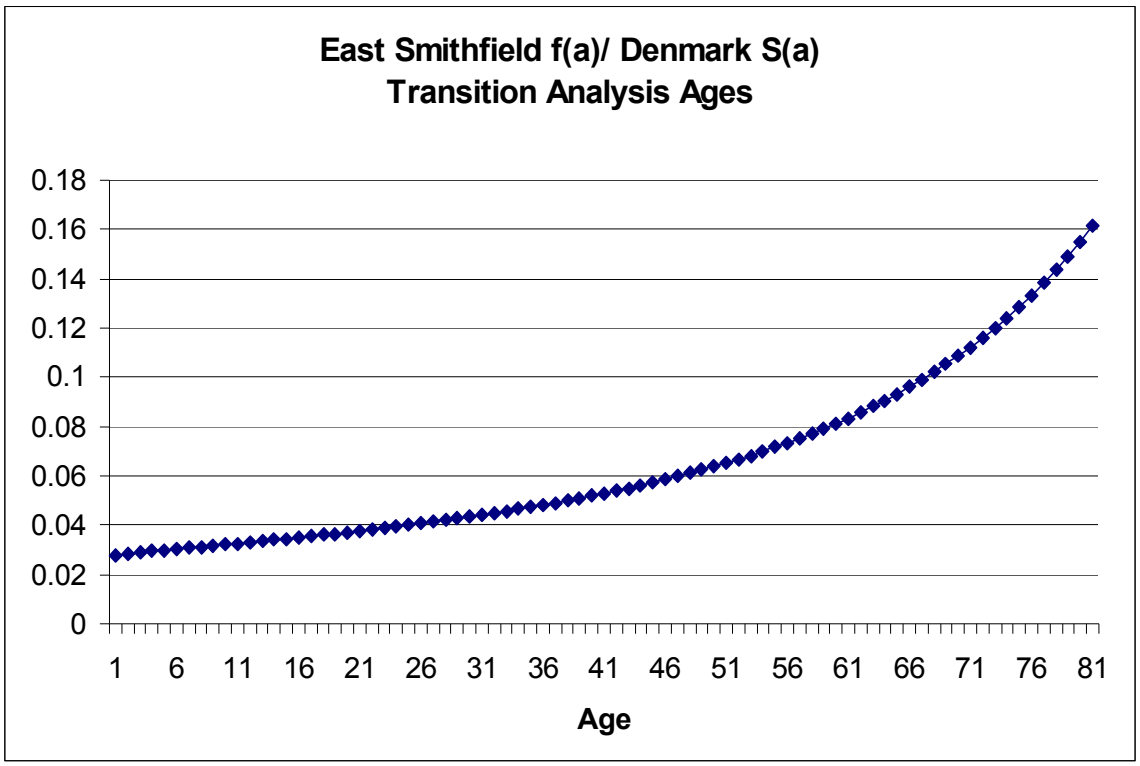

Figure **3-6**: Ratio of the East Smithfield age-at-death distribution *f*(a) and the Danish survival function *S*(a) by age.

Surprisingly, the graph of the Siler hazards suggests that infants and young children below the age of five years were not at a higher risk of death during the epidemic compared to older age groups, whereas children between age zero and five in Denmark were at a slightly higher risk than were older children and young adults. However, the relative risk of death for very young children in the Danish sample is not as high as one might expect, given what is known about high rates of infant mortality in pre-industrial populations. The relative risk for young children in both samples might be underestimated given the relatively poor preservation of children that typically occurs in

cemetery samples. I return to the potential issue of preservation bias at the end of this chapter. To eliminate the potential problem of preservation bias for children, I estimated the Siler model for individuals above the age of four years. The shows the graphs of the Siler hazards for ages five and above are shown in **Figure 3-7**; this analysis yields results similar to those obtained using all ages – i.e. an apparent increase in risk of death with age in both cemetery samples.

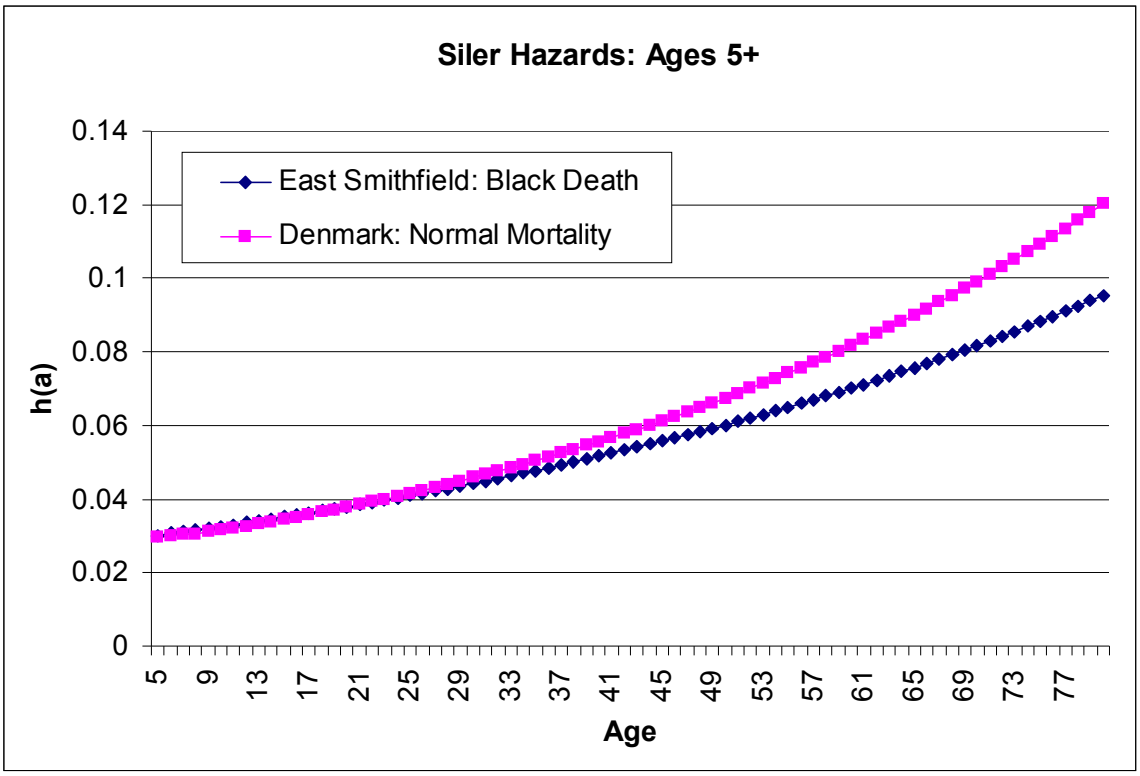

Figure **3-7**: Siler hazard functions for ages five years and above.

The results presented here are consistent with those obtained from historical documents by Razi (1980), Russell (1948), and Wood et al. (2002) who found that the

elderly were disproportionately affected during the epidemic. However, given the fact that only *one* of the Siler parameter estimates (the the β_1 estimate for East Smithfield) was significantly different from zero, and given that I omitted the error associated with adult estimates of age to estimate the Siler parameters, the results are unconvincing. They do not provide strong evidence of selectivity of the Black Death with respect to age, nor do they refute the possibility of such selectivity. Further work is necessary to determine if the age pattern suggested here is real or if it is an artifact of the omission of error associated with age estimates.

Level of Black Death Excess Mortality

To estimate the level of excess mortality associated with the Black Death, I combined the East Smithfield and Danish samples and treated them as a single cemetery ($n = 781$) sharing the same baseline mortality. This is a reasonable approach as in this analysis, I was concerned with the value of the k_{2ES} parameter and not with the exact values of the parameters of the baseline mortality model itself. I considered dying during the Black Death as a risk factor in this application of the Usher model and therefore designated only those individuals from East Smithfield as having the risk factor. In this analysis, the value of k_{2ES} indicates the excess mortality produced by the Black Death relative to normal mortality.

The value of k_{2ES} is 1.04 ($p \leq 0.001$), an estimate that quite surprisingly suggests that mortality increased very little, it at all, during the Black Death compared to pre-

Black Death, Danish mortality conditions. This estimate cannot be believed, given estimates of Black Death mortality from historical documents.

To test the effect of setting k_1 equal to one for these analyses, I conducted sensitivity analyses by running the analyses with varying set values of k_1. For values of k_1 ranging from 0.5 to 9, the estimate of k_{2ES} was very similar to if not identical that reported above, ranging from 0.99 to 1.11. However, setting the value of k_1 close to 0 yielded estimates of k_{2es} much higher than 1.04; for example, setting the value of k_1 to 0.01 yielded a k_{2es} of 9.9. The estimate of k_{2ES} is apparently sensitive to values of k_1 close to zero, but less so to values of k_1 greater than 0.5.

Selectivity With Respect to Frailty

Comparison of Lesion Frequencies

In a previous study of the selectivity of the Black Death, Waldron (2001) simply compared the frequencies of skeletal lesions in East Smithfield to those found in a normal mortality cemetery; he found that lesion frequencies were similar in the two cemeteries, suggesting that the Black Death did not kill indiscriminately. *For the sake of comparison* with Waldron's study, I present a simple comparison of the lesion frequencies in East Smithfield and Danish samples in addition to calculating the relative risk of death associated with lesions using the Usher model. A simple comparison of lesion frequencies is *not* the best approach, as it assumes that lesion frequencies were very

similar in the once-living populations and/or that lesions are associated with the same relative risk in the once-living populations about lesions. These assumptions might not be true, and by using the Usher model to estimate the relative risk of death associated with various skeletal lesions, I did not need to make them.

Because of the varying sample sizes for skeletal lesions within each sample, in order to informatively compare the two cemeteries, I did a direct standardization of the lesion frequencies by age using the Danish samples sizes as the standard population. I first estimated age-specific lesion frequencies within East Smithfield for each skeletal lesions (I used five-year age intervals). I then multiplied these age-specific lesion frequencies by the number of individuals actually within each age interval in the Danish sample; these results represent the number of individuals in the Danish sample within each age group that would be "expected" to have lesions if the lesion frequencies within East Smithfield and Denmark were the same. I then compared the number of individuals in Denmark expected to have lesions to the number within each age group that were actually observed to have lesions.

The sample sizes for each lesion within East Smithfield and Denmark are shown in Table **3-6** at the end of this chapter. Table **3-7** presents the lesion frequencies within both samples; the East Smithfield frequencies are standardized on the Danish sample sizes.

I found significantly higher frequencies (at $\alpha = 0.05$) of proliferative tibial lesions, porotic hyperostosis on the occipital bone, and enamel hypoplasia on the incisor, maxillary canine, and molars in Denmark compared to East Smithfield. The frequency of cribra orbitalia was borderline significantly higher in Denmark than in East Smithfield (α

= 0.09). However, the frequencies of porotic hyperostosis on the left and right parietal bones were significantly *lower* in Denmark compared to East Smithfield. There were no significant differences between the two samples with respect to the frequencies of the remaining lesions.

The majority of the lesions were at a higher frequency in Denmark compared to East Smithfield; however, this result tells me nothing about the selectivity of the Black Death unless I can determine how those lesions are related to risk of death within each cemetery. Fortunately, the Usher model allows me to determine the relationship between lesions and risk of death.

Application of the Usher Model

One problem with the simple comparison of lesion frequencies is that it assumes that the skeletal lesions under consideration had the same effect (or lack thereof) on risk of mortality in England and Denmark. Although this might be the correct assumption, I did not have to make it when I applied the Usher model. The Usher model actually allowed me to determine the risk of mortality associated with each lesion within each sample. The approach I took is therefore more informative than that used in previous studies of East Smithfield.

To determine whether the Black Death was selective with respect to frailty, I applied the Usher model separately to the East Smithfield and Danish samples and estimated the excess mortality associated with several skeletal lesions or stress markers within each sample. If the Black Death killed people indiscriminately, regardless of

frailty, I expected to find that lesions associated with an increased relative risk of death in the Danish cemeteries would not be associated with an increased risk in East Smithfield.

By applying the Usher model to the lesion data, I estimated for each sub-sample of individuals with lesions within East Smithfield and Denmark the five parameters of the Siler model, the risk of making the transition from State 1 to State 2 (k_1) and the excess mortality associated with the lesions (k_2). In this chapter, for clarity, I present only the estimates of the risk of death associated with each lesion, as the other parameter estimates are irrelevant to the current analysis of selectivity with respect to frailty. However, I do provide a complete report of all parameter estimates for each lesion sample in Appendix D. I ran these analyses several times using various start values for the parameters of the Usher model; regardless of the start values, I always obtained the same pattern of results (importantly, the k_2 values were always higher in Denmark than in East Smithfield).

Estimates of the excess mortality k_2 associated with lesions within East Smithfield and Denmark are shown in Table **3-3**; only those results that were statistically significant ($p < 0.001$) are shown. To illustrate the differences between the two cemetery samples, the effects that porotic hyperostosis had on the baseline risk of death within each cemetery are shown in **Figures 3-8 – 3-9**

The presence of all lesions that yielded significant results is associated with an increased risk of death in both cemeteries, but, without exception, the risks are higher in Denmark than in East Smithfield. Further, the majority of lesions which failed to yield significant results revealed this same pattern (see Appendix D).

Table 3-3: Estimates of the excess mortality k_2 associated with osteological lesions and stress markers within East Smithfield and Denmark (only lesions with stastically significant results for k_2 are shown.

	East Smithfield		Denmark	
	k_2	Standard Error	k_2	Standard Error
Tibial Lesions	4.6	1.3	10	4.2
Porotic Hyperostosis: Frontal	1.5	0.4	3.2	1.7
Porotic Hyperostosis: Left Parietal	4.6	2.0	8.6	3.8
Porotic Hyperostosis: Right Parietal	4.2	1.7	9.0	4.1
Porotic Hyperostosis: Occipital	4.4	2.0	5.0	2.2
Cribra Orbitalia	3.6	1.8	4.3	2.3

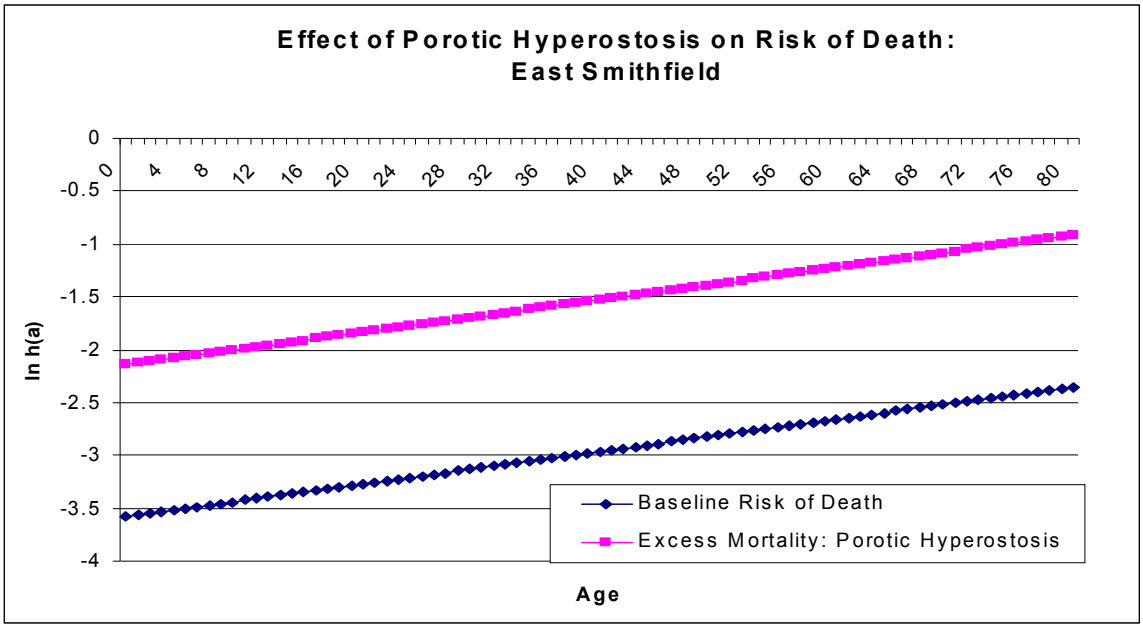

Figure 3-8: Excess mortality associated with porotic hyperostosis on the right parietal bone in East Smithfield.

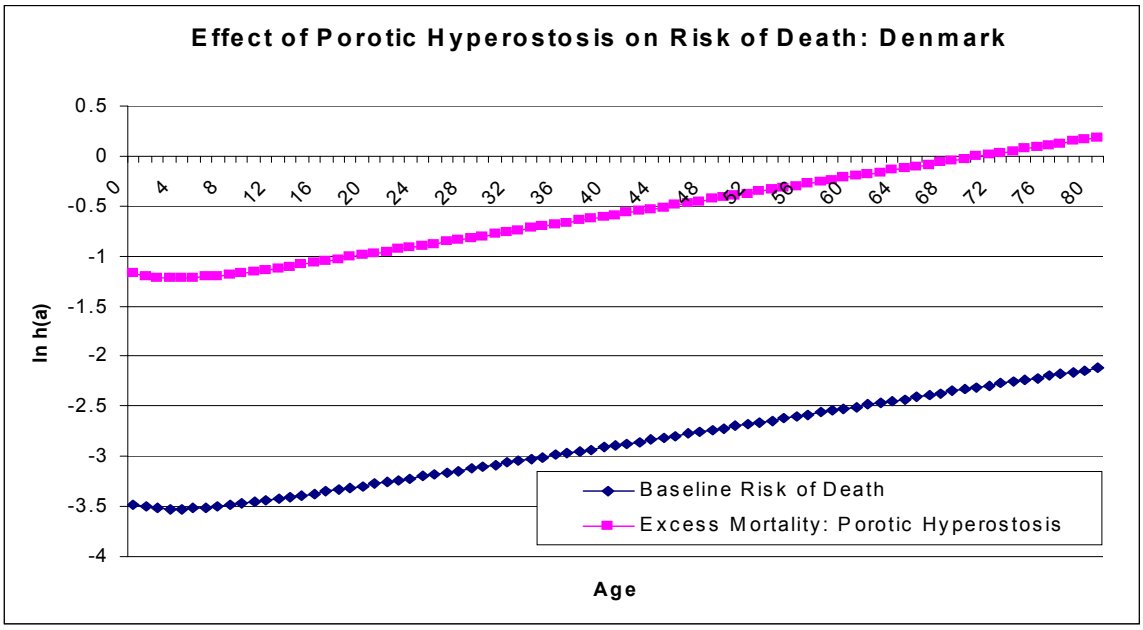

Figure 3-9: Excess mortality associated with porotic hyperostosis on the right parietal bone in Denmark.

Differential Risk Associated with Sex

To determine if there was differential risk of death associated with sex during the Black Death, I applied the Usher model separately to the adults within the East Smithfield and Danish samples. I arbitrarily designated all the adult females in each sample as not having a risk factor, and all the adult males as having a risk factor. The value of k_{2m} indicates the risk of death associated with sex: if k_{2m} is equal to one, men and women were at equal risk of dying; if k_{2m} is greater than one, females were at an increased risk of dying compared to males; and if k_{2m} is less than one, females were an a reduced risk of dying compared to males.

Table 3-4 summarizes the number of adults in the East Smithfield and Danish cemeteries and the proportion of each sample that is female. In both cemeteries, there are more males than females.

Table 3-4: Number of adults and frequency of females.

	Number of Adults	**Frequency of Females**
East Smithfield	298	0.42
Denmark	195	0.44

The estimates of the risk of death for males in each cemetery are shown in **Table 3-5**; both estimates of k_{2m} are statistically significant ($p < 0.001$). In East Smithfield

males and females were at the same risk of dying; in Denmark, males were at a slightly elevated risk of death compared to females; however, the relatively high standard error renders these results unconvincing. These results reveal that during the Black Death and under normal mortality conditions, males and females were at approximately the same risk of dying. The proportions of males and females within each cemetery might suggest that males were actually at a higher risk of death than were females, as there are more males than females in both samples; however, the estimates of the relative risk of death tell a different story and reveal that, there was very little, in any, difference in risk of death for males and females during the Black Death *and* during times of normal mortality.

Table 3-5: Excess mortality for adult males

	k_{2m}	Standard Error
East Smithfield	1.0	0.1
Denmark	1.2	0.2

As described in Chapter 2, for this application of the Usher model, I set the value of k_1 equal to one. To test the effect of setting k_1 equal to one, I conducted sensitivity analyses by running the analyses with varying values of k_1. Values of k_1 ranging from 0.5 to 9.0 yielded estimates of k_{2m} very similar, if not identical to those shown in Table 3-5.

For East Smithfield, the estimates of k_{2m} ranged from 0.97 to 1.0, depending on the set value of k_1. For Denmark, the estimates of k_{2m} ranged from 1.03 to 1.18, depending on the set value of k_1. Values of k_1 below 0.1 yielded estimates of k_{2m} that were higher than those shown in Table 3-5; for example, setting the k_1 value to 0.01 yielded estimates of k_{2m} equal to 8.5 and 7.31 for East Smithfield and Denmark, respectively. As with estimates of the excess mortality of the Black Death, estimates of the differential risk associated with males are apparently sensitive to values of k_1 close to zero, but less so to values of k_1 greater than 0.5.

Preservation

Because differential preservation within and between cemetery samples might complicate interpretation of mortality patterns from cemetery samples, I compared the general levels of preservation within the East Smithfield and Danish samples. I compared preservation with respect to broad age intervals. The results of this comparison, separated by skeletal region (head, torso, arms, and legs), are shown in Figures **3-10, 3-11, 3-12,** and **3-13.** In general, preservation for individuals between the ages of zero and fifteen years appears better in the Danish cemeteries than in the East Smithfield cemetery. In particular, compared to the Danish samples, a larger proportion of individuals in the East Smithfield sample below the age of fifteen is missing relevant bones (i.e. scores of "missing") and a smaller proportion is scored has "good" preservation. Individuals above the age of fifteen are, in general, equally well preserved in the East Smithfield and Danish cemeteries. Ideally, all individuals in the samples

would have good preservation, but as can be seen from the figures, that ideal is not realized in this study. In the interests of extracting as much information as possible from the available skeletal material, I included many individuals who were poorly preserved and were therefore missing many of the relevant data.

Because the adults in the East Smithfield and Danish samples appear equally well-preserved, any differences in the estimated mortality patterns for adults are probably not the result of differences in preservation between the two samples. However, the relatively poor preservation of juveniles in the East Smithfield cemetery might be obscuring the pattern of juvenile mortality during the Black Death. As discussed above, the Siler estimates suggest that children between the ages of zero and five years were *not* at an elevated risk of dying during the Black Death; this result is surprising, given the vulnerability of young children under normal mortality conditions. I strongly suspect that the lack of evidence for an increased risk of death for children in East Smithfield is an artifact of poor preservation, rather than an accurate reflection of mortality patterns during the Black Death.

Chapter Summary

- Siler model parameter estimates indicate that older were at a disproportionate risk during the Black Death. *This conclusion, however, is tentative* given that I omitted the error associated with adult ages for the analysis.

- Individuals who were already in poor health before the Black Death were more likely to die than people who were healthy before the epidemic
- The risk of death for frail individuals compared to healthy individuals was not as high during the Black Death as it was during times of normal, non-epidemic mortality.
- Women and men faced a similar risk of death during the Black Death

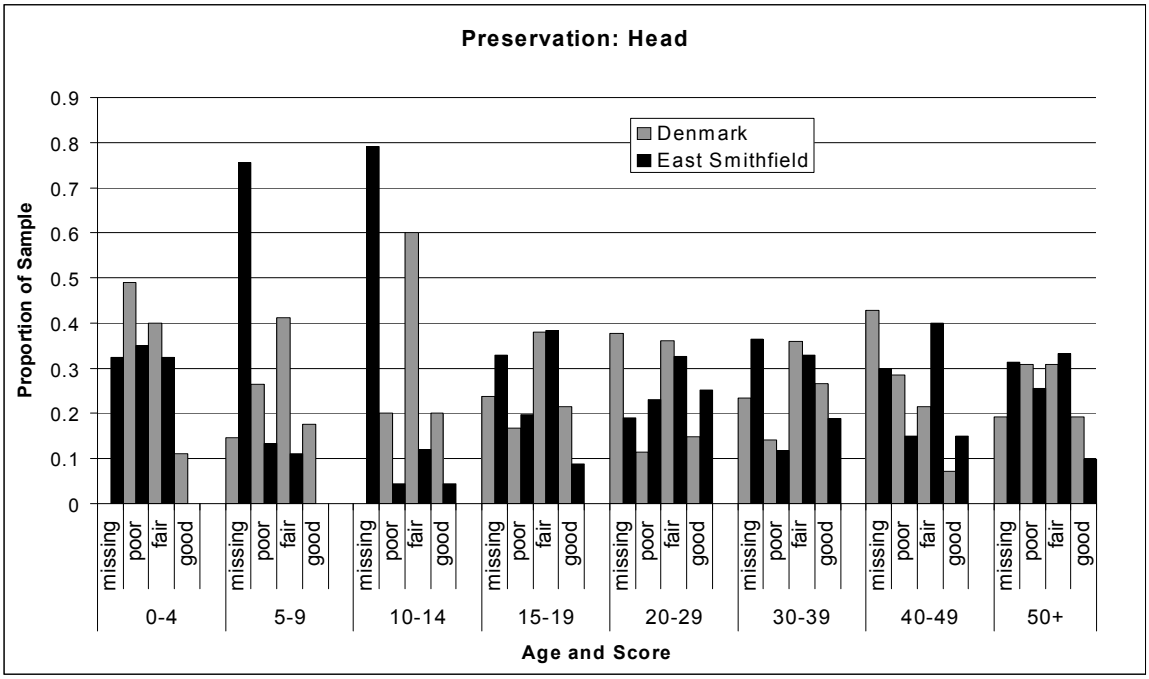

Figure 3-10: Preservation of the head

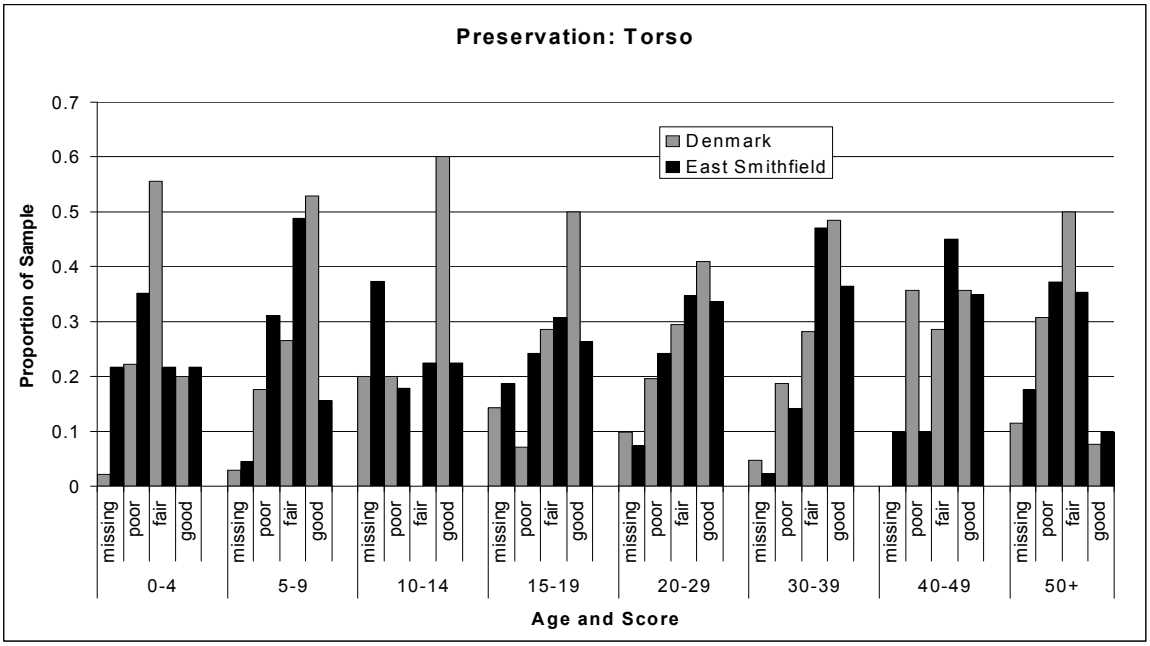

Figure 3-11: Preservation of the torso.

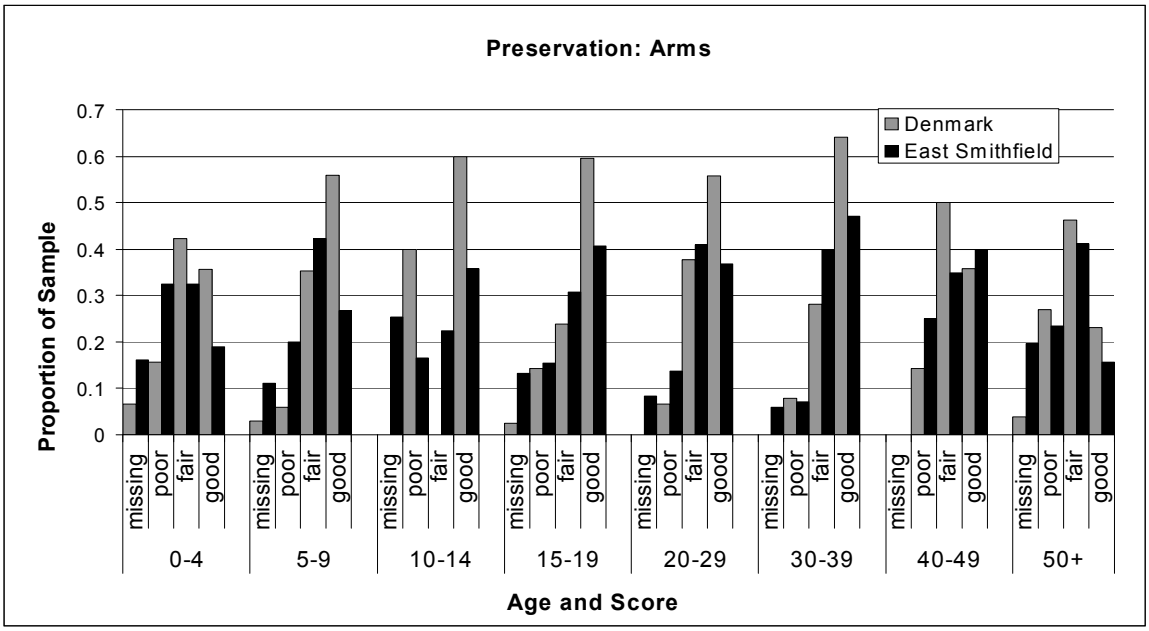

Figure 3-12: Preservation of arms.

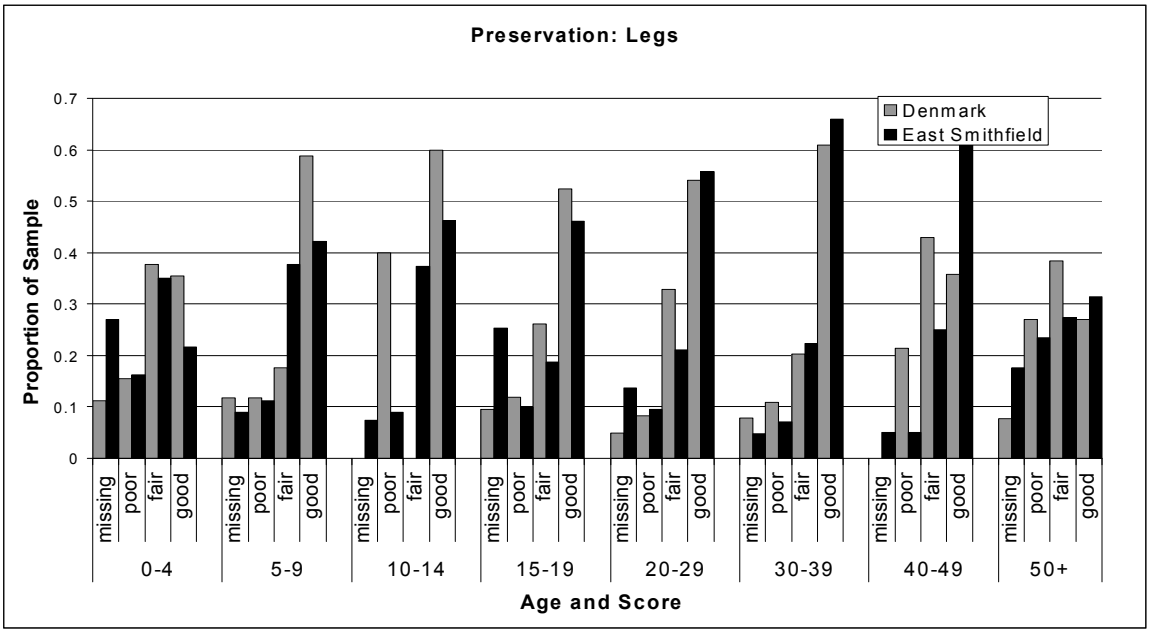

Figure 3-13: Preservation of legs.

Table 3-6: Sample sizes for each osteological lesion or stress marker.

	East Smithfield	Denmark
Tibial Lesions	254	187
Porotic Hyperostosis: Frontal	135	112
Porotic Hyperostosis: Left Parietal	166	125
Porotic Hyperostosis: Right Parietal	163	129
Porotic Hyperostosis: Occipital	159	132
Cribra Orbitalia	143	89
Enamel Hypoplasia: Incisor	85	48
Enamel Hypoplasia: Mandibular Canine	129	54
Enamel Hypoplasia: Maxillary Canine	125	42
Enamel Hypoplasia: M1	109	63
Enamel Hypoplasia: M2	147	44
Femur Length	154	121

Table 3-7: Age-specific lesion frequencies. The East Smithfield (ES) frequencies are standardized on the Denmark (DK) sample sizes.

	Tibial Lesions		Porotic Hyperostosis: Frontal		Porotic Hyperostosis: Left Parietal	
Age	ES	DK	ES	DK	ES	DK
0 - 4.99	22.79	26	0.00	6	5.67	9
5 - 9.99	18.00	20	0.00	1	5.33	2
10 - 14.99	2.95	4	0.00	0	1.50	2
15 - 19.99	9.04	14	2.36	1	10.91	10
20 - 24.99	8.00	12	2.00	2	9.47	9
25 - 29.99	3.14	7	7.30	7	11.41	11
30 - 34.99	10.48	17	7.13	5	14.55	14
35 - 39.99	13.21	25	5.83	10	16.82	14
40 - 44.99	9.00	7	4.00	3	14.00	8
total	96.62	132	28.62	35	89.66	79

	Porotic Hyperostosis: Right Parietal		Porotic Hyperostosis: Occipital		Cribra Orbitalia	
Age	ES	DK	ES	DK	ES	DK
0 - 4.99	8.67	9	3.33	10	5.33	15
5 - 9.99	4.00	3	0.00	2	10.67	12
10 - 14.99	1.29	1	1.50	2	2.00	1
15 - 19.99	12.36	14	10.35	11	2.73	5
20 - 24.99	9.94	8	5.71	5	2.06	1
25 - 29.99	13.13	11	9.75	10	1.13	3
30 - 34.99	16.55	15	13.06	13	2.53	2
35 - 39.99	31.58	26	18.00	15	2.33	1
40 - 44.99			0.00	8	4.50	1
total	97.51	87	61.71	76	33.27	41

Table 3-7 (continued)

	Enamel Hypoplasia: Incisor		Enamel Hypoplasia: Mand. Canine		Enamel Hypoplasia: Max. Canine	
Age	**ES**	**DK**	**ES**	**DK**	**ES**	**DK**
0 - 4.99			2.00	1	1.00	0
5 - 9.99	9.50	9	11.00	9	0.00	6
10 - 14.99	1.00	4	3.08	5	3.00	3
15 - 19.99	5.95	13	14.38	17	8.91	12
20 - 24.99	1.50	1	3.71	3	2.93	3
25 - 29.99	0.60	1	4.05	5	2.80	4
30 - 34.99	0.78	1	2.83	3	2.58	2
35 - 39.99	0.33	0	2.00	3		
40 - 44.99			2.00	2	2.00	2
total	19.67	29	45.05	48	23.22	32

	Enamel Hypoplasia: M1		Enamel Hypoplasia: M2		Femur Length	
Age	**ES**	**DK**	**ES**	**DK**	**ES**	**DK**
0 - 4.99	0.00	5				
5 - 9.99	4.89	4	2.00	1		
10 - 14.99	1.11	1	1.11	1		
15 - 19.99	5.00	8	3.71	8		
20 - 24.99	0.57	2	2.00	4	4.75	1
25 - 29.99	0.94	2	1.92	3	1.17	5
30 - 34.99	0.85	1	1.25	2	3.93	3
35 - 39.99	0.20	0	0.17	0	6.41	6
40 - 44.99					6.50	2
total	13.56	23	12.16	19	22.76	17

Chapter 4

DISCUSSION

The results of the analyses of the age and sex pattern of the Black Death and the selectivity of the medieval disease with respect to frailty reveal that the Black Death was, in fact, selective and did *not* kill people indiscriminately. People with certain skeletal lesions were at an increased risk of death compared to their peers during the Black Death. However, the Black Death was apparently not as strongly selective as normal mortality, as the excess mortality for individuals with lesions was not as high as it would have been during times of normal, pre-epidemic mortality.

Age Pattern of Mortality

The Siler parameter estimates from the East Smithfield and Danish cemeteries (as shown in Tables 3-3 and 3-4 and Figure 3-5) suggest that age had an effect on risk of death within both populations. However, as mentioned in Chapter 3, these results are not convincing, given that I omitted the error associated with adult ages-at-death in the analysis. Any conclusions about the age pattern of Black Death mortality must be viewed as tentative.

The Siler estimates suggest that during the Black Death and under normal mortality conditions, older individuals, above the age of 30 or so, were at an increased risk of death compared to younger individuals. These results are similar to those found by Russell (1948), Razi (1980), and Wood et al. (2002), all of whom found, using data from historical records (described in Chapter 1), that the Black Death differentially affected elderly individuals.

The Siler parameter estimates also show an increased risk of death for very young children in the normal mortality sample, but not in the Black Death cemetery. In Denmark, children below the age of five years were at a higher differential risk of death compared to older children, adolescents, and young adults (but not elderly individuals). The differential risk of death, for infants and young children, as shown in Figure 3-5, is perhaps not as high as one might expect; this seemingly low estimate of the level of juvenile mortality in Denmark might be an artifact of the generally poor preservation of infant skeletal remains typical of most paleodemographic cemetery samples. The Siler estimates suggest that in East Smithfield, children under the age of five years were not at an increased risk of death compared to other age groups.

There are two possible interpretations of these results:

- Infants and young children truly were not at an increased risk of death compared to older individuals during the Black Death. The Black Death might have been so virulent that individuals between the ages of zero and thirty were equally at risk (i.e. older children, adolescents, and young adults were no less vulnerable to the disease than were infants).

- The second possible interpretation of these data is that during the Black Death children under the age of five really were at a higher risk of death than older individuals, just as they were under normal mortality conditions in Denmark, but the relatively poor preservation of young children in East Smithfield has obscured that pattern.

With respect to the second possibility, as discussed in Chapter 3, individuals above the age of fifteen in both East Smithfield and Denmark were equally well preserved; however, young children and adolescents between the ages of zero and fifteen were less well preserved in East Smithfield than they were in the Danish cemeteries. For example, in Denmark, of the children between the ages of 0 and 4.99 years included in the sample, none were missing their skulls, but only about eleven percent had skulls with "good" preservation (see Figure 3-21); in East Smithfield, about 32 percent of the children in the same age group were missing their skulls and none had skulls with "good" preservation. The bones of young children are small, soft, and less mineralized than those of adults, so they are much more likely to disintegrate and thus less likely to be preserved in a cemetery sample for hundreds of years than are adult bones. Many more young children might have originally been interred in both East Smithfield and Denmark than were eventually excavated. This differential preservation would clearly influence observable patterns of juvenile mortality, potentially resulting both in an underestimate of mortality among children, as might be the case in Denmark, or a failure to detect an increased risk at all for children, as might be the case in East Smithfield.

If the pattern of increasing risk of death with advanced age that was observed in both East Smithfield and Denmark is real, then the age pattern for young children in East Smithfield was probably obscured by preservation bias. If the Black Death increased the risk of death for all individuals in a population, while also disproportionately affecting elderly people, why would it not also increase the risk of death for very young children compared to other age groups?

Level of Black Death Excess Mortality

To estimate the excess mortality of the Black Death, I treated the East Smithfield and Danish cemeteries as a single cemetery sharing the same baseline mortality rates; I considered individuals within East Smithfield as a risk factor (dying during the Black Death) and those within the Danish cemeteries as not having the risk factor (dying before the Black Death). By treating the East Smithfield and Danish cemeteries as a single cemetery and determining the value of k_{2es} associated with victims of the Black Death compared to victims of normal mortality, I obtained an estimate of Black Death excess mortality. A value of k_{2ES} equal to one would suggest that the Black Death did not increase mortality significantly (which would have been astonishing given estimates of mortality from historical records); a value of k_{2ES} greater than one would suggest that the Black Death did increase mortality, and the higher the value of k_{2ES}, the greater the excess mortality associated with the epidemic.

The result of this analysis, a k_{2ES} estimate of 1.04, suggests that mortality was increased very little, it at all, during the Black Death compared to mortality under normal,

pre-epidemic conditions. This estimate of excess mortality is unbelievably low, especially in light of estimates of Black Death mortality from historical documents. For example, Wood et al. (2002) estimated annual mortality rates for beneficed priests from bishop's registers and found that they were approximately eleven times higher during the Black Death than in the period immediately before the epidemic.

To test the effect of setting k_1 equal to one for these analyses, I conducted sensitivity analyses by running the analyses with varying set values of k_1. These analyses revealed that estimates of the excess mortality of the Black Death are *not* strogly affected by setting the value of k_1 between 0.5 and 9. However, setting the value of k_1 close to 0 yielded estimates of k_{2es} much higher than 1.04. The estimate of k_{2ES} is apparently sensitive to values of k_1 close to zero, but less so to values of k_1 greater than 0.5.

The k_{2ES} estimate of 1.04 greatly underestimates the true level of excess mortality associated with the Black Death. One possible reason for this underestimation is that I compared victims of the Black Death who accumulated within the East Smithfield cemetery within just a few months to individuals who accumulated over many decades in normal-mortality Danish cemeteries. Because age was estimated in years, the Siler estimates from the Danish cemeteries are essentially annual mortality rates. However, the same is not true for the East Smithfield cemetery given that the epidemic likely lasted only a few months in any given neighborhood in London.

Previous analyses of the time-course of the Black Death have shown that the disease swept through towns and neighborhoods very quickly, lasting only a few months in any one area. For example, Wood et al. (2003) found that in archdeaconries in

Conventry and Lichfield in England, the Black Death only lasted about four to six months; archdeaconries are aggregates of local communities, so the epidemic likely lasted less than six months at lower levels of aggregation. If the Black Death lasted only about four months in any given neighborhood in London (which contributed victims to the East Smithfield cemetery), the mortality rates from East Smithfield should be multiplied by three to make them comparable to the annual rates from Denmark; thus, the estimated value of k_{2ES} should also be multiplied by three, given that k_2 is estimated based on a comparison of East Smithfield and Danish mortality rates. If the Black Death lasted only four months in any given neighborhood of London, the value of k_{2ES} would be 3.12. If the epidemic lasted less than four months in a neighborhood, the estimate of k_{2ES} would be even higher. Unfortunately, we do not know from historical documents exactly how long the Black Death lasted within each of the catchment areas of the East Smithfield cemetery, so I cannot know with certainty the factor by which I should correct the above estimate of excess mortality associated with the epidemic.

Another possible reason for the apparent underestimation of Black Death excess mortality is the potential inclusion of victims of the Black Death in the Danish cemeteries or of non-Black Death victims in East Smithfield. The application of the Usher model to estimate excess mortality associated with the Black Death assumes that East Smithfield includes only Black Death victims and that the Danish cemeteries contain only individuals who died before the Black Death. Deaths from the Black Death were known to have swamped normal mortality (Wood et al., 2003), but it is possible that there are some individuals in the East Smithfield cemetery who died from causes other than the Black Death. Such individuals are likely to be a small minority; nevertheless, their

inclusion will affect estimates of Black Death excess mortality. To obtain a mostly pre-Black Death comparison sample, I chose individuals in the Danish cemeteries with arm positions A and B, which were used predominately before 1350. However, Kieffer-Olsen (1993) and Jantzen et al. (1994) have shown that a small proportion of individuals who died after 1350 were buried with arm position B. It is therefore possible that my Danish sample includes an indeterminable number of Black Death victims, and that the Danish sample is not perfectly representative of pre-Black Death normal mortality conditions. If either or both of the above assumptions are not correct, the differences in mortality patterns between the East Smithfield and Danish cemetery samples will be smaller than expected. It is also possible that the value of k_{2ES} is so small because normal mortality in Denmark was even higher than normal mortality in East Smithfield.

Selectivity with Respect to Frailty

Previous researchers have assumed that *if* the Black Death killed people indiscriminately, lesion frequencies in the East Smithfield cemetery should be similar to those in the original once-living population and thus significantly different from lesion frequencies in normal mortality cemeteries. If the Black Death was not selective, and skeletal lesions normally are associated with an increased risk of death (i.e. the presence of lesions indicates high frailty), one would expect to find a higher frequency of skeletal lesions in the normal mortality cemetery, as normal mortality is expected to be selective with respect to frailty.

Waldron (2001) addressed the question of Black Death selectivity by comparing lesions frequencies in the East Smithfield cemetery to those in the overlying normal mortality cemetery of St. Mary Graces. He found a higher frequency of degenerative lesions such as osteoarthritis in East Smithfield compared to the normal mortality cemetery, but East Smithfield had a lower frequency of dental, traumatic, and infectious lesions; none of these differences was statistically significant. Waldron concluded that lesion frequencies in the two cemeteries were similar, which suggests that the Black Death did not kill indiscriminately.

For the sake of comparison with Waldron's study, I conducted a comparison of lesion frequencies in East Smithfield and Denmark; because the East Smithfield and Danish sample sizes differed, in order to compare the lesion frequencies in the two samples, I standardized the East Smithfield frequencies by age using the Danish sample sizes as the standard population (as described in Chapter 3). A comparison of the standardized lesions frequencies, reveals that there were significantly higher frequencies (at $\alpha = 0.05$) of the following lesions in Denmark compared to East Smithfield: proliferative tibial lesions, porotic hyperostosis on the occipital bone, and enamel hypoplasia on the incisor, maxillary canine, and first and second molars. The frequency of cribra orbitalia was also borderline significantly higher in Denmark ($\alpha = 0.09$). However, there was a significantly *lower* frequency of porotic hyperostosis on the left and right parietal bones in Denmark compared to East Smithfield. Furthermore, there were no significant differences in the frequencies of porotic hyperostosis on the frontal bone, enamel hypoplasia on the mandibular canine, or short femur lengths between the two cemetery samples.

For the majority of lesions examined, the comparison of standardized lesion frequencies reveals a higher frequency of lesions in the Danish sample compared to East Smithfield. Only three of the twelve lesions were at a higher frequency in East Smithfield (two of those differences were statistically significant). This pattern of differences in lesions frequencies *might* suggest that there are differences in selectivity with respect to frailty between the Black Death and normal mortality, such that the Black Death was less selective and killed more indiscriminately than does normal mortality. However, one cannot necessarily make any conclusions about the selectivity of the Black Death from lesions frequencies alone if we do not know the original frequencies of the lesions in either of the once-living populations or how the lesions truly are related to frailty.

A relatively low frequency of individuals with skeletal lesions and, conversely, a relatively high proportion of individuals without those lesions in East Smithfield compared to Denmark *might* suggest any of the following:

- The Black Death killed indiscriminately, or was, at least, less selective than normal mortality.
- There were differences in the frequencies of those lesions in the once-living populations. Perhaps the Black Death was just as selective as normal mortality, but certain lesions were at higher frequencies in the once-living Danish population and therefore appear more frequently in the Danish cemeteries.
- Some skeletal lesions were associated with an increased risk of death (high frailty) in one population (Denmark) but not the other (England); that is, perhaps the risk associated with certain lesions differs from one population to the next.

> Even if the Black Death was as selective as normal mortality, we might detect a difference in selectivity between the East Smithfield and Danish cemeteries if we happen to examine lesions associated with very different risks of death in the different once-living populations.

To make any conclusions about selectivity from skeletal sample lesion frequencies alone, one must assume that lesion frequencies were very similar in the once-living populations and/or that lesions are associated with the same differential risk in the once-living populations.

Because I used the Usher model to estimate the differential risk of death associated with various skeletal lesions, I need not make any of the above assumptions. As described in Chapter 3, I estimated the excess mortality associated with various skeletal lesions and then compared the results from East Smithfield and Denmark to determine whether the Black Death was selective with respect to frailty. If the Black Death killed indiscriminately, I expected to find skeletal lesions associated with significant excess mortality in Denmark *not* to be associated with excess mortality in East Smithfield. That is, if the Black Death was not selective, individuals with lesions (which are shown to indicate frailty in Denmark) should have been at the same risk of death as their peers without lesions in East Smithfield.

All of the lesions included in this study are associated with excess mortality in the normal mortality Danish cemeteries, and many of the estimates of excess mortality are statistically significant. These findings for the Danish samples mean that the skeletal lesions really are telling us something about frailty; i.e. individuals with skeletal lesions

in Denmark had higher frailty than their peers without lesions. All of the lesions were also associated with excess mortality in East Smithfield. This indicates that the Black Death *was* selective with respect to frailty, as individuals who had skeletal lesions before the Black Death were more likely to die during the epidemic than were individuals without those lesions.

However, the level of excess mortality for almost every lesion was higher in Denmark than in East Smithfield; if only those lesions that yielded significant results are considered, this pattern is true for every lesion. This means that the Black Death was *not as strongly selective as normal mortality*. For example, the k_2 estimate for proliferative tibial lesions in the Danish sample was more than twice as high as the estimate in East Smithfield. During the Black Death, frail individuals were more likely to die, but their risk was not as high as it would have been under normal mortality conditions.

There are differences between East Smithfield and Denmark in terms of the excess mortality associated with lesions because a greater number of healthy individuals (i.e. those without lesions) died during the Black Death than under normal mortality conditions. The Black Death was highly virulent and thus killed many otherwise healthy people who would have been less likely to die under normal mortality conditions. This produces a reduction in the excess mortality of individuals with lesions in East Smithfield (compared that in Denmark), not because individuals with lesions were less likely to die during the Black Death than under normal conditions, but because the risk of death for otherwise healthy people increased during the epidemic. In other words, the Black Death did not kill indiscriminately, but it did discriminate less sharply than death normally does.

Differential Risk of Death Associated with Sex

To determine whether men and women were at the same risk of death during the Black Death, I treated "maleness" as a risk factor and estimated the risk of death associated with "maleness" among adults in East Smithfield and Denmark. The estimate of excess mortality for men in East Smithfield, k_{2m}, = 1, indicates that men and women were at the same risk of dying during the Black Death. The estimate in Denmark, k_{2m} = 1.2, suggests that males were at a higher risk of dying compared to women during times of normal mortality. However, the high standard error (± 0.2) associated with the estimate in Denmark renders this result unconvincing. The results therefore suggest that men and women were at approximately the same risk of death both during the Black Death and under normal mortality conditions.

To test the effect of setting k_1 equal to one, I conducted sensitivity analyses by running the analyses with varying values of k_1. Values of k_1 ranging from 0.5 to 9.0 had little or no effect on estimates of k_{2m} for either cemetery sample. For East Smithfield, values of k_1 ranging from 0.5 to 9 yielded estimates of k_{2m} ranging from 0.97 to 1.0. For Denmark, the estimates of k_{2m} ranged from 1.03 to 1.18, depending on the set value of k_1. However, for both cemeteries, values of k_1 below 0.1 yielded estimates of k_{2m} that were higher than those reported above. The estimate of k_{2m} is apparently highly sensitive to values of k_1 close to zero, but relatively insensitive to values of k_1 greater than 0.5.

Assessment of Assumptions

For this study, I have made several assumptions about the cemetery samples and the parameters of the Usher model. As argued in Chapter 2, these assumptions are reasonable and logical, but I do need to examine the possible effects if they are wrong.

As described in Chapter 2, I modeled the hazard of dying from State 2 as proportional to the baseline age-specific risk of dying from State 1. Under this specification, k_2, the constant of proportionality, indicates the proportional difference in risk of death between individuals with and without lesions. By using this proportional hazards model, I assumed that the k_2 parameter is constant across age. That is, the proportional hazards model assumes that the presence of a lesion has a multiplicative effect on the risk of death, so that the difference in risk of death between individuals with and without lesions does not change with age. This assumption might not be true. For example, age at time of lesion formation may have an effect of the differential risk of death. It is possible that the formation of certain lesions might be associated with highly elevated risks of death for young children but less highly elevated or even reduced risks of death for individuals at older ages. Thomas (2003) found, for a sample from medieval Europe, that from the ages of zero to two years, the presence of strong accentuated striae of Retzius (a type of enamel microdefect) was associated with an increased risk of death. Thus, for infants, these enamel defects indicate high frailty. However, between the ages of two and four years, the presence of strong accentuated striae was associated with a *reduced* risk of death during that particular interval; children between two and four with strong accentuated striae were less likely to die during that interval than were their peers without the lesions.

By assuming k_2 is constant, I was not able to observe any age-related variation in the differential risk associated with lesions. The application of the Usher model in this study thus can reveal only very general patterns in the differential risk of death associated with lesions and the differences between East Smithfield and Denmark with respect to that risk. Unless I can determine whether the differential risk associated with lesions really was proportional at all ages, I cannot use estimates of k_2 to infer anything about the risks of death for any one individual in my samples. That is, even though the estimates of k_2 indicate that the presence of lesions was associated with elevated risks of death in both East Smithfield and Denmark, the risk for any given individual with a lesion might actually have been higher or lower than the estimated value for all individuals combined. Also, if the assumption of proportionality is not correct, I cannot interpret the k_2 parameter as a relative risk. As described in the *Future Directions* section below, I will ultimately attempt to determine the age pattern of risk associated with skeletal lesions and test the assumption that k_2 is constant.

In this study, I modeled the hazard of developing lesions (i.e. the hazard of making the transition from State 1 to State 2) as a constant k_1. For simplicity, in this study, the age of onset of lesions is an exponential random variable. Thus, for this study, I assumed that all individuals faced a constant risk of developing a lesion, irrespective of age or frailty. I took this approach because for most skeletal lesions, one does not know the age at which an individual actually developed a lesion nor how long it takes for a lesion to fully develop. If I did know the age at which individuals developed lesions, I could use that data to estimate the age-specific risk of developing lesions instead of assuming the risk is constant. For enamel hypoplasia, it *is* possible to estimate the age at

formation; it would therefore be possible to use data on presence and age-at-formation of enamel hypoplasia to estimate a model of the age-specific hazard of forming such lesions. A comparison of the results obtained by estimating an age-specific hazard versus using a constant k_1 would reveal what effect, if any, the assumption that the hazard of developing lesions is a constant has on estimates of selectivity.

For this study, I assumed that the East Smithfield cemetery contains *only* victims of the Black Death and that the Danish cemeteries contain only individuals who died before the epidemic. However, it is possible that the East Smithfield cemetery contains some individuals who died from causes other than the Black Death. And it is possible that some of the individuals in my Danish samples were victims of the Black Death. According to Kieffer-Olsen (1993) and Jantzen et al. (1994) a small proportion of burials in Denmark with arm position B date to 1350 or later. About 38 percent of my Danish skeletons were buried with arm position B, and given the findings of Kieffer-Olsen (1993) and Jantzen et al. (1994), it is possible that some of those individuals died during or after the Black Death. If East Smithfield includes non-Black Death individuals, and the Danish sample includes victims of the Black Death, differences between the two cemeteries would be reduced. However, I observed a consistent difference in the pattern of selectivity with respect to frailty between the two cemeteries. Therefore, the possibility that East Smithfield does not purely reflect Black Death mortality and Denmark does not purely reflect normal mortality only strengthens my conclusions about selectivity with respect to frailty.

In using the Usher model to compare East Smithfield to the Danish cemeteries, I assumed that immediately before the Black Death, East Smithfield had the same population

growth rate and age-specific mortality rates as did the communities in Denmark. I considered the Danish mortality patterns to be the normal baseline against which I could compare Black Death mortality. This is not an unreasonable assumption given the similarities in socio-economic and demographic conditions between southern England and Denmark during the Middle Ages (see Chapter 1). As I did *not* likely compare across populations with potentially different growth rates, demographic non-stationarity should not pose a problem for this study. However, if the assumption is incorrect, the Danish sample would not represent normal pre-Black Death conditions in East Smithfield, and the comparisons of the two cemetery samples would not be very informative about deviations from normal mortality patterns during the Black Death.

For this study, I assumed that the Danish and East Smithfield populations were stable. As described in Cbapter 2, this assumption is usually reasonable for paleodemographic studies given that most populations closely approximate stable age distributions (Milner et al., 2000). However, because the catastrophic mortality levels of the Black Death would perturb a population's age distribution away from its stable form, assuming the East Smithfield population was stable is problematic *if* the Black Death did not run its course very rapidly. If the Black Death epidemic lasted relatively long in any given area (i.e. several years), and if it was selective, then the population being sampled at the end of the epidemic would have been very different from that at the beginning of the epidemic. This would affect estimates of the age and sex pattern and of selective mortality. For example, imagine that the Black Death disproportionately affected very young children and elderly adults and that the epidemic lasted several years. As the epidemic progressed, the number of young children killed by the disease would remain high because the

"supply" of children was replenished through births. However, there would be no similar replenishment of the supply of elderly people, and the number of elderly killed would decrease over time. Therefore estimates of the age pattern of Black Death mortality from the East Smithfield cemetery would underestimate the differential risk for elderly people.

Estimates of time course of the Black Death show that the time course of the disease was very rapid (Wood et al. 2003), so it is *not likely* that the population affected at the beginning of the epidemic would have differed much from that affected at the end of the epidemic.

Future Directions

Variation in Selectivity

The next step I will take in my ongoing investigation of the epidemiological patterns of the Black Death will be to determine if the patterns of selectivity with respect to frailty vary by age. As described above, for this study, I used a proportional hazards model and assumed that the differential risk associated with lesions remained proportional with age. I looked at the excess mortality associated with proliferative tibial lesions, porotic hyperostosis, cribra orbitalia, and enamel hypoplasias for all ages simultaneously; the only exception was femur length, which I analyzed in adults only. I would like to determine whether the differential risk associated with these lesions is constant across age, as I assumed in this study, or if there is any variation by age. Analyses of the variation in selectivity will be highly informative about heterogeneous

frailty, answering questions about how frailty distributions change by age within populations.

Most of the lesions I examined in this study form during childhood. Porotic hyperostosis and cribra orbitalia are usually associated with childhood illnesses and stressors, and enamel hypoplasias only form during childhood as teeth develop. These lesions may last into adulthood, but the differential risk of death associated with them is not necessarily constant throughout life. For example, young children who develop porotic hyperostosis and also die during childhood might have succumbed to whatever illness caused the lesions, or whatever caused the lesion weakened their immune system and made them more vulnerable to future physiological stressors and they therefore died from other causes. In either case, those children with porotic hyperostosis were at a greatly increased risk of death compared to their peers without such lesions, and the lesions therefore indicate high frailty in children. However, adults with porotic hyperostosis likely survived for many years with such lesions, and the lesions might therefore indicate *reduced* risk of death in adults.

There is reason to suspect that the risk of death associated with at least some lesions might not be constant throughout life. Thomas (2003) analyzed enamel defects in individuals from the cemetery associated with the medieval Danish village of Tirup. She found that when enamel defects were analyzed for all ages simultaneously, the results suggest that the presence of such defects is associated with an increased risk of death (this is the approach taken in the current study). However, when enamel defects are analyzed by age-interval, the results are much more varied. For example, from the ages of zero to two years, the presence of strong accentuated striae (a type of enamel

microdefect) is associated with an increased risk of death. Thus, for infants, these enamel defects indicate high frailty. However, between the ages of two and four years, the presence of strong accentuated striae is associated with a *reduced* risk of death during that particular interval; children between two and four with strong accentuated striae were less likely to die during that interval than were their peers without the lesions. Thomas (2003) also found that individuals who developed *weak* accentuated striae between the ages of one and seven years were less likely to die after the age of seven whereas individuals who developed strong accentuated striae during that same interval were *more* likely to die later in life. Given the patterns observed by Thomas (2003) with respect to enamel microdefects, it is possible that the relationship between other skeletal lesions and frailty might also vary by age.

In addition to analyzing how the risk of death associated with lesions varies by age, I will also examine how the risk of death associated with sex varies by age to determine whether the apparent similarity in risk of death for men and women in Denmark and East Smithfield is constant across all adult ages.

Effect of Error in Age Estimation

As described in Chapter 3, the adult age estimates used in the current analyses are not without error. Using the Rostock Protocol for adult age estimation, I was able to estimate the error associated with each point estimate of age (see Figures 3-3 and 3-4). However, for the analyses of the age and sex patterns of mortality and selectivity with respect to frailty, I used only the point estimates without including the error. In future

analyses, I will attempt to include the error estimates in similar analyses to determine what effect the error has upon estimates of Black Death mortality patterns.

Comparison of Results from Rostock Protocol and Traditional Methods

This project represents one of the few applications, to date, of the Rostock protocol for adult age estimation. Ultimately, I would like to determine if the results of the current analyses were at all influenced by the age estimation method. I will rerun the analyses using adult ages estimated with traditional methods and determine whether they produce the same or similar results. Bill White and his team of researchers at the Museum of London Centre for Human Bioarchaeology have estimated age-at-death for individuals in East Smithfield using traditional methods, and they have age estimates for all of the individuals included in my sample. I will collaborate with them (Bill White, personal communication) and use their age estimates in the Usher model to see if I find the same age and sex pattern of mortality and selectivity with respect to frailty associated with the Black Death as revealed by the current analyses. There have been very few uses of the Rostock protocol, partly because the method is more mathematically complex than traditional methods and it therefore requires the use of a computer program. Some researchers think the method is too complex and may not be worth the time and effort it requires (although with the programming work done by Darryl Holman, Jesper Boldsen, Lyle Konigsberg, and others, the time and effort required is minimal). By comparing the results of the Black Death mortality patterns using traditional methods of age estimation and the Rostock protocol, I can potentially show that the Rostock protocol is not only feasible but also more informative than are traditional methods of adult age estimation.

Conclusion

The results of these analyses reveal that the Black Death did not kill people indiscriminately with regard to frailty (and perhaps age), but also that it was not as selective as normal mortality with respect to those same characteristics. The Black Death was clearly highly virulent and it significantly and dramatically increased the risk of death in affected populations, but it was not so virulent that all individuals in the population experienced exactly the same excess risk of death. The Black Death increased the risk of death for everyone in the population, but as during times of normal mortality, some individuals were at an even higher risk than others during the Black Death. In particular, individuals with certain skeletal lesions were at an increased risk of death compared to others in the population. It is also possible that elderly individuals were disproportionately affected, although further work is needed to confirm this.

We know from contemporary reports that some people were infected with the disease and yet managed to survive the Black Death. Those who survived the Black Death were presumably able to mount an immunological response to the disease. Individuals unable to mount such a response succumbed to the disease; presumably, those already weakened by conditions that are associated with skeletal lesions, had impaired immune function and were therefore unable to fight the disease.

These results show that we cannot assume that selective mortality is never operating, even in a disease as catastrophic as the Black Death. Selective mortality is

likely operating under most if not all mortality conditions. The immediate implication is that skeletal samples are never truly representative of the living populations that produced them. These results are also important because they inform us about the osteological paradox and about emerging diseases in general.

The Osteological Paradox

These results help to resolve one aspect of the osteological paradox: what do skeletal lesions really tell us about individual frailty? Are individuals with skeletal lesions more or less healthy than individuals without those lesions? The results of the current study indicate that mortality, even catastrophic mortality like that of the Black Death, is selective with respect to the lesions included in the analyses. Proliferative tibial lesions, porotic hyperostosis, cribra orbitalia, enamel hypoplasia, and short femur length are all associated with significant excess mortality in both the normal mortality and Black Death cemeteries. The estimate of excess mortality varies by lesion; nevertheless, individuals with any of the above lesions were more likely to die than their peers without such lesions regardless of the mortality conditions.

Given that selective mortality was operating on the lesions included in this study, there are two possible reasons why individuals with lesions were at an increased risk of dying in either population. One possibility is that lesions directly reflect frailty, such that individuals with high frailty were both more susceptible to forming lesions and more likely to die than their less frail peers (Wood et al., 1992; Usher, 2000); individuals with low frailty were better able to fight off infection or otherwise withstand physiological

stress, so they never developed skeletal lesions. Frail individuals developed lesions such as proliferative tibial lesions because they were unable to sufficiently fight off infection. Stress markers such as enamel hypolasias and short femur likely developed in frail individuals because they expended too much energy fighting infection and could not spare energy for growth and development. In the case of the Black Death, individuals with high frailty were perhaps more likely to both develop skeletal lesions before the Black Death and to die during the epidemic than their peers.

The other possible reason for the increased risk of dying associated with lesions is that lesions indicate episodes of past physiological stress that increased frailty (Wood et al., 1992; Usher, 2000). Individuals did not develop lesions because they were frail, rather they had higher frailty because they were exposed to the stressors that caused the lesions (i.e. lesions are a proxy for the cause of frailty rather than the effect thereof). Individual with skeletal lesions survived episodes of stress long enough to form the lesions, but that stress weakened their immune systems, making them more vulnerable to future stress and more likely to die than their peers who were either never exposed to stress or were able to endure it unscathed (Wood et al., 1992; Usher, 2000). Again in the case of the Black Death, individuals who experienced lesion-inducing stressors before the epidemic perhaps had impaired immune function and could therefore not fight off whatever disease caused the Black Death.

Both explanations of increased risk of death associated with lesions are plausible with respect to both Black Death mortality and normal mortality. The current application of the Usher model reveals how lesions are associated with risk of death, but it does not indicate the specific mechanisms responsible for the association; however, the full four-

state Usher model, which includes a frailty distribution for each state, has the potential to reveal more clearly how lesions are related to frailty.

Wood et al. (1992) presented the possibility that some skeletal lesions might actually be indicative of low frailty. As discussed in Chapter 3, visible skeletal lesions take time to form (often weeks or months). It is therefore possible that individuals with certain lesions are actually less frail than their peers without such lesions given that they were able to survive a physiological stressor long enough to develop a lesion. In fact, Usher (2000) found that enamel hypoplasia on the maxillary central incisor was associated with a significantly reduced risk of death, such that individuals with this lesion were about 85 percent *less* likely to die than their peers without them. Among all of the lesions included in the current study, there was not a single example of a lesion that was associated with a reduced risk of dying in either the East Smithfield or Danish cemeteries. Future analyses of the variation in risk by age will reveal if this apparently consistent pattern of increased risk of dying associated with all skeletal lesions used in this study is borne out across all ages. As described above, Thomas (2003) found that if the differential risk is assumed to be constant across age, enamel defects are associated with an increased risk of death; however, she found that if the differential risk is allowed to vary by age, enamel defects are associated with elevated risk of death at some ages and reduced risk at other ages.

Based on the results of this study, we cannot assume that catastrophic cemeteries are representative of living populations. One reason for this is the inherent preservation bias associated with paleodemographic samples (as discussed in Chapter 1). Because of preservation bias, no cemetery (normal or catastrophic) is likely to contain a

representative sample of the once-living population; for example, the greater tendency for juvenile skeletons to disintegrate over time often results in infant underenumeration in paleodemographic studies. The other reason such cemeteries cannot be assumed to be representative, as demonstrated by the current study, is that even extraordinarily catastrophic mortality is selective.

The results of the analyses of selectivity with respect to frailty within the Danish cemeteries are not unexpected; the Danish cemeteries represent normal mortality, which we expect to be selective, so it is not surprising that individuals who appear unhealthy (i.e. have skeletal lesions) were at often greatly increased risks of dying compared to their peers who appeared healthier. The results from the East Smithfield cemetery might surprise some researchers given how deadly the epidemic clearly was. Yet despite the obvious virulence of the medieval epidemic, it did not affect all individuals equally, and cemeteries associated with the Black Death will not provide a perfect "snapshot" of the living population immediately before the epidemic.

The Black Death as an Emerging Disease

The results of the current project are important because not only do they reveal new information about the Black Death, but they also tell us something about emerging or re-emerging diseases in general. Emerging diseases are those that are increasing in frequency after being introduced into a new host population, and re-emerging diseases are those that already exist in a population but are recently and rapidly increasing in incidence or geographical range because of changes in their epidemiology (Woolhouse,

2002; Morens et al., 2004). There are many possible factors affecting the emergence or re-emergence of diseases including microbial evolution, changing ecosystems, economic development and land use (e.g. people encroaching on new environments), international travel and commerce, breakdown of public health measures, and poverty and social inequity (McMichael, 2004; Morens et al., 2004). According to the World Health Organization and the Centers for Disease Control and Prevention, more than fifty diseases have emerged in the past thirty years, including HIV/AIDS, Malaysian Nipah virus, variant Creutzfeldt-Jakob disease, legionnaires' disease, West Nile Virus, Ebola, SARS (Feldmann et al., 2002; McMichael, 2004; Morens et al., 2004). Undoubtedly, new diseases will emerge in the future (Morens et al., 2004); many people are fascinated (if not completely terrified) by ideas of what diseases will emerge next and whether we will be able to cope with them. Understanding how emerging diseases have behaved in the past is a prerequisite for dealing with the challenges presented by such diseases in the future.

According to Wheelis (2002: 971), the Black Death "was probably the greatest public health disaster in recorded history and one of the most dramatic examples ever of emerging or reemerging disease." Morens et al. (2004:242) also describe the Black Death as an important emerging diseases which has "shaped the course of human history". The mortality patterns of the Black Death and the ways in which mortality apparently changed in subsequent outbreaks are informative about the evolution of some emerging diseases (though not all, given the diversity of emerging diseases). As discussed in Chapter 1, in the outbreaks following the original Black Death, excess mortality decreased dramatically and the disease apparently disproportionately affected

young children. Such a change in mortality patterns suggests that Europeans acquired immunity to the disease (i.e. children who had never before been exposed to the disease were most susceptible) and/or the pathogen evolved to become less virulent. The Black Death therefore provides an important historical example of what McMichael (2004:1051) describes as the "amoral, self-interested, coevolutionary struggle" between humans and microbes.

Understanding how emerging diseases behave, now and in the past, has important implications for public health measures in the future. For example, knowing that the Black Death was selective should encourage us to look carefully at what factors increase an individual's risk of morbidity and mortality during an epidemic. The assumption that is often made, that everyone in an immunologically naïve population is at equal risk, might not always be true, and it certainly does not seem to have been the case with the Black Death. Even if future emerging diseases have the potential to cause devastatingly high mortality, they too might be selective. By identifying those individuals or groups who are at highest risk of morbidity and mortality, it might be possible for us to moderate or eliminate their risk factors in an effort to decrease mortality in future epidemics. Such risk factors include poverty, malnutrition, and restricted or no access to health care. Morris and Potter (1997) argue that malnutrition is the leading cause of increasing an individual's susceptibility to infection; studies of malnutrition and diarrheal pathogens in Bangladesh, for example, have shown that malnutrition apparently increases the risk of diarrhea-associated death by thirty times (Santos, 1994). Malnutrition and other risk factors for morbidity and mortality are clearly consequences of poverty; and the alleviation of poverty is likely one of the most important methods for reducing

susceptibility to diseases (McMichael, 2004). Today, people in developing countries are most at risk of morbidity and mortality from infectious diseases, and within developed nations, infectious diseases differentially affect disadvantaged minorities (Feldmann et al., 2002; Morens et al., 2004). McMichael (2004:1050) writes that "poverty perpetuates vulnerability to infectious diseases" and is one of several factors that creates "new opportunities for microbial opportunism."

Identification of those segments of the population most at risk of infectious disease is also important because it may give researchers the opportunity to discover new emerging diseases before they have devastating effects. According to Morris and Potter (1997:440) – "surveillance within populations with increased susceptibility to infection may allow identification of new pathogens before they are recognized within the general population."

The results of this study demonstrate that by using new paleodemographic methods, such as the Usher model, to study infectious diseases, we can more fully understand the emerging diseases that have played an important role in shaping human history and that will almost certainly have important demographic, social, and economic effects in the future.

Chapter Summary

- The Black Death was selective: though the disease clearly increased general mortality during the epidemic, everyone did *not* experience the same risk of death.
- The Black Death was not as strongly selective as normal mortality.

- The selectivity of the Black Death is informative about emerging diseases in general. If a new disease as highly virulent as the Black Death was selective, future emerging diseases might be as well; this should motivate us to identify those segments of the population most likely to be at elevated risk and take measures to decrease that risk.

Bibliography

Allan, BB, R Brandt, JE Seidel, and JF Jarrell (1997) Declining sex ratios in Canada. *Can Med Assoc J* 156:37–41.

Angel JL (1971) *The people of Lerna*. Washington, DC: American School of Classical Studies at Athens and Smithsonian Intitution.

Arneborg J, J Heinemeier, N Lynnerup, HL Nielsen, N Rud, Á Sveinbjsrnsdóttir (1999) Change of diet of the Greenland vikings determined from stable carbon isotope analysis and ^{14}C dating of their bones. *Radiocarbon* 41:157.

Ascenzi, A, A Bellelli, M Brunori, G Citro, R Ippoliti, E Lendaro, and R Ziti (1991) Diagnosis of thalessemia in ancient bones: problems and prospects in pathology. In Ortner, DJ and AC Aufderheide (eds.) (1991) *Human paleopathology: current syntheses and future options.* Washington: Smithsonian Institution Press, pp. 73-75.

Astill F and J Langdon eds. (1997) *Medieval farming and technology: the impact of agricultural change in northwest Europe.* Leiden: Brill.

Aufderheide AC and C Rodriguez-Martin (1998) *The Cambridge encyclopedia of human paleopathology.* Cambridge: Cambridge University Press.

Bannerman WB (1906) The spread of plague in India. *J. Hygiene* 6:179-211.

Bass WM (1995) *Human osteology: a laboratory and field manual (4th edition).* Columbia, MO: Missouri Archaeological Society.

Begier EM, G Asiki, Z Anywaine, B Yockey, ME Schriefer, P Aleti, A Ogen-Odoi, JE Staples, C Sexton, SW Bearden, and JL Kool (2006) Pneumonic plague cluster, Uganda, 2004. *Emerg Infect Dis* 12:460-467.

Benedict C (1996) *Bubonic plague in nineteenth-century China.* Palo Alto: Stanford University Press.

Benedictow OJ (1993) *The medieval demographic system of the nordic countries.* Oslo: Middelalderforlaget.

Benedictow OJ (2004) *The Black Death, 1346-1353: The complete history.* Woodbridge, U.K.: Boydell Press.

Boccaccio G. *The decameron*. Pp 26-34 in Horrox R (1994) *The Black Death*. Manchester: Manchester University Press.

Bocquet-Appel JP, and C Masset (1982) Farewell to paleodemography. *J Hum Evol* 11:321-333.

Boldsen JL (1998) Body proportions in a medieval village population: effects of early childhood episodes of ill health. *Ann Hum Bio* 27:309-317.

Boldsen JL (1998) Livet for døden: Hvad skeletterne fortalte om middelalderens demografi. *Humaniora* 1325-29.

Boldsen JL, GR Milner, LW Konigsberg, and JW Wood (2002) Transition analysis: A new method for estimating age from skeletons. Pp. 73-106 in Hoppa RD and JW Vaupel (eds), *Paleodemography: Age distributions from skeletal samples.* Cambridge: Cambridge University Press.

Boldsen JL and L Mollerup (2006) Outside St. Jørgen: Leprosy in the medieval Danish city of Odense. *Am J Phys Anthropol* 130:344-351.

Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Brooks S and J Suchey (1990) Skeletal age determination based on the os pubis: a comparison of the Ascadi-Nemeskéri and Suchey-Brooks methods. *Human Evo* 5:227-238.

Buikstra JE (1997) Paleodemography: Context and promise. Pp. 367-380 in Paine RR (ed), *Integrating archaeological demography: Multidisciplinary approaches to prehistoric population*. Carbondale, IL: Center for Archaeological Investigations, Southern Illinois University.

Buikstra JE and DH Ubelaker, eds (1994) *Standards for data collection from human skeletal remains.* Fayetteville, AR: Arkansas Archeological Society.

Burnham K and D Anderson (1998) *Model selection and inference: A practical information-theoretic approach.* New York: Springer-Verlag.

Butler T (1989) The Black Death past and present, 1: Plague in the 1980s. *Trans Roy Soc Trop Med Hyg* 83:458-60.

Campbell GL, and JM Hughes (1995) Plague in India: a new warning from an old nemesis. *Ann Intern Med* 122:151-53.

Carpentier E (1971) The plague as a recurrent phenomenon. Pp. 35-37 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Christensen AS (1988) *Middelalderbyen Odense*. Århus: Statens Humanistiske Forskningoråd.

Cleri DJ, JR Vernaleo, LJ Lombardi, MS Rabbat, A Mathew, R Marton, and MC Reyelt (1997) Plague pneumonia disease caused by *Yersinia pestis*. *Semin Respir Infect* 12:12-23.

Coale AJ (1957) How the age distribution of a human population is determined. *Cold Spring Harbor Symposia on Quantitative Biology* 22:83-88.

Cohen MN and GJ Armelagos (1984) Paleopathology at the origins of agriculture: Editor's summation. Pp. 585-601 in Cohen MN and GJ Armelagos (eds), *Paleopathology at the origins of agriculture.* Orlando, FL: Academic Press.

Cohen MN (1989) *Health and the rise of civilization.* New Haven: Yale University Press.

Cohn SK (2002) *The Black Death transformed*. London: Arnold.

Conheeney J (1999) Reconstructing the demography of medieval London from studies on human skeletal material: problems and potential. *Trans London and Middlesex Archaeol Soc* 50:78-86.

Dahlberg, AA (1991) Interpretations of general problems in amelogenesis. In Ortner, DJ and AC Aufderheide (eds.) (1991) *Human paleopathology: current syntheses and future options.* Washington: Smithsonian Institution Press, pp. 269-272.

da Piazza M. *Cronaca.* Pp 35-41 in R Horrox (ed.) (1994) *The Black Death*. Manchester: Manchester University Press.

de Venette J. Chronicles. Pp 54-57 in R Horrox (ed.) (1994) *The Black Death*. Manchester: Manchester University Press.

Dobson MJ (1997) *Contours of death and disease in early modern England.* Cambridge: Cambridge University Press.

Drancourt M, G Aboudharam, M Signoli, O Dutour, and D Raoult (1998) Detection of 400-year-old *Yersinia pestis* DNA in human dental pulp: An approach to the diagnosis of ancient septicemia. *Proc Natl Acad Sci* USA 95:12637-12640.

Dyer C (2002) *Making a living in the middle ages: the people of Britain 850-1520.* New Haven: Yale University Press.

Eisenberg, LE (1991) Mississippian cultural terminations in Middle Tennessee: what the bioarcheological evidence can tell us. Pp. 70-88 in Powell, ML, PS Bridges, and AMW Mires (eds.) (1991) *What mean these bones.* Tuscaloosa: University of Alabama Press.

Ell SR (1984) Immunity as a factor in the epidemiology of medieval plague. *Rev inf dis* 6:866-79.

Elvin SJ, ED Williamson, JC Scott, JN Smith, G Perez De Lema, S Chilla, P Clapham, K Pfeffer, D Schlondorff, and B Luckow. Evolutionary genetics: Ambiguous role of CCR5 in Y. pestis infection. *Nature* 430:417.

Feldmann H, M Czub, S Jones, D Dick, M Garbutt, A Grolla, and H Artsob (2002) Emerging and re-emerging infectious diseases. *Med Microbiol Immunol* 191:63-74.

Fukushima H, M Gomyoda, S Kaneko, M Tsubokura, N Takeda, T Hongo, and FN Shubin (1994) Restriction endonuclease analysis of virulence plasmids for molecular epidemiology of *Yersinia pseudotuberculosis* infections. *J Clin Microbiol* 32:1410-1413.

Gage TB (1988) Mathematical hazard models of mortality: an alternative to model life tables. *Am J Phys Anthropol* 76:429-441.

Gage TB (1991) Causes of death and the components of mortality: Testing the biological interpretations of a competing hazards model. *Am J Phys Anthropol* 3:289-300.

Gasquet FA (1893) *The Great Pestilence (A.D. 1348-9), now commonly known as the Black Death.* London: Simpkin Marshall, Hamilton, Kent and Co.

Gilbert MT, J Cuccui, W White, N Lynnerup, RW Titball, A Cooper, and Prentice MB (2004) Absence of Yersinia pestis-specific DNA in human teeth from five European excavations of putative plague victims. *Microbiology* 150(Pt 2):1-54.

Goodman, AH (1991) Stress, adaptation, and enamel developmental defects. In Ortner, DJ and AC Aufderheide (eds.) (1991) *Human paleopathology: current syntheses and future options.* Washington: Smithsonian Institution Press, pp. 280-287.

Goodman, AH, GJ Armelagos, and JC Rose (1980) Enamel hypoplasias an indicators of stress in three prehistoric populations from Illinois. *Human biology* 52:515-528.

Goodman AH and JC Rose (1990) Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yrbk Phys Anthrop* 33:59-110.

Goodman AH, RB Thomas, AC Swedlund, and GJ Armelagos (1988) Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. *Yrbk Phys Anthrop* 31:169-202.

Gordon CC and JE Buikstra (1981) Soil pH, bone preservation, and sampling bias at mortuary sites. *Am Antiq* 46:566-71.

Gottfried RS (1983) *The Black Death: Natural and human disaster in medieval Europe.* New York: Macmillan.

Grainger I, D Hawkins, and T Waldron (n.d.) *The Black Death cemetery, East Smithfield, London.* London: Museum of London (in press).

Hatch J, P Willey (1974) Stature and status in Dallas society. *Tenn Archaeol* 30: 107-131.

Hatcher J (1977) *Population and the English economy 1348-1530.* London: Macmillan.

Haviland, WA (1967) Stature at Tikal, Guatemala: implications for ancient Maya demography and social organization. *Am Antiq* 32:316-325.

Hawkins D (1990) The Black Death and the new London cemeteries of 1348. *Antiquity* 64:637-642.

Herlihy D (1997) *The Black Death and the transformation of the West.* Cambridge: Harvard University Press.

Hershkovitz I, R Yakar, C Taitz, S Wish-Baratz, A Pinhasov, and B Ring (1993) The human remains from the Byzantine monastery at Khan El-Ahmar. *Liber Annuus* 43:373-85.

Hewlett BS, JMH van de Koppel, and M van de Koppel (1986) Causes of death among Aka pygmies of the Central African Republic. Pp. 45-63 in Cavalli-Sforza LL(ed), *African Pygmies.* Orlando, FL: Academic Press.

Higgins R (2004) Emerging or re-emerging bacterial zoonotic diseases: bartonellosis, leptospirosis, Lyme borreliosis, plague. *Rev Sci Tech Off Int Epi* 23:569-81.

Hinde A (2003) *England's population: A history since the Domesday survey.* London: Hodder Arnold.

Hinnebusch BJ (1997) Bubonic plague: a molecular genetic case history of the emergence of an infectious disease. *J Mol Med* 75:645-52.

Holman DJ (2002) *mle*: *A programming language for building likelihood models*. Seattle (unpublished).

Holman DJ, JW Wood, and KA O'Connor (2002) Estimating age-at-death distributions from skeletal samples: A multivariate latent-trait approach. Pp. 193-201 in Hoppa RD and JW Vaupel (eds), *Paleodemography: Age distributions from skeletal samples.* Cambridge: Cambridge University Press.

Holmes GA (1971) England: A decisive turning point. Pp. 91-99 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Hoppa RD and JW Vaupel (2002) The Rostock manifesto for paleodemography: The way from stage to age. Pp. 1-8 in Hoppa RD and JW Vaupel (eds), *Paleodemography: Age distributions from skeletal samples.* Cambridge: Cambridge University Press.

Horrox R (1994) *The Black Death*. Manchester: Manchester University Press.

Howell N (1979) *Demography of the Dobe !Kung.* New York: Academic Press.

Huss-Ashmore, R, AH Goodman, and GJ Armelagos (1982) Nutritional inference from paleopathology. *Advan Archaeol Meth Theory* 5:395-473.

Jacobzon L (1982) Människor i stadens utkant: Helgeandshusets dyrkogård berätter om liv och död. Pp. 112-123 in G Dahlbäck (ed.) *Helgeandsholmen*. Boräs.

Jantzen C, J Kieffer-Olsen, PK Madsen (1994) De små brødres hus i Ribe. *Mark og montre* 26-36.

Johansson SR and S Horowitz (1986) Estimating mortality in skeletal populations: influence of the growth rate on the interpretation of levels and trends during the transition to agriculture. *Am J Phys Anthropol* 71:233-250.

Kieffer-Olsen J (1993) *Grav og gravskike i det middelalderlige Danmark.* PhD dissertation, Århus University, Århus, Denmark.

Kolman CJ and N Tuross (2000) Ancient DNA analysis of human populations. *Am J Phys Anthropol* 111:5-23.

Komlos J and M Baur (2003) From the tallest to (one of) the fattest: the enigmatic fate of the American population in the 20th century. *Discussion paper 2003-19*, Department of Economics, University of Munich.

Komlos J and P Kriwy (2002) Social status and adult heights in the two Germanies. *Annals of human biology* 29:641-48.

Konigsberg LW and SR Frankenberg (1992) *Am J Phys Anthropol* 89:235-256.

Konigsberg LW, SR Frankenberg, and RB Walker (1997) Regress what on what? Paleodemographic age estimation as a calibration problem. Pp. 64-88 in Paine RR (ed), *Integrating archaeological semography: Multidisciplinary approaches to prehistoric population*. Carbondale, IL: Center for Archaeological Investigations, Southern Illinois University.

Kosminskii EA (1971) The plague deemphasized. Pp. 38-46 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Kremeyer B, S Hummel, and B Herrmann (2005) *Frequency analysis of the delta32ccr5 HIV resistance allele in a medieval plague mass grave*. Anthropol Anz 63:13-22.

Kristensen HK (1987) *Middelalderbyen Viborg*. Århus: Statens Humanistiske Forskningoråd.

Landers J (1993) *Death and the metropolis: Studies in the demographic history of London 1670-1830.* Cambridge: Cambridge University Press.

Larsen, CS (1997) *Bioarchaeology: Interpreting behavior from the human skeleton*. Cambridge: Cambridge University Press.

Lederberg J, RE Shope, and SC Oaks, eds (1992) *Emerging infections: Microbial threats to health in the United States*. Washington: Institute of Medicine.

Lederberg J (1997) Infectious disease as an evolutionary paradigm. *Emerg Infect Dis* 3:417-423.

Lotka AJ (1907) Mode of growth of material aggregates. *Am J Sci* 24:199-216.

Lotka AJ (1922) Contributions to the energetics of evolution. *Proc Natl Acad Sci USA* 8:147-150.

Luders A et al. (eds) *Statutes of the realm 1101-1713.* Pp 287-289 in R Horrox (ed.) (1994) *The Black Death*. Manchester: Manchester University Press.

Lütge F (1971) Germany: the Black Death and a structural revolution in socioeconomic history. Pp. 80-85 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Lütgert SA (2000) Victims of the Great Famine or the Black Death? The archaeology of the mass graves found in the former graveyard of Holy Ghost Hospital, Lubeck (N. Germany), in the European context. *Hikuin* 27:255-265.

Margerison BJ and CJ Knüsel (2002) Paleodemographic comparison of a catastrophic and an attritional death assemblage. *Am J Phys Anthropol* 119:134-43.

Martin, DL, AH Goodman, GJ Armelagos (1985) Skeletal pathologies as indicators of quality and quantity of diet. In Gilbert, Jr., RI and JH Mielke (eds.) (1985) *The analysis of prehistoric diets.* Orlando, FL: Academic Press, Inc.

Massler M, I Schour, and HG Poncher (1941) Development pattern of the child as reflected in the calcification patterns of the teeth. *Am J Dis Child* 62:33-67.

McKern T and TD Stewart (1957) *Skeletal age changes in young American males, analyzed from the standpoint of identification.* Technical report EP-45. Headquarters, Quartermaster Research and Development Command, Natick, Massachusetts.

McMichael AJ (2004) Environmental and social influences on emerging infectious diseases: past, present and future. *Phil Trans R Soc London Bio* 359:1049-1058.

McNeil WH (1971) *Plagues and people.* New York: Doubleday.

Mensforth, RP, CO Lovejoy, JW Lallo, and GJ Armelagos (1978) The role of constitutional factors, diet, and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. 1978. *Med Anthropol* 2:1-58.

Michon LAJ (ed) *Documents inédits sur la grande peste de 1348*. Pp 182-184 n R Horrox (ed.) (1994) *The Black Death*. Manchester: Manchester University Press.

Milner, GR (1991) Health and cultural change in the Late Prehistoric American Bottom, Illinois. Pp. 52-69 in Powell, ML, PS Bridges, and AMW Mires (eds.) (1991) *What mean these bones.* Tuscaloosa: University of Alabama Press.

Milner GR, JW Wood, and JL Boldsen (2000) Paleodemography. Pp. 467-497 in Saunders S and M Katzenberg (eds), *Skeletal biology of past peoples: Research methods*. New York: Wiley-Liss.

Moorees CFA, EA Fanning, and EE Hunt (1963a) Formation and resorption of three deciduous teeth in children. *Am J Phys Anthropol* 21:205-213.

Moorees CFA, EA Fanning, and EE Hunt (1963b) Age formation by stages for ten permanent teeth. *J Dent Res* 42:1490-1502.

Morens DM, GK Folkers, and AS Fauci (2004) The challenge of emerging and re-emerging infectious diseases. *Nature* 430:242-249.

Morris JG and M Potter (1997) Emergence of new pathogens as a function of changes in host susceptibility. *Emerg Infect Dis* 3:435-441.

Morse SS (1995) Factors in the emergence of infectious diseases. *Emerg Infect Dis* 1:7-15.

Müller G, B Love, and RD Hoppa (2002) Semiparametric method for estimating paleodemographic profiles from age indicator data. *Am J Phys Anthrop* 117:1-14.

Musa M and P Bondanella (trans. and eds.) (1977) *Giovanni Boccaccio's* The *decameron*: *A new translation.* New York: W.W. Norton.

Ortner DJ (1991) Theoretical and methodological issues in paleopathology. Pp. 5-11 in Ortner DJ and AC Aufderheide (eds.) *Human paleopathology: current syntheses and future options*. Washington, D.C.: Smithsonian Institution Press.

Ortner DJ (1992) Skeletal paleopathology: probabilities, possibilities, and impossibilities. Pp. 5-13 in Verano, JW and DH Ubelaker (eds) (1992) *Disease and demography in the Americas.* Washington: Smithsonian Institution Press.

Ortner DJ (2003) *Identification of pathological conditions in human skeletal remains.* San Diego: Academic Press.

Owens IPF (2002) Sex differences in mortality rates. *Science* 297:2008-9.

Paine RR (2000) If a population crashes in prehistory, and there is no paleodemographer there to hear it, does it make a sound? *Am J Phys Anthropol* 112:181-190.

Palubeckaitė Ž, R Jankauskas, and J Boldsen (2002) Enamel hypoplasia in Danish and Lithuanian late medieval/early modern samples: A possible reflection of child morbidity and mortality patterns. *Int J Osteoarch* 12:189-201.

Papathanasiou A (2005) Health status of the Neolithic population of Alepotrypa Cave, Greece. *Am J Phys Anthropol* 126:377-90.

Penrose EC (1859) *A History of England from the first invasion by the Romans to the present.* London: John Murray.

Perry RD, Fetherston JD (1997) *Yersinia pestis*: Etiologic agent of plague. *Clin. Microbiol. Rev.* 10:35-66.

Phenice T (1969) A newly developed visual method of sexing in the os pubis. *Am J Phys Anthrop* 30:297-301.

Plague Research Commission (1907) The epidemiological observations made by the

Commission in Bombay City. *J Hygiene* 7:724-798.

Platt C (1996) *King Death: the Black Death and its Aftermath in late-medieval England.* Toronto: University of Toronto Press.

Poos LR (1991) *A Rural Society after the Black Death: Essex 1350-1525.* Cambridge: Cambridge University Press.

Poulsen B (1997) Agricultural technology in medieval Denmark. Pp 115-146 in Astill F and J Langdon eds. (1997) *Medieval farming and technology: the impact of agricultural change in northwest Europe.* Leiden: Brill.

Powell, ML (1988) *Status and health in prehistory.* Washington: Smithsonian Institution Press.

Power, C (1985-86) Diet and disease: evidence from the human dental remains in two medieval Irish populations. *J Irish Arch* 111

Raoult D, G Aboudharam, E Crubézy, G Larrouy, B Ludes, and M Drancourt (2002) Molecular identification by "suicide PCR" of *Yersinia pestis* as the agent of medieval Black Death. *Proc Natl Acad Sci* USA 97:12800-12803.

Razi Z (1980) *Life, marriage, and death in a medieval pParish: Economy, society, and demography in Halesowen 1270-1400.* Cambridge: Cambridge University Press.

Reale B, D Marchi, SM Borgognini Tarli (1998) A case of diffuse idiopathic skeletal hyperostosis (DISH) from a medieval necropolis in southern Italy. *Int J Osteoarch.* 9:369-73.

Reid DJ and MC Dean (2000) Brief communication: the timing of linear hypoplasias on human anterior teeth. *Am J Phys Anthropol* 113:135-139.

Renouard Y (1971) The Black Death as a major event in world history. Pp. 25-34 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Roberts C and K Manchester (1995) *The archaeology of disease.* Ithaca: Cornell University Press.

Robb J, R Bigazzi, L Lazzarini, C Scarsini, and F Sonego (2001) Social "status" and biological "status": a comparison of grave goods and skeletal indicators from Pontecagnano. *Am J Phys Anthropol* 115:213-222.

Roesdahl E, ed. (1999) *Dagligliv i Danmarks middelalder: En arkæologisk kulturhistorie.* Copenhagen: Nordisk Forlag.

Rogers J and T Waldron (2001) DISH and the monastic way of life. *Int J Osteoarch* 11:357-365.

Russell JC (1948) *British medieval population.* Albuquerque: University of New Mexico Press.

Ryan, AS (1997) Iron-deficiency anemia in infant development: implications for growth, cognitive development, resistance to infection, and iron supplementation. *Yrbk Phys Anth* 40:25-62

Sabeti PC, E Walsh, SF Schaffner, P Varilly, B Fry, HB Hutcheson, M Cullen, TS Mikkelsen, J Roy, N Patterson, R Cooper, D Reich, D Altschuler, S O'Brien, and ES Lander (2005) The case for selection at CCR5-Delta32. *PLoS Bio* 3:e378.

Santos JI (1994) Nutrition, infection, and immunocompetence. *Inf Dis Clin North Am* 8:243-267.

Santos, RV and CEA Coimbra, Jr. (1999) Hardships of contact: enamel hypoplasia in Tupí-Mondé Amerindians from the Brazilian Amazonia. *Am J Phys Anthropol* 109:111-127.

Satcher DS (1995) Emerging infections: Getting ahead of the curve. *Emerg Infect Dis* 1:1-6.

Sattenspiel L (2000) Tropical environments, human activities, and the transmission of infectious diseases. *Yrbk Phys Anthrop* 43:3-31.

Sattenspiel L and HC Harpending (1983) Stable populations and skeletal age. *Am Antiq* 48:489-98.

Sawyer B and P Sawyer (1993) *Medieval scandinavia.* Minneapolis: University of Minnesota Press.

Scott S and CJ Duncan (1998) *Human demography and disease.* Cambridge: Cambridge University Press.

Scott S and CJ Duncan (2001) *Biology of plagues: Evidence from historical populations.* Cambridge: Cambridge University Press.

Seal SC (1969) Epidemiological studies of plague in India. I. The present position. *Bull WHO* 23:283-92.

Shell-Duncan B, unpublished field data, University of Washington, Seattle.

Signoli M, I Séguy, J Biraben, and O Dutour (2002) Paleodemography and historical demography in the context of an epidemic. *Population* 57:829-853.

Simonet M, Riot B, Fortineau N, Berche P (1996) Invasin production by *Yersinia pestis* is abolished by insertion of an IS*100*-like element within the *inv* gene. *Infect Immun* 64:375-79.

Simpson JA, Weiner ESC (eds.) (1989) *The compact Oxford English dictionary (second edition)*. Oxford: Oxford University Press.

Smith R (2002) Plagues and peoples: the long demographic cycle, 1250 – 1670. Pp. 177-210 in Slack P and R Ward (eds.) *The peopling of Britain: The shaping of a human landscape*. Oxford: Oxford University Press.

Sokal RR and FJ Rohlf (1981) *Biometry*. NY: Freeman.

Statistical Abstract Relating to British India from 1885-86 to 1894-95. (1896) London: Her Majesty's Stationary Office.

Statistical Abstract Relating to British India from 1894-95 to 1903-04. (1905) London: Her Majesty's Stationary Office

Steckel RH (1995) Stature and the standard of living. *J Econ Lit* 33:1903-40.

Stewart, TD (1979) *Essentials of forensic anthropology.* Springfield, IL: Charles C Thomas, Publisher

Stojanowski CM, RM Seidemann, and GH Doran (2002) Differential skeletal preservation at Windover Pond: causes and consequences. *Am J Phys Anthropol* 119:15-26.

Stuart-Macadam P (1991) Porotic hyperostosis: Changing interpretations. Pp. 36-39 in Ortner DJ and AC Aufderheide (eds), *Human paleopathology: Current syntheses and future options.* Washington: Smithsonian Institution Press.

Thoma L, and HM Goldman (1960) *Oral pathologym*. Fifth edition. St. Louis: Mosby

Thomas RF (2003) *Enamel defects, well-being and mortality in a medieval Danish village.* PhD dissertation, Pennsylvania State University, University Park.

Thompson JAA (1906) On the epidemiology of plague. *J Hygiene* 6:537-569.

Thompson JW (1971) The plage and world war: parallels and comparisons. Pp. 19-24 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Todd TW (1921) Age changes in the pubic bone. I: the male white pubis. *Am J Phys Anthropol* 3:285-334.

Trotter M and GC Gleser (1952) Estimation of stature from the bones of American whites and Negroes. *Am J Phys Anthropol* 10:463-514.

Trotter M and GC Gleser (1958) A re-evaluation of estimation based on measurements of stature taken during life and of long bones after death. *Am J Phys Anthropol* 16:79-123

Twigg G (1984) *The Black Death: A biological reappraisal.* London: Batsford.

Twigg G (2003) The Black Death and DNA. *Lancet Infect Dis* 3:11.

Usher BM (2000) *A multistate model of health and mortality for paleodemography.* PhD dissertation, Pennsylvania State University, University Park.

Vaupel JW, TE Johnson, and GJ Lithgow (1979) The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16:439-54.

Vaupel JW and AI Yashin (1985) Heterogeneity's ruses: some surprising effects of selection on population dynamics. *Am Stat* 39:176-185.

Waldron HA (2001) Are plague pits of particular use to paleoepidemiologists? *Int J Epid* 30:104-108.

Waldron T (1992) Osteoarthritis in a Black Death cemetery in London. *Int J Osteoarch* 2:235-240.

Walker PL, JR Johnson, and PM Lambert (1988) Age and sex biases in the preservation of human skeletal remains. *Am J Phys Anthropol* 76:183-188.

Wells JCK (2000) Natural selection and sex differences in morbidity and mortality in early life. *J Theor Biol* 202:65-76.

Wheelis M (2002) Biological warfare at the 1346 siege of Caffa. *Emerg infect dis* 8:971-975.

White TD (2000) *Human osteology*. San Diego, CA: Academic Press.

Wiechmann I and G Grupe (2005) Detection of *Yersinia pestis* DNA in two early medieval skeletal finds from Aschheim (Upper Bavaria, 6th century A.D.) *Am J Phys Anthropol* 126:48-55.

Widgren M (1997) Fields and field systems in Scandinavia during the middle ages. Pp 173-192 in Astill F and J Langdon eds. (1997) *Medieval farming and technology: the impact of agricultural change in northwest Europe.* Leiden: Brill.

Wood JW (1980) *Mechanisms of demographic equilibrium in a small human population, the Gainj of Papua New Guinea.* PhD dissertation, University of Michigan, Ann Arbor.

Wood JW, DJ Holman, KA O'Connor, and RJ Ferrell (2002) Mortality models for paleodemography. Pp 129-168 in Hoppa RD and JW Vaupel (eds), *Paleodemography: Age distributions from skeletal samples.* Cambridge: Cambridge University Press.

Wood JW, GR Milner, HC Harpending, and KM Weiss (1992) The osteological paradox: Problems of inferring prehistoric health from skeletal samples. *Curr Anthrop* 33:343-370.

Wood JW, RJ Ferrell, and SN DeWitte-Aviña (2003) The temporal dynamics of the fourteenth-century Black Death: New evidence from English ecclesiastical records. *Hum Biol* 75:427-448.

Wood JW and SN DeWitte-Aviña (2003) Was the Black Death yersinial plague? *Lancet Infect Dis.* 3:327-328.

Wood JW, SN DeWitte-Aviña, MD Shriver, RJ Ferrell, SA Matthews, and DJ Holman (n.d.) *The biology of the Black Death 1347-1350.* Cambridge: Cambridge University Press (in preparation).

Woolhouse MEJ (2002) Population biology of emerging and re-emerging pathogens. *Trends Micirobiol* 10:S3-S7.

Wright LE and CJ Yoder (2003) Recent progress in bioarchaeology: approaches to the osteological paradox. *J of Arch Res* 11:43-70.

Ziegler P (1969) *The Black Death*. Harper Collins, Gloucestershire.

Ziegler P (1971) Germany: the Flagellants and the persecution of the Jews. Pp. 65-79 in Bowsky WM, ed (1971) *The Black Death: A turning point in history?* New York: Holt, Rhinehart and Winston.

Appendix A

SCORING PROTOCOL

Sex Determination

I determined sex only in adult individuals. I scored features of the cranium and pelvis using the sex determination standards provided by Buikstra and Ubelaker (1994) and Phenice (1969) and listed in Table A-1. Figures **A-1 – A-3** show how these features vary in adult males and females.

Table A-1: Features used for sex estimation

Region	Feature	Reference
Pelvis	Ventral arc	Phenice (1969) Buikstra and Ubelaker (1994)
	Subpubic concavity	
	Ischiopubic ramus ridge	
	Greater sciatic notch	Buikstra and Ubelaker (1994)
Cranium	Superior nuchal line	Buikstra and Ubelaker (1994)
	Mastoid process	
	Glabella	
	Supraorbital margin	
	Anterior mandible	
	Gonial angle	

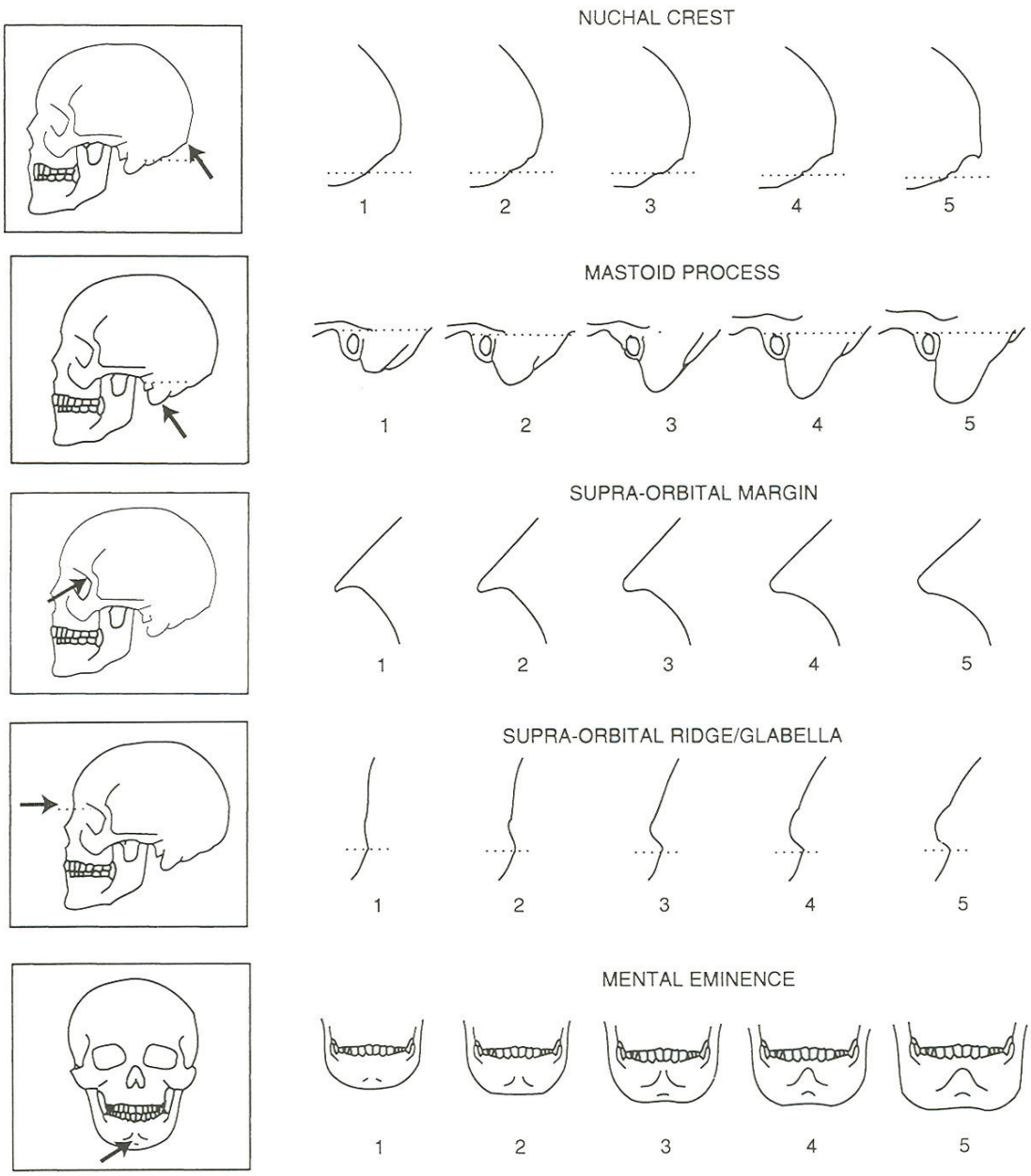

Figure A-1: Features of the skull used in sex-determination are indicated by arrows; scores range from 1 (definitely female) to 5 (definitely male). From Buikstra and Ubelaker (1994).

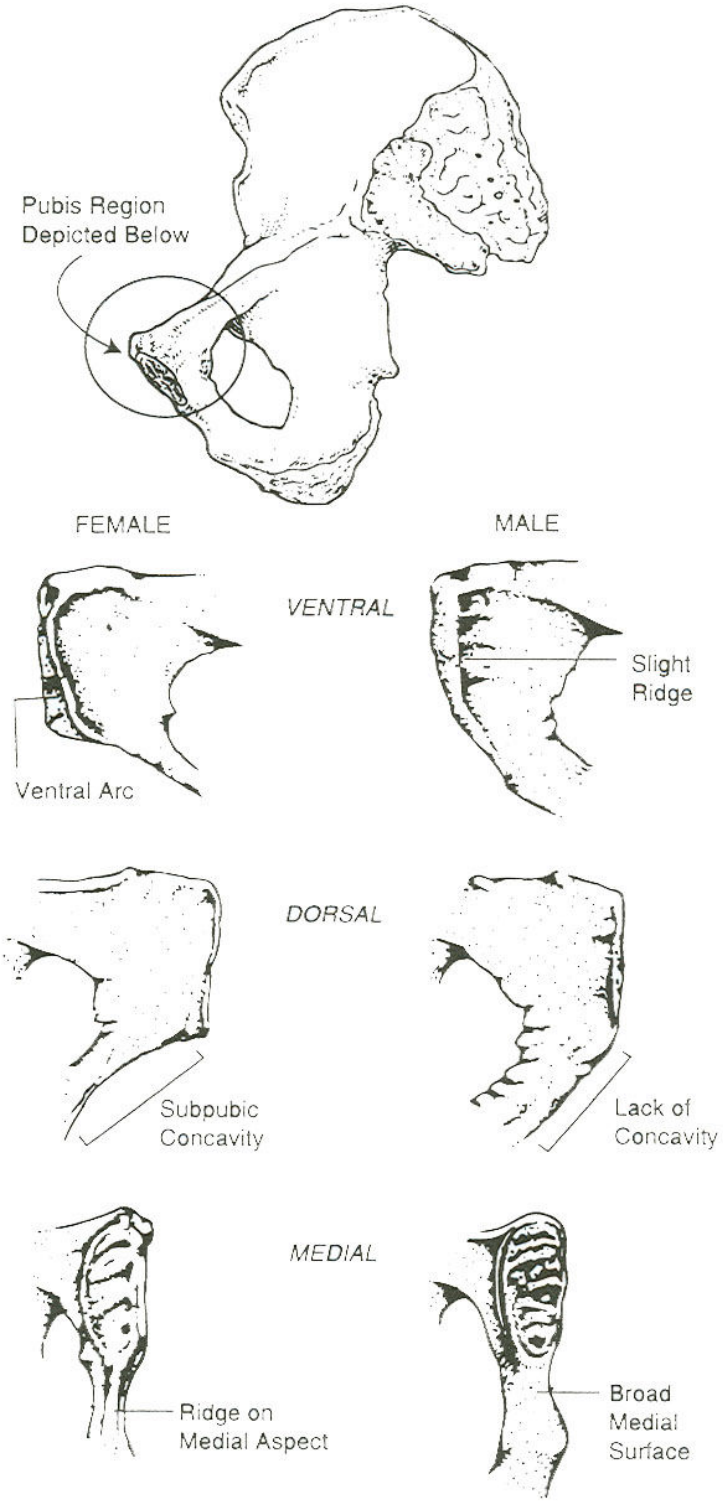

Figure A-2: Typical differences in the male and female pubic region. From Buikstra and Ubelaker (1994).

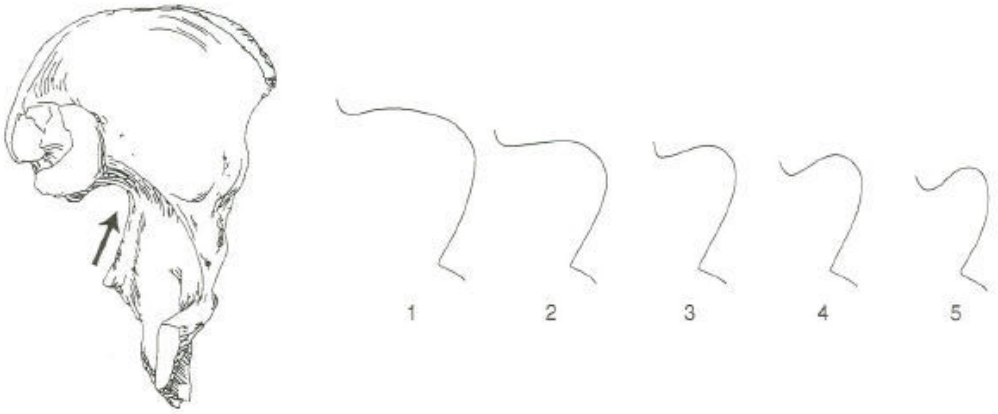

Figure A-3: The greater sciatic notch. Scores range from 1 (definitely female) to 5 (definitely male). From Buikstra and Ubelaker (1994).

Age Estimation

I scored all adults in my samples for the age-related features described by Boldsen et al. (2002) and listed in Table **A-2**. **Figures A-4 – A-3** show examples of age-related changes in the pubic symphysis, iliac auricular surface, and cranial sutures.

Table A-2: Features scored for adult age estimation

Region	Feature	Reference
Pubic symphysis	Symphyseal relief	Boldsen et al. (2002)
	Symphyseal texture	
	Superior apex	
	Ventral symphyseal margin	
	Dorsal symphyseal margin	
Iliac auricular surface	Superior demiface topography	
	Inferior demiface topography	
	Superior surface morphology	
	Apical surface morphology	
	Inferior surface morphology	
	Inferior surface texture	
	Superior posterior iliac exostoses	
	Inferior posterior iliac exostoses	
	Posterior spicules	
Cranial sutures	Coronal pterica	
	Sagittal obelica	
	Lambdoidal asterica	
	Zygomaticomaxillary	
	Interpalatine	

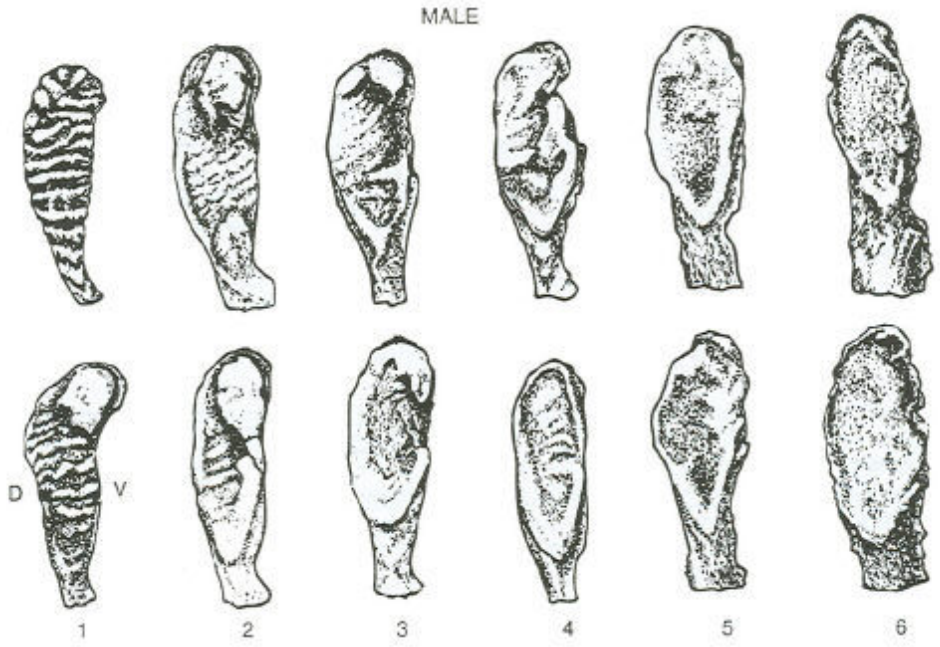

Figure A-4: Age-related changes in pubic symphysis morphology in males. From Buikstra and Ubelaker (1994).

Figure A-5: Iliac Auricular Surface Morphology in a 19-year-old female. From Buikstra and Ubelaker (1994).

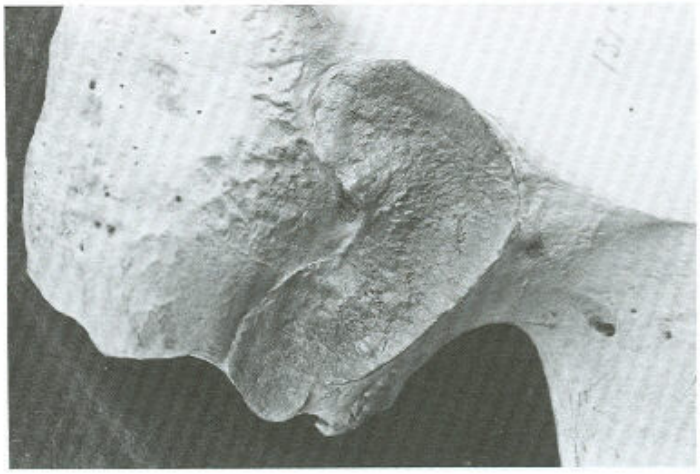

Figure A-6: Iliac Auricular Surface Morphology in a 35-year-old female. From Buikstra and Ubelaker (1994).

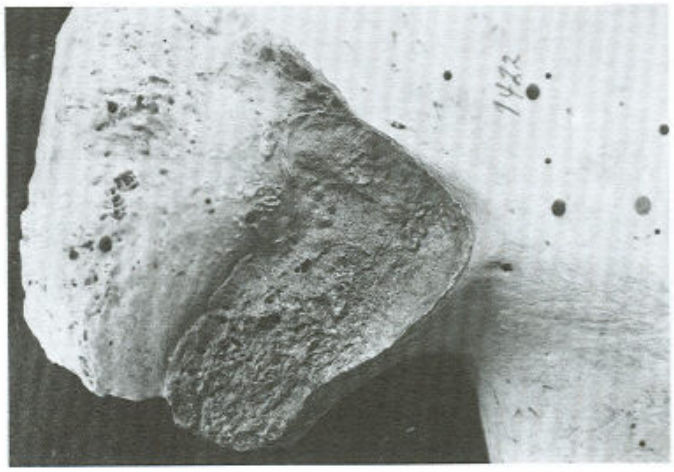

Figure A-7: Iliac Auricular Surface Morphology in a 50-year-old female. From Buikstra and Ubelaker (1994).

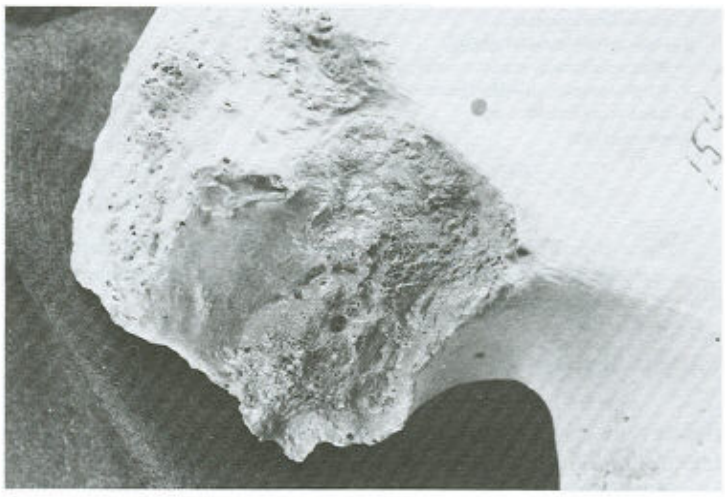

Figure A-8: Iliac Auricular Surface Morphology in a 59-year-old female. From Buikstra and Ubelaker (1994).

Figure A-9: Iliac Auricular Surface Morphology in a 63-year-old female. From Buikstra and Ubelaker (1994).

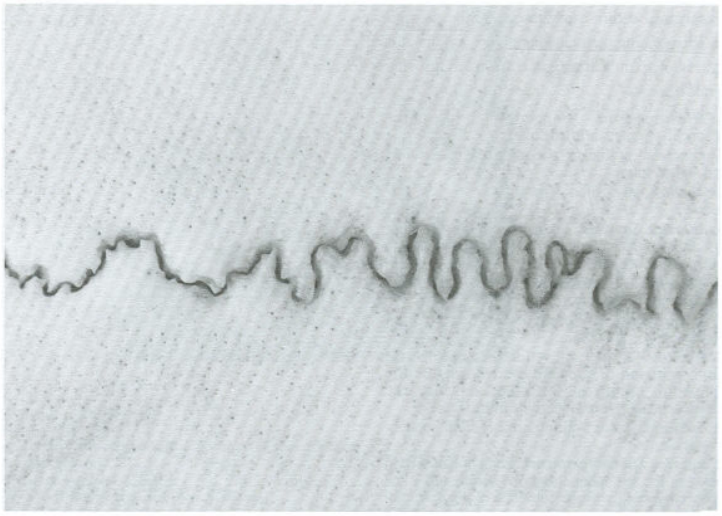

Figure A-10: Open cranial sutures. From Buikstra and Ubelaker (1994)

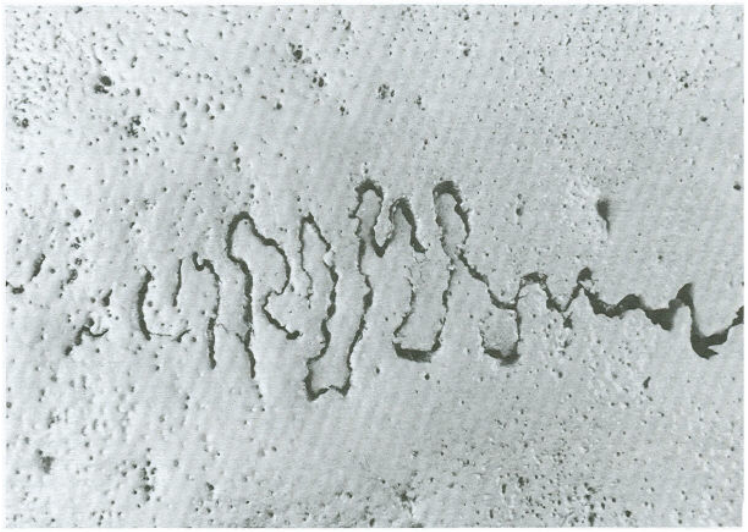

Figure A-11: Cranial sutures with minimal amount of closure. From Buikstra and Ubelaker (1994).

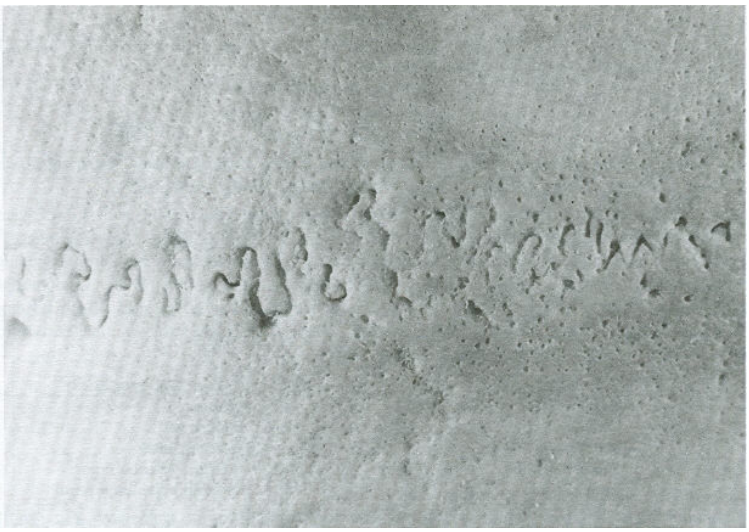

Figure A-12: Partially obliterated cranial sutures. From Buikstra and Ubelaker (1994).

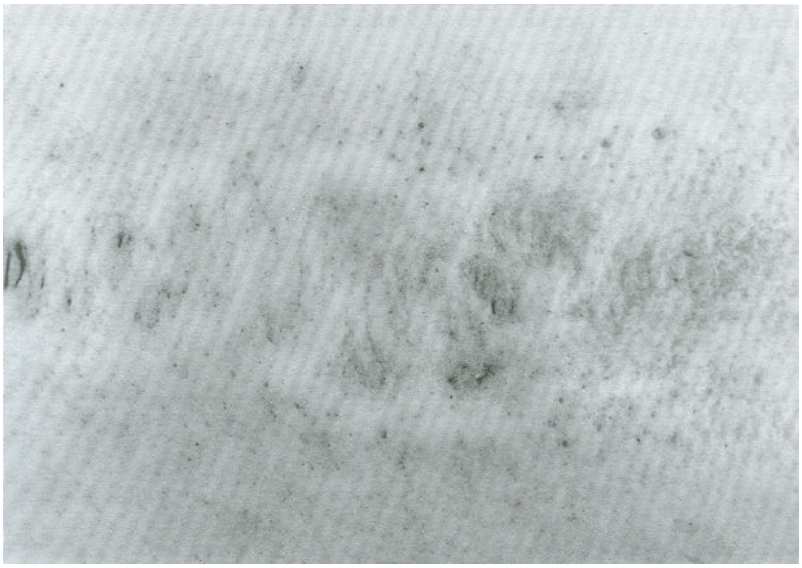

Figure A-13: Obliterated cranial sutures. From Buikstra and Ubelaker (1994).

Osteological Indicators of Frailty

To determine if the Black Death was selective with respect to frailty, I estimated the excess mortality associated with various osteological lesions or stress markers that have been shown to be non-specific indicators of frailty. Table **A-3** lists the skeletal lesions and osteological stress markers included in the study; photos of the lesions are provided in Chapter 2.

Table A-3: Skeletal lesions.

Stress Marker	Reference
Proliferative lesions on the tibia	Buikstra and Ubelaker (1994) Larsen (1997)
Porotic hyperostosis of the frontal, parietals, and occipital bones	Buikstra and Ubelaker (1994) Roberts and Manchester (1995)
Cribra orbitalia	Buikstra and Ubelaker (1994) Roberts and Manchester (1995)
Linear enamel hypoplasia – maxillary central incisor, canine, first and second molars, and mandibular canine	Buikstra and Ubelaker (1994) Palubeckaitė et al. (2002)
Stature – femur length	Buikstra and Ubelaker (1994)

Appendix B

MEASUREMENT ERROR STUDIES

Interobserver Error

I collected all the data used in the current study myself; nevertheless, I conducted an interobserver error study of the age and sex estimation methods I used in order to determine how consistent my scores are with other trained researchers (Ulla Freund and Corey Sparks). I compared my scores for age indicators and sex with those of other researchers for a subset of 63 adult individuals from St. Mikkel cemetery. I calculated Spearman correlations separately for each pair-wise combination of observers. The Spearman correlations (r), significance, and sample sizes for each pair-wise combination are summarized in Tables B-1, B-2, and B-3. Strong correlations range between 0.6 – 1.0, moderate correlations range between 0.4 – 0.6, and weak correlations are less than 0.4. Figures B-1 and B-2 show the regression of Sparks' and Freund's scores on mine for pubic symphysis relief; figure B-4 shows the regression of Freund's scores on Sparks' for the same indicator. The results of the interobserver error study suggest that I am capable of scoring skeletons for sex and age-indicators as well as two highly trained osteologists.

Table B-1: Age-estimation Interobserver Error: Pubic Symphysis

Region	Feature	DeWitte vs. Sparks	DeWitte vs. Freund	Sparks vs. Freund
Pubic Symphysis	symphyseal relief	r = 0.70 p < .0001 n = 41	r = 0.72 p < .0001 n = 36	r = 0.65 p < .0001 n = 36
	symphyseal texture	0.65 < .0001 39	0.60 0.02 36	0.64 < .0001 37
	superior apex	0.70 < .0001 30	0.49 < .0001 22	0.49 0.02 21
	ventral symphyseal margin	0.67 < .0001 38	0.48 0.004 35	0.57 0.0006 32
	dorsal symphyseal margin	0.61 < .0001 37	0.79 < .0001 33	0.77 < .0001 33

Table B-2: Age-estimation Interobserver Error: Cranial Sutures

Region	Feature	DeWitte vs. Sparks	DeWitte vs. Freund	Sparks vs. Freund
Cranial Suture	coronal pterica	r = 0.73 p = 0.0002 n = 21	r = 0.81 p < .0001 n = 17	r = 0.65 p = 0.003 n = 19
	sagittal obelica	0.91 < .0001 29	0.87 < .0001 29	0.89 < .0001 29
	lambdoidal asterica	0.49 0.02 22	0.51 0.02 21	0.57 0.01 18
	Zygomaticomaxillary	0.44 0.06 19	0.33 0.15 21	0.52 0.05 15
	Interpalatine	0.47 0.07 15	0.69 0.003 16	0.71 0.002 16

Table B-3: Age-estimation Interobserver Error: Auricular Surface

Region	Feature	DeWitte vs. Sparks	DeWitte vs. Freund	Sparks vs. Freund
Auricular Surface	superior demiface topography	r = 0.48 p = 0.0002 n = 56	r = 0.62 p < .0001 n = 53	r = 0.63 p < .0001 n = 51
	inferior demiface topography	0.37 0.005 57	0.49 0.0005 46	0.33 0.03 45
	superior surface morphology	0.50 < .0001 55	0.0007 0.9959 53	0.24 0.09 51
	apical surface morphology	0.29 0.03 56	0.42 0.002 53	0.26 0.07 52
	inferior surface morphology	0.66 < .0001 50	0.23 0.16 37	0.33 0.03 45
	inferior surface texture	0.56 < .0001 51	0.28 0.07 42	0.53 0.0002 44
	superior posterior iliac exostoses	0.66 < .0001 51	0.59 < .0001 40	0.46 0.004 39
	inferior posterior iliac exostoses	0.39 0.02 37	0.54 0.001 33	0.13 0.51 29
	posterior spicules	0.12 0.46 38	0.63 < .0001 45	0.39 0.0180 36

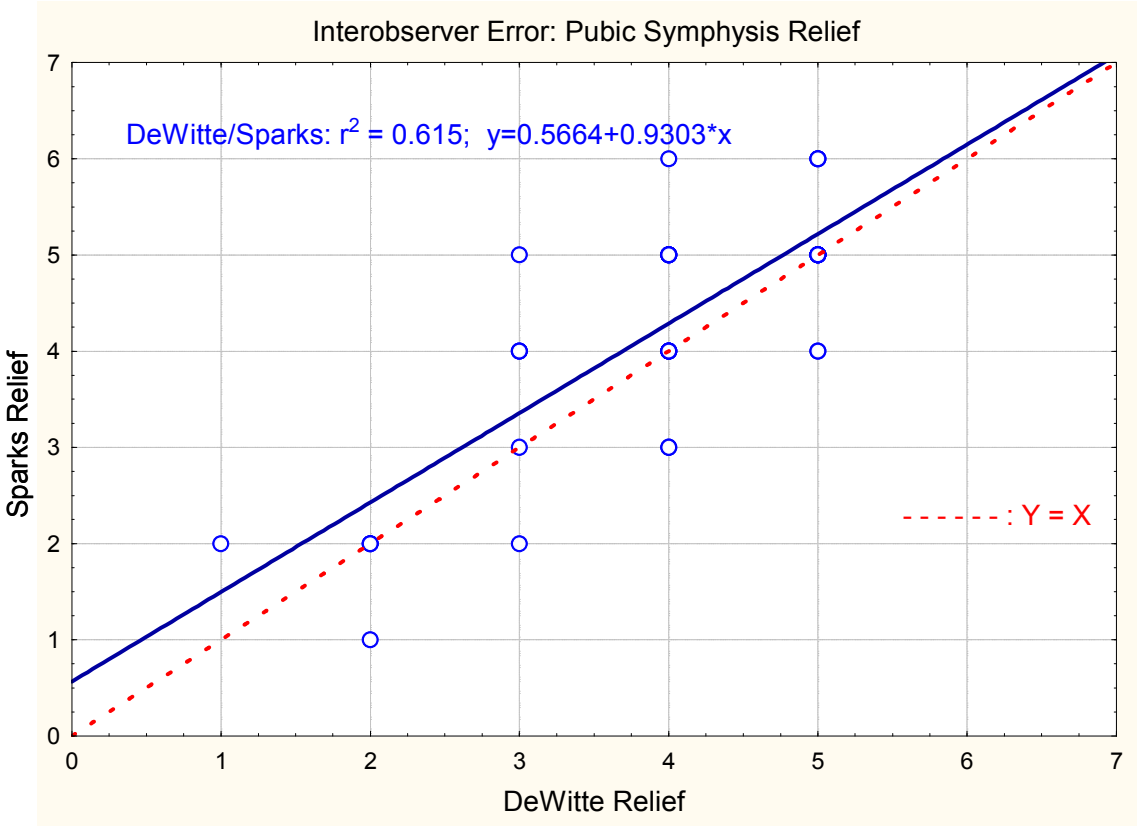

Figure **B-1**: Interobserver error between DeWitte and Sparks: pubic symphysis relief. The true regression line (in blue) is compared to an ideal regression line (in red) that reflects absolutely no error in the repeated measurements (i.e. Y = X with an intercept of zero and a slope equal to one).

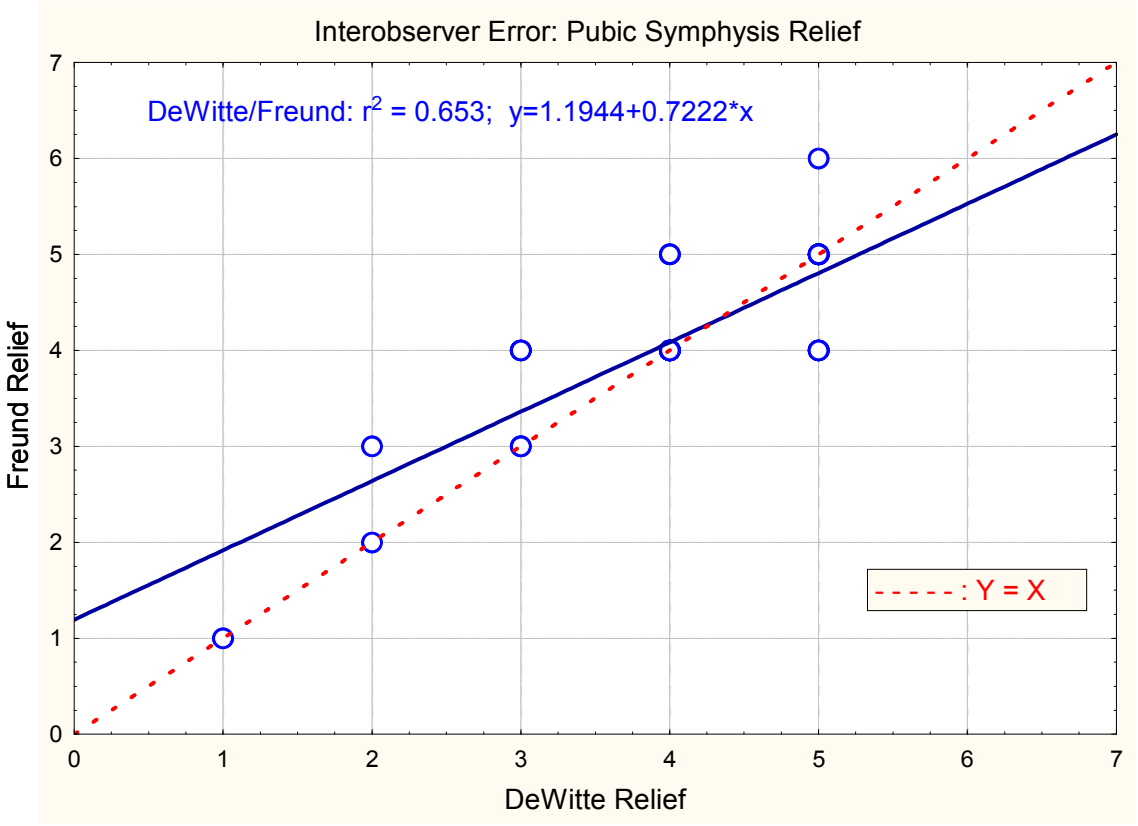

Figure **B-2**: Interobserver error between DeWitte and Freund: pubic symphysis relief. The true regression line (in blue) is compared to an ideal regression line (in red) that reflects absolutely no error in the repeated measurements (i.e. Y = X with an intercept of zero and a slope equal to one).

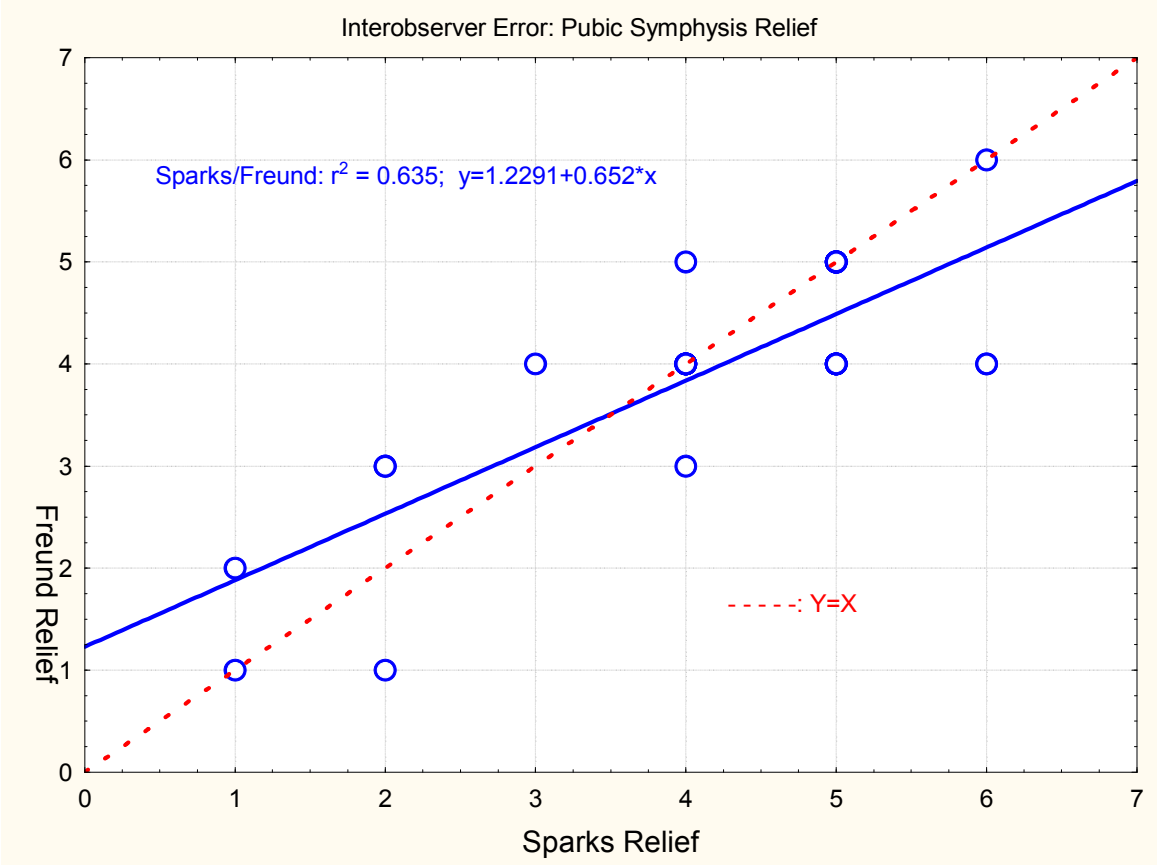

Figure B-**3**: Interobserver error between Freund and Sparks: pubic symphysis relief. The true regression line (in blue) is compared to an ideal regression line (in red) that reflects absolutely no error in the repeated measurements (i.e. Y = X with an intercept of zero and a slope equal to one).

Intraobserver Error

To evaluate the consistency of my age, sex, and lesion scores, I conducted an intraobserver error study using a sample of 32 individuals from the cemeteries of St. Mikkel and Albani. I used the test-retest method, and I scored each individual in my

error study twice; the intervals between first and second measurements ranged from four to ten weeks. I scored each individual for the nineteen skeletal age indicator traits, sex (in adults), cranial and tibial lesions, and I measured their long bones. To analyze the consistency of my scores for sex, age traits and cranial and tibial lesions, I calculated Spearman correlations separately for each pair-wise combination of repeated measurements. Tables **B-4 – 8** summarize the results for age indicators, lesions, and sex. Figure B-4 shows the regression between my original and repeat femur measurements. All of the correlations were significant ($p < 0.05$), and strong to very strong ($0.6 \leq r \leq 1$). The results of the intraobserver error study suggest that I am highly consistent in terms of scoring age-indicators, sex, cranial and tibial lesions, and measuring the femur.

Table B-4: Intraobserver Error: Spearman's Correlations for repeated scores of features of the pubic symphysis used in age-estimation (both right and left pubic symphyses were scored).

	Feature	Repeat Right	Repeat Left
Pubic Symphysis	symphyseal relief	r = 0.996 p < 0.05 n = 25	r = 0.94 p < 0.05 n = 23
	symphyseal texture	0.97 < 0.05 25	0.92 < 0.05 20
	superior apex	0.87 < 0.05 22	0.96 < 0.05 15
	ventral symphyseal margin	0.92 < 0.05 25	0.91 < 0.05 21
	dorsal symphyseal margin	0.97 < 0.05 24	0.95 < 0.05 20

Table B-5: Intraobserver Error: Spearman's Correlations for repeated scores of cranial sutures used in age-estimation.

	Feature	Repeat
Cranial Sutures	coronal pterica	r = 0.94 p < 0.05 n = 15
	sagittal obelica	0.97 < 0.05 21
	lambdoidal asterica	0.89 < 0.05 21
	zygomaticomaxillary	0.86 < 0.05 20
	Interpalatine	0.85 < 0.05 12

Table B-6: Intraobserver Error: Spearman's Correlation for repeated scores of features of the iliac auricular surface used in age-estimation (both right and left auricular surfaces were scored) Traits with no variation between the initial and repeat measures are marked with an asterisk.

	Feature	Repeat Right	Repeat Left
Auricular Surface	superior demiface topography	r = 0.902708 p < 0.05 n = 29	r = 0.840365 p < 0.05 n = 29
	inferior demiface topography	0.859823 < 0.05 25	0.797961 < 0.05 25
	superior surface morphology	0.620135 < 0.05 28	0.605900 < 0.05 28
	apical surface morphology	0.719468 < 0.05 27	0.707864 < 0.05 27
	inferior surface morphology	0.776671 < 0.05 26	0.794397 < 0.05 22
	inferior surface texture	1* <0.0001 24	1* <0.0001 22
	superior posterior iliac exostoses	0.736713 < 0.05 24	0.790139 < 0.05 26
	inferior posterior iliac exostoses	0.875856 < 0.05 18	0.840101 < 0.05 19
	posterior spicules	1* <0.0001 26	0.846114 < 0.05 25

Table B-7: Intraobserver Error: Spearman's Correlations for repeated lesion scores. Traits with no variation between the initial and repeat measures are marked with an asterisk.

	Feature	Repeat Right	Repeat Left
Tibial Lesions	Tibial proliferative lesions	r = 1* p < 0.0001 n = 30	r = 0.844228 p < 0.05 n = 28
	Porotic hyperostosis: frontal	0.833118 < 0.05 21	------
	Porotic hyperostosis: parietal	1* < 0.0001 25	1* < 0.0001 22
Cranial Lesions	Porotic hyperostosis: occipital	0.973262 < 0.05 23	-------
	Cribra orbitalia	0.939336 < 0.05 15	0.901498 < 0.05 18
	Enamel hypoplasia, # of events	0.981840 < 0.05 25	-------

Table B-8: Intraobserver Error: Spearman's Correlations for sex scores. There was no variation between the initial and repeat measures.

	Repeat
Sex	r = 1* p < 0.0001 n = 31

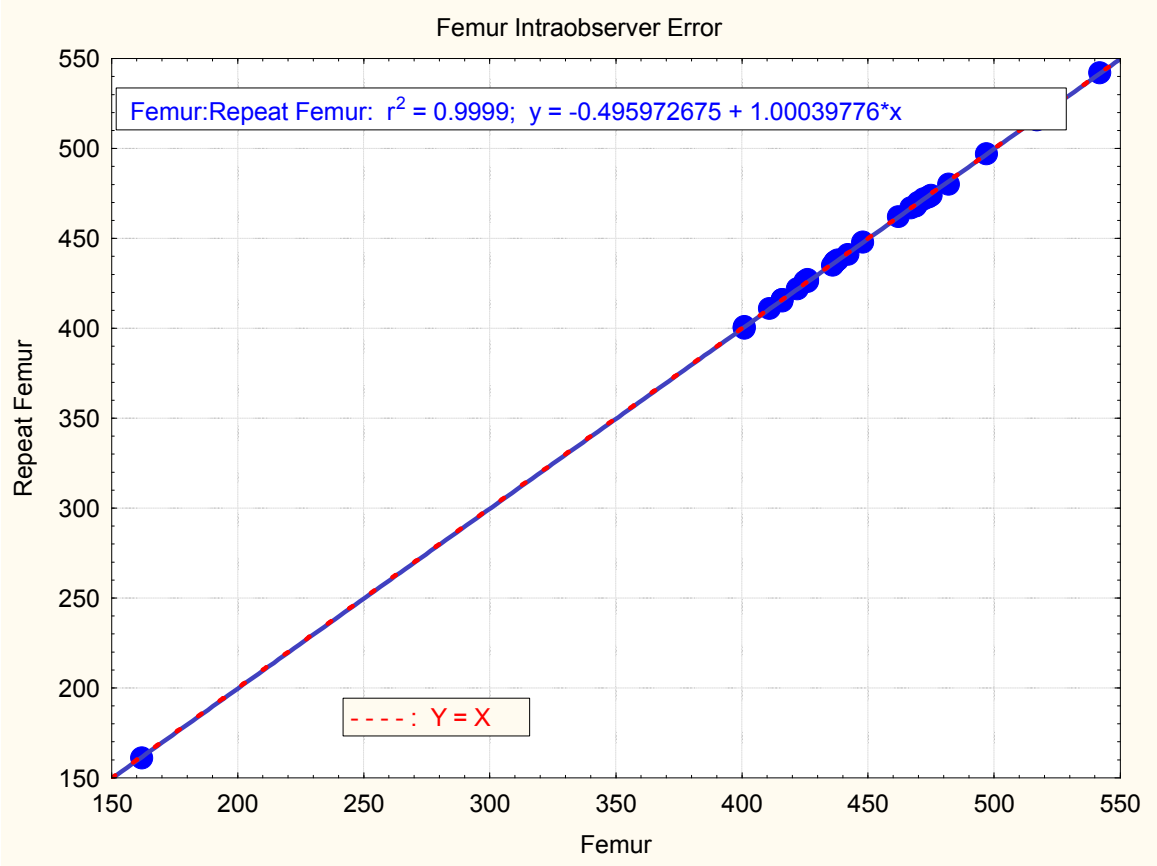

Figure B-4: Femur Intraobserver Error. The true regression line (in blue) is compared to an ideal regression line (in red) that reflects absolutely no error in the repeated measurements (i.e. Y = X with an intercept of zero and a slope equal to one).

Appendix C

Simulation Studies to Assess Parameter Estimation

As described in Chapter 2, Usher (2000) conducted simulation studies to determine if *mle* is able to recover known parameters from a simulated dataset. She did Monte Carlo simulations, and entered probabilistic hazard rates at the beginning of the simulations. She began each simulation with 1000 individuals in State 1 and ran the model until each individual was dead; at the end of the simulation, she knew the age-at-death distribution for the simulated population, and the state each individual was in before death. The dataset, a simulated cemetery sample, was entered into *mle*, and *mle* was always able to recover the k_1 and k_2 parameters. The program performed less well at recovering the parameters of the Siler model, but the recovered estimates were close to the known parameters; such a result is not surprising given that the parameters of the Siler model are correlated with one another, and many different combinations create the same age-at-death distribution. By conducting sensitivity analyses of sample size, Usher found that *mle* was able to recover reasonably precise and unbiased estimates of the *k* values, which are of primary interest in the present study, with samples of 100 to 200 individuals. The results these studies are shown in the following tables.

Table C-1: Simulation studies to test *mle*'s ability to recover parameter estimates for full model. From Usher (2000).

Parameter	Simulation value	*mle* estimate	Standard error
a_1	0.1	0.062113	0.009245
b_1	0.5	0.277246	0.076122
a_2	0	0.000000	0.007855
a_3	0.01	0.008754	0.003545
b_3	0.06	0.063546	0.008235
k_1	0.02	0.019730	0.001115
k_2	1.5	1.419019	0.128594

Table C-2: Simulation studies to test *mle*'s ability to recover parameter estimates for three-state model, with sample size of 100. From Usher (2000).

Parameter	Simulation value	*mle* estimate	Standard error
a_1	0.1	0.062071	0.032159
b_1	0.5	0.277407	0.294004
a_2	0	0.000001	0.034747
a_3	0.01	0.008841	0.016116
b_3	0.06	0.063508	0.036460
k_1	0.02	0.019809	0.003583
k_2	1.5	1.388059	0.400194

Table C-3: Simulation studies to test *mle*'s ability to recover parameter estimates for three-state model, with sample size of 200. From Usher (2000).

Parameter	Simulation value	*mle* estimate	Standard error
a_1	0.1	0.062201	0.021859
b_1	0.5	0.277368	0.195973
a_2	0	0.000000	0.024131
a_3	0.01	0.008759	0.011229
b_3	0.06	0.063547	0.025732
k_1	0.02	0.019731	0.002525
k_2	1.5	1.418761	0.288689

Table C-4: Simulation studies to test *mle*'s ability to recover parameter estimates for three-state model, with sample size of 250. From Usher (2000).

Parameter	Simulation value	*mle* estimate	Standard error
a_1	0.1	0.062136	0.019400
b_1	0.5	0.275213	0.171913
a_2	0	0.000000	0.020840
a_3	0.01	0.008657	0.009559
b_3	0.06	0.063693	0.022191
k_1	0.02	0.019634	0.002251
k_2	1.5	1.446414	0.258768

Table C-5: Simulation studies to test *mle*'s ability to recover parameter estimates for three-state model, with sample size of 500. From Usher (2000).

Parameter	Simulation value	*mle* estimate	Standard error
a_1	0.1	0.062062	0.013594
b_1	0.5	0.276908	0.114815
a_2	0	0.000000	0.011819
a_3	0.01	0.008757	0.005265
b_3	0.06	0.063543	0.012111
k_1	0.02	0.019736	0.001570
k_2	1.5	1.418980	0.181525

Appendix D

ESTIMATES: AGE, SEX, MODEL PARAMETERS

Age and Sex Estimates

Table D-1: Age and sex estimates for the Danish sample. The site code JAH 1-77 refers to St. Mikkel, and the site codes AT 82, AT 83, OBM 9784 and OBM 9785 refer to Albani Church. I determined sex for adults only.

Site code	Grave number	Sex	Age (years)
OBM 9785	606	Juvenile	0.0
OBM 9785	504	Juvenile	0.3
OBM 9785	522	Juvenile	0.3
JAH 1-77	234	Juvenile	0.5
JAH 1-77	247	Juvenile	0.5
OBM 9785	524	Juvenile	0.5
JAH 1-77	120	Juvenile	1.0
JAH 1-77	29	Juvenile	1.3
OBM 9785	508	Juvenile	1.3
AT83	86	Juvenile	1.5
JAH 1-77	122	Juvenile	1.5
JAH 1-77	221	Juvenile	1.5
JAH 1-77	41	Juvenile	1.5
JAH 1-77	93	Juvenile	1.5
OBM 9784	588	Juvenile	1.5
JAH 1-77	145	Juvenile	2.0
JAH 1-77	148	Juvenile	2.0
JAH 1-77	229	Juvenile	2.0
JAH 1-77	257	Juvenile	2.3
JAH 1-77	260	Juvenile	3.0
JAH 1-77	72	Juvenile	3.0
JAH 1-77	86	Juvenile	3.0
JAH 1-77	89	Juvenile	3.0
OBM 9784	558	Juvenile	3.0
OBM 9784	566	Juvenile	3.0
OBM 9784	575	Juvenile	3.0
OBM 9785	539	Juvenile	3.0

OBM 9785	561	Juvenile	3.5
JAH 1-77	15	Juvenile	4.0
JAH 1-77	180	Juvenile	4.0
JAH 1-77	210	Juvenile	4.0
JAH 1-77	215	Juvenile	4.0
JAH 1-77	39	Juvenile	4.0
OBM 9784	540	Juvenile	4.0
OBM 9784	582	Juvenile	4.0
OBM 9785	562	Juvenile	4.0
OBM 9785	802	Juvenile	4.0
OBM 9785	805	Juvenile	4.0
JAH 1-77	138	Juvenile	4.5
JAH 1-77	171	Juvenile	4.5
JAH 1-77	240	Juvenile	4.5
OBM 9784	586	Juvenile	4.5
OBM 9785	530	Juvenile	4.5
OBM 9785	544	Juvenile	4.5
OBM 9785	564	Juvenile	4.5
AT82	1	Juvenile	5.0
JAH 1-77	16	Juvenile	5.0
JAH 1-77	176	Juvenile	5.0
JAH 1-77	223	Juvenile	5.0
JAH 1-77	23	Juvenile	5.0
OBM 9785	549	Juvenile	5.0
AT83	93	Juvenile	5.5
JAH 1-77	11	Juvenile	5.5
JAH 1-77	246	Juvenile	5.5
JAH 1-77	258	Juvenile	5.5
JAH 1-77	30	Juvenile	5.5
OBM 9784	514	Juvenile	5.5
OBM 9785	505	Juvenile	5.5
OBM 9785	532	Juvenile	5.5
AT83	102	Juvenile	6.0
JAH 1-77	137	Juvenile	6.0
JAH 1-77	252	Juvenile	6.5
JAH 1-77	35	Juvenile	6.5
JAH 1-77	37	Juvenile	6.5
JAH 1-77	70	Juvenile	6.5
OBM 9785	808	Juvenile	6.5
JAH 1-77	80	Juvenile	7.5
OBM 9784	536	Juvenile	7.5
OBM 9784	528	Juvenile	8.5
OBM 9785	510	Juvenile	9.0
OBM 9785	705	Juvenile	9.0
OBM 9785	804	Juvenile	9.0
OBM 9785	814	Juvenile	9.0
JAH 1-77	157	Juvenile	9.5

JAH 1-77	3	Juvenile	9.5
JAH 1-77	32	Juvenile	9.5
OBM 9784	503	Juvenile	9.5
OBM 9785	503	Juvenile	9.5
OBM 9785	509	Juvenile	9.5
JAH 1-77	253	Juvenile	10.0
AT83	92	Juvenile	11.0
JAH 1-77	224	Juvenile	12.0
OBM 9784	542	Juvenile	13.0
JAH 1-77	276	Juvenile	14.0
AT83	104	Male	15.0
JAH 1-77	12	Female	15.0
JAH 1-77	130	Male	15.0
JAH 1-77	165	Male	15.0
JAH 1-77	212	Male	15.0
JAH 1-77	230	Male	15.0
JAH 1-77	48	Male	15.0
JAH 1-77	82	Female	15.0
JAH 1-77	99	Male	15.0
OBM 9784	553	Juvenile	15.0
OBM 9784	513	Male	15.0
OBM 9785	501	Male	15.0
OBM 9785	502	Female	15.0
OBM 9784	569	Male	15.1
OBM 9784	602	Male	15.3
JAH 1-77	18	Juvenile	15.5
JAH 1-77	194	Juvenile	15.5
OBM 9785	553	Juvenile	15.5
OBM 9785	603	Juvenile	15.5
OBM 9784	539	Juvenile	16.0
OBM 9785	517	Juvenile	16.0
OBM 9785	807	Juvenile	16.5
OBM 9785	816	Juvenile	16.5
OBM 9784	541	Juvenile	17.0
OBM 9784	564	Female	17.3
JAH 1-77	177	Female	17.4
OBM 9784	583	Female	17.7
OBM 9784	524	Female	17.9
OBM 9784	531	Juvenile	18.0
OBM 9785	525	Male	18.3
AT83	122	Female	18.7
OBM 9784	585	Female	19.0
OBM 9785	604	Female	19.0
OBM 9784	533	Juvenile	19.0
JAH 1-77	228	Female	19.2
OBM 9785	560	Male	19.2
JAH 1-77	90	Male	19.4

OBM 9784	562	Female	19.5
JAH 1-77	238	Female	19.7
OBM 9784	584	Female	19.7
OBM 9784	567	Female	19.8
JAH 1-77	146	Female	19.9
AT83	107	Male	20.2
JAH 1-77	51	Female	20.4
OBM 9784	556	Male	20.5
AT83	90	Male	20.8
JAH 1-77	62	Female	20.8
JAH 1-77	283	Female	21.1
OBM 9785	512	Female	21.3
OBM 9784	593	Male	21.5
JAH 1-77	85	Male	21.5
JAH 1-77	88	Female	22.1
OBM 9785	529	Male	22.3
JAH 1-77	147	Female	22.4
JAH 1-77	285	Male	22.8
AT83	45	Male	23.0
JAH 1-77	254	Female	23.1
JAH 1-77	118	Female	23.1
OBM 9785	712	Male	23.2
OBM 9785	543	Female	23.5
OBM 9785	507	Male	23.7
JAH 1-77	175	Female	23.9
JAH 1-77	121	Female	24.2
AT82	9	Male	24.2
OBM 9785	540	Female	24.4
OBM 9785	716	Female	24.5
OBM 9784	534	Female	25.1
JAH 1-77	102	Male	25.2
JAH 1-77	119	Male	25.3
JAH 1-77	71	Female	25.7
AT83	80	Male	25.7
JAH 1-77	192	Male	25.9
OBM 9784	576	Female	26.0
OBM 9785	516	Female	26.0
OBM 9785	556	Male	26.1
JAH 1-77	100	Female	26.3
JAH 1-77	187	Male	26.3
AT83	82	Male	26.5
OBM 9784	502	Male	26.6
OBM 9785	521	Male	26.6
JAH 1-77	166	Male	27.3
OBM 9785	701	Female	27.4
AT82	10	Male	27.8
OBM 9784	510	Male	27.8

JAH 1-77	141	Male	27.9
JAH 1-77	129	Female	28.0
OBM 9784	535	Male	28.1
JAH 1-77	10	Female	28.1
AT83	87	Male	28.4
OBM 9785	714	Female	28.4
JAH 1-77	104	Male	28.5
OBM 9785	704	Female	28.9
OBM 9784	547	Male	29.1
OBM 9785	810	Female	29.1
OBM 9784	546	Male	29.2
OBM 9785	546	Female	29.3
JAH 1-77	278	Male	29.4
OBM 9785	552	Male	29.4
OBM 9784	526	Male	29.5
JAH 1-77	67	Female	29.6
AT83	37	Male	29.6
JAH 1-77	161	Female	29.6
JAH 1-77	109	Female	29.9
OBM 9784	519	Female	30.0
OBM 9785	555	Male	30.1
JAH 1-77	196	Female	30.2
JAH 1-77	139	Female	30.3
JAH 1-77	149	Male	30.4
AT83	105	Male	30.5
JAH 1-77	22	Male	30.7
JAH 1-77	178	Male	30.7
JAH 1-77	255	Female	30.9
JAH 1-77	170	Female	31.1
JAH 1-77	271	Male	31.2
OBM 9785	815	Male	31.2
JAH 1-77	237	Male	31.2
AT83	128	Male	31.3
JAH 1-77	167	Male	31.4
OBM 9784	590	Female	31.5
OBM 9784	577	Male	31.6
OBM 9784	544	Male	31.8
JAH 1-77	151	Male	32.0
AT83	100	Male	32.0
JAH 1-77	197	Male	32.1
OBM 9784	579	Male	32.1
OBM 9785	703	Female	32.2
JAH 1-77	50	Male	32.3
JAH 1-77	53	Female	32.4
OBM 9785	523	Female	32.4
JAH 1-77	193	Male	32.4
JAH 1-77	156	Female	32.5

JAH 1-77	5	Male	32.8
OBM 9784	518	Male	33.0
OBM 9785	518	Female	33.1
JAH 1-77	232	Male	33.1
AT83	94	Male	33.5
OBM 9784	596	Male	33.6
OBM 9785	547	Male	33.6
AT83	40	Male	33.7
AT83	106	Male	33.9
JAH 1-77	236	Male	34.1
JAH 1-77	245	Male	34.2
OBM 9785	536	Female	34.3
AT83	83	Male	34.4
JAH 1-77	214	Female	34.6
JAH 1-77	169	Male	34.8
OBM 9784	523	Male	35.1
OBM 9784	552	Female	35.2
OBM 9784	538	Female	35.4
OBM 9784	543	Male	35.6
OBM 9785	607	Female	36.2
OBM 9785	531	Male	36.3
OBM 9784	507	Male	36.4
OBM 9784	522	Male	36.5
JAH 1-77	163	Male	36.9
OBM 9785	708	Female	37.2
OBM 9785	542	Male	38.1
JAH 1-77	244	Female	38.5
OBM 9784	578	Male	38.7
OBM 9784	554	Male	38.7
OBM 9784	603	Female	38.8
OBM 9784	506	Male	38.8
JAH 1-77	79	Male	38.9
JAH 1-77	251	Male	39.1
AT83	126	Female	39.4
JAH 1-77	68	Male	39.6
OBM 9784	505	Male	40.0
OBM 9785	534	Female	40.0
JAH 1-77	275	Female	40.3
AT83	118	Male	40.5
OBM 9784	560	Male	40.7
JAH 1-77	42	Female	41.1
OBM 9785	713	Male	41.8
OBM 9785	514	Female	42.4
OBM 9785	806	Female	42.6
JAH 1-77	209	Female	42.7
JAH 1-77	83	Female	43.7
JAH 1-77	66	Male	44.0

OBM 9784	548	Male	47.4
OBM 9785	554	Male	49.4
OBM 9784	572	Female	49.9
JAH 1-77	190	Female	50.2
OBM 9784	563	Female	53.1
OBM 9784	550	Male	54.6
OBM 9784	570	Male	55.7
OBM 9784	561	Female	55.9
OBM 9785	702	Female	57.5
OBM 9784	565	Male	59.0
OBM 9784	599	Female	59.3
JAH 1-77	200	Female	67.3
OBM 9785	541	Male	67.7
JAH 1-77	59	Male	68.7
OBM 9785	551	Female	69.9
OBM 9784	574	Male	71.0
JAH 1-77	133	Female	71.8
JAH 1-77	272	Male	73.1
JAH 1-77	195	Male	73.6
JAH 1-77	183	Female	74.0
JAH 1-77	205	Female	74.5
JAH 1-77	206	Female	74.8
AT82	13	Male	75.0
AT82	4	Male	75.0
JAH 1-77	46	Male	75.9
JAH 1-77	107	Female	77.8
OBM 9784	568	Female	78.9
JAH 1-77	115	Male	79.5
JAH 1-77	213	Female	80.5

Table D-2: Age and Sex estimates for East Smithfield. I determined sex for adults only.

Site code	Grave number	Sex	Age (years)
MIN 86	5840	Juvenile	0.0
MIN 86	11476	Juvenile	0.1
MIN 86	12726	Juvenile	0.3
MIN 86	12592	Juvenile	0.5
MIN 86	12642	Juvenile	0.5
MIN 86	12870	Juvenile	0.5
MIN 86	6075	Juvenile	1.0

MIN 86	8374	Juvenile	1.0
MIN 86	11228	Juvenile	1.3
MIN 86	12875	Juvenile	1.5
MIN 86	8362	Juvenile	1.5
MIN 86	11738	Juvenile	2.0
MIN 86	12578	Juvenile	2.0
MIN 86	11429	Juvenile	2.3
MIN 86	8037	Juvenile	2.5
MIN 86	8115	Juvenile	2.5
MIN 86	8284	Juvenile	2.5
MIN 86	11424	Juvenile	3.0
MIN 86	11845	Juvenile	3.0
MIN 86	12575	Juvenile	3.0
MIN 86	12753	Juvenile	3.0
MIN 86	8365	Juvenile	3.0
MIN 86	11608	Juvenile	3.5
MIN 86	8441	Juvenile	3.5
MIN 86	9965	Juvenile	3.5
MIN 86	11209	Juvenile	4.0
MIN 86	11415	Juvenile	4.0
MIN 86	11739	Juvenile	4.0
MIN 86	11931	Juvenile	4.0
MIN 86	11963	Juvenile	4.0
MIN 86	12582	Juvenile	4.0
MIN 86	5345	Juvenile	4.0
MIN 86	5983	Juvenile	4.0
MIN 86	7405	Juvenile	4.0
MIN 86	8389	Juvenile	4.0
MIN 86	5782	Juvenile	4.5
MIN 86	9844	Juvenile	4.5
MIN 86	11471	Juvenile	5.0
MIN 86	11491	Juvenile	5.0
MIN 86	11939	Juvenile	5.0
MIN 86	11977	Juvenile	5.0
MIN 86	12581	Juvenile	5.0
MIN 86	12795	Juvenile	5.0
MIN 86	12846	Juvenile	5.0
MIN 86	12850	Juvenile	5.0
MIN 86	7196	Juvenile	5.0
MIN 86	8375	Juvenile	5.0
MIN 86	9548	Juvenile	5.0
MIN 86	9599	Juvenile	5.0
MIN 86	9766	Juvenile	5.0
MIN 86	9789	Juvenile	5.0
MIN 86	12625	Juvenile	5.5
MIN 86	11229	Juvenile	6.0
MIN 86	11283	Juvenile	6.0

MIN 86	12796	Juvenile	6.0
MIN 86	5226	Juvenile	6.0
MIN 86	5290	Juvenile	6.0
MIN 86	8461	Juvenile	6.0
MIN 86	11237	Juvenile	6.5
MIN 86	11940	Juvenile	6.5
MIN 86	11736	Juvenile	7.0
MIN 86	12631	Juvenile	7.0
MIN 86	12707	Juvenile	8.0
MIN 86	12854	Juvenile	8.0
MIN 86	9734	Juvenile	8.0
MIN 86	12911	Juvenile	9.0
MIN 86	9547	Juvenile	9.0
MIN 86	9575	Juvenile	9.0
MIN 86	9914	Juvenile	9.0
MIN 86	11113	Juvenile	9.5
MIN 86	11976	Juvenile	9.5
MIN 86	12703	Juvenile	9.5
MIN 86	12792	Juvenile	9.5
MIN 86	12803	Juvenile	9.5
MIN 86	12822	Juvenile	9.5
MIN 86	12847	Juvenile	9.5
MIN 86	12857	Juvenile	9.5
MIN 86	5700	Juvenile	9.5
MIN 86	5731	Juvenile	9.5
MIN 86	7007	Juvenile	9.5
MIN 86	7052	Juvenile	9.5
MIN 86	8020	Juvenile	9.5
MIN 86	11114	Juvenile	10.0
MIN 86	11620	Juvenile	10.0
MIN 86	11621	Juvenile	10.0
MIN 86	11629	Juvenile	10.0
MIN 86	11724	Juvenile	10.0
MIN 86	12583	Juvenile	10.0
MIN 86	12655	Juvenile	10.0
MIN 86	12717	Juvenile	10.0
MIN 86	12722	Juvenile	10.0
MIN 86	12725	Juvenile	10.0
MIN 86	12807	Juvenile	10.0
MIN 86	12820	Juvenile	10.0
MIN 86	5179	Juvenile	10.0
MIN 86	5206	Juvenile	10.0
MIN 86	5293	Juvenile	10.0
MIN 86	7496	Juvenile	10.0
MIN 86	8130	Juvenile	10.0
MIN 86	8364	Juvenile	10.0
MIN 86	8366	Juvenile	10.0

MIN 86	8372	Juvenile	10.0
MIN 86	8400	Juvenile	10.0
MIN 86	8412	Juvenile	10.0
MIN 86	8491	Juvenile	10.0
MIN 86	9652	Juvenile	10.0
MIN 86	9722	Juvenile	10.0
MIN 86	9873	Juvenile	10.0
MIN 86	9894	Juvenile	10.0
MIN 86	9934	Juvenile	10.0
MIN 86	11251	Juvenile	10.5
MIN 86	5317	Juvenile	10.5
MIN 86	8171	Juvenile	10.5
MIN 86	8452	Juvenile	10.5
MIN 86	9898	Juvenile	10.5
MIN 86	11116	Juvenile	11.0
MIN 86	11624	Juvenile	11.0
MIN 86	11735	Juvenile	11.0
MIN 86	12630	Juvenile	11.0
MIN 86	12728	Juvenile	11.0
MIN 86	12739	Juvenile	11.0
MIN 86	8240	Juvenile	11.0
MIN 86	11645	Juvenile	11.5
MIN 86	11892	Juvenile	11.5
MIN 86	12778	Juvenile	11.5
MIN 86	6285	Juvenile	11.5
MIN 86	8263	Juvenile	11.5
MIN 86	9511	Juvenile	11.5
MIN 86	9512	Juvenile	11.5
MIN 86	9932	Juvenile	11.5
MIN 86	11314	Juvenile	12.0
MIN 86	12666	Juvenile	12.0
MIN 86	12914	Juvenile	12.0
MIN 86	5181	Juvenile	12.0
MIN 86	6500	Juvenile	12.0
MIN 86	11626	Juvenile	12.5
MIN 86	11728	Juvenile	12.5
MIN 86	9848	Juvenile	12.5
MIN 86	5864	Juvenile	13.0
MIN 86	6518	Juvenile	13.0
MIN 86	7391	Juvenile	13.0
MIN 86	9961	Juvenile	13.0
MIN 86	11743	Juvenile	14.0
MIN 86	12552	Juvenile	14.0
MIN 86	6552	Juvenile	14.0
MIN 86	8449	Juvenile	14.0
MIN 86	9853	Juvenile	14.0
MIN 86	9915	Juvenile	14.5

MIN 86	8345	Juvenile	15.0
MIN 86	9576	Juvenile	15.0
MIN 86	11740	Male	15.0
MIN 86	11742	Female	15.0
MIN 86	11747	Male	15.0
MIN 86	11914	Female	15.0
MIN 86	12656	Male	15.0
MIN 86	12773	Female	15.0
MIN 86	12815	Male	15.0
MIN 86	5344	Male	15.0
MIN 86	5902	Female	15.0
MIN 86	6177	Female	15.0
MIN 86	6196	Male	15.0
MIN 86	6287	Male	15.0
MIN 86	6368	Male	15.0
MIN 86	6549	Female	15.0
MIN 86	6653	Female	15.0
MIN 86	7064	Male	15.0
MIN 86	7079	Male	15.0
MIN 86	7094	Male	15.0
MIN 86	7366	Male	15.0
MIN 86	7381	Female	15.0
MIN 86	9545	Female	15.0
MIN 86	9702	Female	15.0
MIN 86	9741	Male	15.0
MIN 86	11472	Juvenile	15.5
MIN 86	11619	Juvenile	15.5
MIN 86	12599	Juvenile	15.5
MIN 86	12667	Juvenile	15.5
MIN 86	12793	Juvenile	15.5
MIN 86	12797	Juvenile	15.5
MIN 86	12858	Juvenile	15.5
MIN 86	12900	Juvenile	15.5
MIN 86	5209	Juvenile	15.5
MIN 86	5346	Juvenile	15.5
MIN 86	5701	Juvenile	15.5
MIN 86	6555	Juvenile	15.5
MIN 86	8281	Juvenile	15.5
MIN 86	8329	Juvenile	15.5
MIN 86	9913	Juvenile	15.5
MIN 86	11726	Juvenile	16.0
MIN 86	11971	Juvenile	16.0
MIN 86	12565	Juvenile	16.0
MIN 86	12801	Juvenile	16.0
MIN 86	12843	Juvenile	16.0
MIN 86	5212	Juvenile	16.0
MIN 86	6130	Juvenile	16.0

MIN 86	8291	Juvenile	16.0
MIN 86	8379	Juvenile	16.0
MIN 86	8451	Juvenile	16.0
MIN 86	9675	Juvenile	16.0
MIN 86	8057	Juvenile	16.5
MIN 86	9585	Juvenile	16.5
MIN 86	9796	Juvenile	16.5
MIN 86	5862	Male	16.6
MIN 86	8260	Female	16.6
MIN 86	11252	Male	16.9
MIN 86	11631	Juvenile	17.0
MIN 86	12636	Juvenile	17.0
MIN 86	12744	Juvenile	17.0
MIN 86	6442	Juvenile	17.0
MIN 86	7005	Juvenile	17.0
MIN 86	9815	Juvenile	17.0
MIN 86	8266	Female	17.2
MIN 86	12915	Male	17.3
MIN 86	8299	Male	17.4
MIN 86	11112	Male	17.5
MIN 86	11495	Female	17.5
MIN 86	5283	Female	17.6
MIN 86	9939	Male	17.8
MIN 86	6639	Female	17.8
MIN 86	5728	Male	17.8
MIN 86	8411	Male	17.9
MIN 86	6428	Female	17.9
MIN 86	8382	Female	18.0
MIN 86	11632	Female	18.1
MIN 86	12684	Male	18.2
MIN 86	6418	Female	18.2
MIN 86	11627	Male	18.7
MIN 86	6467	Female	18.7
MIN 86	11625	Male	18.9
MIN 86	6625	Juvenile	19.0
MIN 86	11807	Male	19.4
MIN 86	9807	Female	19.5
MIN 86	9540	Female	19.8
MIN 86	12522	Male	19.9
MIN 86	5281	Male	20.2
MIN 86	6558	Female	20.3
MIN 86	11109	Female	20.3
MIN 86	8217	Female	20.4
MIN 86	8126	Female	20.4
MIN 86	5343	Male	20.5
MIN 86	12690	Female	20.6
MIN 86	11780	Female	20.7

MIN 86	11244	Female	20.8
MIN 86	6644	Female	20.8
MIN 86	6462	Female	20.8
MIN 86	5291	Male	20.9
MIN 86	12525	Male	21.0
MIN 86	6654	Female	21.1
MIN 86	12763	Male	21.1
MIN 86	11016	Male	21.1
MIN 86	8380	Male	21.4
MIN 86	12859	Male	21.4
MIN 86	11028	Male	21.6
MIN 86	11499	Female	21.6
MIN 86	6228	Male	21.7
MIN 86	5170	Female	21.7
MIN 86	12601	Female	21.8
MIN 86	9707	Male	22.0
MIN 86	9525	Male	22.3
MIN 86	8293	Male	22.6
MIN 86	12643	Female	22.7
MIN 86	6398	Female	22.7
MIN 86	6499	Female	22.7
MIN 86	9695	Male	22.7
MIN 86	6409	Female	22.8
MIN 86	9674	Female	22.8
MIN 86	11622	Male	22.8
MIN 86	8378	Female	22.8
MIN 86	8212	Male	22.9
MIN 86	7307	Female	22.9
MIN 86	8414	Female	22.9
MIN 86	12727	Male	23.0
MIN 86	6515	Female	23.0
MIN 86	6545	Male	23.0
MIN 86	11432	Male	23.2
MIN 86	12566	Male	23.9
MIN 86	7065	Male	24.1
MIN 86	7156	Male	24.1
MIN 86	6665	Female	24.1
MIN 86	6108	Female	24.2
MIN 86	6120	Female	24.2
MIN 86	8041	Female	24.2
MIN 86	8424	Female	24.3
MIN 86	11053	Male	24.3
MIN 86	12523	Male	24.4
MIN 86	9832	Male	24.5
MIN 86	5861	Male	24.5
MIN 86	8489	Male	24.6
MIN 86	12839	Female	24.8

MIN 86	11982	Male	25.0
MIN 86	9990	Male	25.0
MIN 86	12506	Female	25.1
MIN 86	12748	Male	25.8
MIN 86	5274	Male	26.3
MIN 86	8277	Male	26.4
MIN 86	11431	Female	26.5
MIN 86	9770	Male	26.5
MIN 86	9797	Male	26.7
MIN 86	5869	Male	27.1
MIN 86	11973	Female	27.2
MIN 86	9849	Male	27.2
MIN 86	11439	Female	27.3
MIN 86	11480	Female	27.5
MIN 86	8202	Male	27.5
MIN 86	6388	Female	27.6
MIN 86	9522	Male	27.6
MIN 86	5779	Female	27.6
MIN 86	5284	Male	28.0
MIN 86	11618	Male	28.0
MIN 86	5265	Male	28.2
MIN 86	11430	Female	28.2
MIN 86	6319	Female	28.2
MIN 86	11115	Male	28.2
MIN 86	11737	Male	28.3
MIN 86	6441	Male	28.3
MIN 86	11254	Female	28.3
MIN 86	5280	Female	28.5
MIN 86	12626	Male	28.7
MIN 86	7332	Male	28.9
MIN 86	9782	Female	28.9
MIN 86	8427	Female	29.0
MIN 86	12553	Female	29.3
MIN 86	12729	Male	29.3
MIN 86	12644	Male	29.4
MIN 86	8450	Male	29.4
MIN 86	6483	Female	29.7
MIN 86	8075	Male	29.8
MIN 86	11426	Male	29.9
MIN 86	8311	Male	29.9
MIN 86	5326	Female	30.5
MIN 86	12632	Male	30.5
MIN 86	8124	Female	30.6
MIN 86	11838	Female	30.6
MIN 86	7163	Female	30.7
MIN 86	5859	Female	30.7
MIN 86	9035	Male	30.9

MIN 86	9823	Male	30.9
MIN 86	12652	Male	31.0
MIN 86	11911	Female	31.1
MIN 86	8082	Male	31.1
MIN 86	5741	Female	31.2
MIN 86	11938	Female	31.3
MIN 86	5275	Female	31.3
MIN 86	5730	Male	31.3
MIN 86	6393	Male	31.4
MIN 86	12782	Male	31.5
MIN 86	11604	Female	31.6
MIN 86	8305	Male	31.6
MIN 86	12856	Female	31.6
MIN 86	7055	Male	31.7
MIN 86	12774	Female	31.7
MIN 86	11449	Male	31.7
MIN 86	5960	Male	31.7
MIN 86	6097	Male	31.8
MIN 86	11110	Male	31.8
MIN 86	11232	Male	31.9
MIN 86	12913	Male	31.9
MIN 86	8341	Male	32.0
MIN 86	7447	Female	32.1
MIN 86	9517	Male	32.2
MIN 86	8343	Male	32.3
MIN 86	11249	Male	32.3
MIN 86	11111	Female	32.3
MIN 86	9731	Male	32.3
MIN 86	9856	Male	32.4
MIN 86	11427	Male	32.5
MIN 86	12903	Male	32.7
MIN 86	12691	Female	32.7
MIN 86	6415	Male	33.0
MIN 86	5285	Male	33.3
MIN 86	11978	Male	33.4
MIN 86	8191	Male	33.4
MIN 86	11118	Male	33.5
MIN 86	12799	Male	33.6
MIN 86	12884	Male	33.6
MIN 86	11117	Female	33.7
MIN 86	12849	Male	33.7
MIN 86	8360	Female	33.9
MIN 86	6405	Male	33.9
MIN 86	12814	Female	34.2
MIN 86	11313	Male	34.2
MIN 86	8392	Female	34.4
MIN 86	11425	Male	34.6

MIN 86	12721	Male	34.8
MIN 86	12710	Male	34.8
MIN 86	12798	Male	34.9
MIN 86	6509	Male	34.9
MIN 86	8317	Female	35.0
MIN 86	12503	Male	35.1
MIN 86	11124	Male	35.2
MIN 86	12700	Female	35.2
MIN 86	11970	Female	35.3
MIN 86	12897	Female	35.4
MIN 86	8416	Female	36.0
MIN 86	11606	Male	36.1
MIN 86	12634	Male	36.1
MIN 86	5871	Male	36.2
MIN 86	6400	Female	36.4
MIN 86	5858	Male	36.9
MIN 86	5829	Female	36.9
MIN 86	12816	Male	37.2
MIN 86	11628	Male	37.6
MIN 86	6443	Male	37.7
MIN 86	11428	Male	38.0
MIN 86	12586	Male	38.3
MIN 86	5805	Female	38.5
MIN 86	12890	Female	38.6
MIN 86	6532	Female	38.8
MIN 86	6452	Female	38.9
MIN 86	5870	Male	39.1
MIN 86	12567	Male	39.3
MIN 86	11951	Female	39.7
MIN 86	12835	Male	39.9
MIN 86	5296	Male	39.9
MIN 86	5860	Female	40.4
MIN 86	11972	Female	41.7
MIN 86	7402	Male	41.8
MIN 86	6102	Female	42.5
MIN 86	9574	Male	42.8
MIN 86	11193	Male	43.0
MIN 86	9032	Male	43.0
MIN 86	11108	Female	43.1
MIN 86	5261	Male	44.1
MIN 86	9819	Female	44.2
MIN 86	6676	Male	44.6
MIN 86	12790	Female	45.9
MIN 86	8038	Female	46.5
MIN 86	11488	Female	46.8
MIN 86	7285	Male	46.8
MIN 86	6313	Male	46.9

MIN 86	9963	Female	47.2
MIN 86	7025	Male	48.0
MIN 86	12906	Male	48.4
MIN 86	11944	Male	48.5
MIN 86	12647	Female	50.1
MIN 86	12723	Female	57.2
MIN 86	12813	Male	57.2
MIN 86	5272	Male	57.5
MIN 86	5702	Male	60.4
MIN 86	5282	Male	62.2
MIN 86	6477	Male	62.3
MIN 86	6481	Male	63.3
MIN 86	6383	Female	63.4
MIN 86	8257	Male	63.5
MIN 86	6431	Male	64.4
MIN 86	9524	Female	64.5
MIN 86	12802	Female	66.3
MIN 86	8235	Male	68.6
MIN 86	7089	Male	68.6
MIN 86	11121	Female	69.9
MIN 86	7432	Male	70.2
MIN 86	6524	Male	70.5
MIN 86	12548	Female	70.8
MIN 86	8161	Female	70.8
MIN 86	7375	Male	71.1
MIN 86	8099	Male	71.9
MIN 86	6475	Female	72.3
MIN 86	5271	Male	72.8
MIN 86	8415	Male	73.1
MIN 86	8393	Female	73.4
MIN 86	5940	Female	73.6
MIN 86	8272	Female	73.7
MIN 86	11496	Female	73.8
MIN 86	7015	Male	73.9
MIN 86	6527	Female	74.0
MIN 86	8251	Female	74.0
MIN 86	5263	Male	74.4
MIN 86	6216	Female	74.9
MIN 86	9066	Male	75.1
MIN 86	5295	Male	75.2
MIN 86	8072	Male	75.9
MIN 86	7311	Male	76.1
MIN 86	12694	Female	76.2
MIN 86	8229	Male	76.4
MIN 86	6628	Female	76.5
MIN 86	11234	Male	76.6
MIN 86	11934	Female	76.7

MIN 86	6512	Male	76.8
MIN 86	9038	Male	77.0
MIN 86	7363	Male	77.2
MIN 86	6412	Male	77.4
MIN 86	6327	Female	77.5
MIN 86	12635	Male	77.7
MIN 86	11857	Female	78.1
MIN 86	5294	Male	79.7

Parameter Estimates

The following tables provide all of the parameter estimates from the analyses of selectivity with respect to frailty. The sample size for each skeletal lesion refers to the number of individuals for whom I was able to score the presence or absence of that lesion. I estimated all seven parameters of the Usher model (the five Siler parameters, and the two *k* parameters) simultaneously using *mle*. For all lesions in both East Smithfield and Denmark, several Siler estimates are not significantly different from zero, likely because of the reduced samples sizes. However, for the analyses of selectivity, the only parameter of importance is the k_2 parameter (the relative risk associated with skeletal lesions), and those estimates were significant in the majority of cases.

Table D-3: Tibial Lesion estimates: East Smithfield (n = 248). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	793371
β_1	0.0	1159366184
α_2	0.0	793371
α_3	0.02	0.2
β_3	0.02	0.07
k_1	0.02*	0.002
k_2	4.6*	1.3

Table D4: Tibial Lesion estimates: Denmark (n = 179). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.005	0.2
β_1	0.004	0.3
α_2	0.0	0.2
α_3	0.007	0.06
β_3	0.02	0.09
k_1	0.04*	0.004
k_2	10*	4.2

Table D-5: Estimates for porotic hyperostosis on the frontal bone: East Smithfield (n = 121). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	0.8
β_1	0.01	10710434307
α_2	0.00	0.8
α_3	0.02	0.2
β_3	0.03	0.1
k_1	0.01*	0.002
k_2	1.5*	0.4

Table D-6: Estimates for porotic hyperostosis on the frontal bone: Denmark (n = 104). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	0.05
β_1	0.3	25209678
α_2	0.00	0.07
α_3	0.02	0.06
β_3	0.02	0.04
k_1	0.01*	0.002
k_2	3.2*	1.7

Table D-7: Estimates for porotic hyperostosis on the left parietal bone: East Smithfield (n = 142). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	0.2
β_1	0.01	5873297725
α_2	0.00	0.5
α_3	0.01	0.2
β_3	0.2	0.2
k_1	0.03*	0.005
k_2	4.6*	2.0

Table D-8: Estimates for porotic hyperostosis on the left parietal bone: Denmark (n = 110). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.02	0.02
β_1	0.3	0.5
α_2	0.00	0.02
α_3	0.005	0.01
β_3	0.03	0.03
k_1	0.03*	0.004
k_2	8.6*	3.8

Table D-9: Estimates for porotic hyperostosis on the right parietal bone: East Smithfield (n = 139). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	353364
β_1	0.00	141259287970
α_2	0.00	353364
α_3	0.009	0.3
β_3	0.02	0.3
k_1	0.04*	0.008
k_2	4.2*	1.7

Table D-10: Estimates for porotic hyperostosis on the right parietal bone: Denmark (n = 114). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.01	0.02
β_1	0.4	0.9
α_2	0.0	0.02
α_3	0.005	0.01
β_3	0.03	0.03
k_1	0.03*	0.004
k_2	9.0*	4.1

Table D-11: Estimates for porotic hyperostosis on the occipital bone: East Smithfield (n = 133). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	335114
β_1	0.0	113875376797
α_2	0.0	335114
α_3	0.009	0.2
β_3	0.02	0.2
k_1	0.03*	0.004
k_2	4.4	2.0

Table D-12: Estimates for porotic hyperostosis on the occipital bone: Denmark (n = 115). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.01	0.02
β_1	0.3	0.8
α_2	0.00	0.05
α_3	0.01	0.04
β_3	0.02	0.04
k_1	0.03*	0.004
k_2	5.0*	2.2

Table D-13: Estimates for cribra orbitalia: East Smithfield (n = 124). Estimates that are not significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	0.06
β_1	0.05	29902724952
α_2	0.0	0.1
α_3	0.02	0.08
β_3	0.03	0.06
k_1	0.01*	0.002
k_2	3.6*	1.8

Table D-14: Estimates for cribra orbitalia: Denmark (n = 75). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.01	8779055
β_1	0.0	19
α_2	0.0	8779055
α_3	0.02	0.2
β_3	0.02	0.09
k_1	0.02*	0.004
k_2	4.34*	2.3

Table D-15: Estimates for enamel hypoplasia on the maxillary incisor: East Smithfield (n = 85). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	3203434
β_1	0.0	13279691470
α_2	0.0	3203434
α_3	0.02	1.3
β_3	0.02	0.5
k_1	0.02*	0.006
k_2	3.8	3.6

Table D-16: Estimates for enamel hypoplasia on the maxillary incisor: Denmark (n = 49). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.00	0.1
β_1	0.0	46069
α_2	0.0	2.22
α_3	0.03	2.2
β_3	0.01	0.3
k_1	0.05*	0.01
k_2	4.0	3.8

Table D-17: Estimates for enamel hypoplasia on the mandibular canine: East Smithfield (n = 94). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	196606
β_1	0.0	17193247028
α_2	0.0	196606
α_3	0.01	1.4
β_3	0.02	1.0
k_1	0.04*	0.02
k_2	5.1	4.9

Table D-18: Estimates for enamel hypoplasia on the mandibular canine: Denmark (n = 55). None of the estimates are significantly different from zero.

Parameter	Estimate	Standard Error
α_1	0.0	1444980
β_1	0.0	9.6
α_2	0.0	1444980
α_3	0.01	0.5
β_3	0.02	0.7
k_1	0.08	0.07
k_2	6.1	11

Table D-19: Estimates for enamel hypoplasia on the maxillary canine: East Smithfield (n = 80). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	3
β_1	0.0	40062683743
α_2	0.0	3.5
α_3	0.0	0.4
β_3	0.03	0.3
k_1	0.04*	0.01
k_2	4.2	2.7

Table D-20: Estimates for enamel hypoplasia on the maxillary canine: Denmark (n = 42). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	55815165
β_1	0.0	25
α_2	0.0	55815165
α_3	0.01	1.1
β_3	0.07	1.2
k_1	0.07*	0.03
k_2	1.7	1.6

Table D-21: Estimates for enamel hypoplasia on the first molar: East Smithfield (n = 108). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	1573526
β_1	0.0	14976740768
α_2	0.0	1573526
α_3	0.02	1.0
β_3	0.02	0.4
k_1	0.01*	0.004
k_2	3.7	2.1

Table D-22: Estimates for enamel hypoplasia on the first molar: Denmark (n = 63). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	10808229
β_1	0.0	3572218774
α_2	0.0	10808229
α_3	0.04	2.3
β_3	0.04	0.8
k_1	0.04*	0.01
k_2	2.1	1.1

Table D-23: Estimates for enamel hypoplasia on the second: East Smithfield (n = 124). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	0.4
β_1	0.2	650856698609
α_2	0.0	0.06
α_3	0.02	0.05
β_3	0.03	0.04
k_1	0.01*	0.003
k_2	3.8	2.1

Table D-24: Estimates for enamel hypoplasia on the second: Denmark (n = 45). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	458920
β_1	0.0	31318021001918
α_2	0.0	458920
α_3	0.02	0.8
β_3	0.06	0.8
k_1	0.03*	0.009
k_2	1.7	1.4

Table D-25: Estimates for short femur length: East Smithfield (n = 154). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	1.8
β_1	0.05	851775492190
α_2	0.0	0.1
α_3	0.01	0.04
β_3	0.04	0.1
k_1	0.004*	0.001
k_2	3.4	2.2

Table D26: Estimates for short femur length: Denmark (n = 120). Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_1	0.0	1.7
β_1	0.2	14353
α_2	0.0	0.05
α_3	0.01	0.02
β_3	0.05	0.03
k_1	0.004	0.004
k_2	4.4	9.7

Table D-27: Estimates for sex: East Smithfield (n = 299). Adult males are considered as having a lesion. Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_2	0.0	0.02
α_3	0.01	0.01
β_3	0.03*	0.02
k_2	1.0*	0.1

Table D-28: Estimates for sex: Denmark (n = 195). Adult males are considered as having a lesion. Estimates that are significantly different from zero are marked with an asterisk.

Parameter	Estimate	Standard Error
α_2	0.0	0.01
α_3	0.01	0.01
β_3	0.04*	0.01
k_2	1.1*	0.1

Appendix E

Hazards Analysis and Likelihood Equations

The Usher model incorporates hazards or survival analysis; the hazard in this application is the risk of dying at a given age, and conversely, survival is the probability of individuals surviving to a given age. In hazards analysis, three related functions are of special importance: the survival function, the hazard function, and the probability density function of ages at death. The survival function $S(a)$ is the probability that an individual survives from birth to age *a:*

$$S(a) = \mathrm{e}^{-\int_0^a \mu(x)dx} \qquad \textbf{E1}$$

where $\mu(x)$ is the age specific mortality rate (the hazard function). Because $S(a)$ is a probability, the value must range between zero and one for any particular age; at birth, the value is one, and the survival function can only go down or remain constant as age increases and individuals die. As age increases, the survival function approaches zero.

The probability density function (PDF) of ages at death is analogous to the age at death distribution for a population. The PDF of ages at death, $f(a)$ can be derived from $S(a)$:

$$f(a) = -\frac{\mathrm{d}S(a)}{\mathrm{d}a} \qquad \textbf{E2}$$

The hazard function $\mu(a)$ is related to the survival function and the PDF in the following way:

$$\mu(a) = \frac{-\mathrm{d}\ \ln S(a)}{\mathrm{d}a} = -\frac{1}{S(a)}\frac{\mathrm{d}S(a)}{\mathrm{d}a} = \frac{f(a)}{S(a)} \qquad \textbf{E3}$$

By rearranging equation E3, one can see that the PDF is determined by the age specific risk of mortality and the number of individuals who survive to each age:

$$f(a) = \mu(a)S(a) \qquad \textbf{E4}$$

Because of the relationships among these three functions, one can determine age-specific mortality rates and survival functions from age-at-death distributions estimated from skeletal samples by transforming the age-at-death distributions into probability density functions (Wood et al., 2002).

As mentioned above, for this study, I specified the baseline risk of death from State 1, $\mu_{13}(a)$, in the three-state Usher model as a Siler mortality model (equation 2.4), and the hazard of developing lesions, $\mu_{12}(a)$, as a constant k_1:

$$\mu_{12}(a) = k_1 \qquad \textbf{E5}$$

I modeled the risk of death for individuals in State 2, $\mu_{23}(a)$, as proportional to the baseline risk of death from State 1:

$$\mu_{23}(a) = k_2 \mu_{13}(a) \qquad \textbf{E6}$$

The survival function for not developing skeletal lesions (i.e. surviving in State 1) until age a_{12}, is:

$$S_{12}(a_{12}) = e^{-\int_0^{a_{12}} \mu_{12}(x)dx} \qquad \textbf{E7}$$

By substituting equation E5 for $\mu_{12}(x)$, equation E7 becomes:

$$S_{12}(a_{12}) = e^{-k_1 a_{12}} \qquad \textbf{E8}$$

The survival function for surviving in State 2 until death at age a_{23} is:

$$S_{23}(a_{23}) = e^{-\int_0^{a_{23}} \mu_{23}(x)dx} \qquad \textbf{E9}$$

By substituting equation E6 for $\mu_{23}(x)$, equation E9 becomes:

$$S_{23}(a_{23}) = e^{-\int_0^{a_{23}} k_2\mu_{13}(x)dx} \qquad \textbf{E10}$$

Equation E10 can be rearranged as follows:

$$S_{23}(a_{23}) = e^{k_2-\int_0^{a_{23}} \mu_{13}(x)dx} = \left(e^{-\int_0^{a_{23}} \mu_{13}(x)dx} \right)^{k_2} \qquad \textbf{E11}$$

The portion of equation E11 in parentheses is equivalent to a survival function, so the equation can be rewritten as:

$$S_{23}(a_{23}) = \left(S_{13}(a)\right)^{k_2} \qquad \textbf{E12}$$

Equation E12 shows the relationship between survival in State 2 and survival in State 1.

The likelihood of dying from State 1 is equivalent to an individual's probability of survival in State 1 until age *a* and risk of dying at exact age *a*. Individuals who survive in State 1 have neither developed skeletal lesions (i.e. have not made the transition to State 2) nor died (i.e. have not made the transition to State 3):

$$L_{a|I=0} = S_{13}(a)\,S_{12}(a)\,\mu_{13}(a) \qquad \textbf{E13}$$

where $I = 0$ indicates that an individual did not have a skeletal lesion at death. Using equation E4 and replacing $S_{12}(a)$ with equation E8, equation E13 can be simplified to:

$$L_{a|I=0} = f_{13}(a)\,e^{-k_1 a} \qquad \textbf{E14}$$

The likelihood of dying from State 2 (i.e. dying after forming a skeletal lesion) is the likelihood that an individual will survive in State 1 until age a_{12}, make the transition from State 1 to State 2 at exact age a_{12}, survive in State 2 until age a_{23}, and then die (make the transition from State 2 to State 3) at exact age a_{23}:

$$L_{a|I=1} = \int_0^a S_{12}(a_{12})\,S_{13}(a_{12})\,\mu_{12}(a_{12})\,e^{-\int_{a_{12}}^{a}\mu_{23}(x)dx}\,\mu_{23}(a)\,da_{12} \qquad \textbf{E15}$$

where I=1 indicates the presence of a skeletal lesion, $S_{12}(a_{12})$ is the probability of survival in State 1 without developing a lesion, $S_{13}(a_{12})$ is the probability of survival in

State 1 without dying, $\mu_{12}(a_{12})$ is the hazard of developing a lesion at age a_{12}, $e^{-\int_{a_{12}}^{a} \mu_{23}(x)dx}$ is a partial survival function for the probability of surviving from the age at which a skeletal lesion develops (a_{12}) until death at age a, and $\mu_{23}(a)$ is the hazard of dying with a skeletal lesion. The likelihood equation must be integrated over all possible ages between 0 and age at death a, because for most skeletal lesions we do not know the age at which the lesion forms. The likelihood equation can be simplified and rewritten in terms of $S_{13}(a)$, $f_{13}(a)$, and $\mu_{13}(a)$; first, the exponential expression can be rewritten:

$$e^{-\int_{a_{12}}^{a} \mu_{23}(x)dx} = e^{-\int_{0}^{a} \mu_{23}(x)dx + \int_{0}^{a_{12}} \mu_{23}(x)dx} \quad \textbf{E16}$$

$$= e^{-\int_{0}^{a} \mu_{23}(x)dx} e^{\int_{0}^{a_{12}} \mu_{23}(x)dx} \quad \textbf{E17}$$

$$= \frac{e^{-\int_{0}^{a} \mu_{23}(x)dx}}{e^{-\int_{0}^{a_{12}} \mu_{23}(x)dx}} \quad \textbf{E18}$$

Both the numerator and denominator of expression E18 are survival functions, so the original exponential expression can be written in terms of the corresponding survival functions:

$$e^{-\int_{a_{12}}^{a}\mu_{23}(x)dx} = \frac{S_{23}(a)}{S_{23}(a_{12})} \qquad \textbf{E19}$$

By using equation E20 in place of the exponential expression in equation E15, the likelihood equation can be simplified as:

$$L_{a|I=1} = \int_{0}^{a} S_{12}(a_{12})\, S_{13}(a_{12})\, \mu_{12}(a_{12}) \frac{S_{23}(a)}{S_{23}(a_{12})} \mu_{23}(a)\, da_{12} \qquad \textbf{E20}$$

Using equations E5, E6, E8, and E12, equation E20 can be written:

$$L_{a|I=1} = \int_{0}^{a} e^{-k_1 a_{12}} S_{13}(a_{12})\, k_1 \frac{\left(S_{13}(a)\right)^{k_2}}{\left(S_{13}(a_{12})\right)^{k_2}} k_2 \mu_{13}(a)\, da_{12} \qquad \textbf{E21}$$

Equation E21 can be rearranged, and the constants and functions not containing the constant of integration a_{12} can be removed from the integral; the likelihood of dying with a skeletal lesion is therefore:

$$L_{a|I=1} = k_1 k_2 \mu_{13}(a)\left(S_{13}(a)\right)^{k_2} \int_{0}^{a} e^{-k_1 a_{12}} \left(S_{13}(a_{12})\right)^{1-k_2} da_{12} \qquad \textbf{E22}$$

All of the above equations are from Usher (2000).

Appendix F

Example of *mle* program

The following *mle* program is designed for estimating the relative risk of death associated with proliferative tibial lesions within the East Smithfield cemetery; it is typical of the programs used in my analyses of relative risk. The program is in bold, and explanations are provided alongside the program. Any statements in the program enclosed in brackets {} are comments or reminders for me, and not actually instructions for the program.

Program	Explanation
MLE	
TITLE = "EStibia.mle"	Title of the *mle* program
DATAFILE("EStibiasiler.dat")	The dataset read by *mle* for this program
OUTFILE("EStibia.out")	Name of the outfile produced when the program is complete
METHOD = SIMPLEX	Maximization method
MAXITER = 1500	Maximum number of iterations permitted in estimating the model parameters
EPSILON = 0.0000001	Convergence criterion: determines how precisely the parameters are to be estimated. Convergence occurs when the change in log-likelihood from one iteration to the next falls below this number

```
INTEGRATE_METHOD = I_TRAP_OPEN
```
Integration method: open trapezoidal

```
INTEGRATE_N = 80
```
Number of interations: 80

```
EXP_HAZARD = FALSE
```
Determines how the proportional hazards are modeled on the baseline hazard: *h(t) = h(t)'p*

```
maxage = 120
```
Maximum age

```
DATA   {data come from East Smithfield}

  age              FIELD 1   {estimated age at death,
                             in years or fractions of years}

  transition_state FIELD 2   {1 means no lesion,
                              2 means lesion}

END {of data statement}
```
The data statement: tells *mle* how to read the data file; data are arranged in columns (fields)

```
{These are the start values.}
a1s = 0.0000000001
b1s = 1.35
a2s = 0.026
a3s = 0.000024
b3s = 0.27
k1s = 1.0
k2s = 1.0
```
The start values tell *mle* where to begin the maximum likelihood estimation. The start value represents the first "guess" of the maximum.

```
MODEL
```
The model statement specifies what is being estimated and any specifications about how the parameters are to be estimated

```
PREASSIGN
    BEGIN
```
The preassign functions allows the parameter values to be pre-computed for efficiency

The following statements define the parameters to be estimated. These are the parameters that are to be changed while *mle* maximizes the likelihood. These statements tell *mle* the minimum and maximum

allowable values and the start values for each parameter.

```
  a1x = PARAM a1 LOW = 0 HIGH = 1 START = a1s FORM = NUMBER  END
  b1x = PARAM b1 LOW = 0 HIGH = 2 START = b1s FORM = NUMBER  END
  a2x = PARAM a2 LOW = 0 HIGH = 1 START = a2s FORM = NUMBER  END
  a3x = PARAM a3 LOW = 0 HIGH = 1 START = a3s FORM = NUMBER  END
  b3x = PARAM b3 LOW = 0 HIGH = 1 START = b3s FORM = NUMBER  END
  k1x = PARAM k1 LOW = 0 HIGH = 10 START = k1s FORM = NUMBER END
  k2x = PARAM k2 LOW = 0 HIGH = 10 START = k2s FORM = NUMBER END
  k2comp = 1 - k2  {pre-compute for efficiency}
  negk1 = -k1  {pre-compute for efficiency}
  k1k2 = k1*k2 {precompute for efficiency}

END {of preassigning}



{ The likelihood goes here}
DATA
```

The DATA function tells *mle* to use data from the DATA statement one at a time in the likelihood

```
 IF transition_state = 1 THEN
```

The likelihood of dying without having a lesion (dying from State 1)

```
    PDF SILER (age, age, 0) a1 b1 a2 a3 b3 END {this is the pdf}
    *e^(negk1*age)

     ELSE
```

The likelihood of dying with a lesions (dying from State 2)

```
        k1k2*PDF SILER (age, age, age) a1, b1, a2, a3, b3 END  {hazard}
         *PDF SILER (age, -1, 0) a1, b1, a2, a3, b3 END^k2
           *INTEGRATE t01 (0, age)
             e^(negk1*t01)*PDF SILER (t01, -1, 0)  a1, b1, a2, a3, b3 END^k2comp
           END  {integrate}

   END {of if-else}
  END {of data function}
 END {of preassign statement}
RUN
 FULL
```

This tells *mle* to estimate the full model (seven parameters)

```
 END {of model}

END {of program}
```

Output

```
254 lines read from file EStibiasiler.dat
254 Observations kept and 0 observations dropped.

NAME          age  transition
MEAN   22.0661772  1.48818898
VAR    124.850962  0.25084809
STDEV  11.1736727  0.50084737
MIN    0.12500000  1.00000000
MAX    41.6950000  2.00000000



EStibia.mle
Program file: EStibia.mle
Input data file name: EStibiasiler.dat
    2 variables read.

Model 1 Run 1 : EStibia.mle

METHOD = SIMPLEX  MAXITER = 1500  MAXEVALS = 100000
Convergence at EPSILON = 0.0000001000
LogLikelihood: -1152.777 AIC: 2319.5545 Del(LL): 0.0000000977
Iterations: 754 Function evals: 972 Time: 00:01:19
Converged normally

Results with estimated standard errors.  (80 evals)
Solution with 7 free parameters
Name Form       Estimate         Std Error          t
a1           7.01674E-0012   0.028788519852     2.4373E-0010
b1          1.999999997793   1566.143807047    0.00127702194
a2          0.023924984096   0.005011434318    4.77407915145
a3          0.000027418801   0.000055672992    0.49249735078
b3          0.256219454108   0.065398321489    3.91782920835
k1          0.027874804617   0.002851443261    9.77568272270
k2          1.516783556644   0.296604753654    5.11382079336
```

VITA

SHARON N. DEWITTE

E-mail: sdewitte@albany.edu

2006 Ph.D. Anthropology, The Pennsylvania State University
2001 M.A., Anthropology, The Pennsylvania State University
1999 B.A., Anthropology, Sonoma State University, CA
1996 A.A., Anthropology, Santa Rosa Junior College, CA

Research Interests: paleodemography, paleoepidemiology, paleopathology, osteology, infectious diseases, the Black Death

Fellowships/Grants:

2005 National Science Foundation Doctoral Dissertation Improvement Grant
2004 Wenner-Gren Individual Research Grant
2004 Research and Graduate Studies Office grant, The Pennsylvania State University
2004 Hill Foundation Fellowship, Anthropology Department, The Pennsylvania State University
2003 Baker Fund, Anthropology Department, The Pennsylvania State University
2002 American Scandinavian Foundation Fellowship

Professional Employment

2005 – 2006 Assistant Professor, Sociology/Anthropology Department, Shippensburg University:
Introduction to Physical Anthropology, Forensic Anthropology

1999-2004 Teaching/Research Assistant, Anthropology Department, The Pennsylvania State University:
Introduction to Biological Anthropology, Lab Methods, Ethnographic Film, Social Organization

Selected Publications and Professional Presentations:

DeWitte, SN (2006) Paleodemographic study of the Black Death. Dissertation. The Pennsylvania State University, University Park.

DeWitte, SN (2005) Paleoepidemiological study of the Black Death. Poster presented at the 20th Annual Pennsylvania State Graduate Exhibition, University Park, PA.

Sparks CS and **SN DeWitte-Aviña** (2004). Population dynamics under catastrophic mortality: the case of the Black Death. Poster presented at the 29th Annual Meeting of the Human Biology Association, Tampa, FL.

Wood JW, RJ Ferrell, **SN DeWitte-Aviña** (2002). The role of infectious diseases in human populations: New evidence from the Black Death in England, 1348-1350. Presented at the annual meeting of the American Association of Physical Anthropologists, Buffalo, N.Y.

Wood JW, RJ Ferrell, **SN DeWitte-Aviña**, DJ Holman (2002). Mortality differentials during the Black Death in England, 1349. Presented at the annual meeting of the Human Biology Council, Buffalo, N.Y.

Wood JW, RJ Ferrell, and SN **DeWitte-Aviña** (2003) The temporal dynamics of the fourteenth-century Black Death: New evidence from English ecclesiastical records. *Human Biology* 75:427-448.