2017

Paleodemographic and Biochemical Analysis of Urbanization, Famine, and Mortality

Brittany S. Walter
University of South Carolina

PALEODEMOGRAPHIC AND BIOCHEMICAL ANALYSIS OF URBANIZATION, FAMINE, AND MORTALITY

by

Brittany S. Walter

Bachelor of Arts
University of Florida, 2010

Master of Arts
University of Central Florida, 2012

Submitted in Partial Fulfillment of the Requirements

For the Degree of Doctor of Philosophy in

Anthropology

College of Arts and Sciences

University of South Carolina

2017

Accepted by:

Sharon N. DeWitte, Major Professor

Charles Cobb, Committee Member

Tosha Dupras, Committee Member

Rebecca Redfern, Committee Member

Cheryl L. Addy, Vice Provost and Dean of the Graduate School

Dedication

Dedicated to the strongest woman I have ever known,

Carol Walter,

for motivating me in every way

Acknowledgements

There are several people that supported and encouraged me while working toward the completion of this dissertation. First and foremost, Sharon DeWitte must be thanked for her unwavering patience, her stellar mentorship, and, most importantly, for teaching me how to fail at things gracefully. Also, I would like to thank my committee members: Drs. Charles Cobb, Tosha Dupras, and Rebecca Redfern for helping me navigate through this large research project and for providing invaluable feedback that, no doubt, greatly improved the design and final state of this dissertation. I would also like to thank some extremely accommodating Brits who made access to the skeletal assemblages used for this project possible: Jelena Bekvalac and Rebecca Redfern at the Museum of London, and Kevin Booth and Simon Mays from English Heritage. Also, a big thank you is due everyone at the Defense POW/MIA Accounting Agency Laboratory at Offutt, particularly Drs. Franklin Damann and Katie Skorpinski, for motivating and enabling me to finish this dissertation in a timely manner.

Also, I must acknowledge the funding sources that made the data collection, sampling, and expensive isotopic analyses for this project possible: the National Science Foundation for the Dissertation Research Improvement Grant (BCS-15440208) and Sharon DeWitte's grant (BCS-1261682), the SPARC grant awarded by the Office of Research at the University of South Carolina, the Ceny Walker Graduate

Scholarship from the Walker Institute at the University of South Carolina, and travel scholarships from the Department of Anthropology at the University of South Carolina.

There were several people that kept me sane through the steps it took to get to this point: my sister, Raquel Walter, for keeping me motivated and supporting me no matter what; Meagan Conway, my archaeology counterpart, for being the unwavering optimist and my own personal cheerleader; Anaïs Parada for her "unique" advice for *all* things; Carrie Healy, Joanna Fletcher, and Shella Mercado for keeping me laughing since my first semester of graduate school; Hannah Rogers, my travel buddy, for making sure I always did more abroad than collect data; and all the Columbia, South Carolina, folks for not letting me take academic life too seriously. Thank you to Hines and Bettis for being the most supportive (and adorable) French Bulldogs an owner could ask for. Finally, the biggest thanks of all to my loving husband, Tyler, who saw me in the thick of it, but understood to just hand me a beer and back away until it was over.

Abstract

Urbanization is a transitional period often associated with deteriorating population health and increased mortality, as the rapid increase of population density in urban centers facilitates the transmission of infectious diseases, unsanitary living conditions, and precarious food supplies. Research on the transition to an urban environment in the past offers a temporal depth to our understanding of the consequences of urbanization that cannot be accomplished through examination of contemporary populations. This project integrates paleodemographic (hazard analysis) and biochemical (stable isotope analysis) approaches to examine the health and diet of inhabitants in late medieval England (c. 1120-1539 CE), specifically the relationship between pathology and nutrition during urbanization and incidences of famine. Skeletal data and samples from the urban St Mary Spital cemetery in London and contemporaneous rural St Peter's cemetery in Barton-upon-Humber, England were analyzed to: (1) determine how survivability patterns in medieval London changed over time as a result of intensive urbanization; (2) evaluate how temporal changes in mortality and survivability of medieval London compare to the rural population of England; (3) investigate the relationship between diet and health during the transition to a more urban environment; and (4) examine how potential biochemical markers of famine are manifested in medieval London using stable isotope analysis, including an

innovative incremental dentine analysis method. The results of hazard and survival analyses suggest that the effects of urbanization on survivability and mortality varied by sex and age. Specifically, results show that, for adults, urbanization may have been more detrimental to health than the rural environment but the implementation of sanitation directives in London as urbanization progressed may have improved living conditions through time. Analyses of stable isotope values from bone collagen samples from St Mary Spital reveal different dietary patterns between age groups and through time. Differences in isotope values between famine- and non-famine periods may be the consequence of famine or migration rather than differences in physiological stress. This project serves as the first bioarchaeological study to investigate urbanization using both paleodemographic and biochemical approaches, providing a comprehensive and nuanced depiction of health and nutrition in an urbanizing environment.

Table of Contents

List of Tables

List of Figures

Chapter 1 - Introduction

Urban intensification is often associated with declines in health and elevated mortality. The rapid increase in population density in urban centers facilitates the transmission of infectious diseases, unsanitary living conditions, and precarious food supplies. The failure of a population to adapt to these changes should be evident in higher rates of mortality, increased levels of physiological stress, and an increased prevalence of metabolic and infectious disease. Moreover, sub-populations may be predisposed to these detrimental effects, increasing risk of death for certain subgroups.

Populations undergoing urban expansion are of considerable interest to current public health studies, with epidemiologists investigating the extent to which urban factors are important in the etiology of chronic and infectious diseases (Kumar et al., 2006; Popkin, 1999; Preston et al., 1981). Research on urbanization in the past provides temporal depth to our understanding of the consequences of urbanism that cannot be accomplished through the examination of contemporary populations. Further, research on preindustrial populations can be informative about how urbanization has affected human populations, and how humans have adapted to and continue to adapt to environmental factors of urbanism (Storey, 2006). Moreover, applying a biocultural approach connects osteological data with evidence for culture, environment, and society and is integral to understanding the inherent dynamic relationship between the

physiological body and the external social and cultural influences that affect adaptability to the environment (Goodman et al., 1988; Roberts, 2000; Zuckerman et al., 2012). This study examines the health and diet of inhabitants in urbanizing Late Medieval London (c. 1120-1539 CE) to investigate the relationship between health and nutrition during the development of urban environments in the medieval period.

1.1 Research objectives and hypotheses

For this project, paleodemographic and biochemical approaches are used to address the following objectives:

Objective 1: Determine how mortality patterns in medieval London changed over time as a result of increased urbanization.

Objective 2: Evaluate how temporal changes in mortality of medieval London compare to those in a contemporaneous rural population.

Objective 3: Investigate the relationship between diet and health during the transition to a more urban environment by evaluating changes in isotope values through time and within different sub-populations.

Objective 4: Examine how potential biochemical markers of famine are manifested in medieval London using stable isotope analysis, including an innovative incremental dentine analysis method.

The focus of this dissertation is on human remains excavated from the cemetery of St Mary Spital (SRP98) (c. 1120-1540 CE), curated by the Museum of London, to investigate how mortality in London changed during urbanization and several famines.

Skeletons from SRP98 were analyzed to reconstruct the demography and overall health of the population within consecutive periods using paleodemographic approaches. Age and sex patterns of mortality were compared within and between different time periods to evaluate differences in mortality during the transition to a more urban environment. These data were compared to a contemporaneous rural population in England (St Peter's cemetery), using the same paleodemographic approaches, to evaluate differences between urban and rural mortality during urbanization. Bone samples from SRP98 were collected for carbon and nitrogen stable isotope analysis to evaluate dietary patterns within the population and across time periods (see 4.2.3 Bone collagen samples for sampling details). These data were integrated with paleodemographic (e.g. age and sex) and paleopathological data from SRP98 to evaluate differences in health patterns within the population that may have been the result of dietary variation. Tooth samples were collected from individuals interred during famine and non-famine periods to conduct incremental dentine stable isotope analysis for examining dietary and metabolic stress experienced prior to death (see 5.2.3.2 Tooth samples for sampling details). These analyses were done to test the following hypotheses:

Hypothesis 1: Given the environmental stressors associated with urbanization, which could have resulted in elevated rates of mortality from both chronic and acute diseases, mortality rates will be higher in the urban sample than in the rural sample.

Hypothesis 2: Given evidence from historic and living populations that migrants experience elevated risks of morbidity and mortality following immigration to

urban centers and that migrants into London during this period were predominantly adolescents and young females, risks of mortality will be highest for subadults and females in the urban sample.

Hypothesis 3: Given evidence from the environmental archaeological record and primary source documents that detrimental environmental factors intensify as urbanization increases over time, survivability will decrease from the earliest to the latest period in the urban sample.

Hypothesis 4: Given that historic and living populations exhibit change in carbon and nitrogen values as a result of nutritional stress and that individuals undergoing famine adapt their diet to available resources, individuals buried in graves associated with famine crises will have different carbon and nitrogen values than those interred in attritional burials.

1.2 Bioarchaeology and paleodemography

Bioarchaeological and paleodemographic analyses of human remains have the potential to provide valuable information concerning past population dynamics and offer a temporal depth to our understanding of human adaptation that cannot be accomplished with living populations (Buikstra and Beck, 2006; Martin et al., 2013). However, issues of preservation biases, differential burial and recovery, inaccuracies associated with age estimation, and hidden heterogeneity and selective mortality within the population are frequently encountered by bioarchaeologists when reconstructing mortality patterns or when interpreting the health of past populations from skeletal

remains. It was not until a few decades ago that osteologists made an effort to go beyond traditional approaches of interpreting mortality patterns from mean age-at-death and making direct conclusions about living populations from lesion frequencies in cemetery collections (Hoppa, 2002). Paleodemographers have begun to apply innovative techniques that account for biases and methodological shortcomings to provide a more accurate depiction of populations in the past, as discussed below. In this section, the fundamental problems and inherent biases of osteological samples that complicate an interpretation of past demographic and health patterns are discussed and also the ways in which osteologists have endeavored to account for these shortcomings.

1.2.1 Intrinsic and extrinsic biases

Before excavated skeletal remains are even considered for paleodemographic analyses, they undergo several postmortem processes that determine whether they make their way into an osteological collection; and if they are selected for analysis, if the remains are preserved well enough to be accurately analyzed (Milner et al., 2007). These postmortem processes inevitably produce a biased sample, leaving osteologists to carefully consider how these biases could have potentially affected the analysis and interpretation of the remains. Differential preservation or excavation (e.g. undernumeration of subadult and old adults) and mortuary practices (who is buried and where they are buried), among other factors, influence which skeletons are available for analysis. Therefore, in many cases, archaeological samples are not representative of the populations from which they are associated, because of intrinsic and extrinsic

preservation, differential mortuary practices, and recovery methods. It is important to recognize these biases and try to account for them because biased samples can largely influence the reconstructed demography of the sample.

Intrinsic influences of preservation do not act uniformly on all human remains. Bone diagenesis (the degradation of bone as a result of taphonomic processes) is related to the intrinsic anatomical features of the skeleton. For example, infant bones are the most vulnerable to detrimental taphonomic processes after burial because they are small, fragile, and less mineralized than adult bones (Bello et al., 2006). Similarly, elderly individuals with fragile bones as a result of osteoporosis may not preserve as well as young adults (Walker et al., 1988). Additionally, extrinsic influences such as variation in soil pH and mineral and water content within a cemetery can cause differential preservation of skeletons. Gordon and Buikstra (1981) found that soil type and preservation of subadult remains were correlated with subadult skeletons undergoing diagenesis at a higher rate in poor soil conditions (i.e. decreased pH) when compared to adult skeletal remains in the same cemetery. On the other hand, a subsequent study considering age and sex differences in preservation at a different site did not find a significant relationship but did find that shallower burials did not preserve as well as deep burials (Stojanowski et al., 2002).

Mortuary practices of the past and recovery methods can also influence which remains become a part of an osteological collection. Infants are often subjected to differential burial treatments that result in their remains being excluded from cemeteries, and subsequently excluded from excavation (Buckberry, 2000; Lucy, 1994).

Some societies may regard infants as not "fully human" until they have reached a specific development stage, and are thus treated differently within the mortuary record. For example, in 17th century Ireland, unbaptized infants and other members of society that were considered unsuitable by the Roman Catholic Church for burial in consecrated ground, were buried away from the general cemetery in separate burial grounds named *Clilliní* (Murphy, 2011). Moreover, subadult graves may be shallower than the graves of their adult counterparts, potentially subjecting them to harsher taphonomic processes than traditional deeper graves (Lucy, 1994). Furthermore, inadequacies in recovery by archaeologists that have not been trained in properly identifying small subadult bones can result in these remains being missed (Bello et al., 2006).

Regarding presence or absence of certain remains in osteological collections, it is important to note that an absence of evidence is not necessarily evidence of absence. Given the poor preservation of most cemetery collections, it is not safe to assume that skeletons of all age groups have been preserved equally well. The issue of preservation must be addressed before a range of cultural or biological explanations are suggested regarding the frequency (or absence) of certain burials at any cemetery site. As emphasized by Walker (1995:40), ''a logical starting point in dealing with this problem is to develop better techniques for assessing the condition of skeletal collections.'' Before remains are analyzed, preservation for each individual must be assessed so the degree of preservation of the entire population can be taken into account when making interpretations using the skeletal sample. Moreover, knowledge gained from the archaeological and historical context about the population could potentially maximize

recovery of skeletal remains or help explain why some subgroups may be missing from the osteological collection.

1.2.2 Analytical influences

In addition to the inherent preservation bias associated with osteological collections, there are several issues concerning analytical methods that can influence how mortality patterns are reconstructed. Ultimately, sex determination and age estimation are the building blocks for any paleodemographic analysis. Preservation can greatly influence which bones are available for analysis, oftentimes leaving osteologists with little more than a few features of the skeleton to assess sex or estimate age, which can lead to sex misclassification or inaccurate age estimation.

With respect to estimating the sex of a skeleton or parts of a skeleton, some skeletal features are more reliable than others (e.g. the pelvis *vs*. long bones), and different populations have different degrees of sexual dimorphism. Although most anthropological populations have a fairly balanced sex ratio, paleodemographic studies often have revealed an overabundance of males in skeletal collections (Weiss, 1972). This may be the result of osteologists favoring cranial features as a means of determining sex, which could lead to classifying all but the most gracile of individuals as males. Poor preservation of pelvic bones, which have a higher degree of accuracy in sex determination methods than other skeletal elements, often necessitates sex determination based solely on the skull or overall robusticity of the bones available. Younger males, who may be more gracile, can be misclassified as females, and older

females, who may develop more masculine cranial features (usually as a result of age), may be misidentified as males (Meindl et al., 1985a). Walker (1995) assessed the accuracy of morphoscopic skull traits (traits that are assessed morphologically with the naked eye) using different populations and determined that interobserver error using these morphological classifications of sex was low with a slight bias toward female classification for all subgroups. He also found that Native American females were frequently classified as more robust when compared to the other subgroups (i.e. looked more male), and that Native Americans, in general, showed markedly less sexual dimorphism. Thus, it is essential for osteologists to have an idea about the degree of sexual dimorphism within the population being in question before analysis. In general, sex determination when using individual skeletal features or a combination thereof have shown to range from 68% to 95% (Phenice, 1969; Sutherland and Suchey, 1991; Walker, 2008; Williams and Rogers, 2006).

While sex determination methods have high accuracy rates, age estimation techniques are not as accurate. Bones and teeth undergo age-related changes throughout life; however, different bones, and boney features, depending on their structure and function, reflect different aspects of the aging process (i.e. epiphyseal fusion is useful for estimating subadult age, while the pubic symphysis is used for adult age estimation). If age-at-death cannot be accurately estimated from skeletal remains, osteologists are not able to make valid statements about any human characteristics that are subject to age-dependent change.

Estimating age-at-death of adults is not as straightforward as age estimation of

subadults, because it is based on assessing the degenerative changes of boney features rather than growth and development patterns (e.g. dental eruption and epiphyseal fusion), which occur at a fairly consistent rate. Degenerative changes, however, are influenced by several factors, including the environment, genetics, and physical activity throughout life (Bello et al., 2006). Moreover, issues with traditional methods that use reference populations to estimate the age of individual, and assign these individuals to predetermined age categories, can bias age-at-death estimates.

Age-at-death of adult skeletons is generally estimated by assessing the degree of degenerative changes that have occurred on non-mobile joints (e.g. cranial sutures, pubic symphyses, and sacro-iliac joints). Usually, age estimates using these features are then aggregated to produce an age-at-death distribution showing how many individuals died at certain age intervals. Techniques that have been developed using skeletal features and age-at-distributions, however, have been found to produce inaccurate results (Bocquet-Appel and Masset, 1982). One example, the Todd method (1921), a widely used aging technique that estimates age using the pubic symphysis, often overages young adults (Brooks, 1955). McKern and Stewart (1957) also used the pubic symphysis to develop an age estimation method from a sample of dead Korean War soldiers (mostly young adult men). Meindl et al. (1985b) determined that McKern and Stewart's method was notably biased for underaging individuals. Rather than blaming the inaccuracy of these methods on the imprecision of degenerative change rates, osteologists began to realize that these biases may actually be the result of imposing an age distribution from the reference collection (the collection used to develop the age

estimation method) on to the target collection (the collection from which age is being assessed using the age estimation method) (Bocquet-Appel and Masset, 1982).

In 1982, Bocquet-Appel and Masset sparked a great debate in biological anthropology concerning the inadequacy of paleodemographic reconstructions. The authors explicitly outlined shortcomings in age estimation techniques that had been briefly mentioned by osteologists in previous papers (e.g. Howell, 1976). They contended (1) that age-at-death distributions from human remains were artifacts of the age distribution of reference samples used to construct the method (often referred to as *age mimicry*), and (2) that age estimation techniques employed by anthropologists were inherently inaccurate because of the low correlation between skeletal age and chronological age. They also argued that paleodemographers incorrectly assumed that age-related changes of skeletal features used to estimate age are constant through time.

Age mimicry is best described mathematically (Konigsberg and Frankenberg, 1992; Konigsberg and Frankenberg, 1994). When a bioarchaeologist is estimating the age of a skeleton, he or she is assessing the probability that the skeleton is a certain age, given that it has age indicator *c [P(a|c)]*. When using a reference sample to determine this, however, one is observing the probability that an individual has age indicator *c* given that they are age *a [P(a|c)]*. In order to determine *P(c|a)* traditional aging techniques use inverse regression to regress age on the age indicator. Determining *P(c|a)* from *P(a|c)* thus necessities the assumption that the target population's age distribution is the same as the reference population's age distribution, which is

extremely unlikely. This improbable assumption results in an imposed age distribution from the reference collection onto the target population. One example of this is McKern and Stewart's (1957) age estimation method mentioned above. A majority of the reference collection was comprised of young males, thus the age-at death distribution developed using McKern and Stewart's method will exhibit a higher young adult mortality and underestimate the age-at-death of individual's within the target sample. Bocquet-Appel and Masset (1982) suggested that paleodemography was doomed because of insuperable methodological issues concerning age estimation of skeletal remains and bid "A Farwell to Paleodemography". However, instead of affirming the premature demise of paleodemography as a result of Bocquet-Appel and Masset's article, osteologists took on the challenge and began to develop innovative methods for correcting issues of age estimation.

In 1999 and 2000, at the Max Planck Institute for Demographic Research, workshops were held to discuss methods for estimating adult age and constructing mortality profiles using human remains (Hoppa and Vaupel, 2002). What came from these meetings was a theoretical framework that attempted to address the issues outlined by critics of paleodemography (Hoppa and Vaupel, 2002). This theoretical framework was dubbed the Rostock Manifesto after its place of origin. There are four major elements to the Rostock Manifesto: (1) osteologists must develop skeletal age indicator stages that can be reliably related to chronological age; (2) osteologists must also develop methods to estimate $P(c|a)$, which is the probability of observing certain skeletal age-indicators given age from a known-age reference sample; (3) osteologists

must recognize that they are interested in calculating *P(a|c),* which is the probability that a skeleton is an age given that a certain age indicator has been observed and that this can be accomplished by applying Bayes' Theorem and the probability distribution of age-at-death for the target population; and (4) osteologists must estimate or model the probability distribution of age-at-death for the target population to calculate *P(a|c)* (Hoppa and Vaupel, 2002). In order to achieve the elements of this framework, four steps to the Rostock protocol must be implemented: (1) use nonparametric methods to estimate *P(c|a,)* which is the probability of observing certain skeletal age-indicators given age death (or *weight functions*); (2) select a parametric distribution that will be used to represent the age-at-death distribution of the target sample; (3) combine the parametric distribution with the weight functions and frequencies of observed osteological age categories to calculate maximum likelihood estimates of the parameters of the age-at-death distribution that maximize the likelihood of the observed age-indicator frequencies within the target sample; and then (4) use Bayes' Theorem to obtain age-at-death for individuals within the target samples (Hoppa and Vaupel, 2002). With traditional methods, individual ages-at-death are first estimated from skeletal age-indicators and these estimates are combined to produce a sample-age-at-death distribution of the target sample. The Rostock protocol, however, is basically the reverse of this, with the age-at-death distribution estimated before age-at-death for any individual is determined.

Müller and colleagues (2002) tested the accuracy of the Rostock protocol to ensure that the methods outlined in the protocol did not impose the age distribution of

the reference sample on the target samples, and to verify that the method was able to obtain accurate estimate of the parameters of the target sample age-at-death distribution. Using a simulated target population and the Gompertz parametric distribution, they found that the estimated target age-at-death distribution was similar to the true target sample distribution and differed from the reference sample. Müller et al. thus successfully demonstrated that the Rostock protocol is able to produce unbiased age estimates, but with a little more work by the osteologist.

Though a theoretically sound method for age estimation and mortality profile reconstruction, the Rostock protocol has its limitations. As the number of age indicators increase, the number of parameter values to be obtained increase as well, which requires a large sample size to allow for compliance of this method (Boldsen et al., 2002). Luckily, an alternative approach – transition analysis – has been developed that accommodates small sample sizes characteristic of osteological samples but still implements elements of the Rostock protocol (Boldsen et al., 2002).

Transition analysis, the age estimation technique used for this project, uses data from a known-age reference sample to obtain the conditional probability $P(c|a)$ of a skeleton exhibiting a certain age indicator state or a suite of age indicator stages, given the individual's known age (i.e. the weight function described above). This conditional probability is then combined with a prior distribution of ages-at-death using Bayes' Theorem to estimate the posterior probability that a skeleton of unknown age died at a certain age, given that the skeleton displays a suite of particular age-indicator stages. Skeletal age indicators used in this method include several features of the pubic

symphysis, iliac auricular surface, and cranial sutures (Boldsen et al., 2002). The difference between transition analysis and the full Rostock protocol is that transition analysis uses an assumed uniform prior distribution or an informative prior distribution of ages-at-death. The uniform prior assumes all ages-at-death have an equal chance of being represented in the age-at-death distribution. An informative prior can also be applied in place of the uniform prior if the osteologist has reason to believe that the age-at-death distribution was similar. This project uses ADBOU (Anthropological Database, Odense University) age estimation software with an informative prior based on seventeenth-century rural Danish parish records to estimation age, as the age-at-death distribution from these records is more similar to medieval English populations than the uniform prior distribution.

In addition to transition analysis accommodating small sample sizes, this method provides point estimates of age and their associated standard errors, rather than the arbitrary age intervals of traditional age estimation techniques. The method also provides more precise age estimates for older adults unlike traditional methods that usually provide open-ended ages for terminal age categories (e.g. 50 and older), limiting information about senescent mortality. Moreover, transition analysis has been successfully used to estimate age-at-death in several archaeological skeletal collections (DeWitte and Wood, 2008; Kreger, 2010; Milner and Boldsen, 2012; Wittwer-Backofen et al., 2008).

1.2.3 Hazard analysis

Limitations to the reconstruction of mortality profiles do not end at biased age estimation methods. Osteologists used to assume that skeletons in a cemetery were reasonably representative of the living populations that produced them (Hoppa, 2002). One method that has been widely used to summarize various aspects of mortality is abridged life tables. Until relatively recently, paleodemographers relied on the construction of life tables, which summarize information such as age-specific mortality rates, survivorship, and life expectancy, to study mortality in skeletal samples. The life table approach, however, relies on several unrealistic assumptions. The application of this method when the assumptions are not true can distort the estimated mortality patterns of the population, resulting in inaccurate interpretations of the population in question (e.g. Lovejoy et al., 1977). One alternative to this approach that has become more popular in paleodemographic studies is the application of hazard analysis, which avoids several of the unrealistic assumptions used in the life table approach (Wood et al., 2002), potentially providing a more accurate depiction of mortality when using skeletal assemblages.

The most obvious limitation when applying the life table approach is that in order to calculate mortality estimates, paleodemographers must assume that the population under study was stationary. A stationary population is closed to migration, has an intrinsic rate of increase (*r*) equal to zero and unchanging age-specific fertility and mortality rates, and a stable age distribution. If all of these criteria are met, the age-at-death distribution of skeletons is equivalent to the number of people who die within

an age interval (${}_nd_x$ column) of a life table (Gage, 1985). The major problem with these criteria, however, is that they are rarely met (Gage, 1985). The assumption that a population was stationary is problematic because we know that populations have undergone periods of increased and decreased growth from environmental and cultural changes (Johansson and Horowitz, 1986).

Assuming stationarity of a population allows osteologists to easily and simply construct abridged life tables based solely on the estimated ages at death of the skeletons, since the proportion of individuals in each category should be constant over time in a stationary population (Johansson and Horowitz, 1986; Sattenspiel and Harpending, 1983). Calculation of life expectancy at birth is then conveniently equal to the mean age-at-death of the skeletal sample. Osteologists would then use the mean age-at-death of the skeletal sample to compare levels of mortality between past populations (Sattenspiel and Harpending, 1983). When this assumption of stationarity is applied and mean age-at-death is used to estimate life expectancy, osteologists could misinterpret a difference in fertility and population growth for mortality (if the stationary assumption is violated by a nonzero intrinsic rate of increase) (Wood et al., 1992). For example, if a population was increasing in size (and thus not stationary because population growth would be present), more individuals would be born into the population, resulting in more individuals alive in each successive birth cohort and thus increasing the number of people at risk of death at each age. Even though the mortality of the population may be the same, more young individuals would be entering the mortality sample, making it seem as though mortality had increased, when it actually

stayed the same. Conversely, a decrease in population size would result in fewer individuals in each successive birth cohort. This would, in turn, result in a greater proportion of adults in the mortality sample, which would then be interpreted as a decrease in mortality. Furthermore, if the population is not stationary, life tables require the estimates of the central mortality for each age interval, which cannot be accomplished from small skeletal samples and without knowledge of the original population risk (i.e. the numbers of people who survive to each age interval and are thus at risk of dying within the interval) (Wood et al., 2002).

Unlike the life table approach, applying parametric mortality models allows the osteologist to assume that the population under study is *stable*- not stationary. Compared to stationarity, the assumption of a stable population is more reasonable, following the same assumptions of stationarity except that zero population growth is not assumed (Lotka, 1922). Because of weak ergodicity, most populations actually approximate the conditions of stability, even if they are open to migration or fertility and mortality rates are changing (Wood et al., 2002).

In addition to the assumption of stationarity, the life table approach requires the assumption that interval estimates for each individual are assumed to be known with the same accuracy or margin of error (Bocquet-Appel and Masset, 1982). Age estimation methods for skeletal remains, however, differ depending on the type of skeleton (i.e. juvenile *vs.* adult skeletons) and the accuracy of age estimation for a skeleton can vary at different times of life. When compared to adults, juvenile age estimates are more reliable and precise because epiphyseal fusion and tooth eruption stages are well known

and consistent over time. On the other hand, adult age estimation, discussed above, is based on degenerative changes of the skeleton and is highly variable.

Furthermore, hidden heterogeneity (i.e. every individual in a population varies in terms of their relative risk of death compared to others in their birth cohort), and selective mortality (i.e. individuals who die at a given age are not likely to be representative of the entire living population at risk of death at that age) prevent direct estimates of mortality rates from skeletal data, as researchers cannot reasonably infer individual risk from aggregate data without prior knowledge of frailty variability in the population (Vaupel and Yashin, 1985; Wood et al., 1992). Hazard analysis addresses these shortcomings by accounting for hidden population heterogeneity through the use of covariates, thus also accounting for the selective mortality that acts upon heterogeneous frailty.

These principles can be extended to research on the health of past populations through skeletal lesions. Hidden heterogeneity and selective mortality could mean that individuals with pathological lesions associated with disease were selected against at a certain age because they were less healthy than their peers, which would lead to an overestimation of disease prevalence in a living population because these individuals were more susceptible to mortality than their peers were (Wood et al., 1992). Frail individuals in the past will not always appear to be so on the basis of lesions, since frailty can exist without leaving marks on skeletal remains. Frail individuals who were selected for by some disease may die so quickly that marks are not left on the skeleton, leaving a *healthy-looking* skeleton within the cemetery sample. Thus the simplistic use of

pathological lesion frequencies to extrapolate disease prevalence of the once-living population is not a reliable approach to assess health in past populations. Prior to Wood et al.'s (1992) paper, paleopathological analyses would assume that higher frequencies of skeletal lesions could be interpreted as unhealthy populations (Hoppa, 2002).

Fortunately, osteologists have begun to address complications associated with assessing mortality and health patterns in the past as a result of hidden heterogeneity and selective mortality. Statistical methods such as parametric or semi-parametric hazards analysis are a powerful way to gain information from small osteological samples. Bio-mathematical hazard models of mortality differ from most mathematical models of mortality in that they attempt to incorporate biological principles into the study of mortality patterns (e.g. Gage, 1988; Gage, 1989; and Wood et al., 2002). These hazard models include models of aging, maturation, mortality selection, and the physiological processes that influence mortality (Gage, 1989). An important benefit for using parametric mortality models instead of life tables is that osteologists only need to assume that the population under study is stable- not stationary, as discussed above. This approach is also advantageous in that it smooths variation that occurs from small samples, characteristic of paleodemographic data, without imposing a particular age pattern.

Model life tables have been valuable resources for estimating mortality when data are incomplete, such as with skeletal data, providing age patterns of mortality (i.e. characteristic patterns for age-specific mortality rates) that aid in the construction of life tables. Age patterns of mortality, however, are different for different populations (Coal

and Demeny 1966). Thus, applying an inappropriate model life table on a population could result in inaccurate reconstruction of the mortality of the population. Gage (1988) argued that model life tables are typically limited and defective when the underlying age patterns of mortality differ from mortality patterns in model life table systems, which is often the case in anthropological applications. He maintained that a method was necessary to affectively smooth the mortality curve but would not impose a particularly morality pattern on the data, and assessed the use of Siler's five-parameter competing hazards model in paleodemographic analysis as a more appropriate model. This parametric mortality model posits that an individual's risk of death at each age is determined by three sets of competing causes- subadult mortality, senescent mortality, and mortality independent of age. The joint effect of these three components varies with age. Using the model, he reconstructed life tables for three different cemetery samples: a New World Late Woodland population (to show that the model can fit anthropological age patterns), a Christian period Nubian (modern day Egypt and Sudan) population (to show that the model corrects for defective data), and the Yanomama (to show that the model can be used on census data). He showed that the model conforms to the age patterns of mortality that are typical of paleodemographic mortality patterns (high adult mortality and low infant mortality), and that the model helps to correct defective data and smooth age-specific mortality of small and defective sample sizes. He also successfully showed that this model can be used as a method of estimating mortality and fertility from census data.

The Siler model is useful in paleodemography because it covers a wider range of

mortality patterns than traditional model life tables and has been shown to span a wide range of mortality patterns reported in paleodemographic literature. The benefit here is that the model is still affective when age-at-death distributions are distorted by selective burial practices, differential skeletal preservation, and biased age estimates (Gage, 1988). Most importantly, it allows mortality patterns to be reconstructed from small samples characteristic of cemetery populations and has been successfully applied to osteological data to reconstruct paleodemographic patterns (DeWitte and Wood, 2008; Kreger, 2010). Gage concludes that using this method on different anthropological data sets (both paleodemographic and ethnographic) can be a useful way to empirically compare life tables, which could shed light on understanding mortality patterns among human populations through time. An example of successfully applying hazard modeling analysis using skeletal remains is DeWitte's (2010) paleodemographic analysis of a Black Death cemetery. She found that risk of mortality during the Black Death increased with adult age, and was similar to the age patterns from a non-epidemic mortality sample. Thus, the Black Death did not kill indiscriminately, and plague Death cemeteries are not representative of the living population, putting the general notion of catastrophic cemeteries as potential cross-sections of the once living population into question.

One thing to note that is often misinterpreted from Wood et al.'s (1992) article is that the authors do not necessarily argue that the increased prevalence of skeletal lesions is directly related to good health; they merely explain that osteologists should not ignore this as a possibility and argue that osteologists must use caution when using skeletal lesions to make interpretations of past population health. Though this is evident

in their article, often osteologists include the Osteological Paradox as a caveat after interpretations have been made without thoughtfully incorporating analytical and methodological approaches that deal with these inherent issues in skeletal samples (see DeWitte and Stojanowski, 2015). This is unfortunate, as the actual application of this perspective provides a more nuanced approach to understanding past population dynamics through skeletal material and could further the field by encouraging other researchers to think more deeply about fundamental assumptions in their research and the variety of interpretations that might account for the patterns they describe in bioarchaeological data.

To conclude, though the inherent biases in osteological collections and shortcomings of analytical approaches within bioarchaeology outlined in this section seem to call into question the practicality of some osteological analytical approaches, there is still much useful information that can be gained from human remains. The realization of preservation biases and appreciation of the complexity of cemetery collections has resulted in the development of innovative techniques that have produced invaluable information concerning populations of the past, while also making paleodemography a stronger discipline. From a biological perspective, paleodemography can provide information on individual health and how health varies throughout time and space, particularly relative to major changes of the environment (i.e. urbanization).

1.4 Stable isotope analysis of human remains

Stable isotope analysis is a well-established technique used for exploring past dietary patterns (Katzenberg, 2007). Stable isotopic analysis of human remains has the potential to provide valuable insights into the dietary physiology of humans and the interactions of humans with the physical and social environments that they live. Moreover, the analysis of isotope values from bone or tooth collagen can be used to deduce an individual's diet and then further used to assess intrasite and intersite dietary patterns.

1.4.1 Isotopes and fractionation

The foundation for diet reconstruction using stable isotopic analysis is based on the fact that characteristic ratios of different stable isotopes, namely carbon and nitrogen, are found within different food webs and the trophic levels are the result of fractionation effects. Carbon and nitrogen are consumed and taken into the body via diet and because isotopic values are passed along through the food chain, the consumer tissues will reflect the diet that was consumed during life. The relative summation of ^{13}C and ^{12}C (expressed as $\delta^{13}C$) stable carbon isotopes is distinct for different ecosystems and varies predictably in isotopic composition (Ambrose and Norr, 1993; DeNiro and Epstein, 1981). Nitrogen stable isotope ratios of ^{15}N to ^{14}N (expressed as $\delta^{15}N$) increase by 3% to 4% with each step up the food chain, allowing the differentiation of protein sources (DeNiro and Epstein, 1978; Schoeninger and DeNiro, 1984). Combining nitrogen and carbon isotope signatures can be used to differentiate between plants, herbivore

proteins, and carnivore proteins to reconstruct the diet for an individual during life. Traditional methods of diet reconstruction (i.e. faunal analysis or paleobotanical analysis), while they contribute very important information, are unable to provide more of a direct reflection of diet .

Isotopes are alternative forms of a chemical element that have different numbers of neutrons, giving them different varying mass numbers (the sum of protons and neutrons within the nucleus). The element carbon, for example, has six protons. A carbon atom with seven neutrons has a mass number of 13 (^{13}C), and a carbon atom with six neutrons has a mass number of 12 (^{12}C). Both of these isotopes are stable (i.e. non-radioactive) because the arrangement of the protons and neutrons within their nuclei do not allow them to decay. The ratio of these isotopes is usually expressed in delta values (δ) and then compared in units per mille (‰) because the values are extremely small (Schoeller, 1999).

Some natural reactions are sensitive to the small variation in mass between isotopes, and this discrimination is referred to as fractionation. Stable isotopes that have low atomic weights show larger differences in mass than heavier elements, making isotopic fractionation more evident (O'Leary, 1981). When the heavier isotope (e.g. ^{13}C) is enriched or depleted during incorporation of another substance, fractionation can be identified. Thus, changes in the ratio of isotopes (e.g. ^{13}C and ^{12}C) between two substances or phases are known as isotopic fractionation. A biochemical reaction, such as photosynthesis, is one example of a reaction resulting in fractionation.

1.4.2 Carbon isotopes

Carbon is prevalent in all organic macromolecules and is constantly replaced during life through tissue synthesis from nutrient cycling. Diet is the means by which carbon is replaced and tissues are regenerated, and thus follows the cycling pathways of the terrestrial food web (Ambrose, 1990). Both ^{12}C and ^{13}C are stable within the environment, meaning that they retain their atomic structure over time. The distribution of carbon isotopes in the biosphere is primarily determined by fractionation as a result of photosynthesis. Most terrestrial plants follow two photosynthetic pathways, which have discrete ranges of $\delta^{13}C$ value: C_3 plants and C_4 plants. A majority of these plants species follow the C_3 cycle, meaning they assimilate CO_2 via C_3; these plants include wheat barley, rye, and oats, nuts, tubers, and most fruits and vegetables. These plants have isotope ratios that are less than atmospheric CO_2, with $\delta^{13}C$ values ranging from -33‰ to -22‰ (DeNiro, 1987). On the other hand, plants that follow the C_4 cycle include millet, maize, sugar cane, and sorghum. Values for C_4 plants range from -16‰ to -9‰ (Smith and Epstein, 1971).

The composition of plants, whether it is C_3 or C_4, or both, is passed on to those organisms that consume them and are thus passed through the food chain. This composition is systematically altered by fractionation as it passes through levels of the food chain, resulting in a small increase of $\delta^{13}C$ values with trophic level. Human tissues and diet differ in isotopic composition by approximately +5‰ due to the fractionation factor between the dietary source and the production of collagen (Lee-Thorp et al., 1989). Thus there is generally an enrichment of ^{13}C in consumer bone collagen relative

to an individual's diet. Vogel and van der Merwe (1977) were the first to confirm that variation in $\delta^{13}C$ values could be used in archaeology by comparing pre-horticultural and horticultural sites in New York state. They demonstrated that the ratios of ^{12}C to ^{13}C in bone collagen of skeletal remains from horticultural sites were enriched in ^{13}C compared to individuals from pre-horticultural sites, reflecting a higher proportion of maize in the horticultural diet.

1.4.3 Nitrogen isotopes

Similar to carbon, there are two sources of nitrogen available to differentiate terrestrial plants. Plants incorporate nitrogen through atmospheric nitrogen (N_2) or through nitrates in the soil (Van Klinken et al., 2002). This divides plants into legume and non-legume groups. The first group, legumes, fix atmospheric nitrogen through symbiosis within their roots, resulting in almost no fractionation; thus, $\delta^{15}N$ values are similar to atmospheric values of 0‰ (Schoeninger, 1995). These nitrogen-fixating plants include beans, clover, peas, etc. Most terrestrial plants, however, are non-legumes and take up soil nitrogen through bacterial degradation of organic material, resulting in plant values that are more enriched than atmospheric values (Schoeninger, 1995). On average, $\delta^{15}N$ values for this group tend to be 3‰ and are thus more enriched than leguminous plants (Schoeninger, 1995).

Like carbon, nitrogen values provide information concerning the trophic level of an organism in the ecosystem. Nitrogen values can also indicate how much plant food was in the diet (Ambrose et al., 1997; Ambrose and Norr, 1993; Lee-Thorp et al., 1989).

Importantly, nitrogen is obtained almost completely from dietary proteins. Generally, isotopic ratios have been shown to increase by approximately 3‰ between food and consumer tissues (Ambrose and DeNiro, 1986; Schoeninger and DeNiro, 1984). For example, a plant with a $\delta^{15}N$ value of 2‰ would lead to the herbivore consuming that plant with a $\delta^{15}N$ value of 5‰. Usually in humans, higher $\delta^{15}N$ values suggest more protein derived from animals or higher trophic levels (e.g. vegans will have lower $\delta^{15}N$ values than those omnivorous humans who eat meat). Consumption of marine resources, however, will generally demonstrate enriched $\delta^{15}N$ values because the marine food chain is much longer than the terrestrial food chain (Schoeninger and DeNiro, 1984).

Environmental factors such as climate, altitude, and soil composition can result in highly variable $\delta^{15}N$ values (Ambrose, 1991). Arid regions have higher $\delta^{15}N$ values than less arid environment because of effects of water or heat stress on urinary nitrogen excretion (Ambrose and DeNiro, 1986), evaporation increase of ammonia from soil (Heaton et al., 1986), or the influence of human plant foraging over time known as "manuring" (Hedges, 2004). Additionally, physiological changes such as pregnancy and illness or metabolic stress as a result of undernutrition can also influence $\delta^{15}N$ values (as discussed below).

1.4.4 Limitations of stable isotope analysis

Isotopic data can provide valuable information about diet in the past, but as all archaeological techniques of reconstruction, there are limitations. One major limitation

of stable isotopic analysis is that this method only provides information about the general makeup of the diet of the consumers, and not specific menu food resources. There are also uncertainties in the characterization of $\delta^{13}C$ and $\delta^{15}N$ values for specific dietary sources, as the values are subject to change across time and space. For this reason, stable isotopic analysis is best applied alongside archaeological evidence so that stable isotopic values may be interpreted as accurately as possible.

Another major issue of stable isotopic analysis is the diagenesis of the material sampled. Different tissues have different responses to the environment. After death, bioapatite of bone collagen undergoes recrystallization and protein undergoes hydrolysis. These changes influence the relationship of protein and bioapatite, increasing the susceptibility of diagenetic agents within the environment on both bone fractions, which could lead to inaccurate isotope values (Ambrose, 1990). Both intrinsic (e.g. bone composition) and extrinsic processes (e.g. burial environment) affect bone diagenesis. Depolymerization from hydrolysis can result in poor sample preservation and loss of collagen. Exogenous carbon and nitrogen in the samples can also occur. Environmental factors such as temperature, weathering, and hydrology can all influence preservation (Hedges, 2002; Nielsen-Marsh and Hedges, 2000). For example, Pate (1994) found that collagen samples at increased depth may result in enriched $\delta^{15}N$ values relative to shallower samples; however, the exact relationship between degradation and depth were not determined. Because diagenetic processes and the sources for diagenesis are complex and often influence one another, it is almost impossible to determine the underlying cause of diagenesis in the sample. It is possible,

however, to assess whether the samples in question are valid for stable isotopic analysis.

Three widely accepted methods of measuring organic preservation of bone include percentage of collagen yield, carbon and nitrogen ratio, and percentage of carbon and nitrogen (Ambrose, 1990). The percentage of collagen in a sample (collagen wt%) is the most straightforward of these validation methods, as it does not require analysis beyond sample preparation. Collagen wt% is the amount of collagen preserved relative to the sample's dry weight (Schoeninger et al., 1989). The upper threshold for collagen yield percentages is approximately 20%, thus values higher than 20% (in the range of fresh bone) indicate uninformative proteins or organic contaminants within the sample (Schoeninger et al., 1989). On the other hand, the lower threshold has been set to as low as 3% to 1% for collagen yield percentages (Tykot, 2006). For further sample validation, atomic carbon and nitrogen ratio may be used. Deniro (1985) recognized that carbon to nitrogen ratios within a range of 2.9 to 3.6 can be considered as not having undergone significant diagenetic alteration and are appropriate for analysis. Carbon and nitrogen weight percentages are also informative about the validity of samples. Ambrose (1990) established that a carbon concentration of 15.3% to 47% and a nitrogen concentration of 5.5% to 17.3% are acceptable for analysis, as these values are found in fresh collagen.

1.4.5 Stable isotope analysis in bioarchaeology

The reconstruction of diet from carbon and nitrogen stable isotopes and an

appreciation of the digenesis of collagen sample permit osteologists to gain valuable and more accurate information about consumptions patterns in the past. Culture, access to resources, and social factors all have an influence on what type of food humans eat. Skeletal material has the potential to allow the reconstruction of diet at both the individual level and the population level, which can corroborate historical evidence of diet or elucidate dietary trends that occurred in the past. Scientific methods for reconstructing diet are not subject to the same biases inherent in historical records. Moreover, isotope data can be examined on a larger scale to identify dietary patterns of different subgroups when paired with paleodemographic data (e.g. dietary differences between males and females).

Because there are no systematic differences in the isotopic composition of male and female body tissues when the body is stable, isotopic differences between the sexes are the result of different diets (Schwarcz and Schoeninger, 1991). In their study of medieval Fishergate, an urban cemetery in York, Müldner and Richards (2007b) found that females consumed less marine foods than males and attributed this dissimilarity to eating practices that were governed by medieval dietary theory concerning differences in the physiological make up of males and females (i.e. the classical writings of Hippocrates and Galen regarding the four humors). Even the absence of differences between sexes is still notable and should be explored, as this could be informative of *similar* eating patterns of male and females.

In addition to sex differentials, stable isotope analysis using carbon and nitrogen can also be informative about socioeconomic status or groups within the population

when paired with contextual data (e.g. burial location or grave goods). For example, in comparing Fishergate lay and monastic cemeteries, Mays (1997) found that marine foods made a greater contribution to the monastic diet than to the lay diet, illuminating foodway differences between these two groups and lending evidence to historical documents that suggested more fish in monastic diets.

Stable carbon and nitrogen isotope values can also be used to deduce breastfeeding patterns in populations by identifying infant $\delta^{15}N$ values above the mean for women of childbearing age (Dupras, 2001). This is valuable because child feeding can provide information about infant and child survival, which ultimately influences population growth and decline, making it a key, but frequently missing, component in bioarchaeological studies. Burt (2013a) reconstructed breastfeeding practices in medieval York, England, and determined that weaning was complete by 2 years of age, which is consistent with historical records in Britain. Burt also found that weaning did not appear to coincide with peak mortality, suggesting that environmental factors may be playing a larger role in child mortality in rural environments. Richards et al. (2002) looked at a contemporaneous neighboring rural cemetery in medieval York, Wharram Percy, and also found weaning was complete by approximately two years of age.

Mobility can also be identified, though less directly than other isotopes (e.g. oxygen and strontium), using carbon and nitrogen isotopic ratios. When evaluating diet variability within a population, individuals with carbon and nitrogen values that are extremely different from the rest of the population are suggestive of having a non-local origin (Beaumont et al., 2013a). In a recent reconstruction of diet in medieval London,

Lakin (2010) identified several individuals with $\delta^{13}C$ and $\delta^{15}N$ values that were significantly different from the rest of the sample, and subsequently performed oxygen and strontium isotope analysis to investigate if these individuals were potential rural-to-urban migrants, based on previous research suggesting that rural inhabitants consumed little marine protein (Fuller et al., 2003). Only one of these individuals, however, could be definitively labeled as a migrant.

In addition to the role of stable isotope data in diet reconstruction, carbon and nitrogen values can also be informative about health in the past. Diet is one the most important mediators of health, and its function as a proximate factor through which several other factors exert their influence (Hill et al., 2011) make the investigation of diet an integral component in the understanding of health dynamics. Improper diet and malnutrition weakens the immune system, making the body more susceptible to disease (Scrimshaw et al., 1968). Pathology-influenced isotopic fractionation can potentially be used to understand physiological health and nutrition (Reitsema, 2013). For example, elevated nitrogen values have been associated with physiological stress on the body. Nitrogen comes from both ingested protein and recycled tissues in the body, making it impossible to definitively distinguish between physiological and dietary differences of $\delta^{15}N$ values. Stress responses and compromised health influence the physiological processes underlying stable isotope fractionation and distribution in the body, and may be useful in identifying changes to health or nutritional changes.

Clinical (Fuller et al., 2004; Fuller et al., 2005; Mekota et al., 2009; Mekota et al., 2006) and recent bioarchaeological research (Beaumont et al., 2013a; Beaumont et al.,

2013b; Beaumont and Montgomery, 2016; Holder et al., 2017; Olsen, 2013; Reitsema, 2013) has demonstrated that, when analyzed with historical evidence, enriched nitrogen isotope values may be suggestive of nutritional stress and starvation. After fractionation, tissues are enriched in ^{15}N and waste, in the form of urea, are enriched in ^{14}N relative to the diet. When there is not enough protein consumption to maintain tissues, the body enters a catabolic state (Hobson et al., 1993). The recycled tissues, as a result of catabolism, repeat the fractionating process causing the body tissues to be more enriched in ^{15}N. One clinical study that tracked dietary changes in isotopic values found that, of anorexia nervosa patients, $\delta^{15}N$ values were highest when BMI levels were low (Mekota et al., 2006). Another clinical study found that nutritional stress as a result of morning sickness resulted in higher $\delta^{15}N$ values, but decreased later in the pregnancy (Fuller et al., 2005). Both studies attributed the high $\delta^{15}N$ values to individuals being in a catabolic state because of nutritional stress. Bioarchaeological research has also investigated elevated nitrogen values and potential dietary stress. A mass grave in Lithuania containing the remains of Napoleonic soldiers believed to have undergone severe starvation exhibited high $\delta^{15}N$ values, suggesting that prolonged dietary stress was the cause (Holder et al., 2017).

Enriched nitrogen values have also been associated with pathological lesions on bones. In comparing pathological bone with normal bone within and between individuals, Katzenberg and Lovell (1999) found that osteomyelitic bone exhibited increased $\delta^{15}N$ values. The authors contributed the elevated values to new tissue depositing during bone repair, as the healing process requires an abundance of energy

that may result in a catabolic state. Similarly, White and Armelagos (1997) found significantly enriched $\delta^{15}N$ values in individuals with osteopenia, a condition in which bone mass is reduced as a result of poor mineralization, when compared to individuals without osteopenia. This difference, however, was only seen in females who suffered from osteopenia. The authors argue that enriched ^{15}N in osteopenic females was most likely a result of altered urea excretion, creating premature forms of the disease in females as a result of physiological demands (i.e. lactation and pregnancy, among others). In a dissertation investigating pathological condition and stable nitrogen analysis, Olsen et al. (2014) found no significant differences in osteomalacia or other degenerative disease and $\delta^{15}N$ values. However, high $\delta^{15}N$ values were evident in *some* samples of fractures bone, periostitis, and unaffected bone. Similarly, in their review of isotopic research and paleopathology, Richards and Montgomery (2012) found some relationship between dental caries and isotopic evidence of plant consumption, but in many cases there were no convincing links between isotopic values and paleopathological conditions.

Another important contribution of stable isotope analysis in bioarchaeological studies is the ability to investigate childhood diet and potential stress of adults who survived childhood. In 1992, Wood et al.'s Osteological Paradox demonstrated how stress experienced in childhood could contribute to the frailty of an individual into adulthood, thus creating hidden heterogeneity with the population and complicating the interpretation of population health from skeletal remains. Stable isotopic signatures, however, can be assessed at different times throughout life, prior to death. In contrast

to bone, primary dentine does not remodel and can provide a time-bound archive of food and drink ingested during tooth formation (Beaumont et al., 2013b). Carbon and nitrogen stable isotopic analysis of teeth that developed during childhood can thus provide information about childhood diet or physiological or dietary stress. When compared with nutritional carbon and nitrogen isotopic values of bone collagen, which is informative about diet years before death, a better understanding of health (using mortality as a proxy for health) during the lifespan can be achieved (Reitsema, 2013). Comparison of childhood diet and stress can also be informative about the frailty of the individual. For example, an individual who experienced enriched $\delta^{15}N$ during childhood possibly associated with dietary stress might live to old age, which would suggest the individual was *hardier* than the individual's counterparts that experienced the same stress in childhood but died earlier in life.

The comparison of tooth and bone sample for isotopic analysis also allows the application of a life history approach looking at diet and health. Reitsema and Vercellotti (2012) compared rib and tooth samples to investigate if poor childhood diet contributed to poor health later in life, evident in adult skeletal stress markers. Results of stable isotope analysis of the second molar suggested that childhood diets of the sample were similar, and thus childhood malnutrition was most likely not the cause for skeletal lesions later in life (Reitsema and Vercellotti, 2012).

An innovative technique wherein a tooth is incrementally sampled and then analyzed for carbon and nitrogen values creates an even more precise picture of childhood health (Beaumont et al., 2013b). Given that the rate of tooth development of

human teeth is well established (Hillson, 2005), accurate age ranges may be attributed to incremental samples with a single tooth, producing a high-resolution isotopic record of dietary and metabolic changes. This method can expand the time scale of an individual's isotopic makeup by including inter-tooth comparison (for teeth mineralizing at different ages), creating a dietary record for most of, and in some cases the entirety, of an individual's lifespan. Through this technique and strategic sampling, osteologists can investigate an individual's childhood diet, and potential stress experienced by the individual during this time if enriched nitrogen is exhibited after weaning (Beaumont et al., 2013b). These data can be paired with bone collagen isotopic data as well to further investigate the effect of childhood diet on health.

To conclude, stable isotope analysis of carbon and nitrogen has been extensively used to assess biocultural behavior through the dietary reconstruction and the investigation of diet variability in populations. Isotopic data can provide valuable information on past human activity such as weaning behaviors, dietary differences between groups and sex, temporal changes in diet, and mobility. Isotope analysis is also an indispensable technique for investigating health in the past. Physiological changes as a result of dietary or pathological stress can be exhibited through enriched or depleted isotope values. Moreover, the comparison of stable isotope analysis of different tissues (e.g. tooth dentine and bone collagen) can provide a more nuanced understanding of diet and health by investigating diet (and potential stress) throughout life. Childhood isotopic data can be used as a window into an individual's past so that the relationship between childhood diet and stress and adult health can be explored. This can, in turn,

provide information concerning the heterogeneity of the population and how this may have affected the overall mortality, thus offering a more accurate depiction of past population health and mortality.

1.5 Urbanization in Late Medieval London

1.5.1 Rescue archaeology in England

Archaeological evidence provides a substantial contribution to the study of urbanization and urban health, particularly the investigation of certain features of past populations that are believed to have facilitated disease. Dense settlements, contaminated water supplies, and inadequate disposal of sewage have been cited as features of urban cities that result in higher morbidity and mortality. Much of London's archaeological data have been the result of rescue archaeology because of the ever-growing nature of the city (Schofield, 2011), which has produced important archaeological finds while allowing urban redevelopment to ensue.

Excavated skeletal collections are the product of several selection processes that render a biased sample (Milner et al. 2000). For example, the reason for which a site is excavated (e.g. planning or development) or the extent to which an area is excavated (often the result of budget constraints) can introduce bias. In England, archaeology is now mostly undertaken as a response to planning applications that hold the developer responsible for funding the preservation of archaeological remains during construction (Darvill and Russell, 2002), which has had a substantial influence on what samples are

available for osteologists to analyze.

After World War II, there was an increase of development in England (Darvill and Russell, 2002). The reconstruction of buildings and towns affected by bombings, modernization of the motorway infrastructure with new roads, and expansion of towns and cities occurred at a fast pace (Everill, 2007; McKinley, 2013). This period of dramatic development resulted in the destruction of valuable, irreplaceable archaeological material. Archaeologists recognized that the preservation of archaeological remains beneath building plots and roadways should be preserved before they were destroyed (Darvill and Russell, 2002). To mitigate additional loss of archaeological material, archaeologists applied innovative techniques to documenting and preserving artifacts and archaeological contextual information. Excavation methods became more efficient and documentation techniques became standardized because of the need to work quickly under development deadlines (Darvill and Russell, 2002). The methods applied during the 1950's and 1960's are now widely used in Cultural Research Management (CRM), and the practice of preserving archaeological sites that were in danger of being destroyed was dubbed "rescue archaeology" (Everill, 2007).

Eventually, the government recognized the necessity of archaeological expertise in the documentation of these sites and created county archaeologist positions (Andrews and Thomas, 1995). After the boom in rescue archaeology, the government began funding excavations as well. Local units with full- and part-time archaeologists were employed under these units (Darvill and Russell, 2002). At the end of the 1970s, however, the funding of archaeological excavations and preservation changed as a

result of government funding changes in which government-funded initiatives became strategically funded though “project funding” (Andrews and Thomas, 1995). Project funding calls for a clear set of objectives of a defined project, a timetable to complete the project, and a transparent budget that accounts for all expenses needed to complete the project (Andrews and Thomas, 1995). Government-funded archaeological projects were allocated funds based on this framework, both locally and nationally. Project-funding also created competition among archaeology firms since the government would choose the firm that could complete the project at a lower cost (Andrews and Thomas, 1995).

During the 1980s, government funding for archaeological excavations was replaced by developer funding (Chadwick, 1991; Everill, 2007). The Polluter Pays Principle, an environmental policy principle that makes the polluter responsible for any cost of pollution control, clean up, or prevention, allowed the government to make the cost of any necessary archaeological work during development to be the responsibility of the developer (Graves-Brown, 1997). The only difference between government-funded projects and developer-funded projects is who is paying for the excavation or preservation of the archaeological material. In an effort to regulate developer funding, the UK government issued formal documents to advise local development planning authorities on the treatment of archaeological material during the planning process (Wainwright, 2000). The first of these documents was the Planning Policy Guidance 16: Archaeology and Planning (PPG16) document released in 1990.

The PPG16 established the rescue archaeology’s first policy on archaeological

remains, recognizing them as an irreplaceable and finite resource vulnerable to damage and destruction. The PPG16 also states that archaeological materials provide evidence of the past development of English civilization and contribute to national identity, education, leisure, and tourism (DoE, 1990). To mitigate damage of these valuable archaeological remains, an evaluation of the site must be conducted before development occurs. If it is found that the site has archaeological potential after the evaluation, the site can either be preserved *in situ* or preserved "by record" (DoE, 1990). *In situ* preservation maintains the archaeological material by allowing the remains to stay in their current location and the structure to be built on top of the archaeological remains (DoE, 1990). If *in situ* preservation is not possible, the site is preserved by record, meaning the site is excavated (either during construction or before construction) and the findings are recorded (DoE, 1990). The developer is responsible for all funds required to complete the archaeological investigation to whatever degree is necessary in order for development to occur (DoE, 1990). The developer may choose which archaeological firm to conduct the work, but a county archaeologist must serve as an advisor during the investigation (DoE, 1990). Once the county archaeologist has submitted a satisfactory site report, the developer may begin their project (DoE, 1990).

The PPG16 has since been replaced by several government documents with similar policies. In 2010, the Planning Policy Statement 5: Planning for the Historic Environment (PPS5) replaced PPG16 (DoE, 2010), and holds that the historic environment should be viewed as a whole, rather than considering each element separately. Generally, the document upheld policies from PPG16 but added that the

archaeological context of the entire site must be preserved and also placed emphasis on publishing the results of archaeological investigations (McKinley, 2013). Two years later, in an effort to make the planning system less complex, the National Planning Policy Framework (NPPF) replaced the PPS5, and is currently in use today. The NPPF is similar to the PPS5, however, the language is more straightforward and places a heavier emphasis on sustainable development and environmental protection (DoE, 2012).

Developer funding has resulted in a great increase in archaeological projects compared to government-funded projects. Government-regulated documents like the PPG16 brought archaeology to the forefront of those making planning decisions (Mellor, 1992), and provided a logical approach to the collection and use of archaeological remains in the decision-making process. These regulations also ensured that sites were excavated to completion rather than being left to decay when funds ran out, characteristic of some research-oriented investigations (Darvill and Russell, 2002). The regulations also influenced sample collection strategies (Orton, 2000) because of the need to provide high-quality briefs and specifications to present to developers (Chadwick, 1991). Moreover, some argue that the competition facilitated by developer funding has resulted in an increase of professionalization in archaeology (Everill, 2007).

Though there are several positives apparent in the shift to developer-funded and government-regulated excavations, there are several critiques as well. The most notable downside of this massive increase in archaeological projects that has resulted from developer-funding is the backlog of unpublished site reports (Darvill and Russell, 2002). Hinton (1992) argued that archaeological investigation is no longer interesting because

it is not driven by research questions, and that work done is less assessable because of confidentiality clauses. Morris (1998) and Brenan (1994) contended that developer-funded archaeology has created a fragmented discipline, different from long-term research projects conducted in the past. Some argue that developers are employing archaeological firms that will complete the job at a lower price, resulting in lower quality work (Hamilakis and Duke, 2007). Furthermore, though precious archaeological material is being saved, commercial archaeology has introduced bias into what osteologists evaluating skeletal collections are available to analyze since developers are only willing to fund what must be preserved or recorded. Full archaeological sequences, thus, may not be excavated because the primary objective had been achieved (though these can still be preserved under the building) (Darvill and Russell, 2002).

One example of a site excavated as a result of development is St Mary Spital. The cemetery at St Mary Spital comprises the largest urban skeletal collection in Europe to date and is currently housed at the Museum of London. In the 1980s, Spitalfields Market, situated in the London Borough of Tower Hamlets outside of the city of London boundaries, was to be moved further from the City and the existing site was to be totally redeveloped with the exception of preserving St Botolph's hall and the market buildings (Connell et al., 2012). The evaluation of the site began in 1991 and excavations were completed in 2007 (Connell et al., 2012). The latest phase of works was conducted for an underground ramp service and the new buildings of Bishop Square. During this phase, the evaluation, excavation, and development of the site, a well-preserved medieval cemetery, was uncovered adjacent to the medieval priory church (Connell et

al., 2012). The church was to be preserved and not fully excavated except for two adjacent areas that required excavation as a result of realignment of the deep-level sewer. As a result of development over the existing chapel for preservation purposes, several burials were excavated in addition to the excavations that were conducted during the previous evaluation of the chapel. Much of the archaeological material recovered was conducted through watching briefs, allowing the archaeologists to identify and excavate (or preserve) necessary archaeological and human remains (Connell et al., 2012).

In the case of St Mary Spital, the required preservation of the church and subsequent preparation of the foundation required the fortunate excavation of thousands of skeletons within the cemetery. As mentioned previously, and as seen with St Mary Spital, excavations commissioned as a result of planning are usually never excavated to completeness and only to what is necessary for redevelopment and preservation purposes. Thus, these excavations generally represent a sample of the potential archaeological resource of the site. Moreover, not all potential archaeological evidence is retrieved or recorded, making it important for the researcher to carefully evaluate what methods were used and what archaeological material was considered necessary for collection (Fulford and Holbrook, 2011).

Several redevelopment projects in the last few decades in London have resulted in a large amount of commercial archaeological preservation projects and thus a high volume of archaeological evidence (Darvill and Russell, 2002). When gathering information concerning urbanization of medieval London through archaeological

evidence, it is important to take into account the potential presence of this information in grey literature or unpublished site reports as a result of watching briefs. For example, in Fulford and Holbrook 's (2011) assessment of the contribution of commercial archaeology to the study of Roman England, they found that a vast amount of information resided only in grey literature. Though commercial archaeology has generated a vast amount of archaeological material and information useful for examining the effects of urbanization, most archaeological information has to be found in sources like the Historic Environment Record (Fulford and Holbrook, 2011). Oftentimes results of commercial archaeological excavation may not be deemed worthy of publication when they are submitted (i.e. reports with only seed and plant remains or information concerning the soil); however, when analyzed with additional archaeological evidence or with a specific research question in mind, mundane site reports could be informative of how people lived in medieval London. For example, insect evidence of a commissioned site may seem unpublishable, but when paired with additional archaeological and historical evidence, could potentially contribute to understanding population density in the past (Kenward, 1978).

The emergence of commercial archaeology in England has produced a vast amount of archaeological evidence. Commercially commissioned excavation as a result of development planning has created a funding source that allows the preservation of valuable archaeological information informative of England's past. However, when analyzing evidence from sites excavated as a result of commercial archaeology, it is important to recognize potential biases that could affect the sample in question. The

excavation history of the site, including what parts of the site were excavated and why other parts of the site were not, should be considered and included in interpretations of archaeological material. The type of archaeological evidence that was deemed worthy for collection or recording at the time should also be considered. Moreover, the researcher should thoroughly investigate the grey literature and unpublished site reports, as this is where a majority of information from commercial excavations resides. Finally, as with any archaeological analysis, either commercially funded or not, a thorough investigation of potential sources of biases is necessary and should be included in interpretations of archeological evidence so that the most accurate depiction of the past can be reconstructed.

1.5.2 An Urbanizing England

In London, there was a clear transition toward expansion and development in the later eleventh and twelfth centuries. A significant number of new parish churches were established, and new streets were subsequently built to accommodate these structures (Schofield, 2011). The placement of the parish churches indicates that the city was built up with a focus on the dockside areas, with several streets radiating out from the River Thames (Thomas, 2002). The docks of the River Thames were expanded because of increased trade. Monastic houses were built, leading to increased settlement around them along the main streets out of the city. With London having the largest port in England, and with the proximity to royalty and the government, London was set as the major power in England, both economically and politically (Schofield, 2011).

By 1100 CE, approximately 20,000 people resided in London (Dyer, 2002). Within a century the population doubled to approximately 40,000 residents, illustrating the extraordinary growth of the city. Urban expansion was inevitable at this rate. The fourteenth century would see another doubling of the population at 80,000 and then to 100,000 inhabitants before the Black Death (Dyer, 2002). Archaeological excavations and historical maps show that the rapid increase in population density is evident in the reconstructed street pattern of the thirteenth and fourteenth centuries, with preexisting blocks hastily further subdivided to accommodate rapid population increase, creating new spaces for housing (Thomas, 2002). Unfortunately, buildings and roads constructed at the end of the medieval period are closer to the modern ground surface, so most have been destroyed by modern buildings (Schofield, 2011). The plan of London for this period, then, is partly conjectural from historic maps.

1.5.3 Infrastructure and sanitation

The majority of buildings in London were made from timber, although the wealthier families built houses of stone (Thomas, 2002). Unfortunately, houses constructed from wood were less likely to survive in an archaeological context than those wealthier stone residences, thus providing less contextual information for people who resided in these wood structures (i.e. individuals with lower socioeconomic status). Houses in London tended to be more than one-story tall, with several houses reaching four stories and several families inhabiting them (Dyer, 1989). The poor generally resided in single rooms with straw as flooring and no latrine, kitchen, or water supply

(Dyer, 1989). At the twelfth century Guildhall site, evidence of people and cattle living together was evidenced by adjacent cesspools (Schofield, 2011). One building at the site, made of timber and thatch, was first thought to be for animal use its structure and lack of a hearth. However, human refuse (e.g. human fecal material, household waste) excavated outside the building, suggests that it functioned as a place of human habitation (Schofield, 2011), which would have been extremely unhygienic for those residents.

With individuals living so closely, means of disposing sewage and waste became problematic, and sanitation became an issue. Londoners would often dispose of waste in the streets or in cesspools beneath the floor of the house (Landers, 1993). For this reason, roads were cambered to allow water runoff into gullies dug along the sides of the streets. All kinds of waste material could be found in these gutters and open drains ran between houses to carry waste away from building further exposing humans to deleterious sewage. This inadequate and unhygienic sewage system existed until the nineteenth century (Smith, 2012).

As early as the thirteenth century, London attempted to improve the cleanliness of the city by implementing measures to clean the streets and regulate the disposal of waste (Schofield, 2011). Sanitation within households was also lacking, as latrines could be found near, and at times within, kitchen areas (Rowsome, 2000). In the thirteenth and fourteenth centuries, however, archaeological evidence of sanitary regulations mentioned in historical records is apparent. For example, latrine pits started to be constructed from stone rather than timber to better contain waste (Schofield, 2011).

According to historical records, windows were required to be higher than 16 feet above ground, to keep Londoners from throwing rubbish and waste on the streets. Schofield (2011), explains that this law must not have been seriously enforced since several excavated residences had windows below 16 feet.

Indoor and outdoor latrines and cesspits were home to an abundance of insects, with a total of over 10 million insects documented at excavations at Coppergate alone (Kenward and Large, 1998). Urbanism created natural habitats that were better suited for some insects than rural areas, and they thrived in this setting (Smith, 2012). These are synanthropic communities of species favored by clearly artificial rather than seminatural conditions (Kenward and Allison, 1994). One predominant characteristic of urban insect faunas is that they all contain an abundance of flies, which contributed to epidemic diseases (Smith, 2012).

The pathogen jump to a new host, which in the case of urbanism is initiated by increased population density, leads to the development of new epidemic diseases (Cohen, 1989). Frequently the new population of hosts, lacking the resistance built up by the close evolutionary association between primary host and parasites, suffers high mortality. Once established in a new host, strains of the pathogen may evolve along other pathways (Busvine, 1976). These strains may be rapidly disseminated pneumonically, or their other hosts become city-domesticated synanthropic species, closely associated with people (e.g. houseflies and rats). Thus, where an intermediate arthropod vector is involved, an additional line of evidence becomes available in that of fleas, ticks and lice recovered from archaeological deposits within urban contexts. Fly

species (e.g. house fly, stable fly, and the lesser housefly) have all been implicated as vectors in a range of diseases, usually causing bacterial diarrhea and fevers through deposition from flies on food (Smith, 2012).

Additionally, bioarchaeological studies of infant health have been used as a measure for sanitary conditions of a community, as the health of infant mortality is mostly attributed to exogenous factors (e.g. infectious disease and poor nutrition) (Goodman and Armelagos, 1989; Saunders and Barrans, 1999). Lewis has conducted several studies focusing on juveniles during the medieval period and post-medieval England to examine urbanization (Lewis, 2002; Lewis and Gowland, 2007; McEwan et al., 2005). Similarly, to determine whether urbanization and industrialization had an effect on health, Lewis (2002) conducted a comparative study of child health in medieval and post-medieval England. Subadult health was examined using skeletal remains from four sites. It was argued that there were health differences between urban and rural sites, with increased mortality in urban environments. Lewis and Gowland (2007) investigated urbanization by comparing infant mortality rates from urban and rural sites in post-medieval England. Acknowledging the limitation that increased fertility poses to studies of infant mortality, the authors found that urban environments impacted neonatal and postneonatal mortality. Neonatal mortality, which the authors consider endogenous, was higher than post-neonatal mortality in the contemporaneous rural sample. In contrast, the urban sample had a greater proportion of postneonatal deaths, which they attributed to environmental or exogenous factors. Lewis and Gowland (2007) also noted that early weaning and poor sanitation may have contributed to post-neonatal mortality

rates and propose that the urban environment negatively impacted infant health.

In addition to the sheer number and density of people in a city and subsequent sanitation, water contamination is typically cited as one of the primary sources of infectious disease in urban areas (French, 1979). Microbial pollution of water supplies is typically characteristic of environments undergoing urbanization, and many pathogens responsible for disease are essentially water-borne (WHO 1992). This type of pollution can occur from humans and animals using water sources to bathe in or through intermediate hosts (French, 1979). Archaeological evidence of this can be seen in parasite eggs in human fecal matter of urban cesspits. The presence of these parasites indicate that maw worm, whip worm, and various other species of tapeworm were common in the intestines of medieval Londoners of all classes, suggesting that during urbanization individuals carried a relatively large load of intestinal parasites (Smith, 2012).

Wealthier Londoners had the option of purchasing drinking water from outside the city via private companies; however, this was costly. Because the poor could not afford this luxury, they had no choice but to acquire water from the polluted Thames (Dyer, 1989). In the mid-thirteenth century, however, Henry III authorized the construction of a large conduit to provide water to Londoners from the neighboring, less-polluted River Tyburn through lead pipes (Rowsome, 2000). This was an important discovery in London archaeology, as the vault of the building was still intact (Thomas, 2002). The structure lay under the juncture of Cheapside and Poultry at the heart of London, with a staircase leading up to the street (Rowsome, 2000).

Pollution was not limited to contaminated water supplies. Though more of an issue after the medieval period, air pollution was also a concern prior to the Industrial Revolution. Chimneys were common, and historical evidence shows that coal was sold by the sack in the mid-fourteenth century (Schofield, 2011). Also, public records of city complaints show that Londoners were concerned about the smoke produced by metalworkers within the city permeating their house (Brimblecombe, 1976). Biomass fuels (e.g. coal and wood) release particulate matter into the environment that can penetrate the upper and lower respiratory system (WHO 1984). Sinusitis is associated with respiratory health and is one indicator of air pollution in the bioarchaeological record (Bernofsky, 2010; Roberts and Lewis, 2002). This disease is caused by the inflammation of air-filled sinuses of the face that can be recognized from bone lesions within the maxillary sinuses and on the ribs (Boocock et al., 1995).

Roberts (2007) found higher frequencies of sinusitis in sites within the Late Medieval Period (thirteenth to sixteenth centuries) when compared to the Early Medieval Period (mid fifth to twelfth centuries), suggesting that biomass fuels were more abundant in the Late Medieval Period or that housing structures were not as well ventilated. Additionally, through comparison of medieval urban and rural cemeteries, Lewis (1995) found high frequencies of sinusitis. Archaeological evidence of an increased frequency of central hearths and several families living in one residence support these studies (Schofield, 2011). Such small spaces would have filled up with smoke more quickly if fires were used, with irritation of respiratory tracts likely to result and the high density of people enabling the transmission of infection. It is important to

note, however, that sinusitis can also be caused by dental infections and so reported frequencies in cemetery collections may be inflated.

1.5.4 Food supply and diet

In addition to providing information about unsanitary conditions and various forms of pollution, archaeological and bioarchaeological evidence can also be informative about diet, consumption habits, and economic influences of food supplies. By the early fourteenth century, most of London's food and fuel was supplied by a complex system of production in surrounding manors and farms (Dyer, 1989). London was also a point of trade to other countries and continents. Manors were able to maintain their own gardens, and larger manors would sell their surplus to markets (Dyer, 1989). Smaller animals were reared within the city (pigs and chickens), while larger domesticates like cattle were reared a greater distance from the city. Cattle bones are not as abundant in zooarchaeological assemblages in London though, as butchers often returned bones because of difficulties in disposing them in town (Albarella, 1999). Standard diet would have comprised of meat, fish, and shellfish from local markets that was supplemented with their own poultry if space permitted (Adamson, 2004). Fruits and vegetables would have been purchased from the market, as London was becoming a more cash-based economy (Carlin, 1998).

Archaeological evidence of faunal material can be useful in corroborating or enriching historical accounts of diet in medieval London. Analysis of faunal material indicates that those in the city ate more meat than their rural counterparts (Thomas,

2002). This may have been because butchers could be found in town, whereas in rural areas, slaughtering a large animal was infrequent because of the inability to consume the carcass before the meat spoiled. Smaller archaeological evidence, such as botanical remains, can contribute to dietary reconstruction as well. Several samples from rubbish pits of tenth and twelfth century sites near Cheapside were analyzed (Schofield, 2011). Fruit seeds and pips were found, and potential garden plants (e.g. flax, celery) were present as well. Weeds were also present, suggesting adjacent cultivated areas.

Londoners, unlike their rural counterparts, did not generally produce their own food, making them more dependent on the market. Wrigley (1969) has argued that a market economy contributes to high levels of morbidity and mortality in urban communities because in years of poor harvests, grain prices increase, and city inhabitants are unable to purchase sufficient food to stave off malnutrition. As a result, they become increasingly susceptible to infectious disease. Mortality crises often resulted from outbreaks of infectious disease that followed a severe food shortage, suggesting that malnutrition causes greater susceptibility to infection (Dyson, 1991; Galloway, 1988; Mielke et al., 1984).

In the fourteenth century, there were a number of famines, most notably the Great Famine, which killed 10 to 15% of the population of England (Jordan, 1997). Back-to-back poor harvests occurred across Europe as a result of excessive rains and flooding (Farr, 1846; Scrimshaw, 1987). In years of poor harvests, high food prices, combined with declining wages for occupational specialists unable to sell their goods, resulted in urban residents unable to purchase food (Jordan, 1997; Wrigley, 1969). Reduction in

food supplies then led to malnutrition, which would have increased susceptibility to infectious disease and poor health. Excess mortality that correlates with consecutive incidences of famine is evident in the archeological record through catastrophic burials in London (Connell et al., 2012).

There are two important dimensions to diet with regard to health: first, the intake of protein calories and macronutrients such as potassium, sodium, calcium etc.; and second, the intake of essential micronutrients such as vitamins and trace metals (Waldron, 1989). Deficiencies of macronutrients result in starvation and growth failure, while micronutrient deficiencies result in deficiency diseases. Several bioarchaeological studies of cemetery collections in medieval England have investigated the detrimental health effects of potential nutritional stress.

For example, height is considered a good index of the general state of nutrition and reflects failure of an individual to achieve its maximum stature potential (Bogin, 1999; Waldron, 1989). Thus, individuals with shorter stature endured dietary stress or some other cause of stress during their early lives. Moreover, the immune systems could also have been compromised, and may have made them frailer than their counterparts who actually ate well during childhood. Roberts and Cox (2003) found that within medieval England, poorer females (i.e. females who were more likely to experience nutritional stress) were on average more than 5 cm shorter than better-off females, and attributed this difference to chronic malnutrition throughout childhood. Though height may be a good indicator for malnutrition during development, it is important to include additional factors that may influence development, such as

socioeconomic status, genetics, and cultural practices that could affect an individual's diet.

Metabolic diseases are also characteristic of dietary stress, and can potentially be identified in skeletal assemblages. Rickets is a childhood disease that results from vitamin D deficiency. This disease can be identified in skeletal remains because it causes noticeable epiphyseal enlargement, thinning of the cranial bones, and bowing of the long bones (Brickley and Ives, 2008; Mays et al., 2006). On the cranium, iron-deficiency can be seen on the orbital roof as cribra orbitalia or on the cranial vault as porotic hyperostosis (Ortner, 2003). However, these types of lesions have been associated with overall stress experienced during life and should not be considered as indicative specifically of malnutrition, and most of these lesions and skeletal changes are only evident on subadult remains. Roberts (2009) finds that frequencies of cribra orbitalia increases with time during the medieval period in England and concludes that this relationship may be the result of the potential intensification of urbanization factors with time or the result of poor nutrition (Walker et al., 2009).

1.5.5 The Black Death

The catastrophic effect of continental-wide famine was not the only major influence to mortality during urbanization of medieval London. The Black Death occurred between 1348 and 1351 and killed one-third to one-half of the European population (DeWitte and Wood, 2008; Gowland and Chamberlain, 2005). This rapid decrease in population density had a serious economic effect on London, allowing

market expansion that resulted in a higher standard of living for Londoners. The fact that one was alive and able to work made these individuals more valuable and their income increased (Goldberg, 2004). The population did not regain pre-Black Death population levels until after the medieval period, causing more money to be spread among fewer individuals (Schofield, 2011). Rural migrants used this opportunity to make their way to the city to adopt new roles in urban life, as the dead left an abundance of employment opportunities for these migrants, which helped with London's market recovery.

New lane and alleys were built to accommodate migrants, and wealthy tradesmen could afford an even more affluent lifestyle (Thomas, 2002). Markets expanded and London recovered easily, maintaining its position as the largest port and city in England. Official positions were no longer passed down generationally, and historical documents show that several mayors and alderman were from outside of London (Thomas, 2002). Housing, however, did not change drastically. Poor individuals still resided in the lowliest sections of London. However, several residences were replaced with stone foundations, which provided more of a barrier from the outside environment than wood (Schofield, 2011). This was also when stone latrines were built instead of timber latrines.

Though post-Black Death London was an opportunistic time for migrants to take advantage of labor opportunities, rural migrants were making their way to London long before the Black Death created advantageous opportunities. Migrants were important to the development of the city, contributing to population growth and economic

expansion of London (Goldberg, 2004; Wrigley, 1969). Migration into London from neighboring rural areas was particularly common of adolescents and young adults, especially women (Goldberg, 2004) because of greater economic opportunities (Dyer, 2002). These women were employed primarily as servants and were charged with sex-specific food-preparation tasks, which could have increased the risk of transmission of disease.

Epidemic or crowd diseases, such as smallpox and measles, are only able to establish themselves in a population once a particular threshold population size has been reached (Manchester, 1992). As these diseases are typically viral in origin and are associated with high mortality rates, infected individuals either die or gain lifetime immunity to subsequent infection. Therefore, a sufficient number of previously unexposed individuals must enter the population for the disease to maintain itself (Milner, 1980). Thus, the influx of migrants provided the conditions necessary for these diseases to become established within London.

1.5.6 Migrants into London

There are two major hypotheses regarding the health of migrants to cities. Rural-urban migrants might have had a greater risk of disease acquisition because of exposure to new pathogens to which they had no immunity. Endemicization of diseases in dense urban population resulted in immunity to those diseases in urban adults, while in less-dense areas, epidemics remained infrequent and prevented rural-dwelling individuals from acquiring immunity during childhood to the diseases present in cities (McNeill,

1980). Thus, rural-urban migrants would have been at increased risk of epidemic disease compared to those who were born and lived in the city. Moreover, migrants were often met with inadequate living conditions and little familial support when arriving to the city further increasing their susceptibility to infection.

An alternative hypothesis suggests that young adult migrants could have been more robust than their urban counterparts because those who migrated were usually healthy, while migrants that fell ill were more likely to return home before death (Davenport et al., 2010). Research exploring this hypothesis in past populations of London, however, used census and burial data (available only after the medieval period), to reconstruct the age distribution of the population, a source wherein migrants are frequently absent; and the population in question was a post-medieval population (1750-1824 CE) a period when London had already experienced high levels of urban-rural migration (Davenport et al., 2010). Thus, the immunological distinction between rural-born and urban-born migrants could have receded with time as epidemics became increasingly frequent and widespread (McNeill, 1980), contributing to the lack of native and migrant mortality differences.

While migrants figured prominently into the fertility and mortality experience of medieval English cities, comparatively little is known about immigration during the Early Modern period (sixteenth to nineteenth centuries) because of the invisibility of migrants in most of the available demographic records. Migrants are also difficult to identify in the archaeological record, though innovative biochemical techniques using skeletal samples, such as stable isotope analysis, are being implemented to potentially identify

these individuals (Beaumont et al., 2013a; Evans et al., 2006; Evans et al., 2010; Montgomery et al., 2005; Müldner et al., 2009). For example, Kendall et al. (2013) use stable oxygen and strontium isotope analysis to identify migrants in a Black Death London cemetery, and found that five of the thirty individuals that were analyzed were likely migrants.

To conclude, the rapid increase of population density and development in London facilitated the transmission of infectious diseases, unsanitary living conditions, and precarious food supplies. These factors are often identified in the archaeological record through the analysis of structures, assessment of zooarchaeological and botanical material, insect and parasite analysis, and reconstruction of population health through skeletal assemblages, among other techniques. Moreover, archaeological and bioarchaeological investigations of urbanization in the past can complement or refute incomplete historical records and provide multiple lines of evidence to enrich our understanding of urbanization. Various archaeological methods and analyses can be used to reconstruct the living environment, allowing researchers to construct a more nuanced interpretation of medieval life during urbanization.

Additionally, most urban health risks experienced by populations in the past currently affect modern populations, including increased levels of infection from overcrowding, and inadequate sanitation services and food supplies (Moore et al., 2003). Bioarchaeological research of urbanization provides a unique temporal depth to our understanding of the hazards of urbanism that contributes to our ongoing understanding of human adaptation to these changes and can be informative for

developing affective planning and prevention strategies for urban expansion of contemporary populations.

1.6 Dissertation structure

The core of this dissertation is separated into four complementary chapters that address the research objectives outlined above. The first part (Chapters 2 and 3) focuses on the use of paleodemographic methods to evaluate the effects of urbanization on mortality, as a proxy for health, in medieval England, while the second half (Chapters 4 and 5) uses stable isotope analysis to investigate the relationship between diet and health during the transition to a more urban environment.

In Chapter 2, I evaluate the mortality of medieval London through survivability analysis of St Mary Spital cemetery (SRP98) (*c.* 1120-1539 CE). Analyses include comparisons of temporal phases, age groups and sex. In Chapter 3, I evaluate differences between urban and rural mortality through paleodemographic comparison (hazard analysis and survivability analysis) of urban St Mary Spital cemetery and rural St Peter's (BOH) cemeteries, including comparisons of contemporaneous temporal phases, age groups, and sex between the two cemeteries. In Chapter 4, I assess dietary patterns within St Mary Spital through stable isotope analysis of bone collagen from rib bone samples, including a comparison of isotope values between temporal periods and sub-populations (i.e. age cohorts, and the sexes). In Chapter 5, I evaluate potential biochemical markers of famine by comparing stable isotope values from bone samples between individuals within single interments and individuals within famine-related mass

interments. Additionally, incremental dentine isotope values of tooth samples are compared to rib bone samples of the same individuals to assess how dietary stress changed throughout the lifespan. The final chapter, Chapter 6, is a concluding chapter that summarizes the findings of the entire project, and discusses contributions and future directions.

Chapter 2 - Urbanizing Medieval London: Temporal Changes in Survivability[1]

[1] Walter, BS and DeWitte, SN. To be submitted to *Bioarchaeology International*.

2.1 Introduction

The progression of transitional periods, such as urbanization, often produces negative health outcomes as new conditions of the changing environment become more prevalent (Armelagos and Cohen, 1984; Roberts and Cox, 2007; Steckel and Rose, 2002). For urbanization, detrimental living conditions (e.g. sanitation issues, pollution, and disease prevalence) are assumed to intensify as population density increases. Failure of a population to adapt to conditions associated with urbanization should be evident in lower rates of survivorship as urbanization increases through time, as increasing mortality is believed to characterize urban populations until the Industrial Revolution (mid-eighteenth to mid nineteenth centuries) produced enough wealth to improve dietary nutrition and public sanitation (McNeill, 1976; Wrigley, 1969). Urban intensification, however, must not be considered simply as linearly detrimental because humans constantly adapt to unfavorable environmental conditions around them (Nelson et al., 2007). One approach humans use to adapt to detrimental conditions is by improving their surroundings through policy implementation. Sanitation and pollution directives employed in urban areas have the potential to mitigate the negative effects of urbanization, improving environmental conditions of these areas for urban residents.

During the Late Medieval period (c. 1100-1600 CE), London underwent intensive urbanization (Magnusson, 2013). In the tenth century, approximately 20,000 people resided in London (Dyer, 2002). By the 1100s, the population doubled to approximately 40,000 residents (Barron, 2000). The fourteenth century witnessed another doubling of the population to 80,000, and then to 100,000 inhabitants just prior to the Black Death

(Dyer, 2002; Holt and Rosser, 1990). London surpassed the population size of any other contemporaneous English city in the fourteenth century, with four times the population of the second largest English city, Norwich (Barron, 2000). Reconstructed street patterns produced by archaeological excavations and historical maps show the rapid increase of population density, with preexisting blocks hastily subdivided to accommodate rapid population increase to create new spaces for housing (Thomas, 2002). Repercussions of high population density are evident in primary documents including complaints about sanitation regulation violations recorded in the *Assize Nuisance* (court documents specifically for private nuisance complaints) (Chew and Kellaway, 1973).

The rapid population growth in London resulted in the need to adapt to potentially deleterious conditions that came with the rapidly increasing population density, (e.g. sanitation issues, precarious food supplies, and elevated risk of infection). As early as the thirteenth century, London officials attempted to improve the cleanliness of the city by implementing measures to clean the streets and regulate the disposal of waste (Rawcliffe, 2013; Schofield, 2011). Concerns about sanitation in medieval London are evident in legal regulations for waste disposal and plans for basic sanitation systems implemented by the mayor of London, aldermen, and councilors of the city (Sabine, 1934; Sabine, 1937). From 1276, London's *Letter Books* show that the number of sanitation orders increased by four times, and that fines for waste disposal infractions also increased (Sabine, 1937). Though several policies pertaining to sanitation in London are evident in the historical record and through archaeological evidence, it is unclear if

these efforts to produce a more hygienic city were affective at mitigating the potential detrimental consequences of urbanization for Londoners.

To date, most bioarchaeological studies of urbanization and past health patterns have considered urbanization as a monolithic process, excluding the temporal component necessary to understand how health patterns changed as humans adapted. A more nuanced approach that incorporates changes in urbanization through time and considers changes in health as a result of adaptation is integral for clarifying the relationship between human health and the urbanizing environment. Though some bioarchaeological studies of urbanization have included temporal changes of health during urbanization (e.g. Morfin et al., 2002 and Storey, 1985), these studies are mostly limited to the New World.

Further, in addition to evaluating changing health patterns for the entire population, groups within the population (e.g. subadults and females) should also be considered, as sub-populations were potentially differentially affected by, and adapted to, the effects of urbanization. For example, in Chapter 3, females from a Late Medieval London cemetery are shown to have experienced elevated risks of mortality and reduced survival compared to females in a contemporaneous rural cemetery, while males faced equal risks in both environments. This pattern can be attributed to a higher proportion of females migrating from rural to urban centers in search of work opportunities, potentially exposing them to poverty and famine, and to pathogens during immigration or upon arrival in the city (this topic is further discussed in section 3.4.1 Possible explanations for adult patterns of mortality and survivorship).

The focus of this study is a comparison between temporal changes in survivability in the medieval urban St Mary Spital cemetery (SPR98) in London (*n* = 386) and the rural St Peter's cemetery (BOH) in Lincolnshire (*n* = 220), to test the hypothesis that urbanization resulted in temporal declines in survival that did not occur in a contemporaneous rural environment. The effect of time period (Early Phase c. 1120-1300 CE *vs.* Late Phase c. 1300-1539 CE) on survivability is evaluated within each cemetery. To gain a more refined understanding of survivability in an urbanizing environment, sub-populations within the cemeteries are also considered by evaluating survivability changes through time for 1) just the adults (with sexes pooled), 2) the sexes separately, 3) and subadults (individuals below 15 years of age-at-death).

2.2 Materials and methods

2.2.1 Skeletal assemblages

2.2.1.1 St Mary Spital cemetery

The urban cemetery analyzed for this study is St Mary Spital cemetery (SRP98) in London, England (Figure 2.1). The cemetery spans a large time period and was in use during the early urbanization of medieval London, from 1120 to 1539 CE (Connell et al., 2012). SRP98 is a large, well-dated skeletal assemblage curated by the Museum of London. The cemetery was used by the general community, and also included burials from the infirmary and some burials from the monastic order (Connell et al., 2012). The

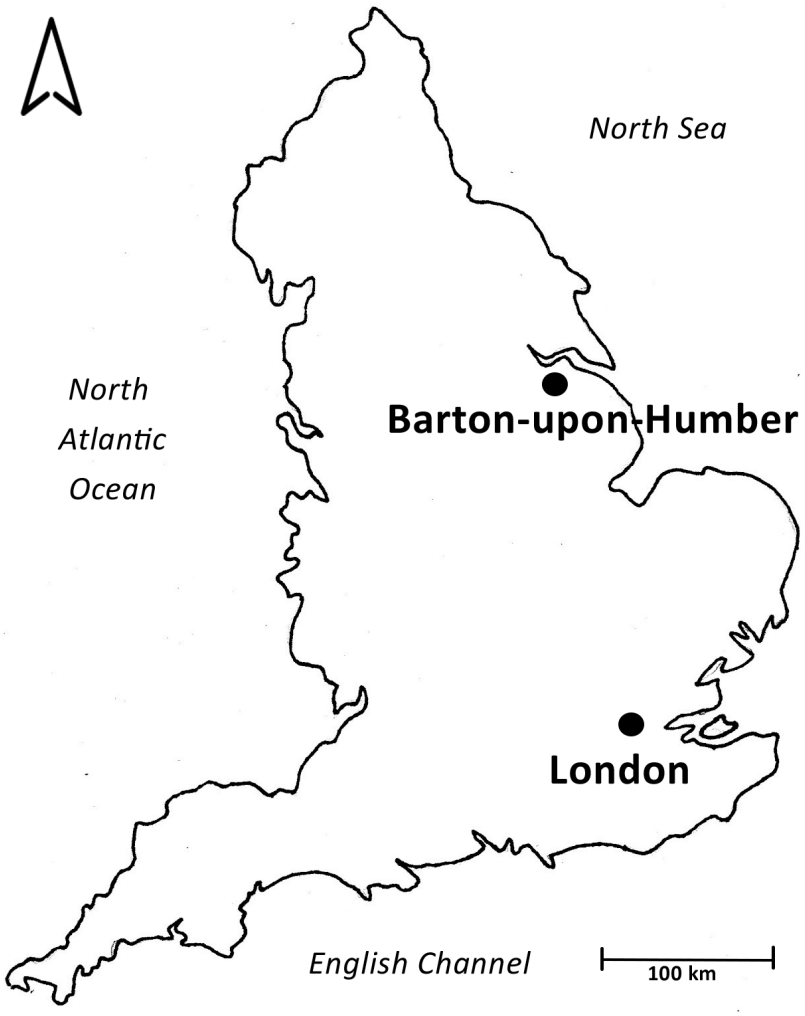


Figure 2.1: Map of England showing locations for London and Barton-upon-Humber

location of the hospital on the outside of London would have attracted travellers and migrants (Connell et al., 2012). The cemetery contains individuals of all age ranges, from infants to mature adults (Connell et al., 2012). Moreover, St Mary Spital was the only hospital in London to accept pregnant women and to care for orphans up to the age of 7 years (Connell et al., 2012). The cemetery includes individuals from various burial contexts, including attritional burials (a single body in a grave, 2-7 bodies horizontally interred in a single grave, and 2-11 bodies stacked in a single grave) and mass burials. The mass burials are associated with catastrophic mortality as a result of famine and the Black Death (Connell et al., 2012).

Though children were interred in the cemetery, there is a notable under-representation of infants and children in the assemblage (Connell et al., 2012). This may be a consequence of preservation (i.e. smaller bones are less dense and would have disintegrate at a faster rate than adult bones (see 1.2.1 Intrinsic and extrinsic biases)). However, this could also be a result of excavation biases. As discussed in section 1.5.1 Rescue archaeology in England, only a portion of SRP98 was permitted to be excavated. It is estimated that there were more than 18,000 burials at SRP98, but only half of these burials were recovered, and then only a portion of these skeletons were recorded (skeletons at or above 30% complete with skeletal characteristics that would included sex and age were recorded) (Connell et al., 2012). Moreover, a concentration of infant burials was found at one location in the cemetery (Connell et al., 2012), which suggests that other infant burials could have been left unexcavated.

For this study, 386 skeletons were randomly sampled from the four temporal phases of SRP98: Period 14 (c. 1120-1200 CE), Period 15 (c. 1200-1250 CE), Period 16 (c. 1250-1400 CE), Period 17 (c. 1400-1539 CE). Temporal periods of the cemetery were determined via high-precision Bayesian radiocarbon dating within a well-defined stratigraphic framework (see Sidell et al., 2007 for details regarding phasing of the cemetery using Bayesian modelling). A stratified random sample of 386 skeletons from skeletons that were at least 50% complete and had the relevant skeletal elements needed for age estimation and sex determination across each of the four temporal phases were selected. For comparison with BOH, individuals from Period 14 and Period 15 are pooled for the Early Phase, and individuals from Period 16 and Period 17 are pooled for the Late Phase. Sample sizes are presented in Table 2.1.

2.2.1.2 St Peter's cemetery

The rural cemetery analyzed for this study is St Peter's cemetery at Barton-upon-Humber (BOH), Lincolnshire, England (Figure 2.1). St Peter's cemetery was in use from 1086 to 1855 CE and is a mixed cemetery with individuals from the village and its hinterland (Waldron, 2007). Barton-upon-Humber was considered a poor rural town during the medieval period, and did not experience significant urbanization until the seventeenth century when factories sprang up along the River Hull (Clapson, 2005). Using radiocarbon dating, dendrochronology, and by establishing relative chronologies

Table 2.1: Sample sizes for St Mary Spital (SRP98) and St Peter's (BOH) cemeteries (Early Phase = c. 1120 – 1300 CE and Late Phase = c. 1300-1539 CE)

			SRP98	BOH	SRP98+BOH
Adult			341	150	491
	Early Phase		174	79	253
		Female	80	38	118
		Male	86	40	126
	Late Phase		167	71	238
		Female	82	37	119
		Male	82	34	116
Subadult			45	70	115
	Early Phase		24	24	48
	Late Phase		21	46	67
All			386	220	606

through localized grave sequences, the cemetery has been divided into five temporal phases: Phase E (c. 950-1150 CE), D (c. 1150-1300 CE), C (c. 1300-1500 CE), B (c. 1500-1700 CE), A (c. 1700-1855 CE) (Waldron, 2007). St Peter's is an ideal rural comparative skeletal assemblage because it is large, provides the approximate temporal phases necessary for comparison with St Mary Spital, and did not experience extensive urbanization during the Late Medieval Period. Individuals from BOH included in this study were randomly sampled from phases that predate urbanization at Barton-upon-Humber (Phases D and C), for a total of 220 individuals. This includes all skeletons from those two phases that were at least 50% complete. For comparison with Early and Late phases of SRP98 (Early: Periods 14 and 15, Late: Periods 16 and 17), Phase D serves as the Early Phase and Phase C the Late Phase. Sample sizes are presented in Table 2.1.

2.2.2 Skeletal analysis

2.2.2.1 Sex determination

Sex was determined for each individual using sexually dimorphic features of the pelvis and skull. Skeletal features of the cranium that were assessed include: supraorbital margin, supraorbital ridge, mastoid process, external occipital protuberance, and mental eminence (Buikstra and Ubelaker, 1994). Skeletal features of the pelvis that were examined include: pubic ventral arc, subpubic concavity, ischiopubic ramus (Phenice, 1969), and greater sciatic notch (Buikstra and Ubelaker, 1994). When available, features of the pelvis were weighed more heavily than those of

the skull, as the former yield more accurate sex estimates (Meindl et al., 1985b; Walrath et al., 2004). Individuals for whom sex determination is not possible or deemed questionable, and for individuals with an age-at-death below 15 years, were not included in analyses evaluating sex differentials.

2.2.2.2 Age-at-death estimation

Age-at-death was estimated using transition analysis if no unfused or fusing epiphyses were present. Transition analysies was used as described by Boldsen et al. (2002), via the ADBOU (Anthropological Database, Odense University) age estimation software. Transition analysis attempts to avoid age estimates that are biased toward a known-age-reference samples, which typically occurs with conventional age estimation methods. For this study, the ADBOU program was set to use an informative prior distribution of age-at-death (the Gompertz-Makeham model) estimated from 17th-century Danish rural parish records, and the conditional probability of known age-at-death estimated from the Smithsonian's Institution's Terry Collection. The parameter estimates for the prior distribution are: $\alpha_1 = 0.01273$, $\alpha_2 = 0.00002478$, and $\beta = 0.1060$. Individuals for whom age could not be estimated are not included in the analyses, and only individuals 15 years of age-at-death and above are included in the analyses for adults (including individuals with indeterminate sex). Subadult age-at-death (individuals below 15 years) was estimated based on dental development and epiphyseal fusion (Buikstra and Ubelaker, 1994; Scheuer et al., 2000).

2.2.3 Statistical analysis

The effect of temporal phase, Early Phase c. 1120-1300 CE *vs.* Late Phase c. 1300-1539 CE, on survival was evaluated within each cemetery using Kaplan-Meier survival analysis with a log rank test. Point estimates of age from the Early Phase and the Late Phase were pooled and "time" was modeled as a covariate affecting survivorship. To assess variation across the lifespan, adult and subadults were analyzed separately within each cemetery. Survival analysis of subadults included adults as right-censored data. To appropriately consider survivability for a specific age group (in this case, subadults), every individual at risk of dying for that specific age group must be included as well, as the age of surviving individuals influence the survivability of the age group as a whole. When considering subadults, adults must be included because they did not die before the age of 15 and survived past childhood, which is the end of the period of observation for subadult survival for this study. For the analysis of subadult survivorship, subadults were coded as having died during the period from 0 to 14.99 years, and adults were coded as not having died during this period (as right-censored). To assess sex differences in adult survivorship, adult males and females were also analyzed separately, using the same approach as above. Kaplan-Meier survival analyses were performed using SPSS Version 21.

2.3 Results

The results of the Kaplan-Meier survival analysis for adults in each cemetery are shown in Table 2.2 and corresponding survival curves for SRP98 are shown in Figure 2.2.

Table 2.2: Kaplan-Meier survival analysis results for all adults, females, males, and subadults

Cemetery	**Phase**	**Mean survival time (years)**	**95% CI**	**Mantel -Cox χ2**	***P***
			Adults		
SRP98	Early	29.48	27.28, 31.68	3.88	*0.05*
	Late	32.99	30.19, 35.80		
BOH	Early	37.35	33.68, 41.03	0.48	*0.49*
	Late	34.63	30.94, 38.32		
			Females		
SRP98	Early	29.52	26.52, 32.52	0.27	*0.60*
	Late	30.74	26.99, 34.50		
BOH	Early	35.97	31.39, 40.55	< 0.00	*0.97*
	Late	34.89	29.41, 40.38		
			Males		
SRP98	Early	30.68	27.32, 34.04	3.59	*0.06*
	Late	35.90	31.71, 40.08		
BOH	Early	39.16	33.43, 44.90	2.57	*0.11*
	Late	34.34	29.38, 39.30		
			Subadults		
SRP98	Early	14.32	14.04, 14.61	0.05	*0.82*
	Late	14.14	13.75, 14.53		
BOH	Early	12.81	11.96, 13.67	6.50	*0.01*
	Late	11.12	11.24, 12.58		

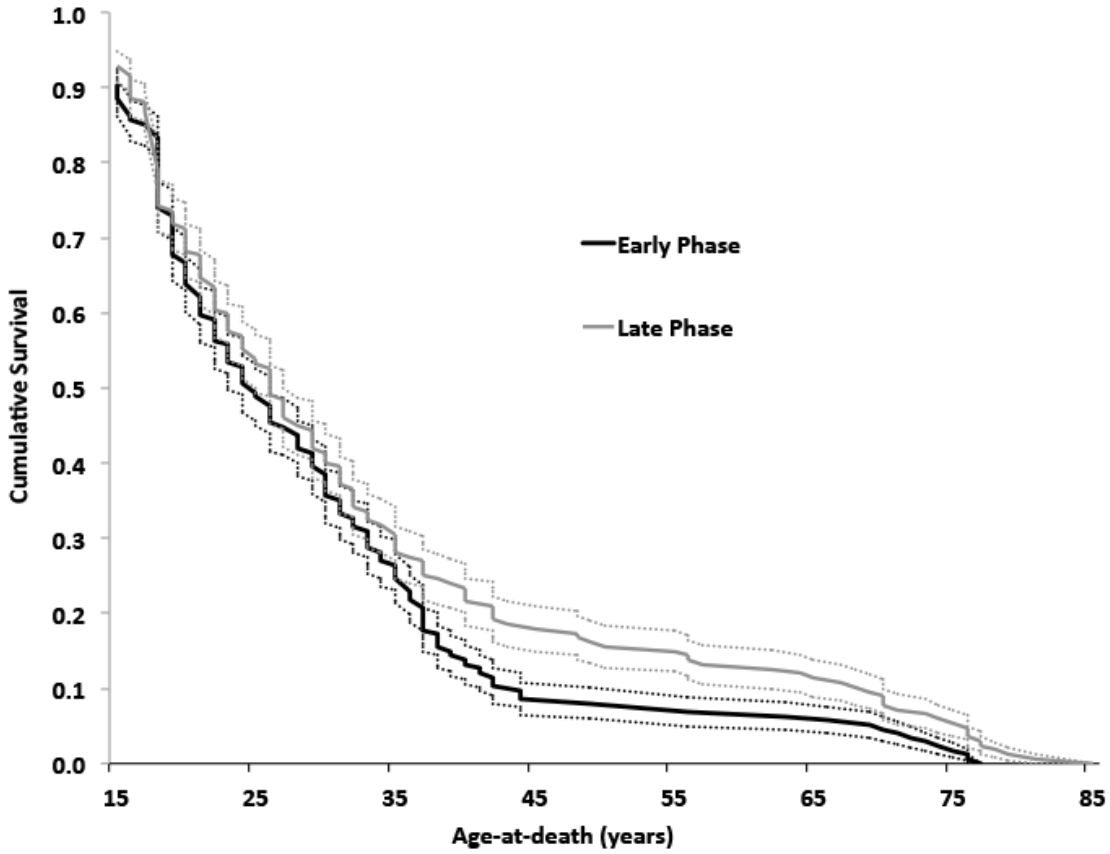


Figure 2.2 Kaplan-Meier survivorship curves of Early and Late Phases with 95% confidence intervals for adults in St Mary Spital

For the urban cemetery, the survival functions reveal a significant difference in mean survival time between the Early and Late Phases (Mantel-Cox *P* = 0.05), and the corresponding 95% confidence intervals for the phases slightly overlap by approximately 1.5 years. Results of the rural cemetery show no statistically significant difference in mean survival time between the phases, and the corresponding 95% confidence intervals overlap substantially. These results suggest that survivorship increased over time for adults in the urban environment, while survivability did not change over time for adults in the rural environment.

The sex-specific Kaplan-Meier results for each cemetery are shown in Table 2.2. For females in both cemeteries, there are no significant differences in mean survival time between the temporal phases, and no significant difference between temporal phases for rural males. However, for urban males, the survival functions are suggestive of a difference in mean survival time between the Early and Late Phases (Mantel-Cox *P* = 0.06), with corresponding 95% confidence intervals overlapping by approximately 2 years (Figure 2.3). These results suggest that as urbanization intensified in London, urban males experienced elevated survivorship.

The results of the Kaplan-Meier survival analysis for subadults in each cemetery are shown in Table 2.2. For the urban subadults, there is no significant difference between the temporal phases. For the rural subadults, survival functions reveal a significant difference in mean survival time between the Early and Late Phases (Mantel-Cox *P* = 0.01) and the corresponding 95% confidence intervals overlap. These results suggest that for urban subadults, survivability did not change with increased

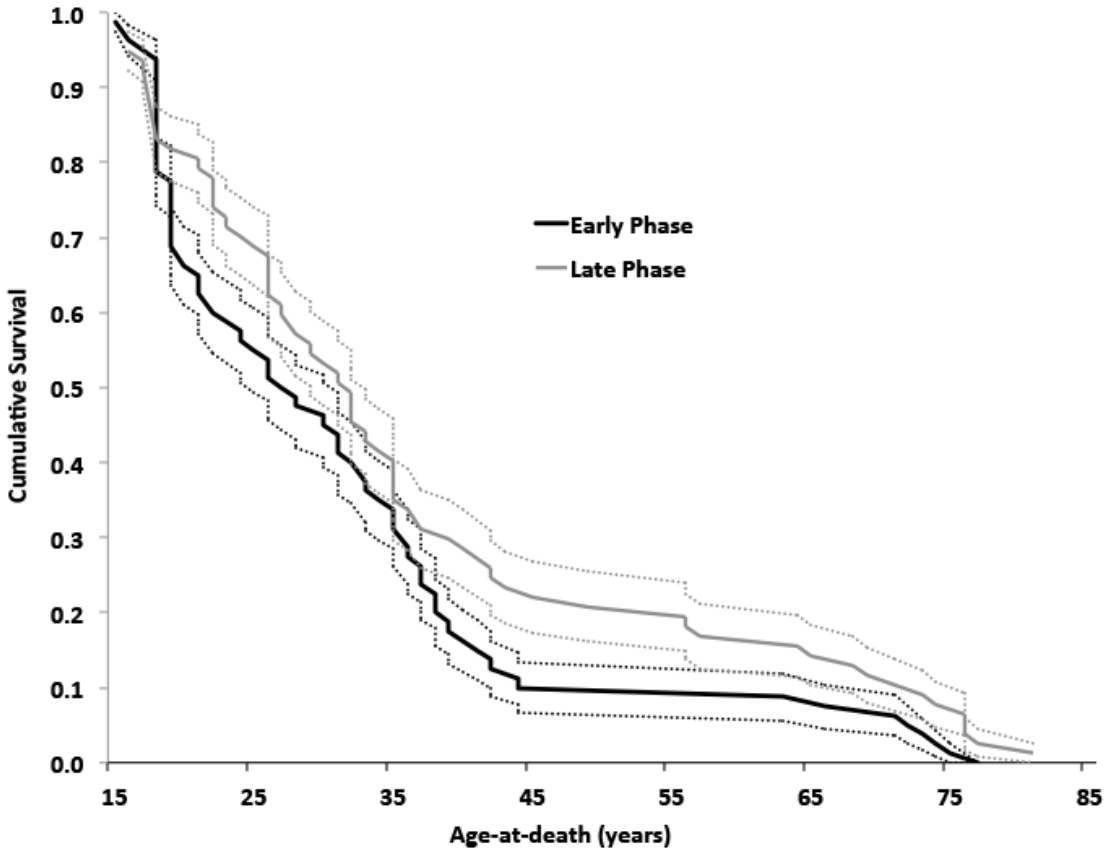


Figure 2.3: Kaplan-Meier survivorship curves of Early and Late Phases with 95% confidence intervals for males in St Mary Spital

urbanization, and in contrast, survivorship for rural subadults decreased in the later phase.

2.4 Discussion

Results of these analyses suggest that, for adults in general, survivorship increased as urbanization intensified in Late Medieval London. Separate analyses of males and females reveal that female survivability did not change as urbanization progressed, while urban males experienced increased survivorship through time. Further, survivability of urban subadults did not change as urbanization increased.

Though in the context of Black Death mortality, previous analysis of survival between temporal periods in Late Medieval London yielded similar results. DeWitte (2014) found in her analysis of London pre- and post-Black Death cemeteries, including SRP98, that there is a significant increase in survival, and thus improved health, after the epidemic, which is consistent with the increased survivability seen in the Late Phase in this study. Unlike DeWitte's 2014 study, however, this chapter focuses on changes in survival in the context of urban intensification in London through time. The inclusion of a rural cemetery in this study allows the comparison survival patterns through time between urbanizing and non-urbanizing environments, rather than only considering survival patterns in London. Moreover, this study includes all burial contexts and temporal periods rather than limiting analyses to only single internments and pre-Black Death temporal periods (i.e. periods prior to 1250 CE) as seen in DeWitte's (2014) study,

which may provide a more accurate depiction of overall survival patterns for different temporal periods.

2.4.1 Adult patterns of survivorship

Increased adult survivorship in an urbanizing London may reflect changes in strategies and implementation of policies to improve sanitation and pollution issues during the Late Phase (as discussed in 1.5.2 An Urbanizing England). As population density increased in London and urbanization intensified, several city-wide regulations and directives were employed to help mitigate the negative effects of urbanization (Rawcliffe, 2013). With individuals living in such close proximity, means of disposing sewage and waste became problematic, causing sanitation issues. London officials attempted to improve the cleanliness of the city by implementing measures to clean the streets and regulate the disposal of waste (Sabine, 1937; Schofield, 2011).

Most evidence for directives to improve sanitation and regulate waste disposal in London were initiated in the thirteenth century (Rawcliffe, 2013). For example, latrine pits were required to be constructed from stone rather than timber to better contain waste and prevent well pollution (Schofield, 2011). By the fourteenth century, most latrines were constructed of stone (Schofield, 2011). In 1277, the London city government ordered that all lanes must be kept clear of dung and other blockages, or a fine would be imposed (Sharpe, 1899). By the end of the thirteenth century, most London roads were paved streets with gutters that facilitated drainage (Nicholas, 2014), and officials were appointed to oversee streets and pavements (Sabine, 1937). Several

ordinances for public sanitation were renewed from the twelfth to fourteenth centuries (Sharpe, 1905), suggesting that there was a deliberate effort to improve living conditions in London.

As urbanization progressed, civic authorities became increasingly committed to communal welfare, and subsequently, the improvement of public health (Rawcliffe, 2013). According to municipal records, Sabine (1934) finds that directives concerning improvement of sanitation in the city increased in the fourteenth and fifteenth centuries (i.e. during the Late Phase for this study). City ordinances of the fourteenth century indicate that residents were responsible for dealing with their own waste and that householders and vendors were responsible for keeping the areas outside of their property clean, paved, and clear from obstruction (Rawcliffe, 2013; Sabine, 1937). Stricter controls were implemented to move hazards such as dunghills and slaughterhouses to the periphery of the city (*Calendar of Close Rolls* 1277-1509). In the fifteenth century, the common council ordered that the Walbrook Stream, which ran through the center of London and was used as an open sewer, be blocked and paved (Sabine, 1934). Subsequently, the River Fleet was also paved over because of sanitation issues (Sabine, 1934). Butchers were now ordered to dispose of their refuse in waterways such as the River Thames, rather than leaving them in gutters, and could be fined for not properly disposing of their waste (Sharpe, 1905). Also during this time, magistrates began implementing legislation that would protect customers against commodities from predatory vendors such as butchers and bakers (Rawcliffe, 2013).

Water contamination is typically cited as one of the primary sources of infectious

disease in urban areas (French, 1979). Wealthy Londoners could purchase drinking water from outside the city via private companies, while most of the poor acquired water from the polluted River Thames (Dyer, 1989). Up to the thirteenth century, the Thames was the ultimate method of waste disposal for London and surrounding areas, with latrines often built over running waterways that eventually emptied into the river (Sabine, 1934). In the mid-thirteenth century, however, Henry III authorized the construction of a large conduit to provide water to Londoners from the neighboring, less-polluted River Tyburn (Rowsome, 2000; Sharpe, 1899), which has been excavated (Schofield, 2011; Sloane, 2004). During the fourteenth and fifteenth centuries, additional springs were incorporated into the conduit and the pipes were extended to serve other parts of the city (Schofield, 2011).

Importantly, the effectiveness of regulations, such as those discussed, hinges on the active participation of the magistrates who implement them and also by the householders who are meant to abide by them (Jørgensen, 2008a). The increase in sanitation ordinances and rate of fines are evident in historical documents, along with the noncompliance of certain Londoners noted in the *Assize de Nuisance*. The question of whether these regulations were appreciated or followed by the residents, however, is up for debate. It is often argued that medieval urban dwellers were indifferent to the deleterious conditions that surrounded them, and that their apathy could be excused by their ignorance of the effects of sanitation and how diseases spread (Pounds, 2005). Williams (1963) suggests that these ordinances were not a communal way of improving

conditions, but actually a necessity to force the “unconcerned community” to be responsible for the condition of the city.

On the other hand, Jørgensen (2014) argues that the increased frequency in sanitation regulations could also be construed as indicators of positive signs of urban cleanliness. Evidence for increased prevalence of ordinances and complaints meant that Londoners were adapting to increasing population density, urban residents were concerned about achieving a clean environment, and that residents had a value for health and dignity of their community (Jørgensen, 2008a; Jørgensen, 2008b). These regulations could also suggest that Londoners and city officials were making connections between disease and cleanliness in the urban environment; city cleaning ordinances and improved sanitation systems were created and implemented mostly because of concerns about the environment on public health (Rawcliffe, 2013). Historical evidence (e.g. building and waste disposal regulations) and archaeological evidence of urban infrastructure (e.g. draining streets and complex conduit systems) indicate that though communities at this time lacked knowledge of how disease spreads, they were still able to devise and implement strategies that improved environmental conditions, and thus health (Bonfield, 2009).

Moreover, views on sanitation at this time, which influenced views on health, were closely governed by aspects of religion. Rawcliffe (2013) argues that initiatives to improve the urban environment were largely a result of moral obligation and religious imperative. Medieval Londoners believed that sinful behavior could be spread much like diseases, and thus epidemics were seen as a form of retribution for an immoral

community (Horden, 2000). It was thus imperative that individuals behaved morally to preserve the wellbeing of the larger community (Rosser, 2000). Magistrates were particularly expected to act morally, as their actions could directly affect the wellbeing of the people they served. This is evident in the Late Phase, when a reform program was launched declaring that officers who participate in sinful activity must be banned from any involvement in civic activities, which would keep immoral officials from achieving spiritual repentance though their civic duties and also protect the spiritual and physical health of the community (Goldberg, 2001).

Most philanthropic gestures to improve the urban environment were actually the result of the elite and merchant class fulfilling their Christian duties or to preserve a place in the next life (Rawcliffe, 2013). The rich were expected to provide funds to build or maintain urban development, particularly sanitation efforts (Rawcliffe, 2013). Construction of sanitation infrastructure as a result of religious obligation from the merchant class and the civic elite increased in the 1300s, resulting in a significant expansion of public utilities in London. These improvements had a large impact on the environmental conditions in London, as most Londoners used these structures. The elite also started leaving funds in their wills to construct and maintain public works such as conduits and public privies in hopes of securing a position in the afterlife (Rawcliffe, 2013).

In addition to the implementation of sanitation and pollution policies in London, the increased adult survivability in the urban cemetery may be a reflection of the consequences of the fourteenth century Black Death. After the Black Death, there was a

substantial decrease in population density, allowing market expansion that resulted in a higher standard of living and subsequent improved health (Bridbury, 1973), which would have occurred in the Late Phase. DeWitte's (2014) analysis of pre- and post-Black Death London cemeteries, including SRP98, suggests increased survivorship following the epidemic, but does not include English cemeteries outside of London. The Black Death, however, affected the entirety of England, and thus the higher standard of living thereafter would not have been unique to London. Therefore, the pattern of increased survivability seen for adults in London should also have been present in the rural sample. Unlike SRP98, there are no mass graves within BOH (i.e. graves characteristic of multiple deaths in a short period of time), but the lack of mass burials in BOH may be due to the smaller size of the population that still experienced the Black Death. For example, in 1593, an incidence of plague resulted in 203 deaths (approximately 26% of the population) and was recorded in parish records (Waldron, 2007). Though there are no parish records for St Peter's prior to the sixteenth century, this suggests that incidences of plague in the fourteenth century most likely reached Barton-upon-Humber but were not evident in the cemetery (i.e. the absence of mass burials does not necessarily mean that there were no plague deaths). Therefore, the lack of increased adult survivability in the non-urban environment, suggests that there are other factors to consider in addition to the improved living conditions after the Black Death that contributed to the increased survivability for urban adults. Additionally, given that plague mortality generally results in an over-representation of adolescents and young adults (15 to 19 years of age) (Castex and Kacki, 2016), the adult survivorship in the Late

Phase, which includes the Black Death, might actually be an underestimation of the elevated survivorship seen in the Late Phase, further underlining the difference in adult survivorship evident between the temporal phases.

The lack of survival differences between temporal phases for the rural adults suggests that this environment may not have experienced environmental changes that affected the mortality of the population. Waldron (2007) notes that, from the skeletal evidence, Barton-upon-Humber provided a stable environment in which to live, and that the population seemed to be adequately nourished and disease frequencies appear to be "unremarkable" (pp.129). As mentioned previously, Barton-upon-Humber did not undergo significant urbanization until the late seventeenth century when factories sprang up along the River Hull (Clapson, 2005). Thus, Barton-upon-Humber did not experience the rapidly increasing population density and sanitation issues experienced in London. Additionally, unlike London, potable water acquisiton in Barton-upon-Humber was not problematic, as freshwater sources were readily available (Rodwell and Atkins, 2011).

2.4.2 Sex-specific patterns of survivorship

Separate analyses of males and females reveal that female survivability did not change during urbanization, while urban males experienced increased survivorship. These results suggest that males may have adapted more successfully to the environmental conditions of urbanization. Though more sanitation and pollution strategies were being implemented as urbanization intensified, female survivability

remained constant. There is no evidence that any sanitation regulations at the time discriminated against women. Men, however, created these regulations, as women did not play any role in urban government and the implementation of these laws (Platt, 1976). Thus, necessary sanitation improvements that would have specifically helped women could have been overlooked.

The unchanging female survivability through time may be a reflection of more young females coming to urban centers seeking better labor opportunities, particularly after the Black Death. Though environmental conditions were improving during the Late Phase, the influx of migrating females into and dying in the city could have obscured the potentially increasing survivability of females. At St Helen-on-the Walls, an English urban cemetery, there was a larger proportion of females aged 25 to 34 compared to males (Dawes, 1980). Similar patterns were also found in Lincoln and York and were attributed to female-led migration (Grauer, 1991). Lewis (2016) and Goldberg (2004) find that, particularly after the Black Death (i.e. during the Late Phase for this study), the number of young females in urban areas increased. This interpretation would complement the assessment in Chapter 3 that the elevated mortality and reduced survivability of urban females compared to rural females may have been due to a higher proportion of rural to urban migration of females looking for labor opportunities.

As stated previously, SRP98 includes individuals from various burial contexts, specifically, attritional and mass graves. Previous research of Late Medieval London cemeteries suggests that there is a lack of difference in mortality between the sexes during catastrophic events such as famine and disease epidemics. DeWitte (2009) and

Castex and Kacki (2016) find that plague mortality was not selective with respect to sex among adults. In an analysis of attritional and famine-related mass graves in SRP98, Yaussy et al. (2016) found a lack of sex differences in mortality during periods of famine, but only prior to the Black Death. This might suggest that the inclusion of mass burials in this study (i.e. the inclusion of individuals who died from catastrophic events) may be obscuring overall survival differences between the sexes, and thus the results from sex-specific analyses may actually be an underestimation of potential differences in survival between the sexes.

2.4.3 Subadult survivorship

The unchanging survivability between temporal periods of urban subadults suggests that subadults were not as affected by urbanization as adults in general. Unfortunately, there is a dearth of evidence in primary documents that could suggest subadults were affected differently by the increase in regulations for sanitation during the Late Phase, as most primary and historical documents typically focus on adults or the population as a whole.

Further, the patterns of survivability for subadults may be an artifact of preservation bias within the different temporal periods. At St Mary Spital, 19.1% of the total recorded cemetery are subadults (Connell et al., 2012), while almost a third of the individuals interred at St Peter's are subadults (Waldron, 2007). St Mary Spital also exhibits a smaller proportion of subadults when compared to other London non-monastic medieval cemeteries like East Smithfield (33.8%) (Cowal et al., 2008), and St

Mary Graces (27.5%) (Bekvalac and Kausmally, 2011). The proportion of subadults at St Mary Spital is particularly low since the hospital served the sick poor, wherein there is usually a high proportion of children dying from malnutrition and infection (Connell et al., 2012). Additionally, subadult bones are more vulnerable to detrimental taphonomic processes after burial (Buckberry, 2000), particularly as time progresses. It is possible that some fragile subadult skeletons, particularly those in the early periods, may not have been considered complete enough to record, which may have resulted in undernumeration of subadults in the cemetery, or that subadult skeletons may have undergone complete diagenesis and could not be recovered. Therefore, this analysis may not have captured some subadults in St Mary Spital, and the constant survivability through time for subadults may actually be an artifact of preservation biases for early periods compared to late periods. Refer to section 2.2.1.1 St Mary Spital cemetery for further discussion regarding preservation and excavation biases.

For the rural subadults, it is expected that, like the adults, the survivability between the Early and Late Phases would remain constant through time, as Barton-upon-Humber did not experience urbanization. The decrease in survivorship for rural subadults reflected in the survivorships functions for this study may actually be an artifact of increased rural-to-urban migration of adolescents and children to urban areas for labor opportunities (e.g. apprenticeships). In Late Medieval England, it was common in England for adolescents to travel from rural areas or small towns seeking apprenticeships and serving positions; children as young as seven could be hired but adolescents hired at twelve to fifteen years of age was more common (Hanawalt, 1995).

Missing adolescents in BOH is consistent with the significant change in survivability age range between the phases (Early: 11.96-13.67 to Late: 11.24-12.58), as less young children would have migrated away from home. Moreover, the increased rural-to-urban migration seen after the Black Death would have meant even more adolescents traveling to urban areas, resulting in an absence of subadults in the non-urban environment during the Late Phase that could be reflected within the cemetery. Further, there is evidence for the trafficking of children into London by criminal gangs, which would have also contributed to the increased migration of subadults (Gilchrist, 2012).

2.5 Conclusion

The results of this study suggest that ongoing urbanization in medieval London varied by age and sex. Comparison of Early and Late phases of urban SRP98 cemetery in London using survival analysis indicates that for urban adults in general, survivability increased as urbanization intensified. The increase in survivorship for urban adults may be a result of an increase in sanitation directives and strategies for improving public health in London.

When the sexes are analyzed separately, survival analysis shows that urban males experienced increased survivability, similar to the survivability pattern of urban adults in general. Urban female survivorship, however, does not change through time. These results suggest that males may have been more successful in adapting to the urbanizing environment. The constant survivability of the urban females through time may be a consequence of a higher proportion of females migrating from rural to urban

areas in search of labor opportunities, particularly after the Black Death, which would have obscured the potential increase in survivability in females.

The constant survivability for urban subadults in this study may be an artifact of preservation biases, as it is expected that sanitation directives in the urban environment would have affected subadults the same was as adults. Preservation limitations of subadults noted within SRP98 may be extended to differential preservation of earlier periods *vs.* later periods in the cemetery. The decrease in survivability through time exhibited for the rural subadults may be a result of an increase in rural-to-urban migration of rural adolescents to urban areas seeking apprenticeships or servant positions.

Bioarchaeological research on urbanization provides a unique temporal depth to our understanding of the hazards of urbanism that clarifies human adaptation to these changes, and can potentially contribute to the development of affective planning and prevention strategies for urban expansion. The results of this project have provided an additional line of evidence, outside of historical documents, to assess whether the increase in sanitation directives in London actually improved health. Additionally, the results of this study supports current literature that challenges the assumption that medieval Londoners were indifferent to the environmental conditions around them, and argues that Londoners were actually making an effort to improve their living conditions, which is evident in the increased survivability of urban adults as urbanization intensified.

Moreover, these results demonstrate that though general comparisons of cemeteries are informative about broad demographic differences or similarities, it is

important to use approaches that allow for the examination of intra-population variation in mortality patterns so that potential disproportionate effects of urbanization on sub-populations may be revealed. Though increased survivability through time is found for adults during urbanization, when females are analyzed separately, it is evident that females actually did experience increased survivability.

Future bioarchaeological analyses of urbanization should consider urbanizing English towns to gain a better understanding of how England experienced urbanization as a whole, prior to the Industrial Revolution (mid-eighteenth to mid nineteenth centuries). London served as a model for urbanizing areas in England (Rawcliffe, 2013), and strategies for improving living conditions during urbanization may be evident in English towns. Further, methods used in this study that incorporate a temporal element to changes in health during transitional periods could be applied to industrialization in Post-Medieval England.

Finally, more research regarding migration in Medieval England is necessary to clarify the effects of migration on changes in fertility and mortality. Though primary documents are available, these documents are often biased against certain subgroups, particularly migrants. Stable isotope analysis using a combination of oxygen, strontium, and lead, can potentially identify migrants within English skeletal assemblages (Montgomery, 2010; Montgomery et al., 2010) and thus contribute to information regarding who was immigrating and where they were coming from.

Chapter 3 - Urban and Rural Mortality and Survival in Medieval England[2]

[2]Walter, BS and DeWitte, SN. 2016. A majority of this study is published in *Annals of Human Biology*: Available online http://dx.doi.org/10.1080/03014460.2016.1275792. Reprinted here with permission (see Appendix F).

3.1 Introduction

Transitional periods, associated with such phenomena as agricultural intensification and urbanization, are of particular interest to bioarchaeologists, as several have concluded that increasing social and economic complexity are associated with dramatic declines in health (Armelagos and Cohen, 1984; Roberts and Cox, 2007; Steckel and Rose, 2002) The United Nations (UN, 1996; UN, 2001a; UN, 2001b) and World Health Organization (WHO, 1998) have made improving living conditions in cities experiencing rapid urbanism a priority, and both call for research exploring the health risks associated with the environmental hazards of urbanism. Most of the urban health risks experienced by populations in the past also affect modern populations, including increased levels of infection from overcrowding and inadequate sanitation services and food supplies (Moore et al., 2003). Bioarchaeological research on urbanization provides a unique temporal depth to our understanding of the hazards of urbanism that clarifies human adaptation to these changes and can contribute to the development of affective planning and prevention strategies for urban expansion.

England underwent intensive urbanization from the 11th to 15th centuries, particularly in its largest city: London (Magnusson, 2013). Most evidence of the medieval London environment comes from primary sources such as court records, complaints, ordinances, or decrees to abate pollution. Environmental archaeology has also contributed to our understanding of urbanization in Late Medieval England, specifically the effects of pollution and urban growth on the landscape (see Hall & Kenward, 1994, and Schofield, 2011). Bioarchaeological research, however, contributes

to our understanding of how the environment may have affected people. By examining the skeletal remains of individuals who experienced urbanization, bioarchaeologists can elucidate patterns of health and mortality associated with urbanism. Analysis of skeletal assemblages of people exposed to urban environmental factors, such as high populations density, potentially elevated risk of infection, and unsanitary living conditions (as described in the Discussion below), can provide unique insights into the effects of urbanization on health and mortality in the past; particularly, when compared to assemblages of people unexposed or less severely exposed to these factors.

Previous bioarchaeological studies investigating health in the context of urbanization primarily assessed raw frequencies of pathological lesions in urban and rural skeletal samples, interpreting higher levels of pathologies in urban samples as evidence for the negative health effects of urbanization (Cohen, 1989; De La Rúa et al., 1995; Lewis et al., 1995; Storey, 1992). However, skeletal lesions should not necessarily be interpreted as direct indicators of health because of heterogeneity in frailty and selective mortality (Wood et al., 1992). Though previous studies of urbanization are potentially informative about broad health changes, they reveal little about underlying heterogeneity within urban or rural populations. In order to more adequately address the potentially complex relationship between urbanization and health, we need to use paleodemographic analyses that allow for intra-population variation in patterns of morbidity and mortality and are thus capable of revealing whether urbanization disproportionally affects certain sub-populations (e.g. males *vs.* females or particular age groups).

Most paleodemographic studies of urbanization have assessed mortality using traditional approaches, such as comparisons of mean age-at-death or life tables (Nagaoka and Hirata, 2007; Steckel, 2005; Storey, 1985). However, the reconstruction of the health consequences of urbanization in the past is inherently complex and might be limited by small sample sizes and traditional methods, given the phenomena of demographic non-stationarity, hidden heterogeneity and selective mortality (Konigsberg and Frankenberg, 2002; Vaupel and Yashin, 1985; Wood et al., 1992). The quantitative models applied in this study, however, allow selective mortality and heterogeneity in frailty to be accounted for. Moreover, the statistical approach used in this study, hazard analysis, accommodates missing data without imposing a particular age pattern on skeletal data (Gage, 1988). Further, this study uses transition analysis, an age estimation technique for skeletal remains that avoids some of the limitations associated with traditional age estimation methods (Boldsen et al., 2002). This age estimation method and hazard analysis are increasingly being used in paleodemography (e.g. Bullock et al., 2013; DeWitte & Wood, 2008; Wilson, 2014). These approaches, however, have not yet been applied to an investigation of urban-rural mortality and survival differences. This study uses hazards and survival analysis to assess demographic differences between the medieval urban St Mary Spital cemetery (SRP98) in London (c. 1120-1539 CE) and the contemporaneous rural St Peter's cemetery (BOH) in Barton-upon-Humber, Lincolnshire, England (c. 1150-1500 CE).

3.2 Materials and methods

3.2.1 Skeletal assemblages

3.2.1.1 St Mary Spital cemetery

St Mary Spital was in use during the early urbanization of medieval London. Medieval London was exceptional in Britain because of the degree to which the area urbanized and how quickly it did so (Thomas, 2002). Urban centers like London attracted poor people, who were presumably vulnerable to infections and exhibited high death rates (Singman, 1999). St Mary Spital was established for treating the general population of London, and occasionally catered to the religious community (i.e. monks and lay sisters) and some wealthy benefactors. Connell et al. (2012) suggest that because there are mass burials within the cemetery and because the burial population is larger than the number of individuals that could have been treated at the hospital, the burial population was not limited to hospital inmates and would have also included individuals from the general community of London.

SRP98 is one of the largest urban skeletal assemblages excavated in Europe to date, with 5,387 skeletons available for analysis, and is curated by the Museum of London (Connell et al., 2012). Using high-precision Bayesian radiocarbon dating within a well-defined stratigraphic framework, the cemetery has been classified into four distinct chronological phases: Period 14: c. 1120-1200 CE, Period 15: c. 1200-1250 CE, Period 16: c. 1250-1400 CE, and Period 17: c. 1400-1539 CE (see Sidell et al., 2007 for details

regarding phasing of the cemetery using Bayesian modeling). A stratified random sample of 386 skeletons from skeletons that were at least 50% complete and had the relevant skeletal elements needed for age estimation and sex determination across each of the four temporal phases were selected; the sample sizes are presented in Table 3.1.

3.2.1.2 St Peter's cemetery

St Peter's cemetery at Barton-upon-Humber (BOH), Lincolnshire, England was in use from 1086-1855 CE; excavation of the cemetery yielded 2,750 skeletons (Waldron, 2007). BOH includes individuals from all levels of society living in the village and its hinterland (Waldron, 2007). Barton-upon-Humber was considered a poor rural community during the medieval period (Clapson, 2005). Though Barton-upon-Humber is often referred to as a small town in the literature, it actually never had a charter in the medieval period or any form of corporate government, attributes that typically differentiate a town from a village (Bryant, 2003). The agricultural village did not experience significant urbanization until the late seventeenth century when factories sprang up along the River Hull (Clapson, 2005), after the period of interest for this study; thus BOH is appropriately considered
"rural" for this study.

The cemetery has been divided into five temporal phases (E: c. 950-1150 CE, D: c. 1150-1300 CE, C: c. 1300-1500 CE, B: c. 1500-1700 CE, A: c. 1700-1855 CE) using radiocarbon dating of coffins and skeletal remains, dendrochronology of coffin boards, and by establishing relative chronologies through localized grave sequences (see Bayliss

Table 3.1: Sample sizes for St Mary Spital (SRP98) and St Peter's (BOH) cemeteries

	SRP98	BOH	SRP98+BOH
Adult (15+)	333	150	483
Male	166	74	240
Female	169	75	244
Subadult (< 15)	53	70	123
All	386	220	606

& Atkins, 2011 for details regarding cemetery phasing). This phasing permitted the selection of a sample that includes only burials that pre-date urbanization at Barton. For this study, 150 individuals were sampled equally from two phases in the assemblage (D and C); the sample sizes are presented in Table 3.1. This includes all skeletons from those two phases that were at least 50% complete. St Peter's provides an appropriate rural comparative skeletal assemblage because it is large, well preserved, and provides the approximate temporal phases necessary for comparison with St Mary Spital.

3.2.2 Skeletal analysis

3.2.2.1 Sex determination

Sex for each individual was determined by examining sexually dimorphic features of the pelvis and skull (per Buikstra and Ubelaker, 1994). See 2.2.2.1 Sex determination in the previous chapter for details regarding sex determination.

3.2.2.2 Age-at-death estimation

Adult age-at-death was determined using transition analysis (Boldsen et al., 2002) via the ADBOU (Anthropological Database, Odense University) age estimation software. Individuals for whom age could not be estimated (i.e. those adults missing or with poorly preserved age-indicators) were not included in analyses. Only adults were included in this study (i.e. individuals with an estimated age-at-death of 15 years and older).

There are several limitations to and biases associated with estimating age-at-death using skeletal remains, particularly for adults. Unlike subadult age estimates, adult skeletal age estimates are typically determined by assessing macroscopic degenerative changes to the skeleton that are also influenced by factors such as environment, genetics, and physical activity throughout life (Bello et al., 2006). In addition to the inaccuracy of degenerative change rates, conventional methods of age estimation risk imposing the age distribution of a known-age reference collection onto the target collection (i.e. age mimicry) (Bocquet-Appel and Masset, 1982). Moreover, traditional age estimation methods assign individuals to broad, predetermined age intervals. An individual with an age-at-death on the low or high end of a predetermined age interval could potentially be categorized into an incorrect age interval, resulting in inaccurate age-at-death distributions. Traditional methods also tend to yield only broad terminal age intervals for the oldest individuals in a sample (e.g. 50 and older), limiting assessment of demographic trends at the oldest adult ages. However, transition analysis, the age estimation method used for this project, attempts to avoid these biases by using some of the criteria articulated in the Rostock Manifesto (see Hoppa & Vaupel 2002).

Transition analysis uses data from a known-age reference sample to obtain a conditional probability $Pr(c_j|a)$ of a skeleton exhibiting a certain age indicator stage or a suite of age indicator stages (c_j), given the individual's known age (a). This conditional probability is then combined with a prior distribution of ages at death using Bayes' Theorem to estimate the posterior probability that a skeleton of unknown age died at a

certain age, given that the skeleton displays a particular suite of age-indicator stages. The ADBOU program uses an informative prior distribution of age-at-death (the Gompertz-Makeham model, see Wood et al. 2002) estimated from 17th-century Danish rural parish records, and the conditional probability of the age indicators given known age-at-death estimated from the Smithsonian Institution's Terry Collection. The parameter estimates for the prior distribution estimated from the 17th-centry Danish records are: $\alpha_1 = 0.01273$, $\alpha_2 = 0.00002478$, and $\beta = 0.1060$. The skeletal age indicators used in this method include several features of the pubic symphysis and iliac auricular surface, and cranial sutures (Boldsen et al., 2002).

Subadult age-at-death (individuals below 15 years of age-at-death) was estimated based on dental development and epiphyseal union (Buikstra and Ubelaker, 1994; Scheuer et al., 2000). Subadult ages were used to assess differences in age-at-death distributions across the lifespan, for survival analysis of subadults, and to estimate the fertility proxy; subadults, however, were not included in hazard analysis, for reasons described below.

3.2.3 Statistical analysis

3.2.3.1 Cox proportional hazards model

The effect of rural *vs.* urban environments on risk of death was evaluated using the Cox proportional hazards model (Cox, 1972) with pooled point estimates of age for adults from both cemeteries and modeling "urban" as a covariate (0 = rural, 1 = urban).

The Cox model is a semi-parametric regression model that estimates relative risk of death, and does not require the specification of the baseline hazard function. The model tests the null hypothesis that the covariate has no effect on the hazard, with the reported hazard ratio indicating the change in risk of death associated with a unit increase in the covariate. Significant ratios ($P < 0.05$) that are greater than 1.0 indicate that the urban covariate is associated with elevated risk of death.

Though the risk of mortality can be assessed across all ages using the Cox model, preliminary analyses using this model and a sample including subadults did not yield significant results; thus, subadults were excluded from this analysis. Subadult individuals, however, were included in survival analyses described below.

Sex differentials in mortality for the urban and rural environments were also evaluated using this approach. To assess whether mortality differences between urban and rural environments are consistent between the sexes, "urban" was modeled as a covariate affecting the Cox model separately for males and females.

3.2.3.2 Kaplan-Meier survival analysis

The effect of rural *vs.* urban on survival is evaluated using Kaplan-Meier survival analysis with a log rank test using pooled point estimates of age from both cemeteries and modeling "urban" as a covariate. Adults (individuals 15 years and above) are included as right-censored data in the survival analysis of subadults (individuals below 15 years), as adults survived past childhood, and thus cannot be excluded from the analysis of urban *vs.* rural differences in subadult survivability (i.e. they did not die

before the age of 15, which is the end of the period of observation for subadult survival). For the analysis of subadult survivorship, subadults were coded as having died during the period 0 to 14.99 years, and adults were coded as not having died during this period. To assess sex differences in adult survivorship in urban and rural environments, males and females were also analyzed separately.

All analyses were performed using SPSS Version 21. It is important to note that these analyses do not take into account the errors associated with estimated ages-at-death; thus the standard errors produced by the Cox proportional hazard analyses and the Kaplan-Meier survival analyses may be underestimated. Therefore, the results from these analyses should be viewed as informative in so far as they indicate general trends, though the numerical estimates themselves should be viewed with caution.

3.2.3.3 Fertility proxy

Changes in fertility (one possible manifestation of demographic non-stationarity) have been found to alter age-at-death distributions irrespective of trends in age-specific mortality (Milner et al., 1989; Paine, 2000; Sattenspiel and Harpending, 1983). In a population experiencing an increase in fertility, for example, increasing numbers of children will be born each year, thus increasing the number of children who die each year, even if age-specific mortality rates do not change. Ultimately, under these circumstances, the resulting cemetery assemblage from this growing population will contain an excess of young individuals relative to older individuals. This phenomenon makes it difficult to infer mortality patterns directly from age-at-death data from

cemetery samples (Milner et al., 2007) and complicates the comparison of two different, contemporaneous assemblages that are potentially derived from populations with different fertility rates.

In this study, fertility was controlled for by examining the number of individuals 30 years of age and above divided by the number of individuals 5 years of age and above (i.e. D_{30+}/D_{5+}). There is a strong negative relationship between D_{30+}/D_{5+} and birth rate (Buikstra et al., 1986), and the corresponding 95% comparison intervals can be informative about whether birth rates differ significantly across samples.

Though the use of this fertility proxy allows the inference of whether differences in estimated survivability and mortality between the rural and urban assemblages are artifacts of differences in fertility between the populations, it does not resolve the potential influence of migration. As is often the case with paleodemographic research, the assumption that populations under consideration were stable (closed to migration and with constant age-specific fertility and mortality rates) is made. This assumption is reasonable, as most populations appear to have stable age structures (Gage et al., 2012). Though previous modeling work has shown that demographic perturbations (resulting, for example, from crisis mortality associated with famine or plague) can have affects on age-at-death distributions that last for several decades (Paine, 2000; Weiss, 1975), substantial effects are relatively short-lived. Given the relatively long period of time across which the samples from both cemeteries are drawn, such potential effects of temporary perturbations in demographic patterns are likely not strong enough to affect conclusions for this study. It is possible, however, that migration during this

period might affect the results. The possible influence of migration on the findings from this study is described in the Discussion.

3.3 Results

3.3.1 Age-at-death distributions

The age-at-death distributions from SPR98 and BOH cemeteries for all adult ages are shown in Figure 3.1. The results of a Kolmogorov-Smirnov test indicate that the two distributions are significantly different ($P < 0.001$). As shown in Figure 3.1, there are more adolescents and young adults (10-19.99 years of age) in SRP98 than BOH. After 20 years of age, the adult distributions are similar until 40 years of age; however, BOH has a higher proportion of individuals 40 to 70 years of age, and SRP98 has a slightly higher proportion of individuals above the age of 70.

3.3.2 Cox proportional hazards model

The results of the Cox proportional hazard analyses are shown in Table 3.2. The estimated hazard ratio, when sex is pooled, is significant and greater than 1.0, with the corresponding confidence interval including only values above 1.0. These results suggest elevated risks of mortality for adults in the urban environment compared to those in the rural environment.

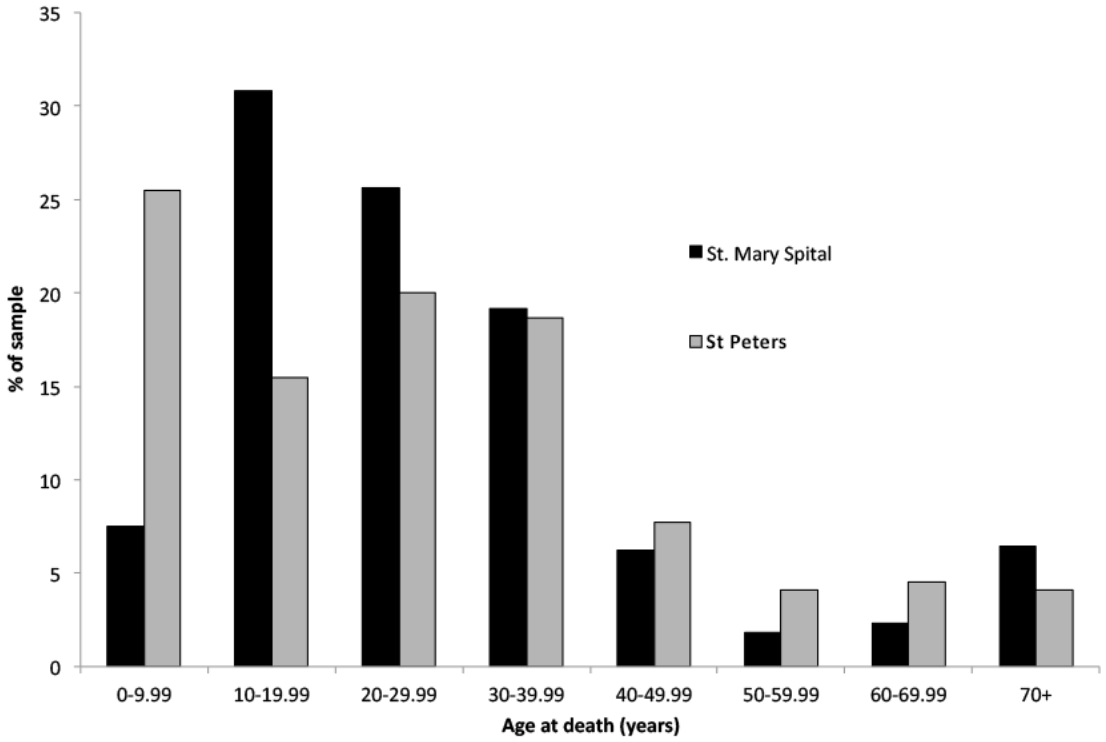


Figure 3.1: Age-at-death distributions from St Mary Spital and St Peter's cemeteries

Table 3.2: Cox proportional hazards results for adults, females, and males, with "urban" as the covariate

	Exp(B)	**95% C. I.**	**Sig.**
Adults	1.225	1.009 – 1.487	0.040
Males	1.104	0.837 – 1.457	0.482
Females	1.349	1.023 – 1.779	0.034

The sex-specific results are also shown in Table 3.2. For females, the hazard ratio is significant and greater than 1.0, with the corresponding 95% confidence interval including only values above 1.0. For males, however, though the hazard ratio is greater than 1.0, it is not significant and the corresponding 95% confidence interval includes 1.0 and values below 1.0. Together, these results suggest that females faced elevated risks of dying in the urban environment, while the risks of dying for males may have been similar in both environments.

3.3.3 Kaplan-Meier survival analysis

The results of the Kaplan-Meier survival analysis are shown in Table 3.3, and the survival curves for adults are shown in Figure 3.2. The survival functions reveal a significant difference in mean survival time between BOH and SRP98 (Mantel-Cox P = 0.04), and the corresponding 95% confidence intervals for the two cemeteries do not overlap. This suggests that urban adults had lower survivorship compared to rural adults.

The sex-specific Kaplan-Meier results are shown in Table 3.3, and the survival curves are shown in Figure 3.3 and Figure 3.4. For females, there is a significant difference in mean survival time between BOH and SRP98 (Mantel-Cox P = 0.04), with slightly overlapping 95% confidence intervals (approximately one year of overlap). There is no significant difference in mean survival time between urban and rural males, and the male 95% confidence intervals overlap substantially. These results suggest that

Table 3.3: Kaplan-Meier survival analysis results for all adults, females, males, and subadults

Sample	Mean survival time (years)	95% CI	Mantel-Cox χ2	*p*-value
		Adults		
SRP98	31.60	29.80, 33.40	4.28	0.04
BOH	36.07	33.46, 38.67		
		Females		
SRP98	31.25	28.77, 33.74	4.07	0.04
BOH	36.00	32.44, 39.56		
		Males		
SRP98	34.51	31.74, 37.28	0.11	0.75
BOH	36.95	33.09, 40.81		
		*Subadults**		
SRP98	14.21	13.98, 14.45	33.48	< 0.00
BOH	11.90	11.24, 12.57		

*Subadult results include right-censored adult data

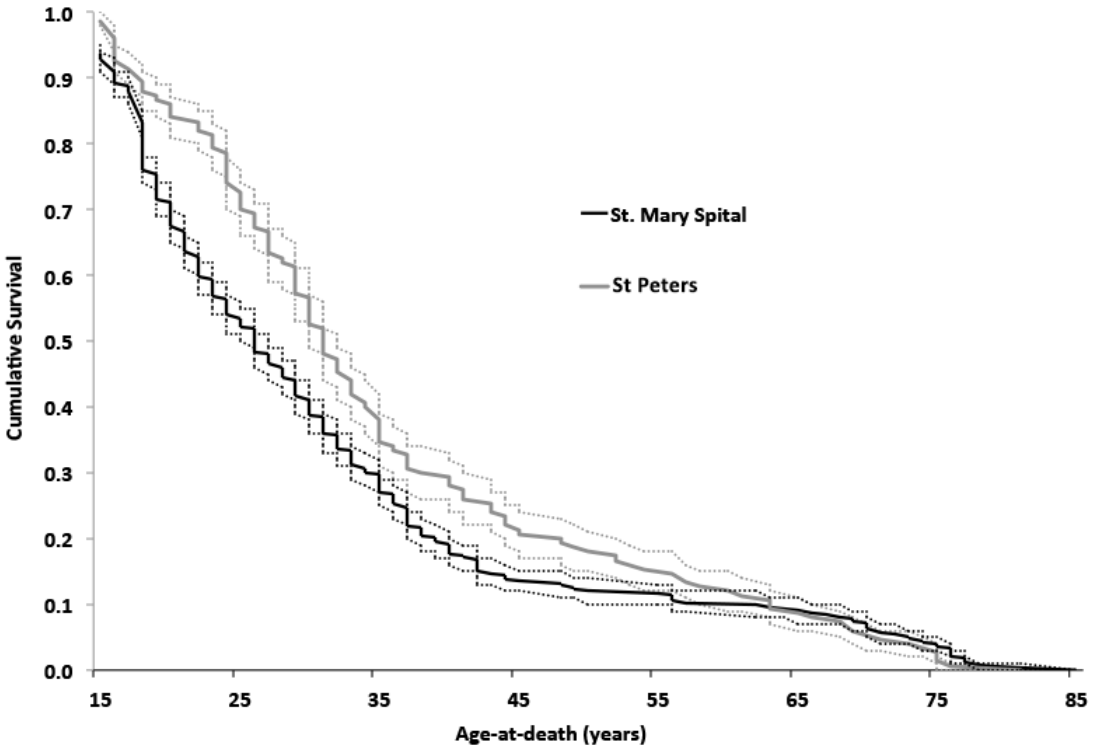


Figure 3.2: Kaplan-Meier survivorship curves with 95% confidence intervals from adults of St Mary Spital and St Peter's cemeteries

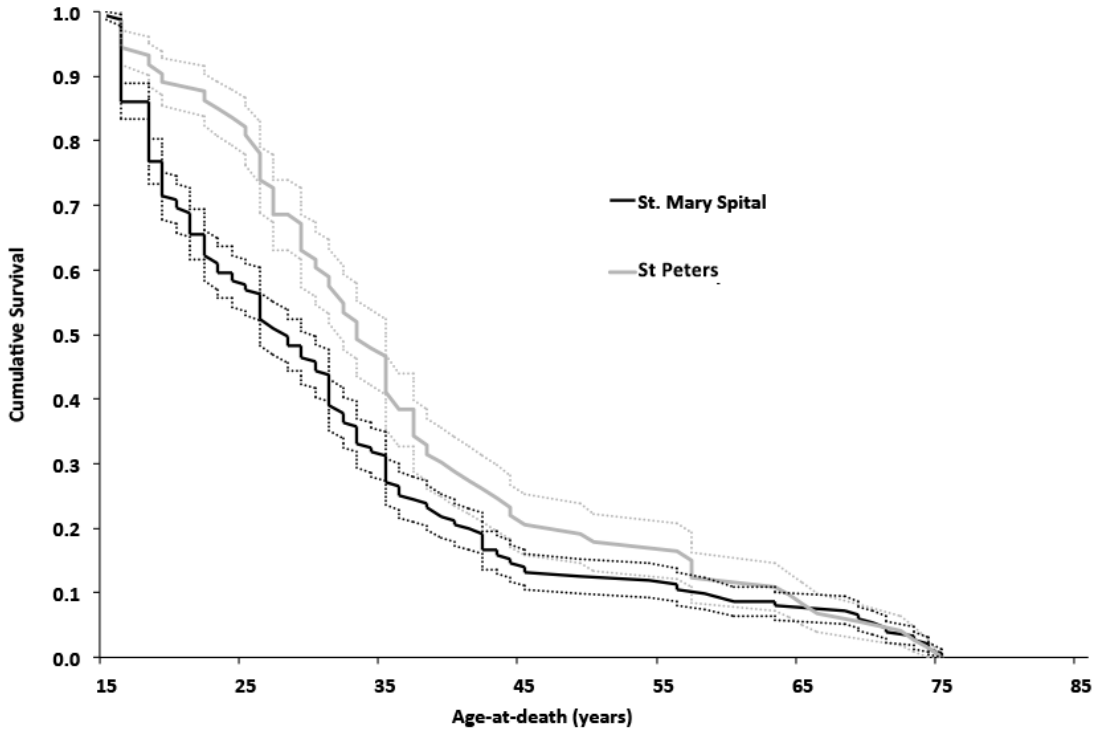


Figure 3.3: Kaplan-Meier survivorship curves with 95% confidence intervals for females from St Mary Spital and St Peter's cemeteries

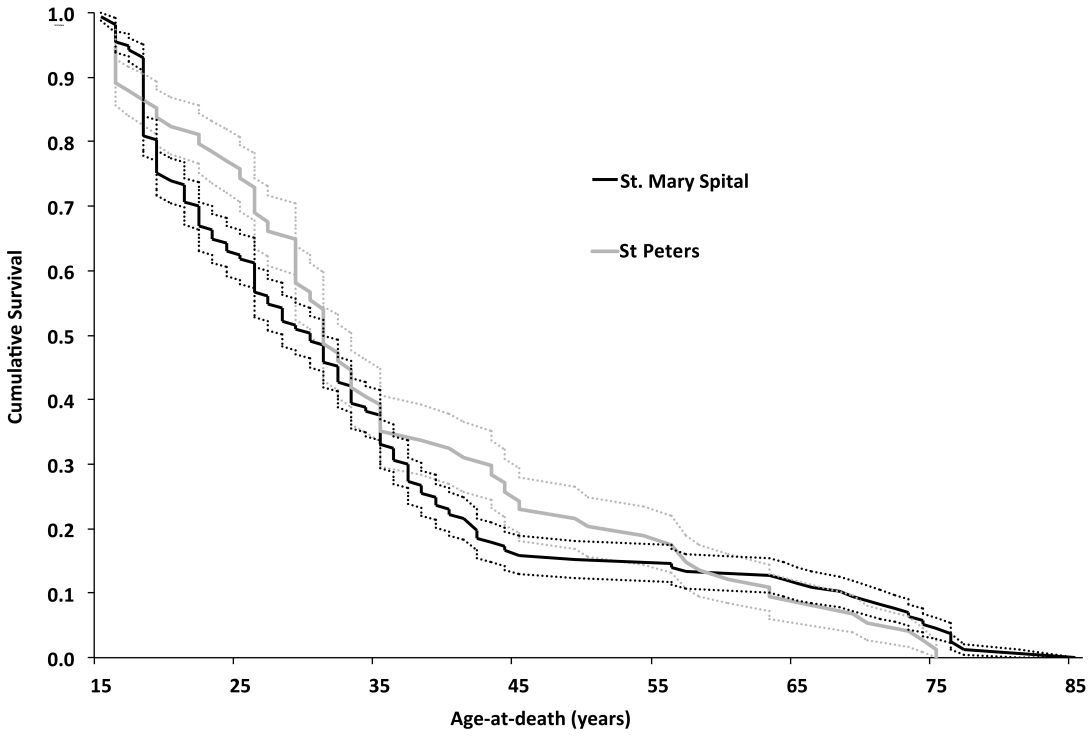


Figure 3.4: Kaplan-Meier survivorship curves with 95% confidence intervals for males from St Mary Spital and St Peter's cemeteries

urban females had reduced survivorship compared to rural females, but males experienced similar survivorship in rural and urban environments.

The results of the Kaplan-Meier survival analysis for subadults are shown in and the survival curves are shown in Figure 3.5. There is a significant difference in mean survival between BOH and SRP98 (Mantel-Cox $P < 0.00$), and the corresponding 95% confidence intervals for the two cemeteries do not overlap. These results indicate that subadults in an urban environment experienced elevated survivorship, compared to subadults in a rural environment.

3.3.4 Fertility proxy

The fertility proxies and their corresponding 95% confidence intervals for both cemeteries are provided in Table 3.4. The D_{30+}/D_{5+} value for BOH is higher than the D_{30+}/D_{5+} value for SRP98, which suggests that birth rates may have been lower in the rural environment compared to the urban environment. However, the comparison intervals for the two cemeteries substantially overlap, indicating a lack of significant difference in birth rates between the two.

3.4 Discussion

The results of these analyses suggest that, for adults in general, there were elevated risks of mortality and reductions in survivorship for individuals in the urban

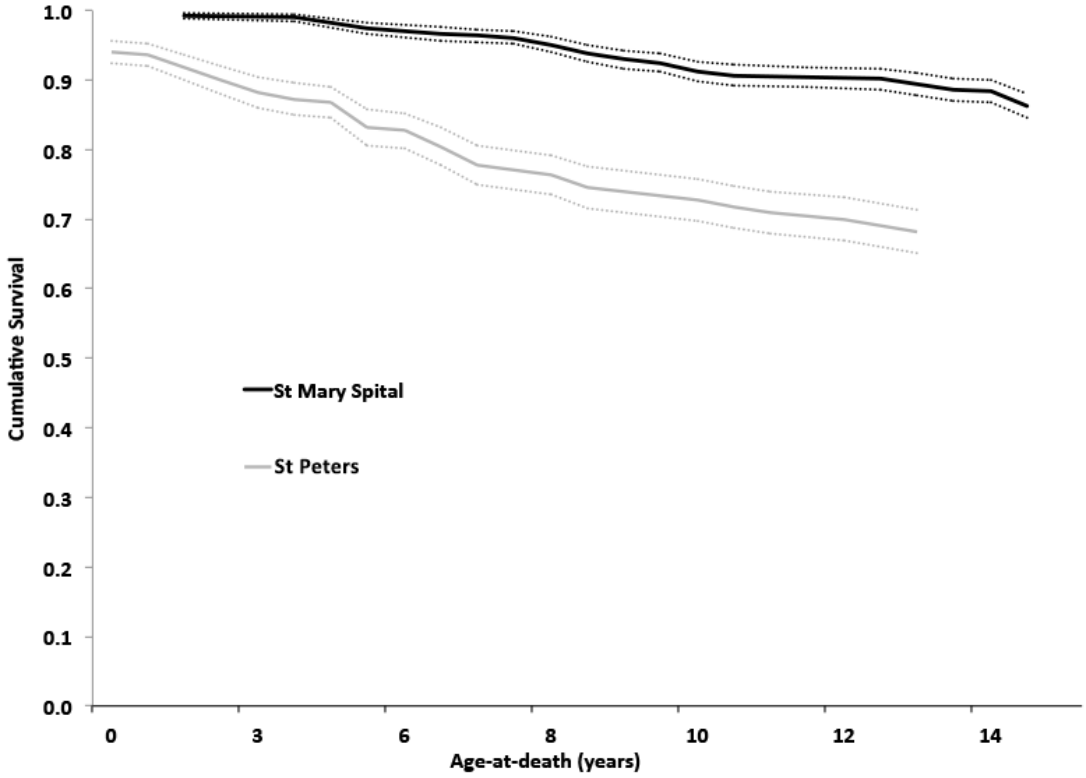


Figure 3.5: Kaplan-Meier survivorship curves with 95% confidence intervals from subadults of the St Mary Spital and St Peter's cemeteries

Table 3.4: D_{30+}/D_{5+} values and 95% confidence intervals for St Mary Spital (SRP98) and St Peter's (BOH) cemeteries

	D_{30+}/D_{5+}	95% CI
SRP98	0.364	0.284-0.444
BOH	0.450	0.337-0.563

environment compared to the rural environment. However, separate analyses of males and females reveal that females faced elevated risks of dying and reductions in survivorship in the urban environment, but the risks for males were similar in both environments. Unlike the adults, subadults faced elevated survivorship in the urban environment compared to the rural environment. The demographic differences between the two cemeteries are not an artifact of changes in birth rates. The inconsistent trends in survivorship suggest that the effects of urbanization in medieval England varied by sub-populations. These demographic differences between the two cemeteries do not appear to be an artifact of differences in birth rates. The inconsistent trends in mortality and survivorship suggest that the effects of urbanization in medieval England varied by sex.

3.4.1 Possible explanations for adult patterns of mortality and survivorship

The observed elevated mortality and, by inference, compromised health in the urban environment for adults in this study are consistent with previous bioarchaeological investigations of urbanization (Lewis et al., 1995; Morfin et al., 2002; Storey, 1985). Comparison of age-at-death distributions between rural Wharram Percy in medieval Yorkshire and urban St Helen-on-the-Walls in York also indicate that urban dwellers did not live as long as rural villagers (Roberts et al., 1998). It is traditionally argued that urban living is inherently risky because of high population density, sanitation issues, water and air pollution, high prevalence of infectious disease, and famine (Moore et al., 2003). Differences in adult mortality and survivorship between the

two cemeteries in this study might reflect detrimental environmental conditions in London compared to Barton-upon-Humber. London was the largest urban center in England during the Late Medieval Period, and experienced rapid population increase, doubling or even quadrupling from the 13th to the 14th century (Schofield, 2011; Williams, 1963) (estimates of the population size at the beginning of the 14th century range from 40,000-80,000 (Holt and Rosser, 1990)). With density increasing rapidly in such a short period of time, waste disposal, and subsequent pollution and water contamination were inevitable problems in London.

Concerns about sanitation in medieval London are evident in the implementation of legal regulations for waste disposal and basic sanitation systems (Sabine, 1934; 1937). City ordinances indicate that London residents were responsible for dealing with their own waste and that householders and businesses were responsible for keeping the areas outside of their property clean, properly paved, and clear from obstruction (Jørgensen, 2008a). In the late 13th century, officials were also appointed to oversee streets and pavements (Sabine, 1937). Several directives for public sanitation were repeated from the 12th to 14th centuries (Sharpe, 1905), which might suggest that efforts to keep the city clean were not successful.

Unlike London, English villages similar to Barton-upon-Humber generally had plenty of space to dispose of their refuse away from residences (Hallam, 1981). Food, household, and stable wastes were often taken to a common area at the periphery of the village that neighbors shared (Miller and Hatcher, 2014), given to pigs owned by the household (Albarella, 2006), or used for agricultural production in fields (Duby and

Postan, 1998; Jones, 2011). At the English village of Wharram Percy, a lack of rubbish pits near households and absence of animal bone near the residential area indicate that household waste was carted to field areas where large quantities of pottery have also been recovered (Beresford and Hurst, 1991).

Sanitary issues and animal waste in the streets of urban areas inevitably led to water contamination. Within medieval London's geology, there was a plentiful supply of well and spring water (Keene, 2001). However, most springs were inconveniently located at the edges of the city, and most wells were within private properties, only accessible to the owners (Schofield, 2011). Londoners without wells most likely used suburban streams for their domestic water needs, which were polluted as result of waste drainage (Keene, 2001) and close proximity to latrines (Schofield, 2011).

Water-borne infections are sustained through pollution of drinking water, which was not prevented in England until the 19th century (Smith, 2002). Waterleaders sold water from the River Thames to residents (Ekwall, 1947), even though it also served as the ultimate method of waste disposal for London (e.g. latrines were built over running waterways that eventually emptied into the River Thames (Keene, 2001)). Boyd's (1981) archaeological study of sediments at the Thames and Fleet Rivers indicate increasing pollution during the same time that Edward III ordered the Mayor of London to clean the area in the early 14th century. In 1237, construction began on the first conduit system, which provided taps at intervals throughout the city (Sharpe, 1899). However, because a fee for using the Conduit was eventually forced, the poor may have still used the Thames as a source of drinking water (Gummer, 2009).

In contrast to London, acquiring fresh water was not an issue at Barton-upon-Humber because several streams traversed the area, flowing into the marshes of the River Humber, and were readily accessible to residents (Rodwell and Atkins, 2011). Residents also used The Beck, which was fed by an artesian spring, to acquire their drinking water (Lyman, 2004; Rodwell and Atkins, 2011). Water supplies at Barton-upon-Humber were likely not at a risk for contamination because rural areas had more space for latrines and waste disposal.

High population density paired with unhygienic conditions made urban dwellers susceptible to infection (Magnusson, 2013). Thus, the prevalence of infectious disease may have been higher in London compared to rural areas. Common diseases noted in Late Medieval London include leprosy, tuberculosis and syphilis (Roberts and Cox, 2003). St Peter's cemetery exhibits remarkably few (skeletally diagnosable) cases of specific infectious diseases, such as tuberculosis, compared to contemporaneous urban areas in medieval England (Waldron, 2007). Waldron (2007) suggests that Barton-upon-Humber may have been immune from the general upsurge in the disease; though it is possible that diseases affecting the population may have been particularly virulent, causing death before the skeleton was affected.

It is possible that the elevated risks of dying and reductions in survivorship in the urban environment reflect the effects of the 14th century Black Death and subsequent outbreaks of plague. Though all of England experienced outbreaks of plague, it might have been more problematic in urban areas, as urban dwellers often fled to rural areas to avoid it (Schofield and Vince, 2003). However, there are conflicting views on whether

towns or rural villages experienced plague differently. Some scholars argue that higher population density caused elevated mortality rates in towns (Britnell, 1994; Wrigley, 1969), while others argue that rural areas experienced higher plague mortality (Benedictow, 2005). Evidence of plague and other short-term fluctuations in mortality are not evident in the BOH skeletal assemblage (e.g. there is no evidence of multiple deaths during a short period of time), but are apparent in parish records (Waldron, 2007). In 1593, an outbreak of plague in Barton-upon-Humber resulted in 203 deaths (approximately 26% of the population) and was recorded in parish records (Waldron, 2007). This level of mortality is consistent with estimates of plague mortality in 17th century London (Cummins et al., 2015), which suggests that plague mortality might not explain the urban *vs.* rural differences observed in this study.

Medical treatment may have been more accessible at Barton-upon-Humber than in London (see Rawcliffe (1995) for a discussion of medieval medical treatment in Late Medieval England), as several doctors and apothecaries are included in the parish registers (Watts, 2013). However, these parish registers did not begin until the 16th century. Care received at SRP98 would have involved spiritual care from the Augustinian canons and nursing care from the lay sisters, with little medical intervention, at least until the 15th century (Thomas, 2000). There is some evidence of physicians at the hospital in the late 15th and early 16th century, but it is not conclusive (Connell et al., 2012).

During the Late Medieval Period, England experienced several famines resulting from changing climate conditions. These adversely affected food production in

neighboring rural areas from which London acquired most of its food (Farr, 1846; Keys et al., 1950). Rural areas like Barton-upon-Humber that were dependent on agriculture, however, may have been better buffered from the famines that occurred during this time. In London, only a few urban residences had land for small gardens (Connell et al., 2012), while village residences could turn to their own fields and livestock rather than selling them for sustenance during times of poor harvest. Thus, the effect of famine could have exacerbated the potentially detrimental effects of urbanization, making nutritional differences, and thus potentially health differences, between urban and rural populations more pronounced.

Rural-to-urban migration is one of the most common types of human movement in all periods of recorded history (Boyce, 1984), allowing cities with high mortality rates to sustain high population densities (McNeill, 1980; Wrigley, 1969). Migrants made up a large proportion of the population in London (Wrigley, 1969). Migrants to urban environments might have faced elevated risk of infection with diseases they did not encounter during their childhoods in rural areas. Some studies find that migrants from rural to urban environments in modern populations have elevated risk of disease compared to urban residents (Baker, 1984; Velimirovic, 1979; Way, 1976). Moreover, rural-to-urban migrants can be exposed to disease during the migration process (Prothero, 1977). Thus, hazardous conditions experienced by rural migrants to London may have contributed to the elevated risk of urban adult mortality for SRP98 observed in this study.

Migration into London from neighboring rural areas was particularly common among adolescents and young adults, especially females, because of greater economic opportunities (Dyer, 2002; Hanawalt, 1995). Evidence for this pattern of rural-to-urban migration is evident in the higher proportion of young adults in the age-at-death distribution of SRP98 compared to BOH (Figure 3.1). According to tax documents, urban cities had more female servants than male servants (Kowaleski, 2014), and females would start formal work away from home at a much younger age than males (Shapland et al., 2015). Moreover, after the Black Death (1348-49 CE), the number of young females in urban areas increased (Lewis, 2016). Female servants were charged with sex-specific food preparation tasks, which could have increased their exposure to zoonotic diseases (Smith, 1997). Osteological evidence of the strain of domestic labor on urban females compared to rural females is evident in medieval English skeletal assemblages as well (Lewis, 2016).

Additionally, a significant proportion of females in medieval London also suffered from poverty (see Leyser, 1995), often living in overpopulated and unsanitary conditions, which would have increased their risk of infection (Taylor, 1983). Moreover, the progression from infection to disease and risk of fatality for reproductive aged females is higher compared to males, at least for some diseases such as tuberculosis, which was increasing in England prior to the Industrial Revolution (Chalke, 1962). Through an analysis of 151 sites in Medieval England, Lewis (2016), found that young urban females showed elevated frequencies of diseases compared to urban and rural male and rural female adolescents (ages 6.5 to 25 years), which she attributes to young

urban females being more vulnerable to disease than other subgroups. All of these factors could have contributed to increased risk of mortality for young females migrating from surrounding rural areas to London, which is consistent with the elevated mortality and reduced survivorship of urban *vs*. rural females in this study. Similarly, at St Helen-on-the Walls, an urban cemetery in York, there was a larger proportion of females aged 25 to 34, compared to males (Dawes, 1980). Similar patterns were also found in St Mark's, Lincoln and York Minster, York and were attributed to female-led migration (Grauer, 1991).

It is important to emphasize the possibility that the elevated mortality and reduced survivorship of females from SRP98 might actually be a reflection of a higher proportion of young females migrating into London from surrounding areas rather than elevated mortality of females after they migrated to London. With respect to the BOH, this sex-specific migration pattern could have resulted in a lower proportion of young females in BOH because they have migrated into urban areas like London and thus would have been buried in urban cemeteries. Not all females were permanent migrants, however, as it was not uncommon for females to return to their rural villages to marry after working in an urban area as an adolescent (Bitel, 2002; Goldberg, 2004), which would underestimate the survival differences between the cemeteries seen in this analysis.

3.4.2 Possible explanations for subadult patterns of survivorship

Contrary to the patterns estimated for the adults in the sample, estimates of survivorship indicate that the urban environment was actually less hazardous compared to the rural environment for subadults. This suggests that all age groups did not experience the detrimental effects associated with urban environments uniformly.

In a study of subadults in medieval England, Lewis (2002) found that the urban environment did not begin to have a substantial detrimental effect on subadult health until the Industrial Revolution. Urban environmental conditions and other factors such as urban employment, socioeconomic status, and infant feeding practices may not have significantly affected subadults until industrialization when these factors would have intensified (Lewis, 2002). Using census data, Preston et al. (1981) found that urban children exhibited lower mortality and elevated survival compared to rural children, similar to the results of this study. The elevated survivability of urban subadults in this study may suggest that environmental conditions associated with urbanization may not affect children as substantially as conditions during intense industrialization.

Perhaps the biggest difference between Barton-upon-Humber and London is that most of Barton-upon-Humbers' residents were engaged in agricultural work for most of their lives, and this might have strongly negatively affected children. According to manorial accounts, children began assisting in fieldwork labor and tending livestock by age ten (Massingberd, 1906), which would have increased their risk of injury and exposure to zoonotic diseases. Zoonotic diseases in particular might have

disproportionately affected young children given that the immune system continues to develop up to 12 years of age (Miyawaki et al., 1981).

Waldron (2007) argued that the population of Barton-upon-Humber likely faced elevated levels of gastrointestinal disease. Diarrhea is most commonly caused by gastrointestinal disease, and can severely affect young children because of their relatively underdeveloped immune systems (WHO, 1968). Skeletal evidence for elevated levels of gastrointestinal disease in rural subadults may be evident in high frequencies of cribra orbitalia in rural skeletal collections given that gastrointestinal diseases are often associated with childhood anemia (Ortner, 2003). Brothwell (1994) found that frequencies of cribra orbitalia were higher in rural Wharram Percy compared to urban skeletal assemblages in northern England. Linear enamel hypoplasia (LEH) is also an indicator of childhood physiological stress, as these enamel defects on teeth reflect growth disruptions during childhood before the age of 6 (Hillson and Bond, 1997). At BOH, Watts (2013) found that about half of the assemblage exhibit LEH, while hypoplastic tooth defects at SRP98 range from 21.6% to 32.9%, which is similar to other London cemeteries (Connell et al., 2012). The difference in the proportion of LEH between these cemeteries suggests that a higher proportion of rural subadults experienced poor living conditions compared to subadults in London, which is consistent with the subadult survivability patterns in this study.

It is important to note that differences in the survivorship of subadults in this study may be an artifact of preservation bias, as seen in the mortality differences for subadults discussed in Chapter 2. At St Mary Spital, 19.1% of the total recorded

cemetery are subadults (Connell et al., 2012), while almost a third of the individuals in BOH are subadults (Waldron, 2007). St Mary Spital also exhibits a smaller proportion of subadults when compared to other London cemeteries like East Smithfield (33.8%) (Cowal et al., 2008), and St Mary Graces (27.5%) (Bekvalac and Kausmally, 2011). The proportion of subadults at St Mary Spital is particularly low considering that the hospital served the sick poor, and it would be expected that a high proportion of poor children died from malnutrition and infection (Connell et al., 2012). Further, subadult bones are more vulnerable to detrimental taphonomic processes after burial because they are small, fragile, and less mineralized than adult bones (Buckberry, 2000). Though 10,516 skeletons were recovered at St Mary Spital, 6,950 of those were preserved well enough and were complete enough to record (Connell et al., 2012). Skeletons considered recordable must have been greater than or equal to 35% complete and must not have consist of only neurocranial fragments, only upper limb, only lower limb elements without pelvic indicators for sex or age (e.g. pubic symphysis or greater sciatic notch), or only a portion of torso less than 30% present with no pelvic skeletal indicators of age or sex (Connell et al., 2012). It is possible that some fragile subadult skeletons may not have been considered complete enough to record, which may have resulted in undernumeration of subadults in the cemetery, or that subadult skeletons may have undergone complete diagenesis and were not recovered.

However, if the subadult pattern of survival is not an artifact of preservation bias and is thus reflective of actual elevated risks of mortality for rural *vs.* urban subadults, then this could have affected the underlying frailty of the rural adults. The abundance of

detrimental childhood hazards and subsequent decreased subadult survival at Barton-upon-Humber may have produced a relatively robust rural adult population. This could be reflected in the reduced mortality and elevated survivorship for rural adults compared to urban adults estimated in this study.

3.5 Conclusion

The results of this study suggest that the effects of urbanization in medieval England varied by sex and age. Comparison of the urban St Mary Spital cemetery in London and rural St Peter's cemetery in Barton-upon-Humber via hazard and survival analysis of pooled-sex samples indicate that urban adults, in general, experienced elevated mortality and reduced survivorship. These results are consistent with most previous bioarchaeological studies comparing urban and rural environments. Environmental factors that are characteristic of urban centers such as high population density, elevated risk of disease, poor sanitary conditions, and famine may have contributed to the detrimental effect of the urban environment on health, and thus mortality and survival.

Analysis by sex, however, suggests that females in the urban sample experienced elevated risks of mortality and reduced survival compared to rural females, whereas males faced equal risks in both environments. These results may be a consequence of a higher proportion of females migrating from rural to urban centers in search of work opportunities. These females may have suffered from poverty, famine, and exposure to

pathogens during immigration or upon their arrival in the city, all of which could have increased their risk of mortality.

In contrast to the patterns estimated for adults, the estimated survivorship of urban subadults is higher than that of rural subadults. This may be a result of childhood agrarian labor and tending livestock in rural areas, leading to increased exposure to animals and thus increased risk of infection from zoonotic disease, and children are particularly vulnerable to zoonotic infection because of their developing immune systems. Rural subadults might also have been at increased risk for gastrointestinal infections, which often led to death during the Late Medieval Period (c. 11^{th}-16^{th} centuries). It must be noted, however, that the survivorship estimates might be an artifact of the undernumeration of subadults in the St Mary Spital cemetery.

This study considers mortality and survival trends along different demographic spectrums (e.g. male/female and adult/subadult) in urban and rural cemeteries, allowing the examination of potential heterogeneity within the urban and rural environments. Heterogeneity across the lifespan is suggested by the inconsistency in the patterns of survival estimated for adults *vs.* subadults, a difference that would have been masked if all ages were pooled for survival analysis. Moreover, the elevated mortality and lower survival for urban females compared to rural females was apparent once the sexes were analyzed separately, and contributed to the overall mortality differences of adults between the two cemeteries. Though general comparisons of cemeteries are informative about broad demographic differences or similarities, it is important to use approaches that allow for the examination of heterogeneity in past

populations so that intra-population variation in mortality patterns can be identified and potential disproportionate affects of urbanization on sub-populations may be revealed. Additionally, this study contributes a unique bioarchaeological line of evidence for the investigation of differential mortality in the context of urbanization beyond primary documents that often exclude the poor and migrants, which comprised the bulk of the population of Late Medieval London.

Finally, urban towns and cities have their roots in rural villages, making the relationship between town and country inherently complex. Migrants contribute to demographic patterns within urban centers but are often invisible from primary historical documents. Future studies using stable isotope analysis to identify migrants in urban centers can offer valuable information regarding where migrants were coming from, who these migrants were (e.g. young females), and how these migrants experienced and adapted to the environmental factors characteristic of urbanism. The integration of biochemical research (e.g. stable isotope analyses) with paleodemography is essential for disentangling the complicated links between mortality, fertility, and migration to better understand medieval life in the face of a changing climate.

Chapter 4 - Dietary Variation in Urbanizing Late Medieval London[3]

[3] Walter BS, DeWitte SN, Beaumont J, and Dupras T. To be submitted to *Journal of Archaeological Sciences*.

4.1 Introduction

Bioarchaeological research on the health effects of urbanization has mostly been limited to skeletal pathologies or mortality rates, generally overlooking the direct contribution of diet to health. In addition to deleterious environmental factors such as sanitation and pollution, urbanizing cities also experience fluctuation of foodstuffs and instances of famines as population density increased (Rawcliffe, 2013). Diet is one the most important mediators of health, and its function as a proximate factor through which several other factors exert their influence (Hill et al., 2011) makes the investigation of diet an integral component in the understanding of health dynamics. Improper diet and undernutrition, often experienced by individuals in urbanizing areas, weakens the immune system, making the body more susceptible to disease (Scrimshaw et al., 1968). The integration of paleodemographic and biochemical data is necessary to reconstruct a comprehensive picture of diet and health, and contributes to the evaluation of synergistic relationships among inequities of nutrition, physiological stress, and disease. Specifically for this study, dietary information from stable isotope data ($\delta^{13}C$ and $\delta^{15}N$) has the potential to provide valuable information about dietary variation experienced by individuals during times of stress, such as urban intensification.

Stable isotope analysis of human tissue has recently gone beyond simple diet reconstruction (Reitsema, 2013) to research about disease patterns (e.g. Richards and Montgomery, 2012), nutrition (e.g. Beaumont and Montgomery, 2016; Neuberger et al., 2013), and physiology (e.g. Fuller et al, 2004; Reitsema et al., 2016; Waters-Rist and Katzenberg, 2010). When interpreted with paleodemographic data, archaeological

evidence, and historical documentation, stable isotope analysis has the potential to be informative about differential access to food sources, social practices, and mobility patterns as well. This is particularly useful in the context of urbanization, as constant migration, fluctuations in food supplies, and disease epidemics are characteristic of this transitional period.

Long-term deprivation of nutrients from insufficient diet, such as a lack of protein and vitamin B, can cause conditions such as anemia and scurvy that leave pathological changes on the skeleton (Brickley and Ives, 2010; Ortner, 2003). Individuals that were more frail than their peers (i.e. individuals with a higher risk of death) may have died more quickly, not surviving long enough for boney lesions as a result of undernutrition to be exhibited on the skeleton (Wood et al., 1992). Stable isotope analysis has the potential to identify dietary trends as a result of undernutrition or differential consumption habits that may not be evident using only osteological analyses of a skeletal assemblage.

During the Late Medieval Period, London underwent extensive urbanization, as the population density increased rapidly from the 11th to 16th centuries (Magnusson, 2013). As discussed in Chapter 2, urbanization is a complex phenomenon that does not simply progress in a linear manner, in part because of the way in which humans adapt to changing environmental conditions. Thus, the inclusion of a temporal element in isotopic analyses of urbanization is necessary to gain a nuanced understanding about how populations adapted to dietary changes during progressing urbanization.

In addition to incorporating a temporal element into stable isotopic analyses of

urbanization, it is essential to consider different sub-populations, as intra-population differences in isotope values may have implications for health disparities. Though isotopic analyses for the cemetery as a whole are informative about population-wide consumption patterns, it is important to use approaches that allow for the examination of heterogeneity in past populations so that intra-population variation in dietary trends can be identified and potential disproportionate effects of urbanization may be revealed.

This chapter evaluates patterns of isotope values during urbanization in Late Medieval London (c. 1120 - 1539 CE) to assess whether an unstable urban environment with increasing population density and instances of famine affected diet by comparing $\delta^{13}C$ and $\delta^{15}N$ values of Londoners across time periods. To assess whether urbanization affected the diet of different subgroups, age cohorts and the sexes are also compared.

4.2 Materials and methods

4.2.1 St Mary Spital cemetery

St Mary Spital cemetery is a large, well-dated skeletal assemblage curated by the Museum of London, yielding 5,387 skeletons that have been analyzed to date. The cemetery spans a long time period (c. 1120-1539 CE) and was in use during the early urbanization of medieval London, when the city experienced increasing population density and multiple instances of famine (Connell et al., 2012). The cemetery is mixed, containing individuals of all age ranges, with burials from the general community, in

addition to patients from the infirmary and members of the associated religious community (i.e. monks and lay sisters) (Connell et al., 2012).

St Mary Spital cemetery is particularly appropriate for this study, as it provides the temporal control necessary to investigate trends in isotope values through time. Using high-precision Bayesian radiocarbon dating within a well-defined stratigraphic framework, burials in the cemetery have been classified into four distinct chronological phases: Period 14 (c. 1120-1200 CE), Period 15 (c. 1200-1250 CE), Period 16 (c. 1250-1400 CE), and Period 17 (c. 1400-1539 CE) (see Sidell et al., 2007 for details regarding phasing of the cemetery using Bayesian modeling). The cemetery also includes individuals from various burial contexts, including attritional burials (a single body in a grave, 2-7 bodies horizontally interred in a single grave, and 2-11 bodies stacked in a single grave) and mass burials (8-45 bodies in a single grave cut). The mass burials are associated with catastrophic mortality as a result of famine and the Black Death (Connell et al., 2012).

Skeletons were initially randomly sampled approximately equally from the four temporal phases for paleodemographic analyses presented in Chapter 2 and 3, and were then selected for bone sampling, further detailed below.

4.2.2 Skeletal analysis

4.2.2.1 Sex determination

Sex was determined for each individual using sexually dimorphic features

of the pelvis and skull (Buikstra and Ubelaker, 1994). See 2.2.2.1 Sex determination in the previous chapter for details regarding sex determination. Individuals for whom sex determination was not possible or deemed questionable, and individuals with an age-at-death below 15 years, were not included in analyses evaluating sex differentials.

4.2.2.2 Age-at-death estimation

Adult age-at-death was determined using transition analysis as articulated by Boldsen et al. (2002) via the ADBOU (Anthropological Database, Odense University) age estimation software. Subadult age-at-death (individuals below 15 years) was estimated based on dental development, epiphyseal union, and (Buikstra and Ubelaker, 1994; Scheuer et al., 2000). To assess whether diet from different age cohorts was affected differently during urbanization, individuals were divided into age cohorts that reflect different stages across the lifespan. Ages for each cohort and sample sizes are provided in Table 4.1.

4.2.3 Bone collagen samples

Sample sizes for rib bone included in this study are available in Table 4.2. To represent temporal groups equally, an approximately equal number of individuals were randomly sampled from each period. Rib samples were obtained from well-preserved bone with no evidence of pathological bone formation. Bone collagen samples were prepared using the modified Longin method (Brown et al., 1988) at the University of Bradford Stable Light Isotope Laboratory (UBSLI). Samples were measured in duplicate

Table 4.1: Age ranges and sample sizes for age cohorts

Age Cohort		***N***
Subadult	0 - 14.99 years	19
Young adult	15 - 24.99 years	65
Adult	25 - 39.99 years	49
Old adult	40 - 59.99 years	13
Mature adult	60 – 100 years	15
All		161

Table 4.2: Sample sizes for adults and the sexes by temporal period

Periods		**14**	**15**	**16**	**17**	
Adult		37	42	35	33	147
	Female	16	19	15	18	68
	Male	20	21	19	15	75

and compared with UBSLI and international standards (see Beaumont et al., 2013a for preparation and analysis details used by UBSLI). The average of the results of the two samples for each individual was used for $\delta^{13}C$ and $\delta^{15}N$ if both samples were considered viable.

Samples considered viable for analysis have a collagen yield > 1% (Van Klinken, 1999), C:N range between 2.9 and 3.6 (DeNiro, 1985), nitrogen content (%N) between 5 and 17% (Ambrose, 1990), and carbon content (%C) between 15 and 47% (Ambrose, 1990). Carbon and nitrogen ratio, nitrogen content, and carbon content for each sample used in this analysis (i.e. viable samples) are provided in Appendix H. Two samples did not produce collagen yield higher than 1% and were excluded from the analyses (SRP98-30515 and -30865). Three samples did not meet the minimum %N requirement (SRP98-24010, -24022, and -30135) and two of those samples did not meet the %C requirement (SRP98-24010 and -24022); all three samples were excluded. Isotope values of rib samples from Lakin's (2010) previous isotopic analysis of St Mary Spital (*n* = 13) are included if they were analyzed previously for demographic data, and follow the sample viability requirements above. There are no correlations between any of the collagen viability indicators (e.g. C:N, %N, and %C), indicating that there is minimal diagenetic alteration of the stable isotope signatures (Ambrose, 1990). The results are expressed using the delta (δ) notation in parts per thousand (per mil or ‰). Analytical error for bone collagen is ± 0.2‰.

4.2.4 Statistical analyses

To assess differences in stable isotope signatures between temporal phases, age cohorts (with pooled temporal phases), the sexes (with pooled temporal phases), and between temporal periods for each sex, nonparametric tests were used: Mann-Whitney U tests for pairwise comparison and Kruskal-Wallis test for comparison of more than 2 groups. The Mann-Whitney U and Kruskal-Wallis tests were used rather than parametric comparison tests because these tests do not require an assumption of approximate normality. Because distributions are similar between all compared groups (an assumption necessary for these tests to compare medians rather than distribution between groups), these tests appropriately assess differences of the medians rather than the distributions across subgroups (Kruskal and Wallis, 1952; Mann and Whitney, 1947). Post-hoc Mann-Whitney U tests were performed for significant pairwise comparisons from Kruskal-Wallis tests. Analyses were conducted separately for $\delta^{13}C$ and $\delta^{15}N$ values.

Regression analysis for the different temporal periods was performed to assess if individuals from each period consumed more varied or less varied protein sources. By evaluating the correlation of $\delta^{13}C$ and $\delta^{15}N$ values, it is possible to evaluate whether a population consumed more varied *proportions* of protein sources or more varied *types* of protein sources. Specifically, by comparing the correlation of $\delta^{13}C$ and $\delta^{15}N$ in different temporal periods, it is possible to elucidate dietary variability patterns through time and $\delta^{13}C$ and $\delta^{15}N$ correlations can also be used to compare cemeteries to evaluate geographic differences in protein consumption. The Pearson's correlation coefficient

measures the linear correlation between two variables (in this case, $\delta^{13}C$ and $\delta^{15}N$) providing a value between 1 and -1 to indicate the correlation (0 = no correlation, 1 = perfect correlation) and direction of correlation (-1 = negative correlation, 1 = positive correlation) between the variables, and thus estimating the amount of variation within the data. Specifically, for this study, this analysis assesses whether the $\delta^{13}C$ values are affected by the $\delta^{15}N$ values. Per Richards and Hedges (1999), the regression line is a line of best fit through the isotope data and represents a hypothetical mixing line, assuming that individual diets are comprised of two end-members. The two end-members in this case are marine protein and terrestrial C_3 protein, consistent with protein sources found in the Medieval English diet (Müldner and Richards, 2007a). Thus any deviation seen from the line of best fit would indicate variation in the consumption of protein. For example, an r^2 closer to 1 would mean that the differences in $\delta^{13}C$ and $\delta^{15}N$ values between individuals within the population is due to diets in differing proportions of marine and terrestrial foods, while an r^2 closer to 0 would mean that the differences between $\delta^{13}C$ and $\delta^{15}N$ is due to different types of marine and terrestrial foods consumed. All analyses were performed using SPSS Version 21.

4.3 Results

Carbon and nitrogen values for bone collagen for all individuals are shown in Appendix H and samples sizes are available in Table 4.2. Isotope values for all individuals are plotted in Figure 4.1. Diet is consistent with a previous isotopic study of SRP98 (Lakin, 2010), though Lakin's study was limited to Periods 15 and 16.

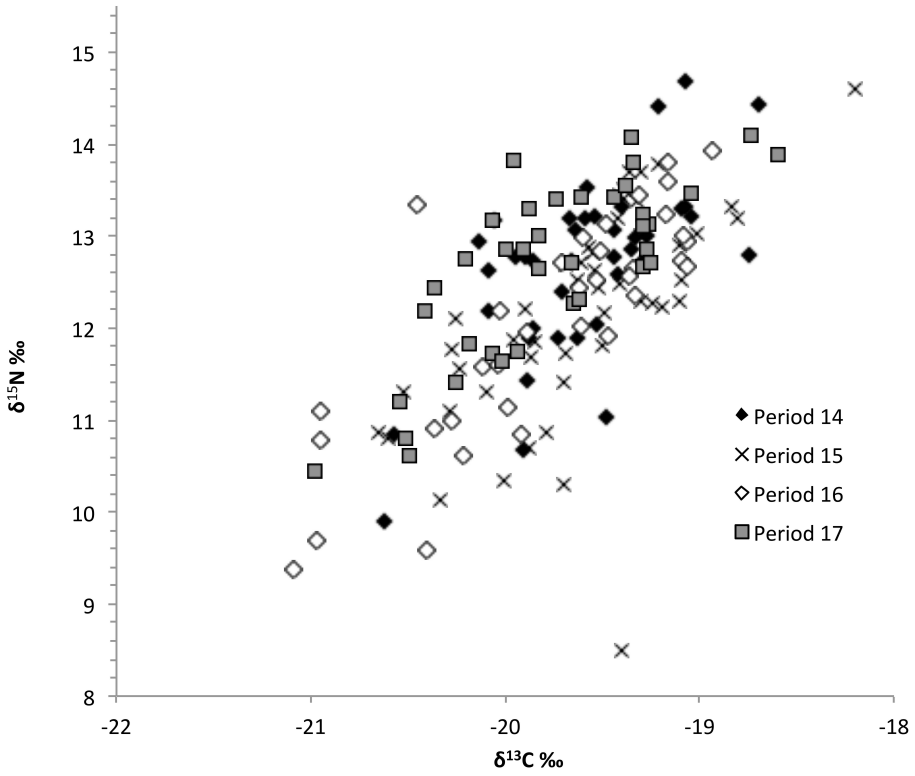


Figure 4.1: Plotted bone collagen $\delta^{13}C$ and $\delta^{15}N$ stable isotope data for all individuals by temporal period

4.3.1 Temporal period

Carbon and nitrogen isotope values for bone collagen are available in Appendix C. Analyses were conducted for all individuals and separately for adults (individuals 15 years and older). Subadults were not analyzed separately because of small samples size. Mean isotope values by period are plotted in Figure 4.2 and boxplots of $\delta^{15}N$ by temporal period are provided in Figure 4.3. Results evaluating differences in isotope values by temporal period using a Kruskal- Wallis test are presented in Table 4.3, and results of post-hoc Mann-Whitney U tests for temporal period with statistically significant pairwise comparisons from the Kruskal-Wallis test are shown in Table 4.4.

For all individuals, the results reveal a difference between temporal periods for $\delta^{15}N$ ($P = 0.07$), but not for $\delta^{13}C$. Post-hoc pairwise comparisons for periods with statistically significant differences indicate that Period 15 exhibits significantly lower $\delta^{15}N$ values compared to Periods 14 and 17. Period 16 does not display a statistically significant difference with any temporal period, and has $\delta^{15}N$ and $\delta^{13}C$ median values falling between increasing $\delta^{15}N$ and $\delta^{13}C$ values in temporal Periods 14 and 17, suggesting a steady increase in isotope values through time. When subadults are excluded, there is no significant difference between temporal periods for $\delta^{15}N$ or $\delta^{13}C$, and thus no post-hoc comparisons were conducted.

Results of the Pearson's regression analysis of $\delta^{13}C$ and $\delta^{15}N$ values are presented in Table 4.5. All correlations are statistically significant and r^2 values indicate moderate correlation between $\delta^{13}C$ and $\delta^{15}N$ for the different temporal periods. Notably, Periods 16 and 17 exhibit correlations closer to 1 compared Periods 14 and 15.

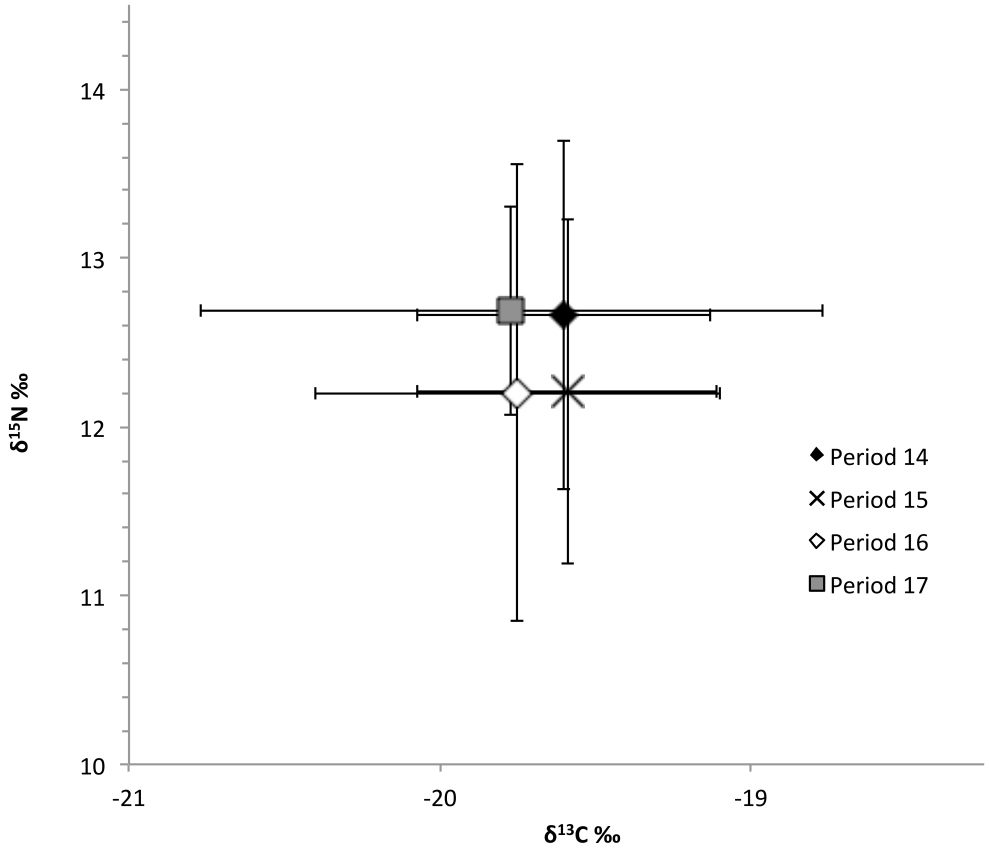


Figure 4.2: Plotted bone collagen $\delta^{13}C$ and $\delta^{15}N$ means (± 1 σ bars) by temporal period for all individuals

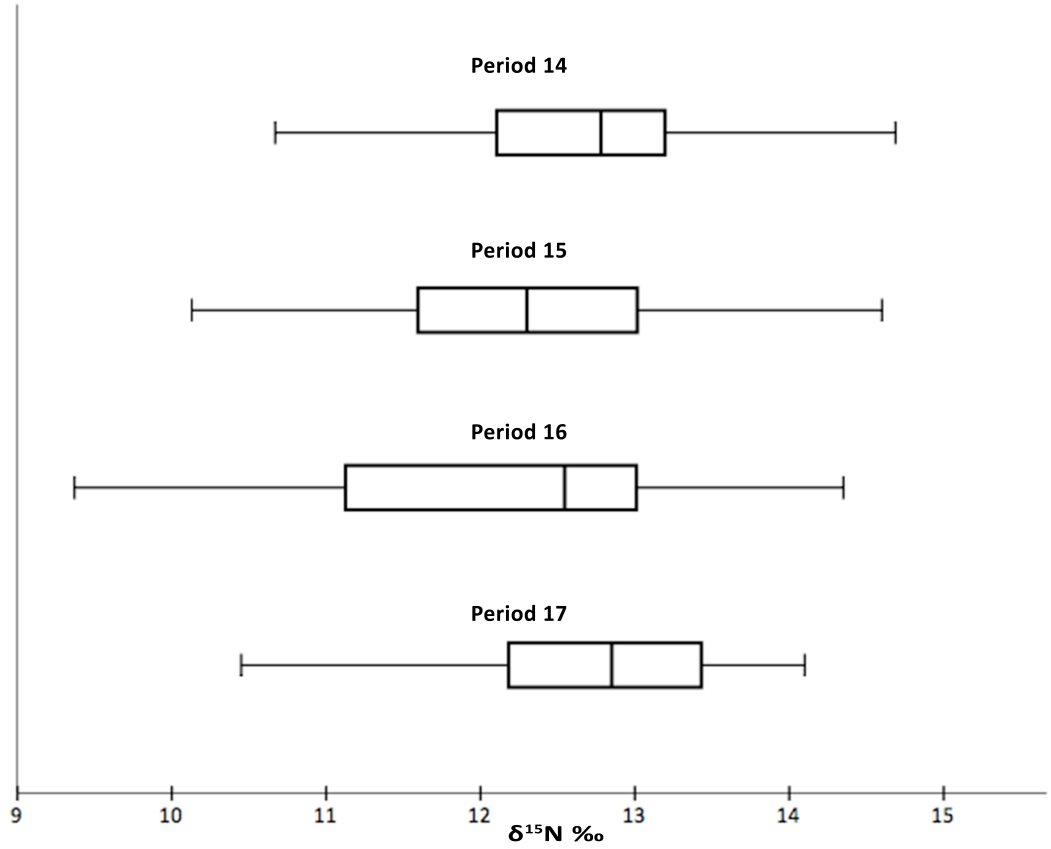


Figure 4.3: Boxplot of $\delta^{15}N$ by temporal period for all individuals

Table 4.3: Results of Kruskal-Wallis comparisons of $\delta^{15}N$ and $\delta^{13}C$ for all individuals and adults by temporal period

	All			
	$\delta^{15}N$ Median	$\delta^{15}N$ *P*	$\delta^{13}C$ Median	$\delta^{13}C$ *P*
Period 14 (1120-1200 CE)	12.78	0.07	-19.09	0.45
Period 15 (1200-1250 CE)	12.30		-19.04	
Period 16 (1250-1400 CE)	12.55		-19.12	
Period 17 (1400-1539 CE)	12.85		-19.33	
	Adults			
Period 14 (1120-1200 CE)	12.78	0.11	-19.13	0.94
Period 15 (1200-1250 CE)	12.47		-19.08	
Period 16 (1250-1400 CE)	12.56		-19.11	
Period 17 (1400-1539 CE)	12.85		-19.24	

Table 4.4: Results of post-hoc Mann-Whitney U tests comparing $\delta^{15}N$ for temporal periods

Compared periods	$\delta^{15}N$ *P*
14 (1120-1200 CE) & 15 (1200-1250 CE)	0.06
15 (1200-1250 CE) & 17 (1400-1539 CE)	0.03

Table 4.5: Results of Pearson correlation of $\delta^{13}C$ and $\delta^{15}N$ values for all periods and for each period

Period	r^2	P
All (1120-1539 CE)	0.49	< 0.00
14 (1120-1200 CE)	0.44	< 0.00
15 (1200-1250 CE)	0.43	< 0.00
16 (1250-1400 CE)	0.68	< 0.00
17 (1400-1539 CE)	0.57	< 0.00

4.3.2 Age cohort

Results evaluating differences in isotope values by age cohort for all temporal periods using a Kruskal-Wallis test are presented in Table 4.6, and results of post-hoc Mann-Whitney U tests for ago cohorts with statistically significant pairwise comparisons are shown in Table 4.7. The results indicate that there is a significant difference between age cohorts for $\delta^{15}N$ ($P = 0.03$), but not for $\delta^{13}C$ ($P = 0.26$). Post-hoc pairwise comparisons for periods with statistically significant differences indicate that adults exhibit higher $\delta^{15}N$ values compared to younger age groups (subadults and young adults). Plotted isotope values for subadult, young adult, and adult age cohorts are provided in Figure 4.4.

4.3.3 Sex differentials

Results of analyses between the sexes of pooled temporal periods using the Mann-Whitney U test are provided in Table 4.8, and boxplots of $\delta^{15}N$ values by sex are provided in Figure 4.5. Results comparing isotope values between the sexes reveal that there is no significant difference ($\delta^{15}N$: $P = 0.24$, $\delta^{13}C$: $P = 0.86$) in stable isotope values between males and females. Results for sex-specific analysis of isotope values between time periods using the Kruskal-Wallis test are provided in Table 4.9. These results reveal that there is no difference in isotope values through time for females or for males.

Table 4.6: Results for Kruskal-Wallis comparisons for bone collagen $\delta^{15}N$ and $\delta^{13}C$ values by age cohort

	$\delta^{15}N$ Median	$\delta^{15}N$ *P*	$\delta^{13}C$ Median	$\delta^{13}C$ *P*
Subadult	12.23		-19.07	
Young adult	12.30		-19.24	
Adult	12.86	0.03	-18.94	0.16
Old adult	12.63		-19.12	
Mature adult	12.67		-18.84	

Table 4.7: Results of post-hoc Mann-Whitney U tests comparing $\delta^{15}N$ for age cohorts

Compared age cohorts	$\delta^{15}N$ *P*
Subadults & Adults	< 0.00
Young adults & Adults	0.01

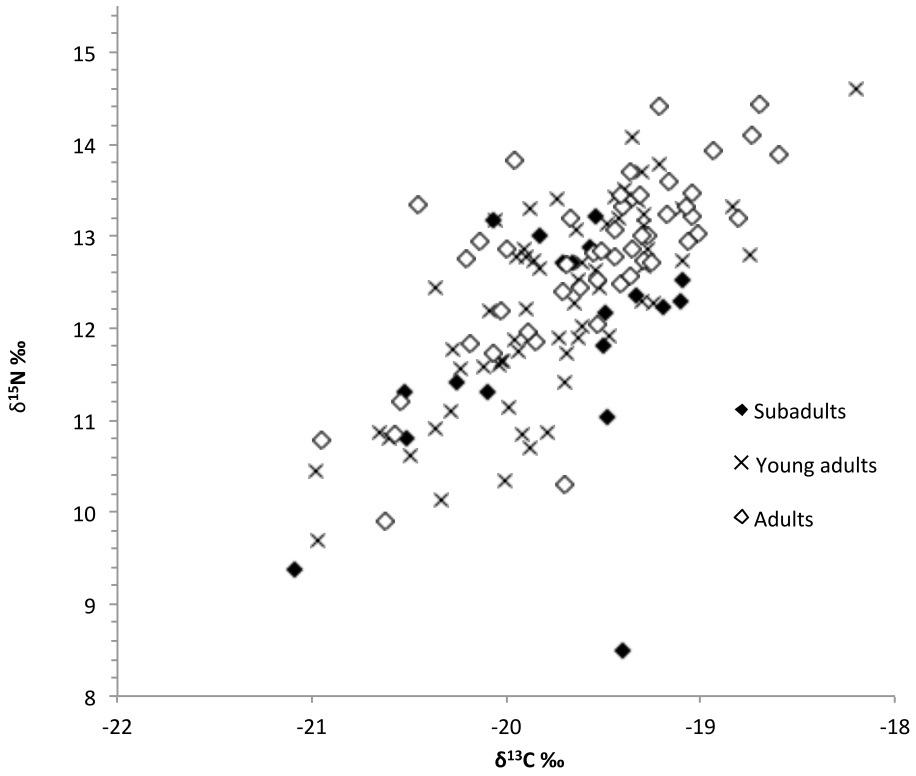


Figure 4.4: Plotted isotope values of subadults, young adults, and adults

Table 4.8: Results of Mann-Whitney U tests comparing isotope values between the sexes

	$\delta^{15}N$ Median	$\delta^{15}N$ *P*	$\delta^{13}C$ Median	$\delta^{13}C$ *P*
Females	12.67	0.14	-19.12	0.86
Males	12.89		-19.10	

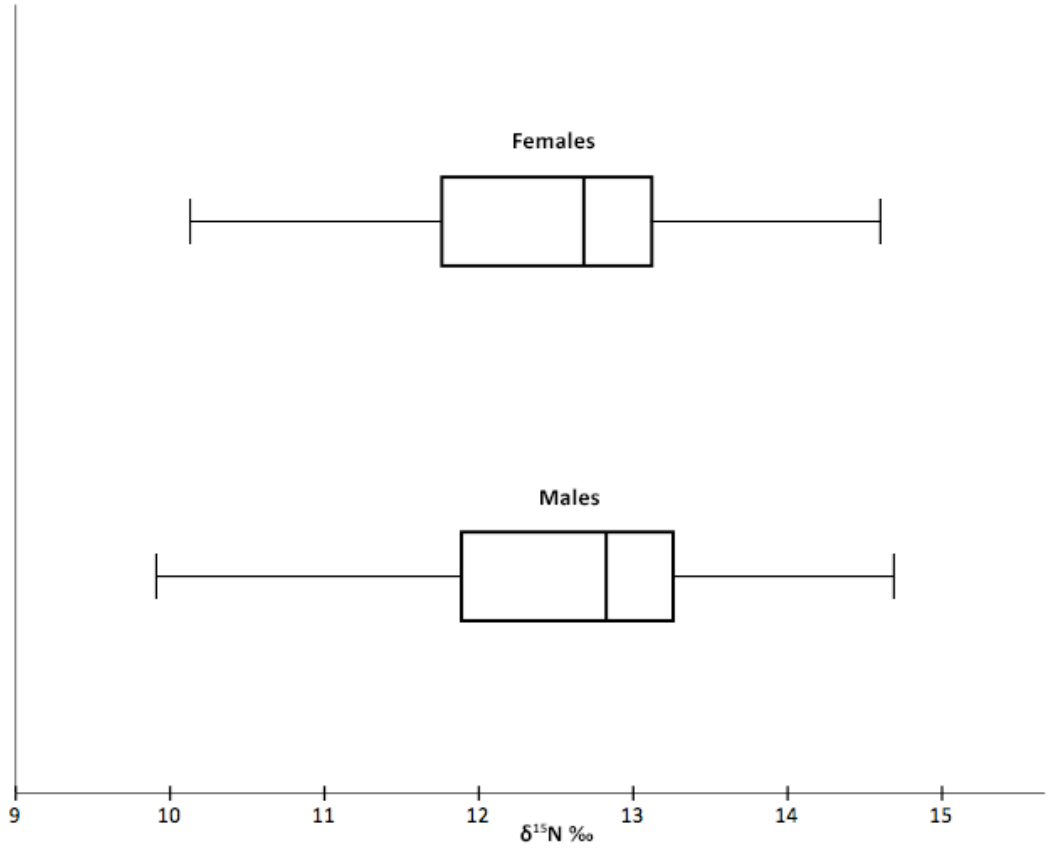


Figure 4.5: Boxplots of $\delta^{15}N$ stable isotope data for all periods by sex

Table 4.9: Results of sex-specific comparisons of $\delta^{13}C$ and $\delta^{15}N$ values between temporal periods using the Kruskal-Wallis test

	Females			
	$\delta^{15}N$ Median	$\delta^{15}N$ *P*	$\delta^{13}C$ Median	$\delta^{13}C$ *P*
Period 14 (1120-1200 CE)	12.66	0.65	-19.11	0.53
Period 15 (1200-1250 CE)	12.20		-19.20	
Period 16 (1250-1400 CE)	12.67		-18.86	
Period 17 (1400-1539 CE)	12.80		-19.13	
	Males			
Period 14 (1120-1200 CE)	12.96	0.64	-19.14	0.57
Period 15 (1200-1250 CE)	12.63		-19.02	
Period 16 (1250-1400 CE)	12.43		-19.30	
Period 17 (1400-1539 CE)	13.12		-19.33	

4.4 Discussion

4.4.1 The London diet

The $\delta^{13}C$ and $\delta^{15}N$ isotope values in St Mary Spital, for all temporal periods, are similar to other Medieval English sites, with the exception of Wharram Percy (Figure 4.6), a poor rural community in Yorkshire that is different from most Medieval English sites because of fewer marine foods in the diet (Fuller et al., 2003), which may be a result of the villages location away from the coast and a lack of trade. The diet in SRP98 is characteristic of a medieval English diet, with differing proportions of terrestrial protein and marine protein C_3 protein and marine protein (Müldner and Richards, 2005; Müldner and Richards, 2007a; Müldner and Richards, 2007b).

High $\delta^{15}N$ values, such as those exhibited in SRP98, are attributed to the consumption of fish and omnivore protein, which are both high in $\delta^{15}N$ (Müldner and Richards, 2007b). Moreover, the isotopic signature of SRP98 is similar to the medieval town of York (Fishergate) (Müldner and Richards, 2007b). When all temporal periods are considered, however, SRP98 displays more variable $\delta^{15}N$ values and a correlation of $\delta^{13}C$ and $\delta^{15}N$ that is notably closer to 0, compared to Fishergate ($r^2 = 0.81$), suggesting that Londoners consumed different species of terrestrial animals and fish compared to those in York. Results from this study are similar to those from a previous isotopic analysis of Periods 15 (c. 1200-1250 CE) and 16 (c. 1250-1400 CE) in SRP98 (Lakin, 2010), but with slightly more negative $\delta^{13}C$ values (-0.60‰). Another London cemetery, St Nicholas Shambles, also exhibits more negative $\delta^{13}C$ values in addition to $\delta^{15}N$ values compared

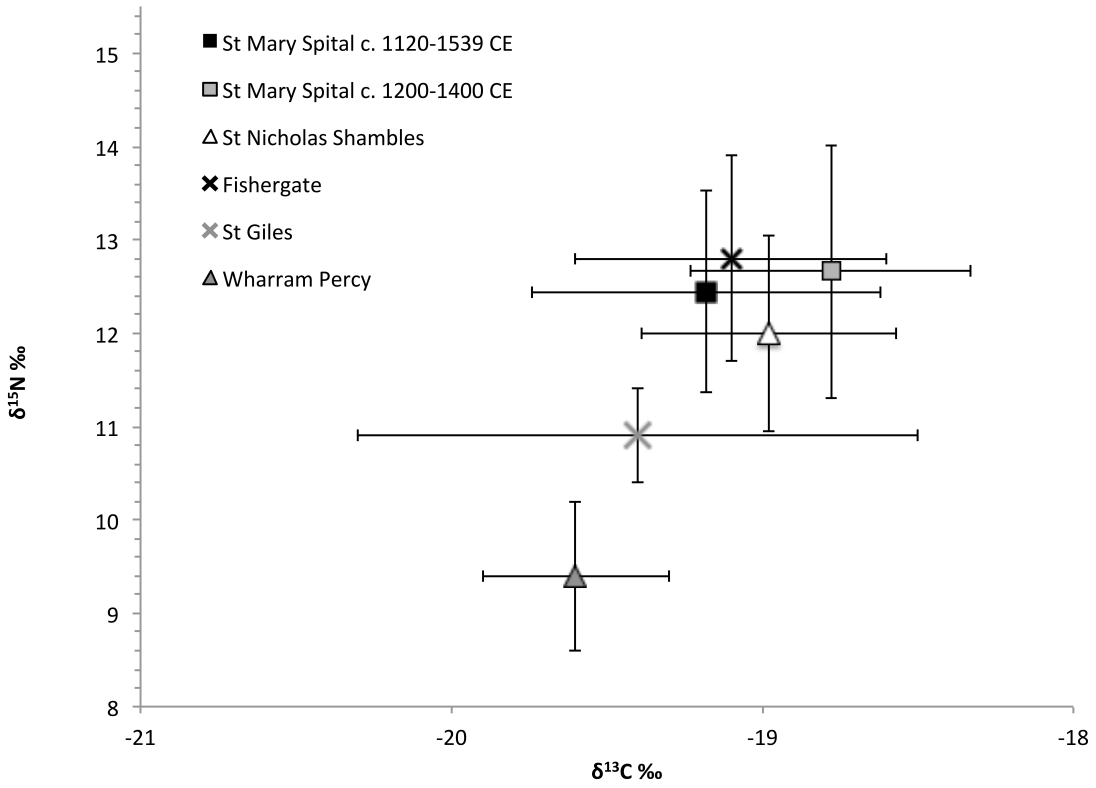


Figure 4.6: Plotted $\delta^{13}C$ and $\delta^{15}N$ values (1 σ bars) in SRP98 and contemporaneous Medieval English cemeteries (Data from St Mary Spital c. 1200-1400 CE and St Nicholas Shambles from Lakin (2010); Fishergate from Müldner and Richards (2007a); St Giles from Müldner and Richards (2005); Wharram Percy from Fuller et al. (2003))

to SRP98, but this may be a result of St Nicholas Shambles consisting of mostly local traders (Schofield, 1997) or because it contained individuals interred before marine consumption was more widespread in England (Barrett et al., 2004b).

The elevation in $\delta^{13}C$ and $\delta^{15}N$ values seen in English cemeteries after approximately 1000 CE is generally agreed to be a reflection of increased marine protein in the diet, referred to as the fish event horizon (Barrett et al., 2004a). Around this time there was a drastic increase in marine fishing and trade, which occurred at the same time that fasting was promoted by the Church (Woolgar 2000). In addition to isotopic evidence from English skeletal assemblages, the fish event horizon is supported by historical documents, and the increase of marine skeletal remains at inland and coastal sites in England dated after c. 1000 CE. This pattern is also present in SRP98, with most individuals exhibiting a diet with a marine component (Figure 4.7). Zooarchaeological evidence of herring and cod in the 11th century is evident in urban English sites, including London (Serjeantson and Waldron, 1989).

During the Late Medieval Period, fish were a relatively cheap source of protein, particularly cheaper preserved fish (Barrett et al., 2004a), but were still more costly than protein sources like beef and pork. At the markets in London, fish were available from a wide variety of sources and could be purchased fresh or preserved (Woolgar, 2000). Fish could also be purchased prepared as part of a meal from vendors at food stalls in the market (Carlin, 1998). Moreover, the role of fish during religious fasting also contributed to the increase in the fish market (Simoons, 1994). Fasting for religious purposes (i.e. abstention from foods, usually meat, as a symbol of faith) became a regular practice

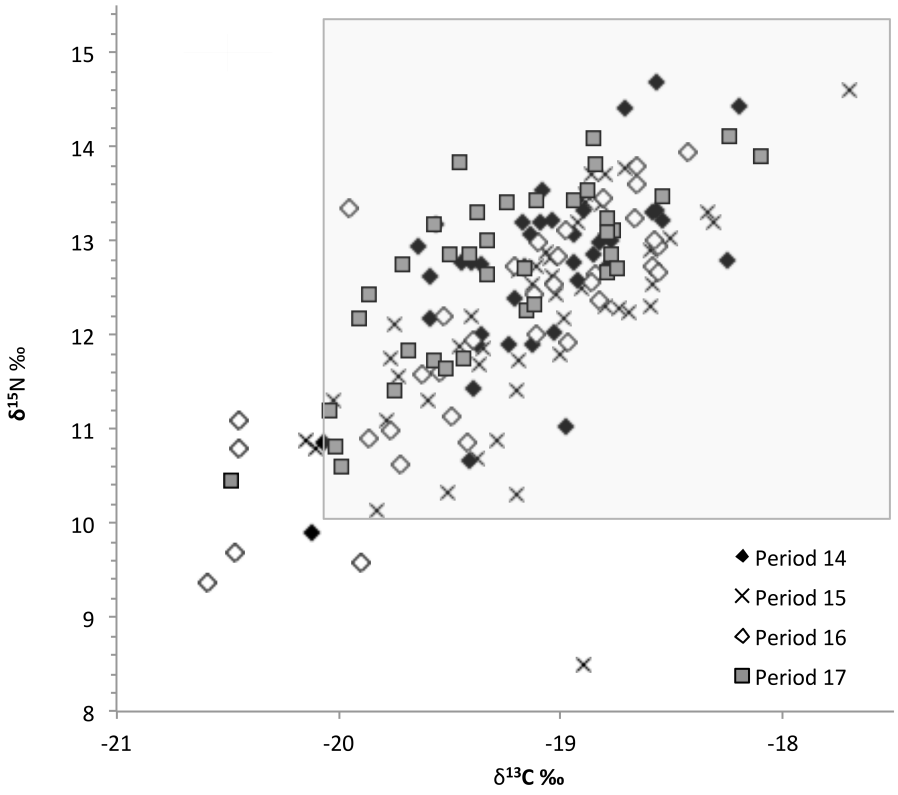


Figure 4.7: Plotted $\delta^{13}C$ and $\delta^{15}N$ values by temporal period with square overlaying values from Richards and Hedges (1999) indicative of marine protein component included in the diet

around the beginning of the 14th century, with some religious leaders calling for abstinence as many as 3 times a week (Woolgar, 2000). Paired with saints' days, fasting days could have amounted to almost half the year for the truly ascetic (Tames, 2003). On fasting days, animal meat was forbidden, although eggs were eventually permitted, and were often substituted with fish as a source of protein (Tames, 2003). With such a high prevalence of fasting, there was a great demand for fish (Simoons, 1994). Though the poor, who constituted the great majority of the population, did not strictly adhere to fasting regulations, they still practiced abstention from meat consumption when possible and ate cheaper permitted protein like salted fish, eels, and oysters (Tames, 2003).

Based on isotope analysis of faunal remains at Spital Square, an adjacent medieval site near St Mary Spital, Lakin (2010) determined that the δ^{15}N-rich protein characteristic of SRP98, mostly came from omnivore protein (e.g. pigs and domestic fowl), and some herbivore protein and fish. Omnivore protein and fish have mostly C_3 terrestrial δ^{13}C values and high δ^{15}N values that would be exhibited as such in humans. Richards and Müldner (2007a) propose that the diet at Fishergate in York was based on terrestrial plant and herbivore protein, with differing quantities of marine and omnivore protein, which is consistent with the results from SPR98. Omnivore protein consumed in medieval London was most likely comprised of pork and fowl. Similar to fish, these sources of protein, along with beef and mutton, were also available fresh or cooked at market stalls (Carlin, 1998). Dyer (1989) notes that pork was often cited in employment agreements to laborers, and was also considered to be a source of protein for low-

status individuals, particularly preserved as bacon. The ease in preserving pork as bacon or cured ham, along with the high fat content of the meat, made pork a common option for the lower classes (Jørgensen, 2013). Pigs were also plentiful in urban areas because they were used as a means of keeping areas clean by allowing them to scavenge through the streets (Jørgensen, 2013; Thrupp, 1989).

In addition to fish and pork as sources of protein, beef and mutton were also components of the Londoner's diet. In general, zooarchaeological evidence of cattle in England is more prevalent in urban areas than rural areas (Thomas, 2002). Cattle were often raised in rural areas, and then sent to towns to be butchered (Tames, 2003). Given of the abundance of meat yielded by such a large animal, rural villages did not have enough people or preservation resources regularly to adequately use the animal (Albarella, 2005). For this reason, butchers were more prevalent in towns where the butchering and selling took place (Woolgar, 2006). Mutton was consumed just as much as beef, but these protein sources were not as available as pork (Sykes, 2006). Though there is an increase in sheep remains in urban areas after the 13th century, this is attributed to the growing wool industry during this time, rather than increased consumption by the population (O'Connor, 1989).

Whether fish, pork, fowl, beef, or mutton, all varieties of protein prepared in different ways were available at food stalls, also referred to as cook shops, in the market. From the 12th century on, the number of food stalls increase throughout London (Carlin, 1998), further increasing the range and access of different protein sources available to the population. Poor urban workers were the principle clientele of

cookshops because they were easily accessible and did not require fuel or supplies necessary for food preparation (Carlin, 2008). Because poor urban workers comprised the largest proportion of the London population, the consumption of different animal meat from these food stalls most likely contributed to the varied protein sources seen in SRP98. Pottage, a thick soup, was also a major source of protein for the poor, serving as the "major vehicle for the consumption of meat" (Tames, 2003:16). Low-status pottage was made from animal by-products, such as lungs, blood, lard, and marrow, from different animals (Tames, 2003). In addition to protein from meat, dairy products would also have contributed to the isotopic signature at SRP98. Woolgar (2006) argues that dairy was actually one of the most common foodstuffs in Late Medieval England. Unfortunately, it is not possible to differentiate quality of protein consumed (e.g. pottage *vs.* prime rib), the portion of the animal consumed (e.g. cheese *vs.* meat), or how the meat was prepared (e.g. preserved or roasted) using stable isotope analysis. Thus, it is necessary that any interpretation of the consumption of animal protein must be conducted using historical and archaeological evidence.

As previously mentioned, SRP98 exhibits a wide range of $\delta^{13}C$ and $\delta^{15}N$ values compared to other medieval English sites. This variation in isotope values paired with the low correlation between $\delta^{13}C$ and $\delta^{15}N$ values indicates substantial dietary variation of fish and animal meat sources in London. Also, though the typical London diet was comprised of foods found in villages and towns, Londoners had more variety. London relied on its hinterland for its food sources (Galloway and Murphy, 1991; Tames, 2003), and resulting foodstuffs came from a variety of places outside of London, which would

exhibit different isotopic signatures. This would have contributed to the varied diet seen in SRP98. As discussed, fish comprised a portion of the protein in the typical London diet. Both marine and freshwater fish, which exhibit different isotopic signatures (marine fish with less negative $\delta^{13}C$ values compared to freshwater fish), were consumed. Marine fish would have been more common in the diet, however, because it was cheaper (Dyer, 1988). Several species of marine fish could be found in London, although the most common were herring and cod. In the thirteenth and fourteenth centuries there was an increase in cod-related species from more distant sources such as Norway (Barrett et al., 2004b), which would have contributed to the already diverse isotopic signature of Londoners. Marine fish that came in from farther distances were usually salted or cured for preservation. Preservation was also useful to sustain the market and keep prices from increasing when fish stock was low (Woolgar, 2000). Cheaper sources of marine protein such as oysters and eel, which were usually farmed locally, were also consumed (Tames, 2003). Moreover, London's function as the largest port in England would also have introduced exotic foods and consumption practices to the city, though these foods would generally not have been accessible to the majority population, the poor.

Migrants would have also contributed to the variation seen in the isotope values. As discussed in previous chapters, migrants made up a large proportion of the London population (Wrigley, 1969). These individuals would exhibit isotope values characteristic of their place of origin, which would be different from isotopes values of a typical London resident. Because bone tissue from ribs takes four to ten years to fully remodel

for adults (Valentin, 2002), these nonlocal isotopic signatures would potentially be averaged in the diet of the individual consumed prior to age-at-death (i.e. the London diet). Given that a large proportion of the population were migrants and that nonlocal isotopic signatures would be evident in these individuals, variation in isotopes for migrants seen in bone collagen would have also added to the isotopic variation evident in SRP98.

In addition to London in general, St Mary Spital itself served a diverse range of individuals drawn from the London region, which would subsequently result in an assortment of consumption practices, and thus, isotopic values. Inmates from the infirmary would have been interred in the cemetery (Connell et al., 2012). Members of the religious community (i.e. monks and lay sisters) who served as staff for the hospital were also buried in the cemetery, along with wealthy benefactors (Thomas et al., 1997). Unlike most medieval hospitals, St Mary Spital took in poor pregnant females and cared for children until the age of 7 (Thomas et al., 1997). Moreover, St Mary Spital's location at the edge of the city would have resulted in travelers and pilgrims interred in the cemetery who may have died during their travels.

Moreover, the inclusion of individuals from all burial contexts within the cemetery may introduce variation into the results. As mentioned, SRP98 is comprised of different burial types, including attritional burials and mass burials associated with famine and Black Death mortality. Most individuals interred in mass graves in SRP98 likely died as a result of famine-related causes (Connell et al., 2012), and thus may have different diets, compared to individuals that did not experience famine prior to death.

Differences between burial types in the cemetery are evaluated in Chapter 5.

4.4.2 Temporal period

Results comparing isotope values indicate that individuals from Period 15 (1200-1250 CE) exhibit significantly higher $\delta^{15}N$ values compared to Periods 14 (1120-1200 CE) and 17 (1400-1539 CE), and Period 16 (1250-1400 CE) exhibits no significantly different isotope values compared to the other temporal periods. When individuals below the age of 15 are excluded from the analysis, the differences in $\delta^{13}C$ and $\delta^{15}N$ between periods for adults are not significant, suggesting that $\delta^{15}N$ values for younger individuals contribute to the difference seen between periods when all individuals are included. Isotopic research has indicated that young, growing animals generally exhibit lower nitrogen values compared to their older counterparts (Sponheimer et al., 2003b). The difference in nitrogen balance that could occur during growth, however, may not be detectable because of the slow turnover rates in bone tissue (Waters-Rist, 2010), which suggests that any isotopic differences seen between young and old age groups are actually a reflection of dietary differences. Isotopic differences between age cohorts, including young and older groups, are further discussed below.

The plotted isotope values illustrate that the differences between temporal periods for $\delta^{13}C$ and $\delta^{15}N$ are not substantial, as the median's plot closely together and the ranges overlap considerably. Though $\delta^{15}N$ values for Period 15 (1200-1250 CE) are significantly lower than Periods 14 (1120-1200 CE) and 17 (1400-1539 CE), the difference is minimal (by -0.48‰ compared to Period 14, and by -0.55‰ compared to

Period 17) and is not substantive enough to suggest any major dietary shifts. However, the significantly lower $\delta^{15}N$ values in Period 15 compared to Period 14 and Period 17 are interesting because Period 15 has the shortest timespan of approximately 50 years (1200-1250 CE). Moreover, the correlation for $\delta^{13}C$ and $\delta^{15}N$ is closer to zero than the other periods, indicating that there was more variety in the species consumed during this period compared to other periods. Given the short time period, one would expect the r^2 to be closer to 1, allowing less time for new species to enter and exit the food circuit. Sources of produce are less likely to change during a 50-year period compared to a 100 or 150-year period (i.e. the length of the other periods in SRP98). The low correlation of $\delta^{13}C$ and $\delta^{15}N$ may be influenced by an individual in Period 15 with a unique isotopic signature, SPR98-31022 (an individual 10 years of age at death in an attritional burial), with a moderate $\delta^{13}C$ value (-18.90‰) but low $\delta^{15}N$ value (8.50‰). Post-hoc regression without this individual reveals a significant ($P < 0.00$) and increased r^2 of 0.60. The exclusion of this outlier reveals an r^2 that is more consistent with the other periods and the short time span of Period 15 compared to other periods (e.g. closer to zero).

With the outlier excluded, Pearson correlations for the $\delta^{13}C$ and $\delta^{15}N$ values of the different temporal periods reveal a trend of increasing correlation between $\delta^{13}C$ and $\delta^{15}N$ values through time for the first three periods in SPR98 (Periods 14 to 16). This could be reflective of a decrease in the variation of food sources prior to the Black Death as urbanization increased in London, and may be reflecting changes in trade strategies for food supplies in London. During the expansion of trade in London, produce may have

become more regulated, restricting outside foodstuffs from certain locations (Galloway and Murphy, 1991). This trend is consistent with London's shift to a specialist-produce trade network that expanded during the fourteenth century (Galloway and Murphy, 1991). The slight reduction for r^2 seen in Period 17, the period after the Black Death, may be reflective of changes in the food trade reverting back to more varied food sources exhibited in earlier periods, possibly as a result of an unstable market. Moreover, after the Black Death, there was an increase in migrants from rural areas taking advantage of the abundance of labor opportunities left by the dead in London (Barron, 2000). These individuals would exhibit nonlocal isotope values that might contribute to reduced correlation between $\delta^{13}C$ and $\delta^{15}N$ seen in this period.

Given that Period 16 experienced the most severe and numerous famines during the Late Medieval Period, which included the Great Famine of 1315-1317 CE and the Bovine Pestilence of 1319-1350 CE, it is expected that this period would exhibit different isotopic patterns compared to the other periods. Individuals would have needed to adapt to the limited access to food supplies during times of famine, which presumably would have left isotopically varying diets throughout the period, as access to food supplies fluctuated and the population adapted to the uncertainty of food accessibility. Though exhibiting low $\delta^{15}N$ values, Period 16 does not exhibit a significant difference when compared to any of the other periods in SRP98. This period, however, has the highest standard deviation compared to the other periods, suggesting that there were more varied diets during this period, which may be a consequence of famine. Moreover, Period 16 displays the highest r^2 value among the periods, suggesting that protein was

consumed from less varied sources. This may be a reflection of the limited supplies available, leaving fewer sources of certain proteins to choose from. The Black Death also occurred in this period, and the low correlation between $\delta^{15}N$ and $\delta^{13}C$ values could be a reflection of decreased trade during the epidemic.

4.4.3 Age cohorts

Results evaluating differences in isotope values between age cohorts for pooled temporal periods indicate that there is a significant difference between age cohorts for $\delta^{15}N$, but not for $\delta^{13}C$. Post-hoc pairwise comparisons for age cohorts with statistically significant differences indicate that the adult cohort exhibits elevated $\delta^{15}N$ values compared to the younger age cohorts (subadults and young adults). These results may be reflective of physiological changes affecting isotope values of growing individuals, migration, and differential access to meat protein.

The difference in subadult and adult isotope patterns may exist because subadults generally exhibit lower isotope values compared to adults, as discussed previously. Though not significant, subadult and young adult cohorts display lower $\delta^{15}N$ values compared to the other adult age cohorts (old adults and the mature adults), indicating that the general difference between subadult and young adult cohorts *vs.* adult cohorts is not unique to younger ages and the adult age cohort. Moreover, because the two older adult age cohorts have small sample sizes, the samples may not be capturing the actual difference between these cohorts and subadults. Low nitrogen values for younger individuals, have also been identified in English skeletal assemblages

(e.g. Beaumont, 2012; Henderson et al., 2014), and other skeletal assemblages (e.g. Eerkens et al., 2011; Richards et al., 2002; White and Schwarcz, 1994), suggesting that this pattern is not unique to St Mary Spital or London.

The low $\delta^{15}N$ values seen in younger compared to older ages-at-death are discussed above concerning isotopic differences between temporal periods. When an individual is experiencing growth he or she is usually in a state of nitrogen imbalance, meaning that there is an imbalance in total nitrogen intake and excretion in the body (Schurr, 1988). Specifically, during growth, the body is in a state of positive nitrogen imbalance, with more nitrogen being consumed by the body for tissue growth than is being excreted, resulting in low nitrogen values (Schurr, 1988; Trueman et al., 2005). Alternatively, Ponsard and Averbuch (1999) argue that there should not be depletion in nitrogen during growth because younger individuals generally consume more than adults, relative to their body weight, which would compensate for the nitrogen needed for tissue growth. Whether the low nitrogen values of younger individuals occur during nitrogen balance or imbalance, it is important to note that the individuals here died at young ages and are thus likely not to have had sufficient nutrition or may have experienced some kind of physiological stress that caused these individuals to die young.

Rather than being a reflection of physiological changes or stress, the difference in $\delta^{15}N$ values between adults and subadults may also be a reflection of differences in diet. Subadults may have consumed less animal protein than their older counterparts. The lower $\delta^{15}N$ values could also be reflective of undernutrition, namely lack of protein that could have contributed to their early death. Reitsema et al., (2016) found, through

comparison between isotope values in subadults and adults in medieval Italy, that incorporating animal protein in the diet during growth (i.e. during childhood) enhanced the ability of subadults to survive to adulthood. It is also possible that weaning could have introduced effected the isotope values for the younger adults. There are only two individuals, however, sampled for bone collagen with an age-at -death below the age of 8, an age at which high $\delta^{15}N$ values as a result of breastfeeding would likely not be reflected in bone collagen samples (Haydock et al., 2013), as bone remodels rapidly during early childhood (Valentin, 2002).

In addition to subadults and adult cohorts, the young adult cohort also exhibits significantly lower $\delta^{15}N$ values compared to the adult cohort, and this may also be influenced by nitrogen imbalance during growth. Unlike the subadult cohort, however, the significantly lower $\delta^{15}N$ values in young adults should not be totally attributed to growth status because most of the individuals in this cohort are not growing or the growth rate of body tissues has decreased substantially (Scheuer et al., 2000). It is possible that because of slower tissue turnover rate in bone tissue compared to the subadults, the low $\delta^{15}N$ values produced during childhood may have been averaged in with the high $\delta^{15}N$ values produced prior to death as a young adult. This could have resulted in an underestimation of the actual $\delta^{15}N$ values prior to death.

It is also plausible that migration could have contributed to the differences seen between age cohorts. Migrants who were coming into London for labor opportunities were mostly adolescents and young adults. Thus, the isotope values exhibited by the young adult cohort could be a reflection of nonlocal diets. Given that the London

isotope signature has characteristically high nitrogen values and that most migrants coming into London were young adults, this suggests that the elevated values seen in adults may be a reflection of a high proportion of young adults migrating into London. Bone tissues from individuals who died within a few years of coming to London, would exhibit an isotopic signature that included both the nonlocal diet and the London diet consumed prior to death because of slower bone turnover rate. The higher nitrogen values of the adult cohort could be a reflection of a longer period of time spent in London since migrating in as an adolescent or young adult, as these individuals would have had a longer period of time to incorporate the London diet into their tissues before death. Previous isotopic analysis of SRP98 reveal that young adults, specifically females, exhibit lower $\delta^{15}N$ values compared to older individuals in the sample, and subsequent oxygen and strontium analysis suggests that some of the females with lower values were most likely migrants (Lakin, 2010).

The higher adult cohort isotope values seen for adults could also be a reflection of food accessibility. Adults may have had better access to animal protein and fish compared to other age groups. Though not significant, adults exhibit higher $\delta^{15}N$ values and more negative $\delta^{13}C$ values than the other age cohorts, suggesting a higher proportion of animal meat in the diet. This is consistent with historical documents from urban areas (e.g. wage accounts and maintenance agreements) documenting foods such as bacon, dairy products, and eggs as a component of compensation for labor (Bennett, 1937; Dyer, 1989). Urban workers were also known to patronize the food market stalls, which provided cooked meals containing different meats (Carlin, 2008). It is possible

that a higher of the proportion of the adult cohort were urban workers with better access to animal protein in their diet, which, in turn, would result in elevated $\delta^{15}N$ values and reduced $\delta^{13}C$ values for the cohort.

4.4.4 Sex differentials

Analyses of differences between the sexes within pooled temporal periods reveal that there is no significant difference in stable isotope values between males and females. Further, results for sex-specific analysis of isotope values for time periods indicate that there is no significant difference in isotope values through time for females or for males. These results suggest that the sexes were eating a similar diet consistently through the Late Medieval Period.

Previous analysis of SRP98 including only Periods 15 and 16 (c. 1200-1400) find a significant difference in the $\delta^{15}N$ values of bone collagen between the sexes, with females exhibiting lower $\delta^{15}N$ values compared to males (Lakin, 2010). The difference between the means is small (0.30‰), however, and ranges overlap substantially. The analysis for this study, which includes all periods in SRP98, also reveals lower $\delta^{15}N$ values for females, though the difference is not significant. The inclusion of data from the other periods of SRP98 suggests that the sexes may have had slightly different diets during Period 15 and 16 compared to Periods 14 and 17. Differences may be so minimal in these periods, however, that they cannot be identified isotopically because of slow bone turnover rate. For example, in an early Anglo-Saxon cemetery in Oxfordshire, Privat et al. (2002) find no significant differences between $\delta^{13}C$ and $\delta^{15}N$ for bone

collagen between the sexes even though there is textual and archaeological evidence that depicts rigidly distinct diets. This suggests that the evidence indicating differences in eating practices may not be accurate, or, more plausibly, that major differences in diet must occur to actually capture these differences biochemically. Privat et al. (2002) attribute the lack of isotopic differences between the sexes to the inability to differentiate the parts of the animal eaten (e.g. females ate flesh, while males ate dairy) or the quality of the food being consumed (e.g. females ate preserved meat, while males ate fresh meat).

Lakin (2010) also compared isotope values between the sexes for St Nicholas Shambles, a medieval London cemetery dated from the 11th to 16th century, and finds no significant difference in isotope values between the sexes. Given that there are no significant differences for $\delta^{13}C$ and $\delta^{15}N$ values between the sexes in SRP98 (when periods are pooled) and for St Nicholas Shambles, this suggests that the lack of sex differences was sustained throughout the Late Medieval Period in London. However, in an analysis of dentine collagen from a mixed post-medieval industrial era cemetery (c. 18th-19th centuries) in Southwark, England, Henderson et al. (2014) found a difference in $\delta^{13}C$ and $\delta^{15}N$ values for the sexes during childhood, with females exhibiting slightly elevated $\delta^{15}N$ values, which is the opposite trend seen in this study. Results of Henderson's (2014) study and this study, suggest that there may have been a change in dietary practices between males and females after the Medieval Period. Analysis of bone collagen in older individuals representing diets after childhood from the Post-Medieval cemetery is needed to assess whether this trend is actually a reflection of

isotope changes through life.

Unlike SRP98, comparison of isotope values between the sexes at Fishergate, a Late Medieval English urban cemetery in York, shows a significant difference between the sexes, with females exhibiting lower $\delta^{15}N$ values (Müldner and Richards, 2007b). These results are similar to the lower $\delta^{15}N$ values for females seen at SRP98. Specifically, Müldner and Richards (2007) found that females consumed less marine foods than males and attributed this dissimilarity to eating practices that were governed by medieval dietary theory concerning differences in the physiological make up of males and females (i.e. the classical writings of Hippocrates and Galen regarding the four humors). Comparison of the isotope results from Fishergate suggests a stronger difference in consumption practices related to sex in English towns compared to London. Females may have played a more equal role in large cities like London than in towns, reflected in the lack of isotopic differences seen in SRP98. Unfortunately, any historical or archaeological evidence of sex-related differences for diet in England is sparse, as these sources generally regard the consumption patterns of the population as a whole and not by sex. It is important to note, however, that it is not possible to differentiate food quality, which may have been differed between the sexes. This is further discussed below.

A pattern of lower $\delta^{15}N$ values in females compare to males is not specific to English skeletal assemblages. Isotopic research on skeletal assemblages from urbanizing environments at other geographic locations and temporal periods also find this pattern. For example, Tsutaya et al.'s (2013) isotopic analysis of urbanizing Japan (c. 1657-1683

CE) reveals that females exhibit lower $\delta^{15}N$ values compared to males. Additionally, analysis of an Imperial Roman cemetery in southern Italy (c. 100-200 CE) indicates significant, though minimal (<1‰), difference in isotope values between sexes (Craig et al., 2009).

There are no metabolic differences between the sexes that would cause variations in $\delta^{13}C$ and $\delta^{15}N$ when individuals are at a steady state (DeNiro and Schoeniger, 1983). Non-steady states, however, have been found to alter isotope values. Pregnancy has been shown to result in low $\delta^{15}N$ values (Fuller et al., 2004). Unlike other London hospitals, SRP98 took in pregnant females (Connell et al., 2012); however, to produce a significant difference in isotope values, a substantial portion of the sample would need to include pregnant females, which is unlikely. Moreover, lower $\delta^{15}N$ values exhibited during pregnancy would likely have occurred for too short a period of time to be captured in bone collagen values because of slow turnover rates.

4.5 Conclusion

This study provides a temporal analysis of diet in urbanizing Late Medieval London, including analyses of age cohorts and the sexes. Overall, the diet for St Mary Spital is consistent with other Medieval English sites but demonstrates more variability. Analyses comparing isotope values for sub-populations reveal that isotope values varied both through time and by age cohort, and underline the importance evaluating isotopic differences within sub-populations to achieve a more nuanced depiction of diet in the context of urbanization.

The diet in SRP98 is characteristic of a medieval diet, with terrestrial C_3 protein and differing amounts of marine protein. The nitrogen rich protein characteristic of SRP98 mostly comes from omnivore protein (e.g. pigs and domestic fowl), some herbivore protein (e.g. beef and mutton), and fish. The cemetery exhibits a wide range of $\delta^{13}C$ and $\delta^{15}N$ values compared to other medieval English sites. This variation in isotope values paired with the low correlation between $\delta^{13}C$ and $\delta^{15}N$ values indicate substantial dietary variation in London, which is reflective of the city's reliance on the hinterland for food sources, migration, and the mixed nature of the cemetery.

Comparison of isotope values between temporal periods indicates that individuals from Period 15 exhibit significantly higher $\delta^{15}N$ values compared to Periods 14 and 17, though the differences between these periods is minimal and not substantive enough to suggest any major dietary shifts. Post-hoc analysis for regression of $\delta^{13}C$ and $\delta^{15}N$ indicates that the different temporal periods reveal a steady increase in $\delta^{13}C$ and $\delta^{15}N$ values for the first three periods. This trend could be reflective of a decrease in the variation of food sources as a result of changing import strategies prior to the Black Death as urbanization increased in London.

Results evaluating differences in isotope values between age cohorts for pooled temporal periods indicate that there is a significant difference between age cohorts for $\delta^{15}N$, but not for $\delta^{13}C$. Post-hoc pairwise comparisons for periods with statistically significant differences indicate that the adult cohort exhibits elevated $\delta^{15}N$ values compared to the younger age cohorts (subadults and young adults). These results may

be reflective of physiological changes affecting isotope values of growing individuals, migration, and differential access to different types of protein sources.

Analyses between and within the sexes suggest that males and females were eating approximately the same diet consistently through the Late Medieval Period. This lack of difference between the sexes suggests a stronger difference in consumption practices related to sex in English towns compared to London. Comparison of isotope values from other English cemeteries indicates a stronger difference in consumption practices related to sex in English towns, suggesting that females may have played a more equal role in large cities like London than in towns.

Analysis of medieval faunal assemblages, particularly from London, could help clarify the variation in isotope values exhibited in the cemetery. Though previous isotopic analysis of faunal evidence from Spital Square yields an informative baseline to analyze skeleton remains from London (Lakin, 2010), the sample size is small. Additional contemporaneous faunal material should be analyzed to elucidate the variation in $\delta^{13}C$ and $\delta^{15}N$ values and help explain the different sources of protein consumed by Londoners. Additionally, analysis of enamel from faunal remains could be informative of where medieval Londoners were obtaining their protein sources.

Moreover, the identification of migrants in the cemetery through additional isotope analyses (e.g. strontium, oxygen, and lead) using teeth could clarify differences in dietary patterns exhibited by the different age cohorts in London (Montgomery et al., 2010). Though not entirely conclusive, stable isotope analysis using $\delta^{13}C$ and $\delta^{15}N$ helps to identify trends in an urbanizing population when paleodemographic information (e.g.

age and sex) is integrated. For example, in this chapter, $\delta^{13}C$ and $\delta^{15}N$ values reflecting different proportions of animal and marine protein in the diet may suggest better access to animal meat sources for adults.

Finally, analyses conducted in this chapter include individuals from all burial contexts, including mass graves as a result of famine-related or Black Death mortality, which may have obscured some of the comparisons. Differences in consumption practices during periods of famine or plague epidemic could have influenced results comparing age cohorts or the sexes. In Chapter 5, isotope values are compared between attritional and famine-related mass graves in an effort to clarify patterns related to consumption during periods of famine and non-famine.

Chapter 5 - Assessment of Metabolic Stress Between Attritional and Famine Burials using Stable Isotope Analysis[4]

[4] Walter BS, DeWitte SN, Beaumont J, and Dupras T. To be submitted to *American Journal of Physical Anthropology*.

5.1 Introduction

Late Medieval London (*c.* 12th to 16th centuries) experienced several instances of famine (Keys et al., 1950; Rawcliffe, 2013) most notably the Great Famine, which killed 10-15% of the population in England (Jordan, 1997). Londoners, unlike their rural counterparts, did not generally produce their own food, making them more dependent on the market (Galloway and Murphy, 1991). Back-to-back poor harvests (Farr, 1846; Scrimshaw, 1987) throughout the Late Medieval Period occurred across Europe as a result of excessive rains and flooding. High food prices, and declining wages for occupational specialists unable to sell their goods reduced the ability of urban residents to purchase food (Jordan, 1997; Wrigley, 1969). Moreover, mortality crises often result from outbreaks of infectious disease that follow a severe food shortage, suggesting that undernutrition would have caused greater susceptibility to infection (Duncan et al., 1993; Dyson, 1991; Galloway, 1985; Galloway, 1988; Mielke et al., 1984). Excess mortality that correlates with consecutive incidences of famine is evident in the archeological record through catastrophic mass burials in London (Connell et al., 2012).

Several bioarchaeological studies of cemetery collections in medieval England have investigated the detrimental health effects of potential undernutrition. Height is considered a useful index for the general state of nutrition, reflecting the failure of an individual to achieve its maximum stature potential (Bogin, 1999; Waldron, 1989). DeWitte and Yaussy (2015) found that individuals interred in famine burials in SRP98 exhibited shorter stature compared to those in attritional burials in a medieval London cemetery. Similarly, Roberts and Cox (2003) found that within medieval England, poorer

females (i.e. females who were more likely to experience nutritional stress) were on average more than 5 cm shorter than better-off females, and attributed this difference to chronic malnutrition throughout childhood. In addition to short stature as a potential indicator for undernutrition, metabolic diseases identifiable in skeletal remains may also reflect potential dietary stress. Rickets is a childhood disease that results from vitamin D deficiency and can be identified by noticeable epiphyseal enlargement, thinning of the cranial bones, and bowing of the long bones (Brickley and Ives, 2010; Mays et al., 2006). Additionally, lesions as a result of possible iron-deficiency can be seen on the orbital roof as cribra orbitalia or on the cranial vault as porotic hyperostosis (Ortner, 2003), but these lesions have also been linked to other etiologies associated with undernutrition (e.g. scurvy) (Walker et al., 2009). Roberts (2009) reported that frequencies of cribra orbitalia increase with time during the medieval period in England and concluded that this relationship may be the result of poor nutrition, among other factors. These types of lesions, however, have been associated with overall stress experienced during life and should not be considered as indicative specifically of undernutrition. Moreover, individuals that were more frail than their peers (i.e. individuals with a higher risk of death) may have died more quickly, not surviving long enough for boney markers of poor health to be evident on the skeleton (Wood et al., 1992); thus, frailer individuals who died from famine may not exhibit skeletal lesions characteristic of undernutrition macroscopically on their bones.

Like skeletal indicators that have been associated with undernutrition, stable isotope data from bone and tooth samples contribute to our understanding of

nutritional stress and health. Specifically, isotope analysis may be informative about how $\delta^{13}C$ and $\delta^{15}N$ values (carbon and nitrogen isotope ratios in ‰ comparative to international standards (Tykot, 2006)) are manifested among individuals that experienced nutritional stress and also how nutritional stress may be exhibited biochemically in human tissue. Further, integrating isotope data from teeth and bone can provide a more nuanced understanding of nutrition and health throughout the life course (Reitsema and Vercellotti, 2012).

Clinical (Fuller et al., 2004; Fuller et al., 2005; Mekota et al., 2009; Mekota et al., 2006), bioarchaeological, and forensic research (Beaumont et al., 2013a; Beaumont et al., 2013b; Beaumont and Montgomery, 2016; Holder et al., 2017; Neuberger et al., 2013; Olsen, 2014 Reitsema, 2013) have demonstrated that enriched $\delta^{15}N$ may be indicative of nutritional stress and starvation. Under metabolic stress, enriched $\delta^{15}N$ is exhibited when there is not enough nitrogen consumption to maintain tissues, causing the body to enter a catabolic state (Hobson et al., 1993). Prolonged protein depravation causes changes in the body's metabolic production and breakdown of protein in tissues, resulting in the enrichment of $\delta^{15}N$ in these tissues which serve as amino acid pools used in the maintenance of other bodily functions (Orten and Neuhaus, 1982). For example, a clinical study that tracked dietary changes in isotopic values found that in anorexia nervosa patients $\delta^{15}N$ was highest when BMI levels were low (Mekota et al., 2006). Thus, it is expected that individuals who died while undergoing nutritional stress would exhibit high nitrogen values in their bones and teeth.

To evaluate whether individuals experiencing nutritional stress exhibit enriched nitrogen compared to individual who did not, $\delta^{15}N$ values from bone collagen of individuals interred in famine-related mass burials and attritional burials from St Mary Spital cemetery in Late Medieval London were compared. Given that nutritional carbon can also be informative about changes in eating practices influenced by limited access to food (Beaumont and Montgomery, 2016), or possibly from the breakdown of body fat deposits in starving individuals (Neuberger et al., 2013), $\delta^{13}C$ values were also analyzed.

Previous research on skeletal assemblages has not yet identified a definitive association between elevated nitrogen values and famine using bone collagen because of slow bone turnover rates (Beaumont et al., 2013a; Beaumont and Montgomery, 2016). However, given that London experienced several instances of famine during the Late Medieval Period (Rawcliffe, 2013) rather than a single, catastrophic famine event, and that most of those buried in SRP98 were the working poor (i.e. individuals disproportionately affected by famine (Tames, 2003)), it may be possible to capture elevated $\delta^{15}N$ values as a result of undernutrition in this skeletal assemblage.

Additionally, dentine collagen samples from the different burial types, described below, were compared to bone collagen samples from the same individual to assess how diet, and potentially nutritional stress, changed throughout the life course. Specifically, using an innovative technique, incremental dentine analysis, patterns in isotope values for individuals from childhood (≈0.5-3.5 years) to adolescence (≈13.5-15.5 years) were evaluated, and dentine isotope values, reflecting diet early in life, were compared to bone collagen values, reflecting diet prior to death. Finally, pathological

information (age of enamel hypoplasia formation) of individuals was compared to patterns in incremental dentine collagen profiles to assess whether fluctuations in isotope values are the result of dietary change or of physiological stress.

5.2 Materials and methods

To evaluate potential biochemical markers of famine, bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values were compared between individuals within attritional interments and famine-related mass interments. To evaluate differences between the sexes, males and females were analyzed separately. The Mann-Whitney U test, a non-parametric test for pairwise comparison, was used rather than a Student's t-test because this test does not require an assumption of normality. Analyses were conducted separately for $\delta^{13}C$ and $\delta^{15}N$ values, and all analyses were conducted using SPSS version 21.

5.2.1 St Mary Spital cemetery

St Mary Spital cemetery is a large, well-dated skeletal assemblage curated by the Museum of London. The cemetery spans 1120-1539 CE and was in use during the urbanization of medieval London, when the city experienced multiple periods of famine (Connell et al., 2012). The cemetery was used by the general community in addition to burials from the infirmary, officials, and benefactors of the hospital, and contains individuals of all age ranges (Connell et al., 2012). For the paleodemographic analyses presented in Chapters 2 and 3, skeletons were equally sampled from the four temporal phases of SRP98, which include: Period 14 c. 1120-1200 CE, Period 15 c. 1200-1250 CE,

Period 16 c. 1250-1400 CE, and Period 17 c. 1400-1539 CE). Samples were then randomly selected for bone and tooth sampling for this study, further detailed below. The temporal periods of the cemetery were determined via high-precision Bayesian radiocarbon dating within a well-defined stratigraphic framework (see Sidell et al., 2007, for details regarding phasing of the cemetery using Bayesian modelling).

Within SRP98, different burial types correlate with periods of attritional mortality patterns (types A, B, and C) and crisis mortality patterns (8 - 45 bodies in a single grave cut; type D) (Connell et al., 2012). Paleodemographic patterns between the burial types and the correlation of these burials with documented years of famine indicate that type D burials are the result of famine-related crises; however, the 14th century Black Death also resulted in mass graves in SRP98 (Connell et al., 2012). Because the cause of the mass graves cannot be determined for interments during Period 16 (i.e. the period which includes the Black Death), analyses both including and excluding Period 16 were conducted.

5.2.2 Skeletal analysis

Sex was determined for each individual using sexually dimorphic features of the pelvis and skull (Buikstra and Ubelaker, 1994; Phenice, 1969). See 2.2.2.1 Sex determination in the previous chapter for details regarding sex determination.

For this study, epiphyseal closure of bones and dental development was used to differentiate subadults from adults (per methods in Buikstra and Ubelaker (1994) and Scheuer et al. (2000)). Individuals with an estimated age-at-death of 15 years or older

are considered adults, and those below 15 years are considered subadults.

5.2.3.3 Enamel hypoplasia

Enamel hypoplasia is a tooth enamel defect caused by disruptions in the metabolism of enamel-forming cells as a result of physiological stress (Huss-Ashmore et al., 1982). These defects appear as a ring or as pits on enamel, and have been found to be a good indicator of physiological stress during childhood (Goodman, 1991). The age at which the hypoplasia formed can be estimated by evaluating the location of the lesion on the tooth (Reid and Dean, 2000). However, Neiburger (1990) argues that stress is often experienced differently by individuals and that enamel hypoplasias could be the result of other causes, rather than stress; thus, enamel hypoplasias may not actually be reflective of physiological stress during childhood for all individuals. Though enamel hypoplasia may have a multifactorial origin, it is still used here with caution because it is the only potential indicator of stress that can be linked to an age at development.

To assess whether fluctuation in isotope values is the result of dietary change or of physiological stress, the presence and age at which enamel hypoplasias formed were included in the analysis of incremental dentine profiles. Only the maxillary and mandibular canines were assessed for presence of enamel hypoplasia, if present, as this tooth type is highly sensitive to physiological stress (Huss-Ashmore et al., 1982). The age-at-formation of enamel hypoplasia was estimated using guidelines from Reid and Dean (2000).

5.2.3 Sampling for isotope analysis

5.2.3.1 Bone samples

To obtain a sample that equally represents the four temporal groups, an approximately equal number of individuals were sampled from each period (Period 14 *n* = 41, Period 15 *n* = 41, Period 16 *n* = 39, and Period 17 *n* = 39). Rib samples were obtained from well-preserved bone with no evidence of pathological bone formation. Bone collagen samples were prepared using the modified Longin method (Brown et al., 1988) at the University of Bradford Stable Light Isotope Laboratory (UBSLI). Samples were measured in duplicate and compared with UBSLI and international standards (see Beaumont et al. (2013a), for preparation and analysis details used by UBSLI). The average of the results of the two samples for each individual was used for $\delta^{13}C$ and $\delta^{15}N$ if both samples were considered viable.

Samples considered viable for analysis have a collagen yield > 1% (Van Klinken, 1999), C:N range between 2.9 and 3.6 (DeNiro, 1985), nitrogen content (%N) between 5 and 17% (Ambrose, 1990), and carbon content (%C) between 15 and 47% (Ambrose, 1990). Carbon and nitrogen ratio, nitrogen content, and carbon content for each sample used in this analysis (i.e. viable samples) are provided in Appendix H. Two samples did not produce collagen yield higher than 1% and were excluded from the analyses (SRP98-30515 and SRP98-30865). Three samples did not meet the minimum %N requirement (SRP98-24010, -24022, and -30135) and two of those samples did not meet the %C requirement (SRP98-24010 and SRP-24022); all three samples were excluded. Isotope

values of rib samples from Lakin's (2010) previous isotopic analysis of St Mary Spital were included if the individual had been analyzed previously for demographic data, and follow the sample viability requirements above. There are no correlations between any of the collagen viability indicators (e.g. C:N, %N, and %C), indicating that there is minimal diagenetic alteration of the stable isotope signatures (Ambrose, 1990). The results are expressed using the delta (δ) notation in parts per thousand (per mil or ‰). Analytical error for bone collagen is ± 0.2‰ or better.

5.2.3.2 Tooth samples

In addition to comparison of $\delta^{13}C$ and $\delta^{15}N$ of attritional and mass burial types, incremental dentine analysis for carbon and nitrogen isotopes was conducted for tooth samples (*n* = 30) from individuals that were also sampled for rib bone. In contrast to bone, primary dentine does not remodel and can provide a time-bound archive of isotopes ingested during tooth formation (Beaumont et al., 2013b). Because the rate of human tooth development is well established (Hillson, 2005), accurate age ranges may be attributed to incremental samples with a single tooth, producing a high-resolution isotopic record of dietary and metabolic changes during childhood. Changes in $\delta^{13}C$ and $\delta^{15}N$ values from lack of food can be biochemically evident in hair tissue within days (Neuberger et al., 2013) and thus could be reflected in incremental tooth samples if experienced during childhood.

Sample sizes for teeth are summarized in Table 5.1. Like the bone collagen

Table 5.1: Sample sizes of teeth by burial type, including and excluding Period 16

	All Periods	Period 16 Excluded
Attritional	15	10
Mass	15	10
	30	20

samples, an approximately equal number of individuals were sampled from each period. Samples were obtained from teeth with no pathologies and minor tooth wear. Only loose teeth or teeth that could be steamed out of the mandible or maxilla were collected.

For this study, second molars were sampled, as this tooth type begins to mineralize after weaning age (approximately 2 years of age for medieval subadults (Richards et al., 2002; Shahar, 1990)) up to approximately 15.5 years of age (AlQahtani et al., 2010). If the second molar was not available, the first or second premolar was sampled because these tooth types follow similar mineralization patterns of the second molar (see Table 5.2 for approximate years of tooth mineralization). If those tooth types could not be collected (because of preservation, antemortem tooth loss or tooth wear), the first molar was sampled, as it has a different but still informative mineralization span of approximately 0.5 to 9.5 years (AlQahtani et al., 2010).

For each tooth, the full length of a single root was sampled and prepared for incremental dentine collagen analysis. Per Beaumont and Montgomery (2015), teeth were cut into sections and each section was assigned an approximate age by averaging the number of sections with the total mineralization span of the tooth. $\delta^{15}N$ and $\delta^{13}C$ values from each section were then assigned to that approximate age. Though there is some change in growth rates of the root length in certain areas of the tooth, root dentine secretion in permanent teeth is relatively consistent (Dean and Scandrett, 1995). Beaumont et al. (2013b) reported that though there is some averaging of the isotope values because of the direction in which the dentine is formed, the differing

Table 5.2: Tooth types sampled with approximate years of mineralization per AlQahtani et al., 2010

Tooth Type	Mineralization Years
First Molar	≈ 0.5 - 9.5
Second Molar	≈ 2.5 - 15.5
First Premolar	≈ 2.5 - 13.5
Second Premolar	≈ 3.5 - 14.5

rate of dentine growth is not significant.

Like the bone collagen samples, dentine collagen samples were prepared using the modified Longin method (Brown et al., 1988) at UBSLI. Samples were measured in duplicate and compared with UBSLI and international standards. The average of the results for the two dentine samples was used for $\delta^{13}C$ and $\delta^{15}N$, if both samples were considered viable.

All incremental dentine sections for each tooth produced collagen yields > 1 %. Following the same sample viability as the bone collagen, all dentine sections used yielded a C:N range between 2.9 and 3.6 (DeNiro, 1985), nitrogen content (%N) between 5 and 17% (Ambrose, 1990), and carbon content (%C) between 15 and 47% (Ambrose, 1990). The results are expressed using the delta (δ) notation in parts per thousand (per mil or ‰). Analytical error for dentine collagen is ± 0.2‰ or better.

5.3 Results

5.3.1 Bone sample results

Carbon and nitrogen isotope values for rib bones are available in Appendix H and are plotted by burial types, excluding Period 16, in Figure 5.1. Though analyses were conducted separately for all temporal periods and for temporal periods excluding Period 16, plotted data presented in this study excludes Period 16 because mass burials in this period cannot be differentiated between famine and Black Death mass burials; thus, the

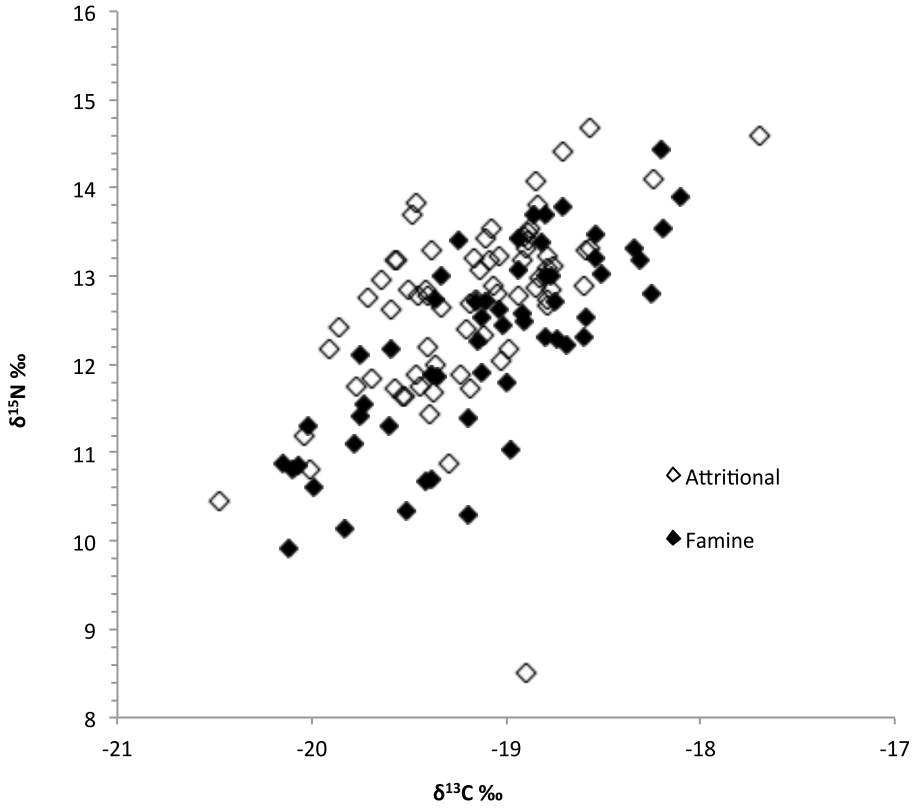


Figure 5.1: Plotted $\delta^{13}C$ and $\delta^{15}N$ bone collagen data for all individuals by burial type excluding Period 16

plotted data provide a potentially more conservative depiction of difference in isotope values between burial types. Sample sizes for rib bone included in this study, and a summary of the mean and ranges of the isotope data for the different subgroups are provided in Table 5.3. The famine burial isotope values show a higher standard deviation ($\delta^{15}N$ σ = 1.09, $\delta^{13}C$ σ = 0.54) than the attritional burials ($\delta^{15}N$ σ = 1.00, $\delta^{13}C$ σ = 0.44), indicating slightly more isotopic variability in the famine burials.

Results of the pairwise comparisons of bone collagen $\delta^{15}N$ and $\delta^{13}C$ values and medians (in accordance with nonparametric statistics) between burial types are shown in Table 5.4. Boxplots by burial type of $\delta^{15}N$ values for all individuals, excluding Period 16, are provided in Figure 5.2. Comparison of isotope values between attritional and famine burials for all individuals reveal a significant difference in $\delta^{15}N$ between burial types, with elevated $\delta^{15}N$ values in the attritional burials, but no significant difference in $\delta^{13}C$ values. Similar results for all individuals and for just the adults are found with Period 16 included in the analyses.

Carbon and nitrogen isotope values of bone collagen for the sexes are plotted by burial type, excluding Period 16, in Figure 5.3 and Figure 5.4. A summary of the mean and standard deviation of the isotope data for the sexes are provided in Table 5.3. Sex-specific pairwise comparisons of isotope values between burial types are shown in Table 5.4. Boxplots by burial type of $\delta^{15}N$ values for the sexes, excluding Period 16, are provided in Figure 5.5 and Figure 5.6. When Period 16 is included in the analysis of females, there is a significant difference in $\delta^{15}N$ between the burial types, with

Table 5.3: Mean and standard deviation of bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for attritional and famine burials for all individuals, adults (individuals 15 years of age and above), and the sexes (including and excluding Period 16 1250-1400 CE)

		All Periods			
		$\delta^{15}N$		$\delta^{13}C$	
	N	Mean	SD	Mean	SD
All	166	12.44	1.08	-19.18	0.56
Attritional	86	12.65	1.03	-19.21	0.56
Mass	80	12.21	1.10	-19.15	0.58
Adults	147	12.52	1.04	-19.17	0.57
Attritional	79	12.71	0.94	-19.21	0.57
Mass	68	12.29	1.11	-19.13	0.57
Females	68	12.43	1.01	-19.17	0.48
Attritional	36	12.69	0.83	-19.13	0.45
Mass	32	12.16	1.12	-19.22	0.53
Males	75	12.60	1.08	-19.19	0.64
Attritional	43	12.73	1.03	-19.27	0.65
Mass	32	12.43	1.14	-19.07	0.62
		Period 16 Excluded			
		$\delta^{15}N$		$\delta^{13}C$	
	N	Mean	SD	Mean	SD
All	128	12.48	1.06	-19.15	0.49
Attritional	71	12.68	1.00	-19.18	0.44
Mass	57	12.24	1.09	-19.11	0.54
Adults	112	12.49	1.06	-19.15	0.49
Attritional	65	12.77	0.86	-19.17	0.45
Mass	47	12.30	1.16	-19.10	0.55
Females	53	12.43	1.03	-19.20	0.47
Attritional	31	12.68	0.86	-19.14	0.46
Mass	22	12.10	1.17	-19.28	0.48
Males	56	12.10	1.17	-19.28	0.48
Attritional	34	12.85	0.87	-19.20	0.44
Mass	22	12.48	1.22	-18.97	0.59

Table 5.4: Results of Mann Whitney U tests of bone collagen comparing isotope values between attritional and famine burials for all individuals, adults, and the sexes (including and excluding Period 16)

		All Periods				Period 16 Excluded			
		Attritional Burial Median	Mass Burial Median	*U-Statistic*	*P*	Attritional Burial Median	Mass Burial Median	*U-Statistic*	*P*
All	$\delta^{15}N$	12.85	12.40	-2.68	0.01	12.85	12.44	-2.33	0.02
	$\delta^{13}C$	-19.16	-19.04	0.97	0.33	-19.16	-19.04	1.02	0.31
Adults	$\delta^{15}N$	12.85	12.53	-2.20	0.03	12.85	12.53	-1.86	0.06
	$\delta^{13}C$	-19.17	-19.04	0.99	0.33	-19.17	-19.04	1.02	0.31
Females	$\delta^{15}N$	12.85	12.38	-2.02	0.04	12.77	12.23	-1.75	0.08
	$\delta^{13}C$	-19.13	-19.11	-0.25	0.81	-19.14	-19.18	-0.71	0.48
Males	$\delta^{15}N$	12.85	12.69	-0.89	0.37	12.90	12.87	-0.77	0.44
	$\delta^{13}C$	-19.19	-19.03	1.41	0.16	-19.18	-18.91	1.46	0.14

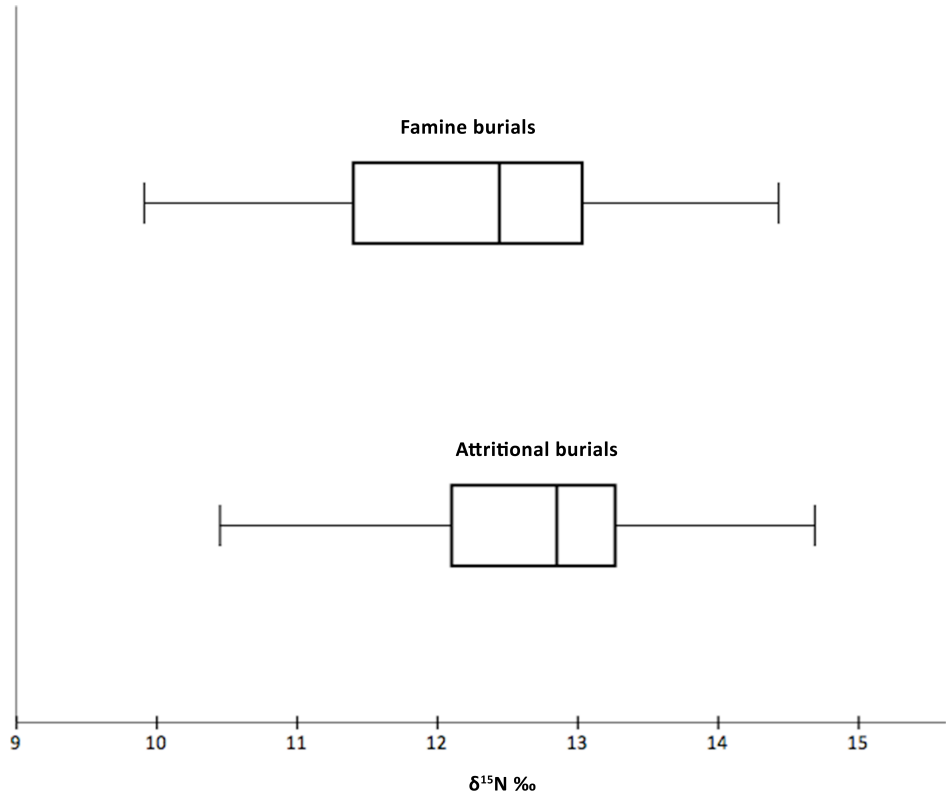


Figure 5.2: Boxplots of $\delta^{15}N$ bone collagen for all individuals by burial type, excluding Period 16

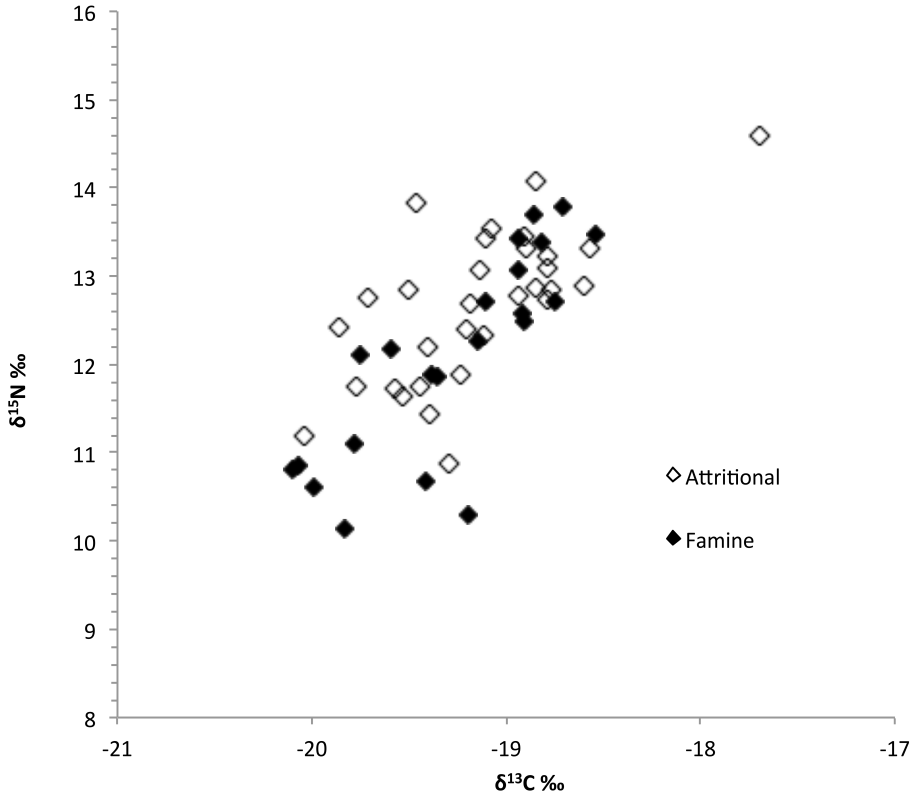


Figure 5.3: Plotted $\delta^{13}C$ and $\delta^{15}N$ bone collagen data for females by burial type excluding Period 16

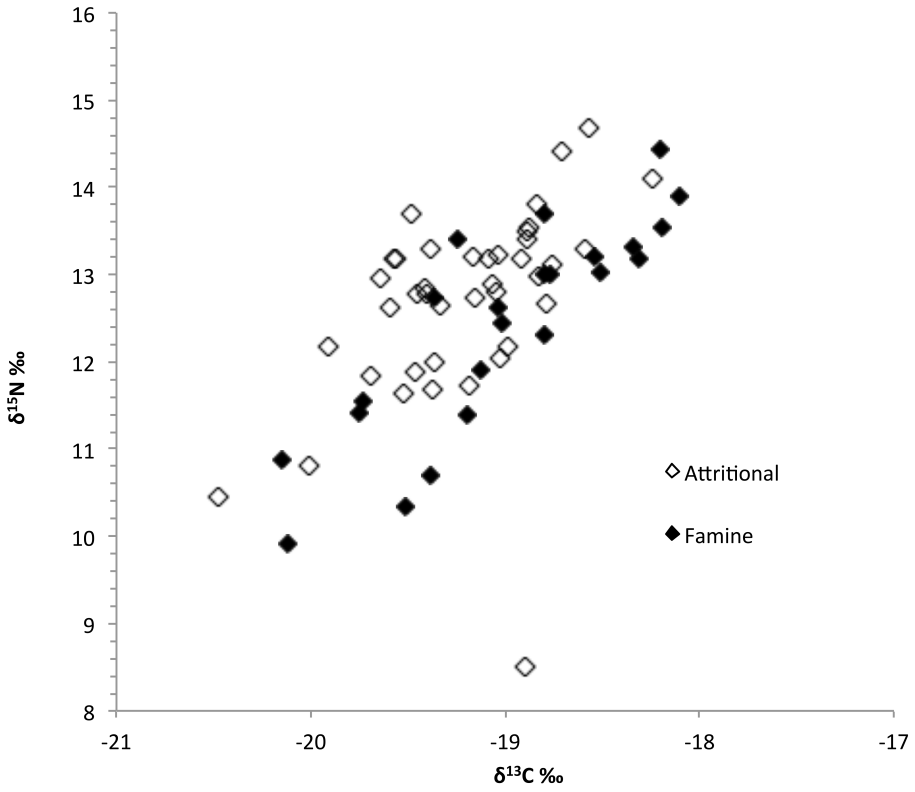


Figure 5.4: Plotted $\delta^{13}C$ and $\delta^{15}N$ bone collagen data for males by burial type excluding Period 16

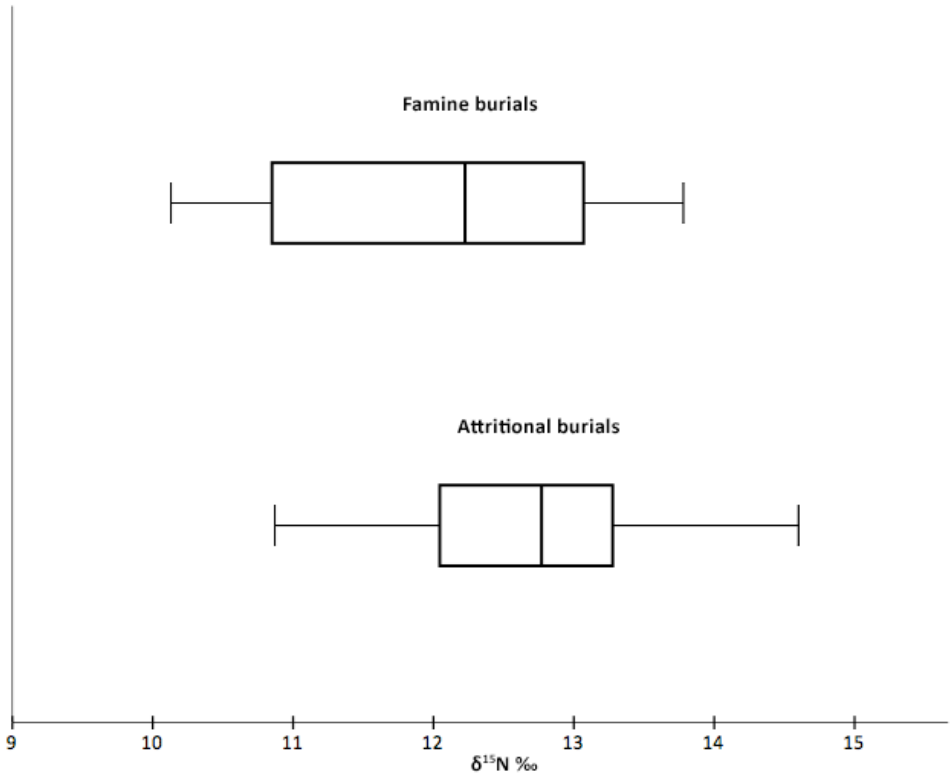


Figure 5.5: Boxplots of $\delta^{15}N$ bone collagen for females by burial type, excluding Period 16

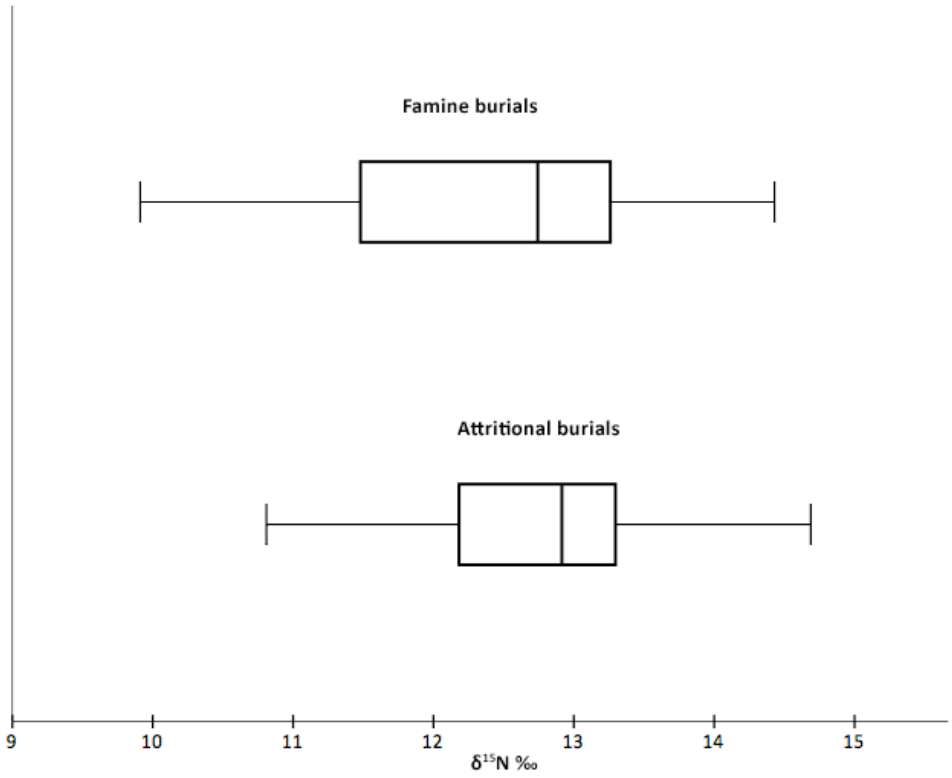


Figure 5.6: Boxplots of $\delta^{15}N$ bone collagen for males by burial type, excluding Period 16

elevated $\delta^{15}N$ values in attritional burials, but no difference in $\delta^{13}C$. Results are similar when Period 16 is excluded ($P = 0.09$ is suggestive of a difference). For males, there are no significant differences in $\delta^{13}C$ or $\delta^{15}N$ between the burial types when Period 16 is included or excluded.

5.3.2 Incremental dentine results

Incremental dentine isotope values are provided in Appendix I, with corresponding graphs of plotted isotope values for each individual in Appendix J. Bone collagen results are plotted with the dentine collagen results only for comparison and should not be considered reflective of age for these results. That is, bone collagen values are not reflective of age on the horizontal axis in these graphs, but of time prior to death. Results show that all individuals exhibit changes in diet throughout childhood, regardless of burial type. A detailed analysis and discussion of the results is provided in the Discussion section below.

5.4 Discussion

5.4.1 Bone samples and burial type

Analyses of rib bones comparing isotope values between attritional and famine burials suggest that individuals interred in attritional graves exhibit higher $\delta^{15}N$ values compared to individuals in famine graves. The results are the same for separate analysis

of adults. Though a difference in $\delta^{15}N$ values between burial types was hypothesized, it was expected that the famine burials would exhibit higher $\delta^{15}N$ values than the attritional burials because of physiological responses to nutritional stress. The results, however, show that the famine burials actually exhibited significantly lower $\delta^{15}N$ values than the attritional burials. Previous research comparing bone collagen isotope values of burial types in SRP98 similarly reveals elevated $\delta^{15}N$ values for single burials; however, this difference was not statistically significant and only Periods 15 and 16 were sampled (Lakin, 2010). Additionally, the median for $\delta^{13}C$ values is consistently lower in the attritional compared to the famine burials, though this difference is not significant. This is to be expected, as $\delta^{13}C$ values are generally less variable than $\delta^{15}N$ values unless changing, for example, from a C_3- to C_4-based diet or vice versa. All individuals show carbon values below -18‰, indicating a C_3-based diet (Smith and Epstein, 1971).

Rather than reflecting differences in nutritional stress experienced between the burial types, these results may actually reflect differences in diet that could have been a consequence of famine. That is, individuals from attritional burials may have been eating different proportions of foods (particularly more protein sources) compared to individuals from the famine mass burials. The high $\delta^{15}N$ is most likely indicative of more protein in the diet such as fish and/or animal meat. Because most of the available nitrogen in marine environments is produced by bacterial denitrification, a process that creates more ^{15}N than N_2 soil fixation, marine organisms have elevated ^{15}N values compared to terrestrial plants, which is then passed through trophic levels. Thus, individuals with more fish in their diet will exhibit higher $\delta^{15}N$ values compared to

individuals who lack fish in their diet (Schoeninger and DeNiro, 1984). Müldner and Richards (2005) find more fish in high-status *vs*. low status individuals because of better access to food, and suggest that the lack of a marine signature may be indicative of a low-status diet. Similarly in SRP98, individuals during non-famine periods would have had better access to foods with high protein (Rawcliffe, 2013), such as fish, compared to individuals experiencing famine, which would be reflected in the elevated $\delta^{15}N$ values in individuals from attritional burials.

In addition to fish, individuals from non-famine periods may have consumed more animal meat, as individuals with a higher proportion of animal meat in their diet also exhibit higher $\delta^{15}N$ values (Pearson et al., 2003; Sponheimer et al., 2003a; Sponheimer et al., 2003b). Through analysis of faunal remains in London, Lakin (2010) found a high herbivore $\delta^{15}N$ baseline in London compared to other medieval English assemblages. The difference may reflect better access to animal meat, such as beef, or dairy sources for individuals not undergoing famine (i.e. attritional burials), compared to individuals who experienced a lack of accessibility to animal meat during famine. For example, cattle plagues, such as the Great Bovine Pestilence of 1318, which claimed over half of the bovine population in Europe, including England, caused beef shortages and a scarcity of milk resources for decades (Slavin, 2012). This catastrophic event was particularly detrimental because it occurred just after the Great Famine of 1315-17, prolonging inadequate food supplies in England, and also because dairy products were an important source of protein for the working poor (Tames, 2003). Moreover, the slightly higher standard deviation in the famine burials compared to the attritional

burials may be a reflection of more dietary variability in the famine burials, as individuals adapted to limited accessibility to food supplies during famine.

Fasting for religious purposes (abstention from foods, usually meat, as a symbol of faith) became a regular practice during the Late Medieval Period (Woolgar, 2000). On fasting days, consumption of animal meat was forbidden, but fish was consumed as a permissible alternate source of protein (Tames, 2003). Purposeful fasting, however, was most likely not practiced during periods of famine, as individuals would have consumed whatever food supplies were available. Thus, the high $\delta^{15}N$ values seen in burials from non-famine periods (i.e. attritional burials) may be reflective of fish consumed as result of religious fasting. Though most individuals interred in this cemetery were mostly likely poor and the poor did not participate in fasting as strictly as high-status individuals, the poor still consumed $\delta^{15}N$ enriched salted fish, eels and oysters to fast when they could (Tames, 2003).

Additionally, it is possible that bone collagen samples from young individuals may capture isotopically enriched $\delta^{15}N$ from breastfeeding because of slow bone turnover rates. For this project, however, there are only two individuals sampled for bone collagen with an age-at -death below the age of 8, an age at which high $\delta^{15}N$ values as a result of breastfeeding would likely not be reflected in bone collagen samples, as bone remodels rapidly during early childhood (Valentin, 2002). Moreover, both of these individuals were interred in mass burials and would thus not have contributed to the high $\delta^{15}N$ values evident in the attritional burials.

Results from the rib bone collagen may not reflect nutritional stress experienced just prior to death because of slow tissue turnover rates. Hedges et al. (2007) argue that isotopic values derived from human bone collagen may actually reflect the average adult diet for the last 20 to 30 years before death, rather than the estimate of 4 to 10 years by Valentin (2002). Bone turnover rates, however, generally decrease with age (Hedges et al., 2007; Klepinger, 1984), making it difficult to estimate the timespan that the isotope values are actually reflecting. Unless chronically affected by famine throughout life, food deprivation experienced only years or months prior to death, which would presumably also increase an individual's risk of dying, may not be captured in bone collagen. Thus, individuals who died quickly as a result of famine or who only experienced famine for a short time may not exhibit higher nitrogen values in bone collagen. Similarly, in an analysis of individuals that experienced the Great Irish Famine (1845-1852 CE), Beaumont and Montgomery (2013a) expected higher $\delta^{15}N$ values for bone collagen samples, but found no difference in $\delta^{15}N$ values, and attribute this slow turnover rate in bone that averaged short-term changes in $\delta^{15}N$ values.

Compared to bone tissue, teeth are more informative about temporal fluctuations in isotope values, as tooth mineralization occurs at known ages in life (AlQahtani et al., 2010; Hillson, 2005). However, interpretations of isotope analysis from teeth are limited to years prior to adulthood because all teeth, except the third molar, are fully mineralized by 15.5 years of age (ALQahtani et al., 2010). Thus, dentine collagen is generally not informative about nutritional stress or dietary changes occurring just prior to death unless the individual died during or soon after tooth

mineralization (i.e. during childhood or adolescence). Incremental dentine, however, may be useful to identify dietary patterns or nutritional stress occurring during childhood that could have increased an individual's risk of death during famine. Unlike bone and teeth tissues, hair samples are more informative about nutritional stress experienced prior to death, as samples of hair near the root can provide isotope information as recently as two weeks, and then for several months when the hair is sampled farther away from the root (Neuberger et al., 2013). For example, Neuberger et al. (2013) conducted segmental analysis of hair and found an increase in $\delta^{15}N$ values as BMI decreased. Though hair samples are useful for studying potential nutritional stress in living populations, preserved hair is rare in skeletal assemblages, except in certain environments, and thus may be of limited use in bioarchaeological research.

Additionally, the difference in $\delta^{15}N$ values for bone collagen between burial types may reflect the presence of migrants in the sample. As discussed in previous chapters, rural-to-urban migration for labor opportunities was prevalent in Late Medieval London (Dyer, 2002). Migrants who entered the city may exhibit different isotope values than individuals who lived in London for longer periods of time, and the diet migrants consumed in their place of origin would be averaged in with their diet prior to death (i.e. the London diet). Specifically, because bone typically takes at least 10 years, and possibly longer (Hedges et al., 2007), to remodel in adults, recent migrants would exhibit different isotope values than those residing in London longer than a decade. It is not possible to know conclusively whether migrants are more likely to be buried in attritional or mass interments without additional isotopic analysis (e.g. oxygen,

strontium, and lead (Montgomery, 2010; Montgomery et al., 2010)). However, rural-to-urban migrants might have experienced increased susceptibility to infection (McNeill, 1980), which may have been exacerbated during famine, potentially making them more likely to be in famine graves. Moreover, the slightly higher variability of isotope variables seen in the famine burials, compared to the attritional burials, may be a reflection of more migrants in the famine burials. Further, the high $\delta^{15}N$ values seen in SRP98 (Lakin, 2010) would most likely be a result of higher $\delta^{15}N$ values from London residents (who, per the discussion above, might have been more likely interred in attritional burials) rather than migrants, as London residents would have been consuming the London diet for a longer period of time. Incremental dentine analyses can be used to show patterns of dietary change that may indicate if the individual was a migrant (e.g. drastically different isotope values from childhood compared to adulthood).

Similar to the analysis for all individuals, sex-specific results of bone samples reveal that females in attritional burials exhibit higher $\delta^{15}N$ values compared to females in famine burials. Males, however, did not show a difference in $\delta^{15}N$ or $\delta^{13}C$ isotope values between burial types. Previous isotopic analysis of SRP98 show that, in Period 15, young females plotted differently than males, with lower $\delta^{13}C$ and $\delta^{15}N$ values, which Lakin (2010) attributed to female-led migration. As discussed in previous chapters, it was common for adolescents and young adults, particularly females (Goldberg, 2004), to travel to urban centers because of greater economic opportunities (Dyer, 2002; Hanawalt, 1995). According to tax documents, urban cities had more female than male

servants (Kowaleski, 2014). Particularly after the Black Death, even more females were present in urban areas (Barron, 1989; Lewis, 2016), filling positions once held by males (Goldberg, 2004). If a high proportion of migrants were interred in famine graves (per the discussion above) and if more females were migrating into London, there would thus be a higher proportion of female migrants in famine graves compared to males. This is consistent with the lack of difference in isotope values between burial types for males.

5.4.2 Isotope values and the life course

Results of incremental dentine analysis show that all individuals exhibit changes in diet throughout childhood, regardless of burial type. Depending on the individual, $\delta^{15}N$ values remain steady or drastically change through childhood, while $\delta^{13}C$ values generally do not vary much throughout childhood, staying within a range of -20.09‰ to -18.19‰. All individuals exhibit values during childhood indicative of a C_3-based diet ($\delta^{13}C$ less than -18‰). Dentine collagen results from SRP98 are much more variable than the consistent $\delta^{13}C$ and $\delta^{15}N$ values from dentine collagen seen at Wharram Percy, a contemporaneous rural English cemetery (Fuller et al., 2003). The variation in diet between the different individuals seen during childhood could be a reflection of London having a greater chance for multiple pathways to be co-existing than in rural areas (i.e. migration, famine, and urban intensification). For example, a migrant would most likely exhibit a nonlocal isotopic signature in dentine collagen, rather than having isotopic values with the range for London. Moreover, an individual experiencing famine or

substantial changes in diet may exhibit a fluctuating pattern in dentine values throughout childhood because of precarious food supplies. The rapid increase in population density in London at this time would also have affected access to food supplies.

5.4.2.1 Potential markers of famine

A higher proportion of attritional burials exhibit a steady childhood diet (less than 2‰ increase or decrease in isotope values of dentine collagen sections) (SRP98-6571, -9420, -10765, -11471, -12379, -17539 -18138, -21273, -26522, and -29698), than in the famine burials (SRP98-2487, -24010, and -32302). This complements the higher variability seen in the famine burials compared to the mass burials for isotope values in bone collagen. This pattern may be reflective of a steady diet and adequate nutrition during childhood for individuals in attritional burials. This may also reflect individuals adapting to the limited accessibility of food supplies during famine by incorporating other foods into the diet. Also, most of the individuals with a steady childhood diet have dentine values within the $\delta^{15}N$ range for the cemetery in general (11.36 to 13.52‰), which is consistent with living in or near London during childhood. Further, all except two of these individuals (SRP98-10765 and SRP98-11471) have bone collagen $\delta^{15}N$ values, which generally reflect diet for at least the last 10 years of life, that are within 1‰ of the oldest dentine $\delta^{15}N$ value (≈9.5 to 14.5 years of age). This suggests that the steady diet seen in childhood from incremental dentine analysis likely continued into adulthood, and possibly, that the individuals resided in London until their death.

Using modern hair samples, Neuberger et al., (2013) found that during starvation, in addition to increasing $\delta^{15}N$ values, $\delta^{13}C$ values decrease at the same time. The authors suggest that because body fat is already depleted with ^{13}C unlike other tissues like muscle, a body experiencing undernutrition will recycle carbon from fat deposits, thus integrating more ^{12}C into the newly synthesized tissues, which would result in reduced $\delta^{13}C$ values. Additionally, when no new carbohydrates are being consumed, as is typical during periods of undernutrition, the $\delta^{13}C$ level in the body is not preserved, resulting in reduced $\delta^{13}C$ values. Mekota et al. (1999) also found this pattern in hair samples of anemic patients, with $\delta^{15}N$ values increasing and $\delta^{13}C$ values decreasing as BMI decreased. Further, when the patients began to recover and BMI levels increase, the $\delta^{15}N$ values would decrease and $\delta^{13}C$ values would decrease, creating a bubble appearance (Mekota et al., 1999). Beaumont and Montgomery (2016) also reported this pattern in their analysis of individuals affected by the Great Irish Famine via incremental dentine analysis, though this may have been a result of shifting from a C_3 to C_4-based diet, and label this pattern as *opposing covariance*. They also suggest that a lack of change in $\delta^{13}C$ while $\delta^{15}N$ increases could be construed as potential nutritional stress, as carbon often takes longer to be reflected in isotope values. For example, Figure 5.7 shows a dentine collagen profile that exhibits a pattern of opposing covariance at ages ≈2.5 to 5.5 and ≈9.5 to 13.5. Conversely, SRP98-10765 shows a dentine profile of potential dietary change with consistent decreases in $\delta^{15}N$ and $\delta^{13}C$ throughout childhood (≈3.5 – 14.5 years).

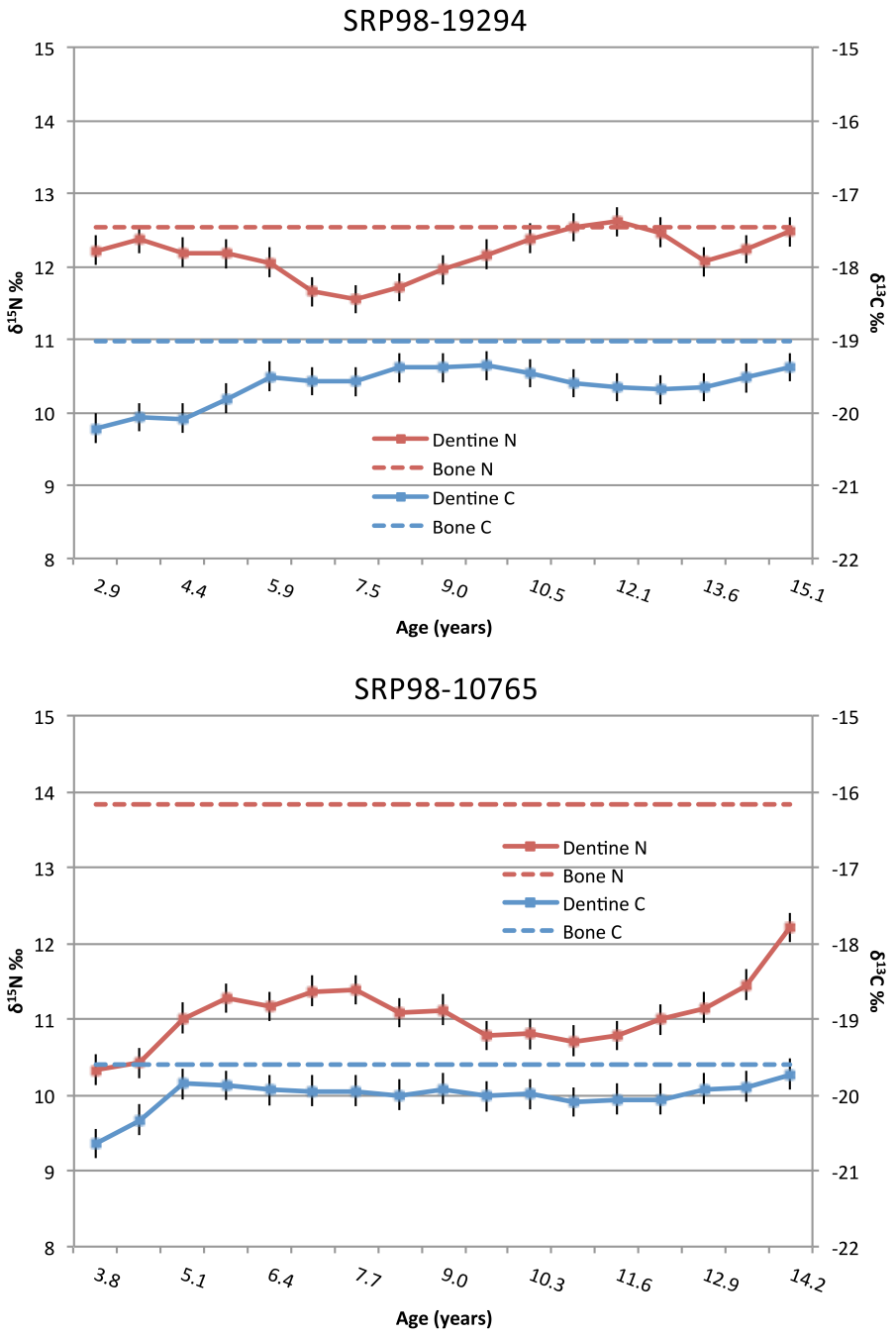


Figure 5.7: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-19294 (top- adult male) and SRP98-10765 (bottom- adult female)

Several individuals from famine and mass burials exhibit the opposing covariance pattern (SRP98-9632, 9789, -19294, -20682, -24010, -30920, -32302), and some individuals show multiple instances, suggesting that these individuals may have experienced multiple periods of undernutrition during childhood. For example, SRP98-30920 (an young adult male) shows covarying opposition from approximately 3 to 6 years of age, with a corresponding linear enamel hypoplasia at approximately 3 years of age on the mandibular canines (Figure 5.8). Given that enamel hypoplasias indicate the experience of some form of physiological stress during mineralization (Hillson and Bond, 1997) and that this person exhibits patterns of opposing during enamel hypoplasia formation, this suggests that the isotope values are more likely reflecting undernutrition rather than dietary change. Not all individuals have enamel hypoplasias when the opposing covariance pattern is present; however, this may be an artifact of when the canine mineralizes (between 1.5 and 6.2 years of age (Reid and Dean, 2000)) or of undernutrition that was not severe enough to affect the mineralization of the tooth (Neiburger, 1990). Moreover, patterns of opposing covariance in $\delta^{13}C$ and $\delta^{15}N$ values are also present in some attritional burials (SRP98-5287, -12379, -18138, and -21273) though they are not as distinct, indicating that this pattern is not unique to famine burials and that individuals from attritional burials may have experienced nutritional stress during childhood as well.

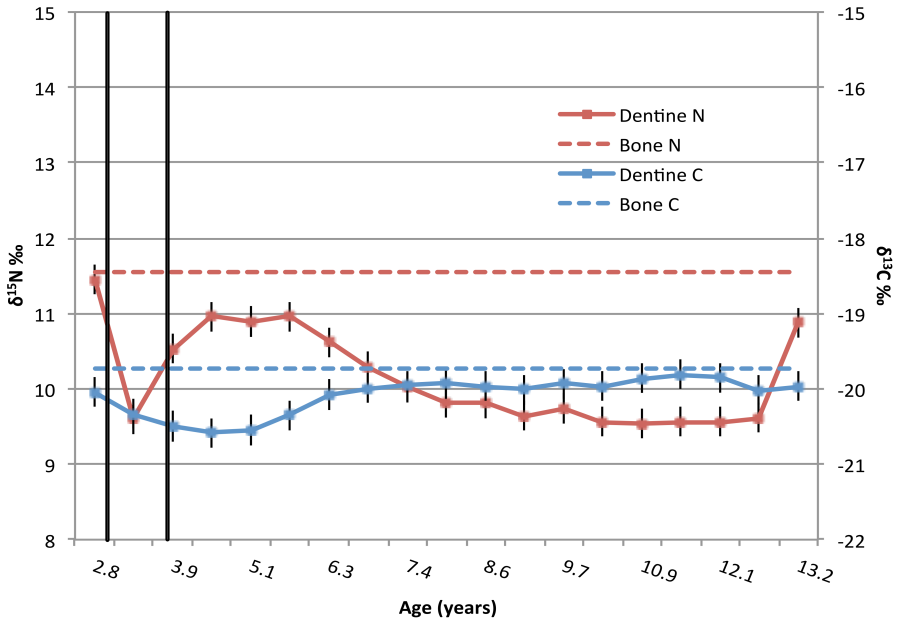


Figure 5.8: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-30920 (young adult male) with black line showing enamel hypoplasia formation age

5.4.2.2 Unique isotope patterns

Two famine burials (SR989-3934 and SRP98-29058) exhibit particularly unstable isotope patterns throughout childhood (Figure 5.9). SRP98-3934 is an adult female with a drastic increase in $\delta^{15}N$ (+3.58‰) and $\delta^{13}C$ (+2.09‰) from approximately 7 to 9 years old. After 9 to 13.5 years old, both the $\delta^{15}N$ and $\delta^{13}C$ decrease ($\delta^{15}N$ = -3.16‰, $\delta^{13}C$ = -1.36‰). Given that the $\delta^{13}C$ does not covary with $\delta^{15}N$ and because enamel hypoplasias do not coincide during these ages, this change more likely reflects changes in diet or possibly migration (i.e. residing in a place with a higher trophic level diet with a marine component from 7 to 9 years of age). The bone collagen results for $\delta^{15}N$ are above the London average, which may reflect chronic undernutrition prior to death or a diet that was higher in protein than most Londoners. SRP98-29058, a young adult male, exhibits a pattern inverse to that of SRP98-3934, with a drastic decrease in $\delta^{15}N$ (-3.85‰) from about 3 to 5.5 years old , and then a steady increase (+1.30‰) to 11 years of age. This individual also exhibits three enamel hypoplasias estimated at ages 3.4, 4.3, and 4.8. Given that enamel hypoplasias are estimated at the same time as the rapid decrease in $\delta^{15}N$ seen in the dentine collagen profile, this pattern is more likely reflecting undernutrition or some other physiological stress that could possible be associated with diet.

SRP98-20682 (an adult male) also exhibits a unique isotopic pattern during childhood but is interred in an attritional burial. From approximately 2.5 to 4.5 years old there is a decrease in $\delta^{15}N$ (- 2.81‰) and increase in $\delta^{13}C$ (+ 0.17‰) (Figure 5.10). After 5.5 years of age, $\delta^{15}N$ begins to increase significantly, by 2.0‰, to age 11.5. Unlike the

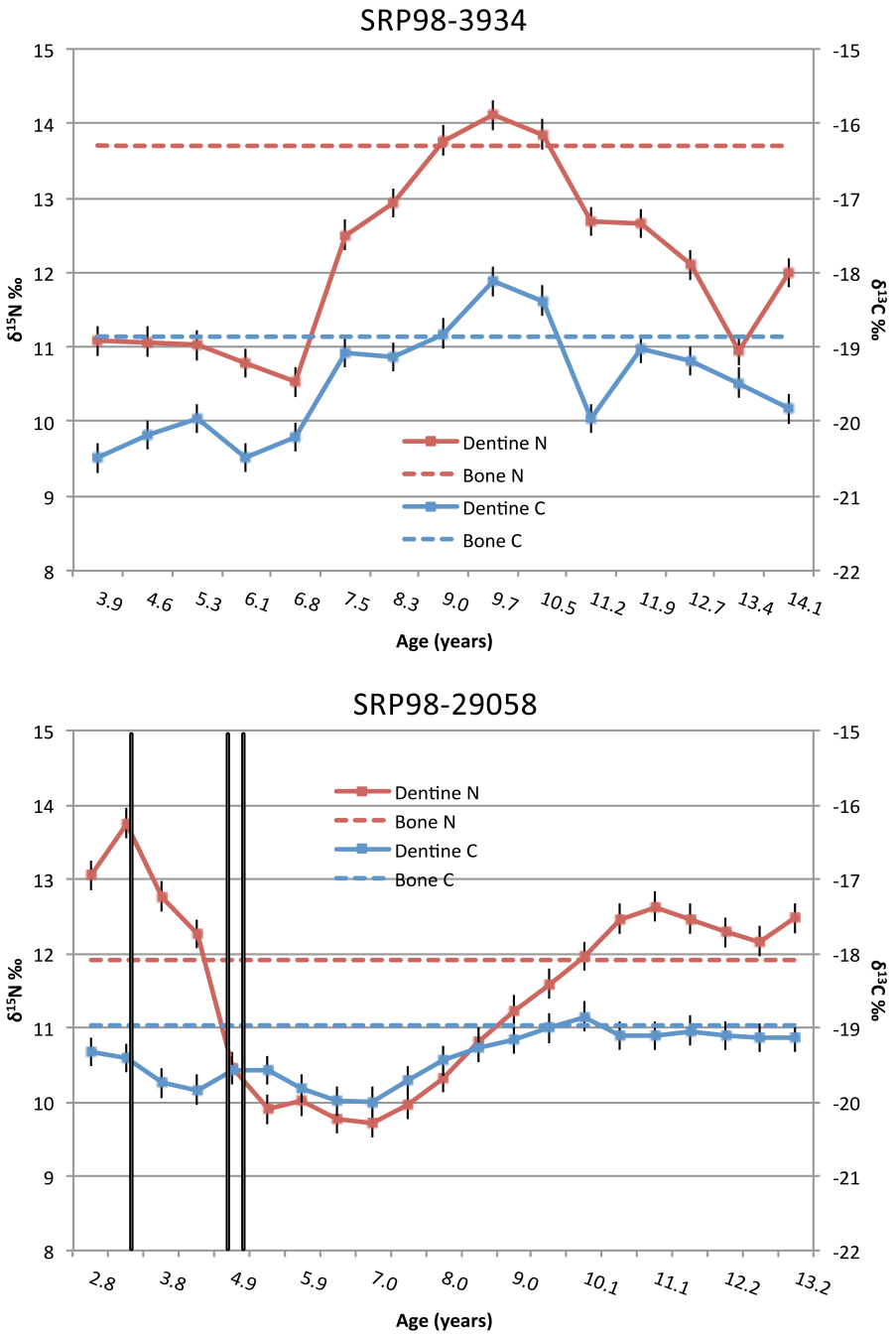


Figure 5.9: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-3934 (top- adult female) and SRP98-29058 (bottom- young adult male) with black line showing enamel hypoplasia formation age

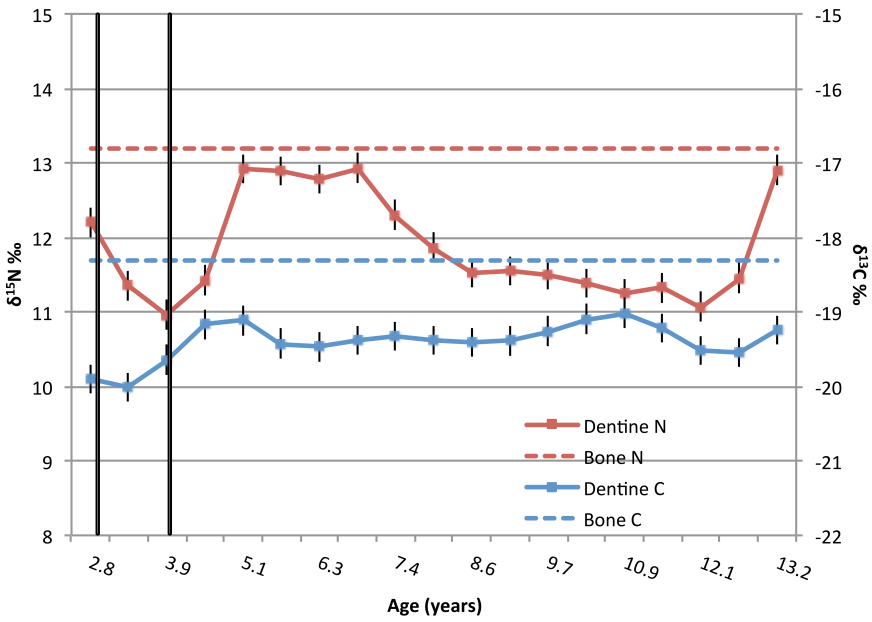


Figure 5.10: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-20682 (adult male) with black line showing enamel hypoplasia formation age

gradual changes prior to 11.5 years old, there is a sudden decrease of 1.25‰ within months, and subsequently an increase of 2.20‰ within months as well. Two enamel hypoplasias are present and are estimated to have formed during the decrease in $\delta^{15}N$, from 2.5 to 4.5 years old, which suggests that this decrease is associated with some physiological stress such as undernutrition. It is not possible to know if some physiological stress was experienced during the other isotopic fluctuations, as enamel hypoplasias are not exhibited after approximately 6 years of age. The bone collagen isotope values suggest that the final increase prolonged into adulthood, and is within the average for the cemetery values.

Additionally, samples of the first molar are able to capture isotope values in earlier ages because of the time at which this tooth begins to mineralize (≈0.5 years (AlQahtani et al., 2010)). For these individuals (SRP98-2679 and SRP98-12379), a decrease in $\delta^{15}N$ from approximately 0.5 to 2 years of age suggests that these individuals were consuming less isotopically enriched breast milk and more weaning foods during this time, which is consistent with weaning age in Medieval England of about 2 years old (Fuller et al., 2003; Haydock et al., 2013). Some individuals for whom the second molar and first premolars are sampled (teeth that begin mineralizing at 2.5 years old (AlQahtani et al., 2010)) exhibit values that may suggest late or prolonged weaning (SRP98-30920, -19147, and -22199) (i.e. a decrease in $\delta^{13}C$ and $\delta^{15}N$ after 2.5 years of age), though this could also be the result of some other dietary change just after weaning (e.g. consumptions of foods with a different origin).

It is important to note that there is no skeletal evidence to suggest that these individuals experienced poor health or physiological stress during life, and thus the interpretations made here are based solely off of the isotopic profile, demographic information from the skeleton (e.g. sex and age), and burial context.

5.4.2.3 Potential migrants

The integration of data from both dentine collagen and bone collagen may be informative of potential migrants in the sample. For example, SRP98-24185, an older female from a famine burial, has lower isotope values from approximately 2.5 to 9 years of age (average is $\delta^{15}N$ = 10.37‰, $\delta^{13}C$ = -18.60‰) and a sharp increase at 9 years to higher isotope values (average is $\delta^{15}N$ = 12.35‰, $\delta^{13}C$ = -18.64‰) that are within the cemetery average (Figure 5.11). The sudden increase in isotope values (by $\delta^{15}N$ = +2.84‰, $\delta^{13}C$ = +0.83‰) may reflect a dietary change, or more specifically, a dietary change as a result of migration to London. In England, there is evidence for females as young as 7 traveling to urban areas for work opportunities, though it was more common for adolescents around twelve to fifteen years of age (Hanawalt, 1995). Also, as discussed previously, females were more likely to migrate into London for labor opportunities (Goldberg, 2004). This individual may have migrated into London earlier than other females (possible with her family rather than alone for work), at approximately 9 years old and resided in London until late adulthood. It is also possible, that this individual could have become a lay sister, as the individuals were often buried at SRP98 (Connell et al., 2012). Bone collagen results suggest that this person's diet

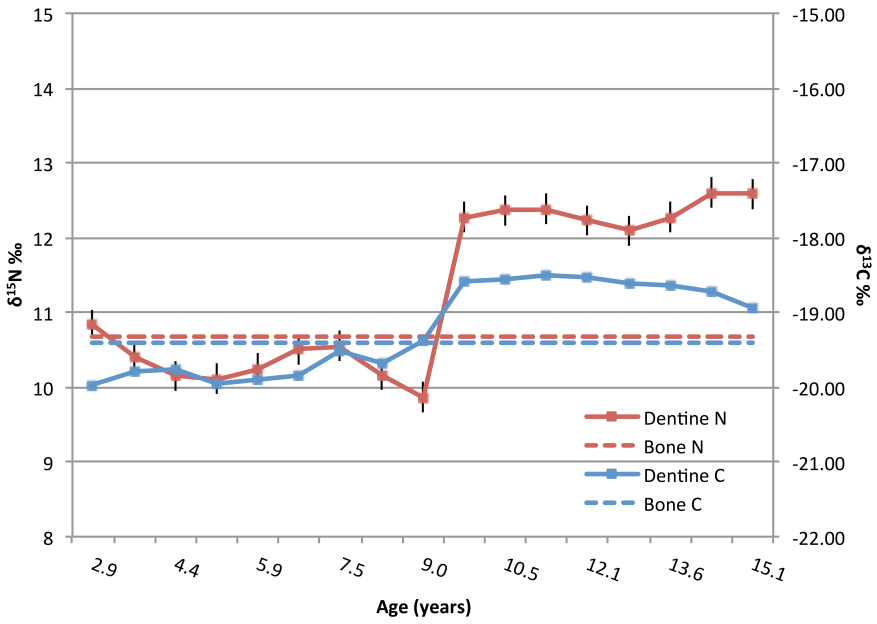


Figure 5.11: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-24185 (mature adult female)

changed prior to death at around 50 to 60 years of age, with $\delta^{15}N$ (10.67‰) and $\delta^{13}C$ (19.41‰) values below the cemetery average. Perhaps this individual's diet changed a result of famine with less protein in the diet prior to death.

SRP98- 30920 is a young adult female from a Period 15 famine burial exhibiting consistent $\delta^{15}N$ values during childhood, with changes within 1‰ from approximately 3 to 12 years of age (Figure 5.12). At approximately 12 years of age, there is a sudden 1.26‰ increase in less than a year, and the $\delta^{15}N$ values for the bone collagen are within the average for the cemetery. These results suggest that this individual had a dietary change around the age of 12 that was sustained prior to death, which is consistent with an adolescent female migrating into London from another area. SRP98-9789 is a male interred in a Period 14 famine burial who exhibits a similar pattern as SRP98-30920, with a childhood diet of $\delta^{15}N$ values between 8.86‰ and 11.25‰, and $\delta^{13}C$ values between -20.98‰ and -19.58‰. The bone collagen results reflect a different diet prior to death, with values that are higher than any of the dentine collagen results ($\delta^{15}N$: 13.00‰, $\delta^{13}C$: 10.48‰) but are within the average for the cemetery. The difference between the average dentine collagen results and bone collagen results is a substantial decrease of 3.15‰ for $\delta^{15}N$ and 1.30‰ for $\delta^{13}C$. This difference may reflect a change in diet from childhood to years prior to death, which, in turn, could be a result of migration into London. $\delta^{15}N$ values begin to increase around the age of 10, which may be when this individual's diet started to change (i.e. when the individual migrated).

SRP98-32302 also exhibits a notable difference between dentine and bone collagen (Figure 5.13), which may be indicative of migration. From approximately 3.5 to

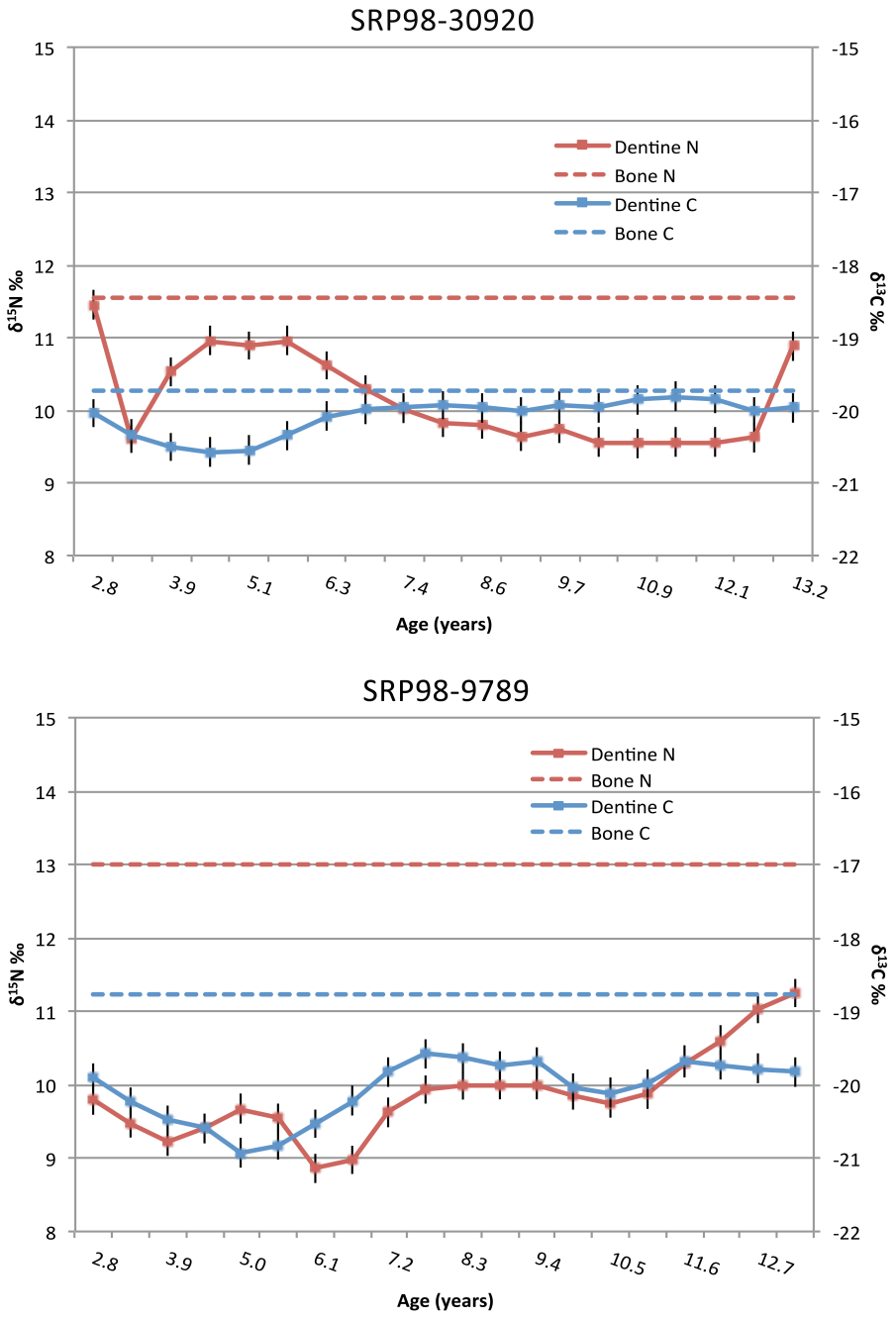


Figure 5.12: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-30920 (top- young adult male) and SRP98-9789 (bottom- adult male)

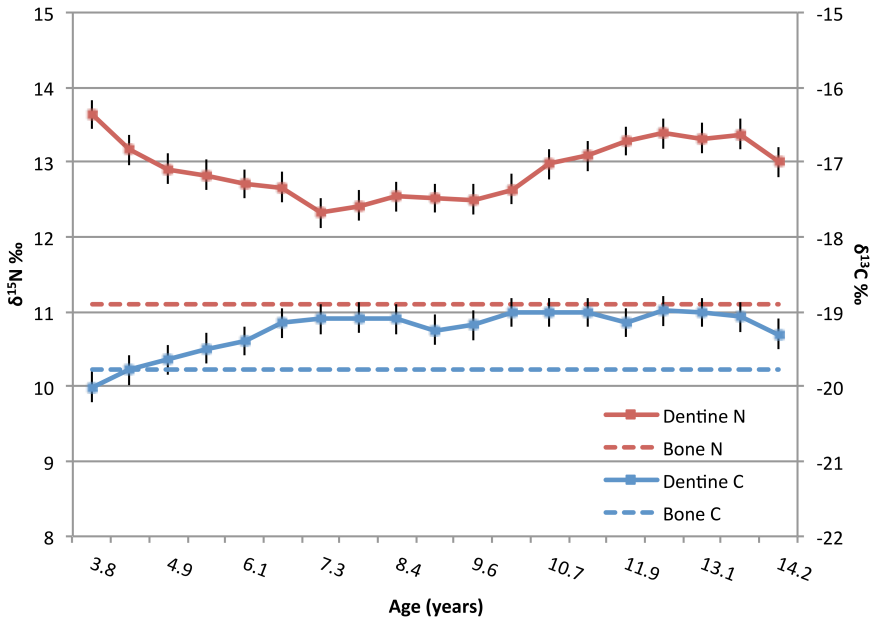


Figure 5.13: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-32302 (young adult female)

14.5 years old, the individual shows steady isotope values (average dentine $\delta^{15}N$ = 12.91‰, $\delta^{13}C$ = -19.25‰) that are within the average range for the cemetery. However, bone collagen results show a diet prior to death with a 1.81‰ decrease in $\delta^{15}N$ values (i.e. most likely from less protein in the diet). Given that the individual is estimated to have died around the age of 20 and that bone collagen values generally reflect the last 10 years of life, the change in diet was drastic and must have occurred just after 14.5 years of age. It is possible that this individual migrated to London around the age of 15, which was the common age to travel into urban areas in search of work (Hanawalt, 1995). The individual may have experienced famine when entering the city, and then died approximately 5 years later, as the low bone collagen values are consistent with the consumption of less protein and may be a reflection of inadequate access to animal protein or fish.

In addition to famine burials, there are also individuals interred in attritional burials with patterns that may suggest an origin outside of London. SRP98-11471 displays a substantial difference in $\delta^{15}N$ and $\delta^{13}C$ values between dentine collagen and bone collagen ($\delta^{15}N$ = 3.39‰, $\delta^{13}C$ = 0.86‰). Unlike the dentine collagen values, isotope values from bone collagen are within the average for the cemetery. $\delta^{15}N$ values begin to increase around the age of 9 until approximately 14 years old (Figure 5.14). Bone collagen results are higher than the oldest dentine collagen value, suggesting that the increase in $\delta^{15}N$ must have persisted just prior to death (age 25). However, it Is important to note that this individual is observed as having scoliosis, which may have had an affect on mobility or possibility even consumption practices during life. SRP98-

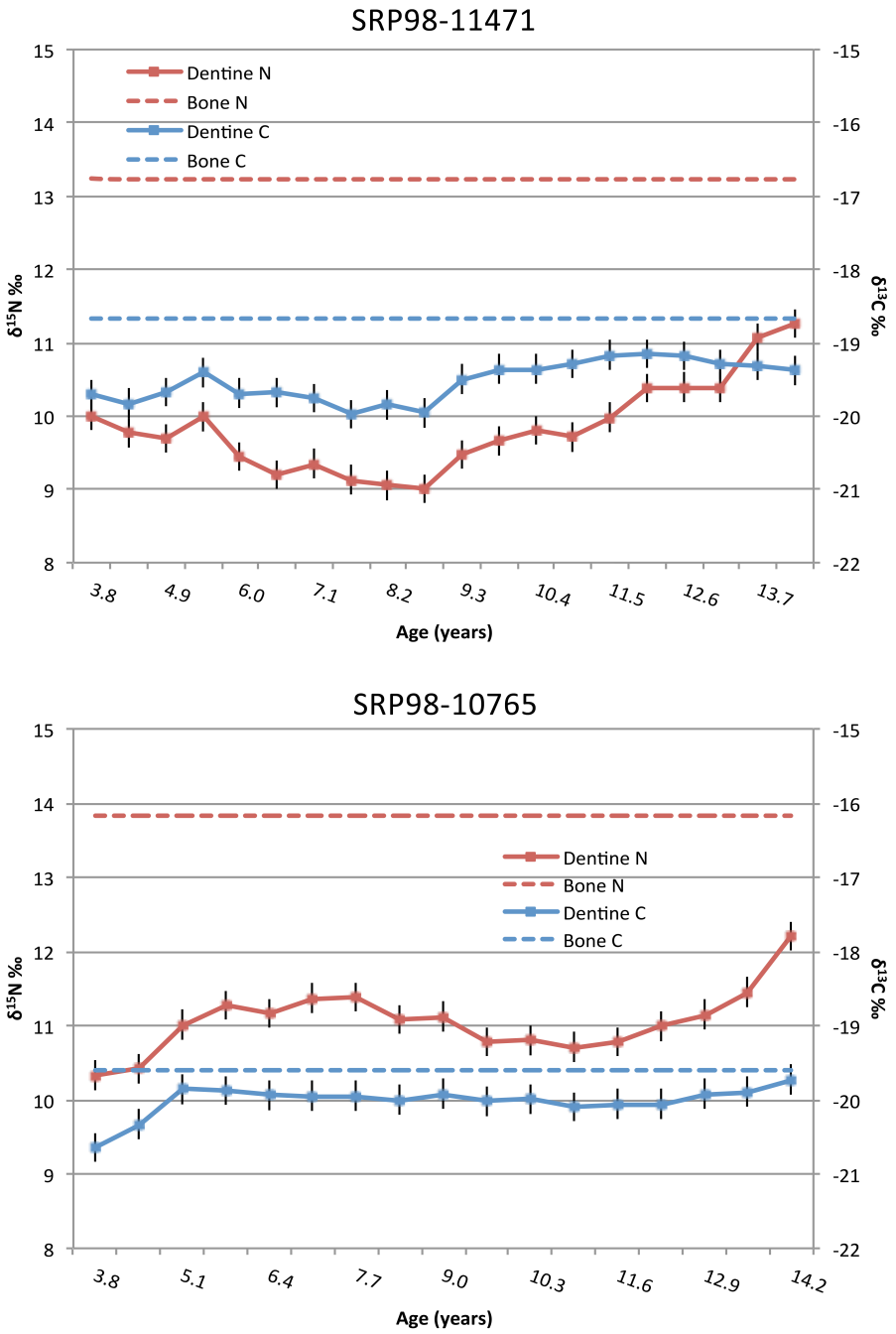


Figure 5.14: Plotted dentine and bone collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-11471 (top- adult female) and SRP98-10765 (bottom- adult female)

10765 also exhibits a considerable difference in $\delta^{15}N$ and $\delta^{13}C$ between bone collagen and average dentine collagen ($\delta^{15}N$ = 2.77‰, $\delta^{13}C$ = 0.62‰). The bone collagen $\delta^{15}N$ value, however, is outside the average for the cemetery, suggesting that this individual ate more fish than the average Londoner or, possibly, that this individual did not reside in London long enough for the change in diet to be captured in bone collagen. Additionally, this individual is noted as have boney lesions indicative of tertiary syphilis, which may have influenced the bone collagen isotope values.

The incorporation of different tissues for stable isotopic analysis can be informative about dietary changes and mobility through the life course. Specifically, the comparison of bone collagen with dentine collagen allows the identification of potential migrants in the cemetery. The higher proportion of potential "migrants" in famine burials compared to attritional burials is consistent with the preceding argument that more migrants were interred in famine burials. Lakin (2010) also used carbon and nitrogen isotope values to identify potential migrants in her isotopic study of SRP98, though only bone collagen is used. She isolated individuals with the most negative $\delta^{13}C$ and $\delta^{15}N$ values and attempted to confirm that they were actually migrants through strontium and oxygen isotope analysis, determining that at least one of the individuals may be a migrant.

It is important to note that the individuals for whom isotopic dentine profiles in this section are discussed are used only as examples of unique dentine isotopic profiles, and are not meant to serve as comprehensive case studies of individuals in SRP98.

5.5 Conclusion

Results of this study may be more reflective of differences in diet or mobility between burial types rather than nutritional stress. Analyses of bone collagen comparing $\delta^{13}C$ and $\delta^{15}N$ values between attritional and famine burial types indicate that individuals interred in attritional graves exhibit significantly elevated $\delta^{15}N$ values compared to individuals in famine graves, with no significant difference in $\delta^{13}C$. Given that it is expected for famine burials, rather than attritional burials, would exhibit enriched $\delta^{15}N$, these results may actually reflect differences in diet that occurred as a consequence of famine (e.g. less protein in the diet during famine because of limited access to food supplies) or migration (e.g. isotope values from a migrant's origin captured in bone). Sex-specific results for bone collagen reveal that females in attritional burials exhibit higher nitrogen values compared to females in famine burials. Males, however, did not show a difference in isotope values between burial types. The difference between burial types for females may be an artifact of migration as well, as a higher proportion of female migrants may have been interred within famine burials, compared to males.

Analyses of incremental dentine collagen profiles reveal that individuals from famine burials exhibit more variable $\delta^{13}C$ and $\delta^{15}N$ values during childhood compared to attritional values, though no specific pattern unique to famine burials is identified. Similar to the bone samples, changes in diet as a result of famine and the abundance of potential migrants in the population may be obscuring elevated nutritional values often associated with metabolic stress. In Beaumont and Montgomery's (2016) study, even

when the timing of famine is well-documented and incremental dentine from mineralizing teeth is used, evidence of enriched nitrogen during starvation was still not obviously exhibited in the samples. They deduced that the duration of famine paired with the averaging of the dentine sections representing weeks or months result in only a minor rise in nitrogen values that may not be identifiable. However, some individuals in this sample exhibit patterns that have been identified in previous incremental analyses of nitrogen and carbon in the context of undernutrition. Specifically, some individuals from famine burials exhibit an opposing covariance pattern of $\delta^{13}C$ and $\delta^{15}N$ that suggests the individual may have experienced famine during childhood.

Additionally, results from the rib bone collagen may not reflect nutritional stress experienced just prior to death because of slow tissue turnover rates. Estimates for isotope values from bone collagen range from the last 10 (Valentin, 2002) to 30 years (Hedges et al., 2007) prior to death, unless chronically affected by famine throughout life, food deprivation experienced only years or months prior to death, which would also increase an individual's risk of dying, may not be captured in bone collagen. Thus, individuals who died quickly as a result of famine or who only experienced famine for a short time may not exhibit high nitrogen values in bone collagen. Compared to bone tissue, teeth are more informative about temporal fluctuations in isotope values, as tooth mineralization occurs at known ages in life (AlQahtani et al., 2010; Hillson, 2005), but are limited to the lifespan before adulthood. Thus, tooth collagen is generally not informative about nutritional stress or dietary changes occurring just prior to death unless the individual died during childhood or adolescence. However, incremental

dentine may be useful for identifying dietary patterns or nutritional stress occurring during childhood that could have increased individual's risk of death during famine.

Though patterns of undernutrition could not be definitively identified using bone and dentine collagen, the integration of these data may be informative of potential migrants in the sample. Several individuals, more from famine burials, exhibit dentine collagen values (reflecting childhood diet) that are substantially different than bone collagen values (reflecting at least 10 years of diet prior to death). Though these individuals may have resided in a location that was different than their origin, in this case London, further isotopic analysis (e.g. strontium, oxygen, and lead (Montgomery et al., 2010) is necessary for confirmation.

Finally, this project underlines the importance of integrating multiple tissues and lines of evidence in stable isotope analyses in the context of famine. Using different tissues can tell us about diet throughout the life course, rather than limiting analyses to broad averages of diet prior to death (Beaumont 2013a; 2013b). For example, by comparing bone and tooth collagen samples, it is possible to identify potential migrants within SRP98. Further, by including pathological information, such as enamel hypoplasias, in stable isotope analyses, it may be possible to clarify patterns of fluctuating isotope values. In this project, for example, by including the age at which enamel hypoplasias form with isotope values changes, fluctuations in isotope values could be attributed to potential undernutrition rather than dietary change. Further, integrating pathological data and isotope values could also be informative of the relationship between frailty and diet (i.e. if individuals who died from famine exhibit

fluctuations in isotope patterns during childhood related to physiological stress), which could contribute to current research evaluating frailty and famine mortality (e.g. Yaussy et al., 2016).

Chapter 6 - Conclusion

This research project incorporates multiple lines of evidence and several analytical methods to present a nuanced depiction of health and nutrition in urbanizing Late Medieval London. According to historical and archaeological sources, the rapidly increasing population density during this period resulted in elevated risk of disease and poor sanitary conditions. Additionally, recurring poor harvests caused several instances of famine during this time that contributed to fluctuations in food sources and subsequent poor health. These environmental factors compounded with constant human mobility during this period created a complex environment in which to assess health and mortality, requiring the application of analytical approaches that could accommodate multiple interacting factors and the integration of different lines of evidence. Specifically, the application of paleodemographic and biochemical data was used to reconstruct a comprehensive picture of diet and health during urbanization, and contributed to the evaluation of potential inequities for sub-populations. In Chapters 2 and 3, paleodemographic data from skeletal remains were used to assess variability in mortality and survivability both within and between urbanizing and non-urbanizing English cemeteries, including an analysis of age groups and the sexes; and in Chapters 4 and 5, biochemical data, specifically stable isotope values, were used to assess dietary variation through time and to evaluate potential isotopic markers of famine in an

urbanizing medieval London cemetery.

In Chapter 2, the assumption that urbanization was linearly detrimental for Londoners was tested by assessing temporal changes in survivability in St Mary Spital cemetery. Adult survivability was shown to increase as urbanization intensified, was attributed to Londoners adapting to detrimental environmental conditions by improving their surroundings through policy implementation. Paleodemographic approaches were also used in Chapter 3 to assess the effect of urbanization on health by comparing survivability and mortality between urbanizing London and an English cemetery not undergoing urbanization. Results showed that there were elevated risks of mortality and reductions in survivorship for adults in the urban environment compared to the rural environment, suggesting that deleterious conditions associated with urbanization in London were hazardous for adults.

In addition to survivability and mortality patterns assessed in Chapters 2 and 3, dietary patterns in St Mary Spital were also evaluated. Diet is one the most important mediators of health, making the investigation of diet a necessary component in the understanding of health dynamics. Inadequate diet and undernutrition, often experienced by individuals in urbanizing areas, weakens the immune system, making the body more susceptible to disease (Scrimshaw et al., 1968). In Chapter 4, stable isotope values of Londoners were evaluated to investigate dietary patterns in the urban environment, including separate analysis of temporal periods, age cohorts and the sexes. Results indicate that the diet for St Mary Spital is consistent with other Medieval

English sites, but demonstrates more variability in protein sources, and that isotope values varied both through time and by age cohort.

The effect of famine, a frequent occurrence during the Late Medieval Period, was also included in stable isotopic analysis of St Mary Spital. In Chapter 5, an assessment of how nutritional stress could be exhibited biochemically in human tissue was conducted by comparing isotope values of individuals from non-famine and famine periods. Results were contrary to what was hypothesized, with significantly lower isotope values in the famine-related burials. These results may reflect differences in diet that occurred as a consequence of famine (e.g. less protein in the diet during famine because of limited access to food supplies) or migration (e.g. isotope values from a migrant's origin captured in bone tissue), rather than differences in physiological stress. Additionally, isotope values from incremental dentine collagen were used to evaluate potential nutritional stress at the level of the individual. Some individuals in the sample exhibited patterns that have been previously identified in incremental analyses of nitrogen and carbon in the context of undernutrition, which may be supported by pathological data, and suggesting they may have experienced undernutrition.

6.1 Tissue type and isotope analysis

Though stable isotope analyses in this project are informative about dietary changes within subgroups and through time, there were some limitations. Particularly for bone collagen, estimates for isotope values from rib tissue ranges from the last 4 to 10 years prior to death (Valentin, 2002). Individuals who died quickly as a result of

famine or who only experienced famine for a short time may not exhibit this event isotopically in bone collagen. Thus results from the rib bone collagen samples in SRP98 may not reflect nutritional stress experienced just prior to death because of slow tissue turnover rates. Additionally, dentine collagen is generally not informative about nutritional stress or dietary changes occurring just prior to death unless the individual died during or soon after tooth mineralization (i.e. during childhood or adolescence). Incremental dentine isotope data, however, were useful for identifying potential dietary changes or nutritional stress occurring during childhood (see Chapter 5). Though definitive patterns of undernutrition could not be identified using bone or dentine collagen, the integration of these data is informative about potential migrants in the sample. Several individuals, more from famine burials, exhibit dentine collagen values (reflecting childhood diet) that are substantially different than the individual's bone collagen values (reflecting at least 10 years of diet prior to death). Differences in isotope values between these tissues suggests that these individuals may have resided in a location that was different than their origin, in this case London. Further isotopic analysis (e.g. strontium, oxygen, and lead (Montgomery et al., 2010), however, is necessary for confirmation.

In addition to using collagen from different tissues, it is also useful to include other types of data (e.g. paleodemographic and paleopathological data) when analyzing stable isotope patterns. For example, in Chapter 5, pathological information (age of enamel hypoplasia formation) for individuals was compared to patterns in incremental

dentine collagen profiles to assess whether fluctuations in isotope values could have been the result of dietary change or of physiological stress.

6.2 Sub-populations and the individual

Rather than focusing on mortality and dietary changes in cemeteries as a whole, this project assessed variation within and between sub-populations, including analyses of the sexes and different age groups. Primary documents generally focus on those who paid taxes or those with high socioeconomic status, resulting in the exclusion of subadults, the poor, migrants, and often females (Hinde, 2003). Skeletal data used in this project, however, includes these subgroups, and thus provides a more accurate depiction the population in urbanizing London compared to parish registers or tax documentation. Moreover, these subgroups were potentially affected by, and adapted to the effects of urbanization differently. To more adequately address the complex relationship between urbanization and health, it is necessary use methods that allow for intra-population variation in patterns of mortality and that are capable of revealing whether urbanization sub-populations were disproportionally affected.

Chapters 2 and 3 identified differences in mortality and survivability when sub-populations (the sexes and different ages groups) were analyzed separately. In Chapter 3, the inconsistency in patterns of survival estimated for adults *vs.* subadults suggests heterogeneity across the lifespan, a difference that would have been masked if all ages were pooled for survival analysis. Also in Chapter 3, the elevated mortality and lower survival for urban females compared to rural females became apparent once the sexes

were analyzed separately, and may have contributed to the overall mortality differences of adults between the two cemeteries.

6.3 Influence of migration

Urban centers like London attracted the poor, who were presumably more vulnerable to infections and exhibited high death rates compared to their rural counterparts because of the effect of high population density (Singman, 1999). Rural-to-urban migration is one of the most common types of human movement in all periods of recorded history (Boyce, 1984), allowing cities with high mortality rates to sustain high population densities (McNeill, 1980; Wrigley, 1969). Migrants made up a large proportion of the population in London, but are often invisible from primary historical documents. Migrants to urban environments might have faced elevated risks of infection with diseases they did not encounter during their childhoods in rural areas, or may have been exposed to disease during the migration process (Prothero, 1977). Thus, hazardous conditions experienced by rural migrants to London may have contributed to the elevated risk of urban adult mortality in SRP98 observed in Chapter 3.

Separate analyses for females and subadults in this project generally display a unique mortality or isotopic trend that can be attributed to rural-to-urban migration. The unchanging female survivability through time in Chapter 2 may be a reflection of more young females coming to urban centers seeking better labor opportunities, particularly after the Black Death. These females may have suffered from poverty, famine, and exposure to pathogens during immigration or upon their arrival in the city,

all of which could have increased their risk of mortality. This interpretation would complement the interpretation in Chapter 3 that the elevated mortality and reduced survivability in urban females compared to rural females may have been due to a higher proportion of rural to urban migration of females looking for labor opportunities.

In Late Medieval England, it was common for adolescents to travel from rural areas or small towns seeking apprenticeships and serving positions; children as young as seven could be hired but adolescents hired between twelve and fifteen years of age was more common (Hanawalt, 1995). Analysis of subadults in Chapter 2 revealed a decrease in survivability through time in rural subadults, and this is attributed to the increase in rural-to-urban migration of rural adolescents to urban areas seeking apprenticeships or servant positions.

In addition to paleodemographic analyses of St Mary Spital, migration also affected analyses using stable isotope data in Chapters 4 and 5. Typically, a migrant will exhibit stable isotope values unique to their place of origin in the individual's bone tissue. These values are averaged in with their diet prior to death as bone remodels. Depending on how long the individual was consuming the London diet (i.e. how long he or she lived in London prior to death), stable isotope values from bone collagen reflect of a combination of stable isotope values characteristic of the individual's origin *and* London. This phenomenon makes it difficult to assess isotope values unique to London. Additionally, in Chapter 5, rural-to-urban migration could have influenced isotopic differences between burial types because migrants were more likely to be interred in famine burials. Comparing dentine and bone collagen and including paleodemographic

data from skeletal remains allowed the identification of potential migrants in the sample.

More research regarding migration in Medieval England is necessary to clarify the effects of migration on changes in mortality, particularly because mortality is often used as a proxy for health. Future studies using stable isotope analysis to identify migrants in urban centers can offer valuable information regarding where migrants were coming from, who these migrants were (e.g. young females), and how these migrants experienced and adapted to the environmental factors characteristic of urbanism. The integration of stable isotope analyses such as those conducted in Chapters 4 and 5, with paleodemography is essential for disentangling the complicated links between mortality and migration to better understand medieval life in the face of a changing climate. Stable isotope analysis using a combination of oxygen, strontium, and lead, can potentially identify migrants within English skeletal assemblages (Montgomery et al., 2010), and thus contribute to information regarding who was immigrating and where they were coming from.

6.4 The Black Death

In addition to migration, the effects of the Black Death played a large role in the interpretations in this project. After the Black Death, there was a substantial decrease in population density, allowing market expansion that resulted in a higher standard of living and subsequent improved health (Bridbury, 1973). Previous analyses of pre- and post-Black Death English cemeteries suggest increased survivorship following the

epidemic (DeWitte, 2014). These findings complement the results found in Chapter 2, with an increase in adult survivability in London from the Early to Late Phases. This adult survivability pattern is not evident in the rural cemetery analyzed for this project, even though the village was likely affected by the plague. Thus, the lack of increased survivability for adults in the rural environment suggests that there are other factors to consider in addition to the improved living conditions after the Black Death that contributed to the increased survivability for urban adults.

Additionally, it is possible that the elevated risk of dying and reductions in survivorship for adults in the urban environment seen in Chapter 3 might reflect the effects of the Black Death. Though all of England experienced outbreaks of plague, it is possible that it might have been more detrimental in urban areas. There are, however, conflicting views as to whether towns or rural villages experienced plague differently. Parish records indicate that later plague epidemics reached Barton-upon-Humber and caused more deaths compared to periods without plague, suggesting that plague mortality might not explain the urban *vs.* rural mortality and survival differences observed in this project.

In Chapter 4, the changing food market that occurred after the Black Death (Galloway and Murphy, 1991) may be reflected in changing isotope values between temporal periods. After Period 17, the period after the Black Death, there is a reduction in correlation between $\delta^{13}C$ and $\delta^{15}N$, which is indicative of a decrease in variation of food sources. This can be attributed to the food trade reverting back to more varied food sources exhibited in earlier periods of urbanization because of an unstable market.

Finally, in Chapter, 5, to control for the effect of the Black Death in the comparison of isotope values in attritional and mass burials, separate analyses were performed that excluded the period in which the Black Death occurred (Period 16). Differences between analyses included and excluding this period, but the results were not substantially different.

6.5 Contributions

This project contributes a unique osteological line of evidence to the investigation of health and nutrition in the context of urbanization. Most evidence of the medieval London environment comes from primary sources such as court records, complaints, ordinances, or decrees to abate pollution. Environmental archaeology has also contributed to our understanding of urbanization in Late Medieval England, specifically the effects of pollution and urban growth on the landscape. Bioarchaeological research, however, contributes to our understanding of how the environment may have affected people. By examining the skeletal remains of individuals who experienced urbanization, bioarchaeologists can elucidate patterns of health and mortality associated with urbanism. Analysis of skeletal assemblages of people exposed to urban environmental factors, such as high population density, potentially elevated risk of infection, and unsanitary living conditions can provide unique insights into the effects of urbanization on health and mortality in the past; particularly, when compared to assemblages of people unexposed or less severely exposed to these factors.

Further, results from this project challenge the assumption that urbanization is a linearly detrimental process resulting in declining health as it progresses. Humans constantly adapt to unfavorable environment conditions around them (Nelson et al., 2007), and this is consistent with increasing adult survivability as urbanization intensified in London seen in Chapter 2. The increase in adult survivability through time in St Mary Spital can be attributed to the increase in sanitation and pollution policies evident in historical and primary sources. These results also support current literature that challenges the assumption that medieval Londoners were indifferent to the environmental conditions around them, and argues that Londoners were actually bettering their health by improving their living conditions.

Additionally, this project used innovative analytical methods (e.g. hazard analysis, transition analysis, and incremental dentine isotope analysis) to an investigation of urbanization in the past. Most paleodemographic studies of urbanization have assessed mortality using traditional approaches, such as comparisons of mean age-at-death or life tables (Nagaoka and Hirata, 2007; Steckel, 2005; Storey, 1985). The reconstruction of the health consequences from urbanization in the past is inherently complex and is limited by small sample sizes and traditional methods, given the phenomena of demographic non-stationarity, hidden heterogeneity and selective mortality (Konigsberg and Frankenberg, 2002; Vaupel and Yashin, 1985; Wood et al., 1992). The quantitative models applied in this project allowed selective mortality and heterogeneity in frailty to be accounted for by analyzing different groups within the cemeteries. Moreover, the statistical approaches used in this study, hazard and survival

analysis, accommodates missing data without imposing a particular age pattern on skeletal data (Gage, 1988), providing a more accurate depiction of mortality for the cemeteries. Finally, transition analysis, the age estimation technique used for this study, avoids limitations associated with traditional age estimation methods by using some of the criteria articulated in the Rostock Manifesto.

Additionally, the effect of changes in fertility in skeletal assemblages spanning long periods of time was also taken into account when comparing the rural and urban cemeteries in Chapter 3. A population experiencing an increase in fertility, increasing numbers of children will be born each year, thus increasing the number of children who die each year, even if age-specific mortality rates do not change. Under these circumstances, the resulting cemetery assemblage from this growing population will contain an excess of young individuals relative to older individuals, making it difficult to infer mortality patterns directly from age-at-death data (Milner et al., 2007) and complicates the comparison of two different, contemporaneous assemblages that are potentially derived from populations with different fertility rates. A fertility proxy was used to show that the demographic differences between the two cemeteries were not an artifact of changes in birth rates, allowing the mortality patterns of the cemeteries to be compared.

Finally, stable isotope data from this project and from other urban English cemeteries contribute to the construction of a dietary landscape in England during urbanization in the Late Medieval Period, and the analyses of stable isotope data from

BOH would complement the paleodemographic comparative analyses between SRP98 and BOH in this project.

6.6 Future directions

Future analyses will incorporate the use of paleopathological data originally collected for this project to evaluate temporal changes in frailty during urbanization and to compare potential frailty and selective morality in the urban and non-urban cemeteries. In addition, the elevated mortality exhibited by females in this project will be further explored; specifically, how migration could have influenced mortality patterns for females.

Methods used in this study that incorporate a temporal element to changes in health during transitional periods could be applied to industrialization in Post-Medieval England and other geographic areas. Industrialization was different than urbanization in that it produced enough wealth to substantially improve dietary nutrition and public sanitation (McNeill, 1976; Wrigley, 1969). Studies of subadults in urbanizing and industrializing environments, however, suggest that the urban environmental conditions may not have significantly affected subadults until industrialization when these factors would have intensified (Lewis, 2003).

The inclusion of additional data from London cemeteries would provide a more accurate depiction of health patterns in London during urbanization. Preservation issues in St Mary Spital, particularly for subadults (see Chapters 2 and 3) may have obscured actual mortality and survivability patterns for this group. In addition to London, future

bioarchaeological analyses of urbanization in England should consider urbanizing English towns to gain a better understanding of how England experienced urbanization as a whole prior to the Industrial Revolution. London served as a model for urbanizing areas in England (Rawcliffe, 2013), and strategies for improving living conditions during urbanization may be evident in English towns. Also, the inclusion of skeletal data from other English non-urban cemeteries would be useful to determine the degree to which urban living detrimentally affected health.

References

Adamson MW. 2004. Food in Medieval Times. Westport, Connecticut: Greenwood Publishing Group.

Albarella U. 1999. The Mystery of husbandry': medieval animals and the problem of integrating historical and. Antiquity 73(282):867-875.

Albarella U. 2005. Meat production and consumption in town and country. In: Giles K, and Dyer C, editors. Town and Country in the Middle Ages: Contrasts, Contacts and Interconnections, 1100-1500. Leeds: Maney Publishing. p 131-148.

AlQahtani SJ, Hector MP, and Liversidge HM. 2010. Brief Communication: The London Atlas of Human Tooth Development and Eruption. Am J of Phys Anthrop 142:481-190.

Ambrose SH. 1990. Preparation and characterization of bone and tooth collagen for isotopic analysis. J Archaeol Sci 17(4):431-451.

Ambrose SH. 1991. Effects of diet, climate and physiology on nitrogen isotope abundances in terrestrial foodwebs. J Archaeol Sci 18(3):293-317.

Ambrose SH, Butler BM, Hanson DB, Hunter-Anderson RL, and Krueger HW. 1997. Stable isotopic analysis of human diet in the Marianas Archipelago, Western Pacific. Amer J Phys Anthrop 104(3):343-361.

Ambrose SH, and DeNiro MJ. 1986. The isotopic ecology of East African mammals. Oecologia 69(3):395-406.

Ambrose SH, and Norr L. 1993. Experimental evidence for the relationship of the carbon isotope ratios of whole diet and dietary protein to those of bone collagen and carbonate. In: Lambert JB, and Grupe G, editors. Prehistoric Human Bone. Berlin: Springer-Verlag. p 1-37.

Andrews G, and Thomas R. 1995. The Management of Archaeological Projects. In: Carman J, Cooper M, Firth A, and Wheatley D, editors. Managing Archaeology. London, UK: Routledge. p 184-202.

Armelagos GJ, and Cohen MN. 1984. Paleopathology at the origins of agriculture. Orlando, FL: Academic Press.

Baker P. 1984. Migrations, genetics, and the degenerative diseases of South Pacific Islanders. In: Boyce AJ, editor. Migration and Mobility. London: Taylor and Francis. p 209-239.

Barrett JH, Locker AM, and Roberts CM. 2004a. 'Dark Age Economics' revisited: the English fish bone evidence AD 600 - 1600. Antiquity 78:618-636.

Barrett JH, Locker AM, and Roberts CM. 2004b. The origins of intensive marine fishing in medieval Europe: the English evidence. Proceedings of the Royal Society Biological Sciences 271:2417-2421.

Barron C. 1989. The 'golden age' of women in medieval London. Reading Medieval Studies 15:35-58.

Barron C. 2000. London, 1300-1540. In: Palliser DM, editor. The Cambridge Urban History of Britain. Cambridge. p 395-440.

Bayliss A, and Atkins C. 2011. Specialist Studies, 1: Interpreting Chronology. In: Rodwell W, and Atkins C, editors. St Peter's Barton-upon-Humber, Lincolnshire: A Parish Church and its Community, History, Archaeology, and Architecture Part 2. Oxford: Oxbow Books. p 753-788.

Beaumont J, Geber J, Powers N, Wilson A, Lee-Thorp J, and Montgomery J. 2013a. Victims and survivors: stable isotopes used to identify migrants from the Great Irish Famine to 19th century London. Amer J Phys Anthrop 150(1):87-98.

Beaumont J, Gledhill A, Lee-Thorp J, and Montgomery J. 2013b. Childhood diet: A closer examination of the evidence from dental tissues using stable isotope analysis of incremental human dentine. Archaeometry 55(2):277-295.

Beaumont J, and Montgomery J. 2015. Oral histories: a simple method of assigning chronological age to isotopic values from human dentine collagen. Ann Hum Biol 42(4):405-412.

Beaumont J, and Montgomery J. 2016. The Great Irish Famine: Identifying Starvation in the Tissues of Victims Using Stable Isotope Analysis of Bone and Incremental Dentine Collagen. PLoS ONE 11(8):1-21.

Bekvalac J, and Kausmally T. 2011. Human Bone. In: Grainger I, and Phillpotts C, editors. Excavations at the Abbey of St Mary Graces, East Smithfield, London Museum of London Archaeology.

Bello SM, Thomann A, Signoli M, Dutour O, and Andrews P. 2006. Age and sex bias in the reconstruction of past population structures. Amer J Phys Anthrop 129(1):24-38.

Benedictow Ø. 2005. The Black Death: The Greatest Catastrophe ever. History Today 55(3):42-49.

Bennett HS. 1937. Life on the English Manor: A Study of Peasant Conditions, 1150-1400. Cambridge: Cambridge University Press.

Beresford MW, and Hurst JG. 1991. Wharram Percy: Yale University Press.

Bitel L. 2002. Women in Early Medieval Europe 400–1100. Cambridge: Cambridge University Press.

Bocquet-Appel J-P, and Masset C. 1982. Farewell to paleodemography. J Hum Evol 11(4):321-333.

Bogin B. 1999. Patterns of Human Growth. Cambridge: Cambridge University Press.

Boldsen JL, Milner GR, Konigsberg LW, and Wood JW. 2002. Transition analysis: a new method for estimating age from skeletons. In: Hoppa R, and Vaupel JW, editors. Paleodemography: age distributions from skeletal samples. Cambridge: Cambridge University Press. p 73-106.

Bonfield C. 2009. Medical Advice and Public Health: Contextualising the Supply and Regulation of Water in Medieval London and King's Lynn. Poetica 72:1-20.

Boocock P, Roberts CA, and Manchester K. 1995. Maxillary sinusitis in medieval Chichester, England. Amer J Phys Anthrop 98(4):483-495.

Boyce AJ. 1984. Migration and Mobility: Biosocial aspects of human movement.

Boyd P. 1981. The paleoecology of estuarine deposits associated with archaeological sites, with particular reference to the City of London. In: Brothwell D, and Dimbleby G, editors. Environmental Aspects of Coasts and Islands: BAR. p 87-88.

Brenan J. 1994. PPG 16 and the restructuring of archaeological practice in Britain. Planning Practice and Research 9(4):395-405.

Brickley M, and Ives R. 2010. The bioarchaeology of metabolic bone disease. New York: Academic Press.

Bridbury AR. 1973. The Black Death. Econ Hist Rev 26(4):577-592.

Brimblecombe P. 1976. Attitudes and responses towards air pollution in medieval England. Journal of the Air Pollution Control Association 26(10):941-945.

Britnell R. 1994. The Black Death in English Towns. Urban History 21(2):195-210.

Brooks ST. 1955. Skeletal age at death: the reliability of cranial and pubic age indicators. Amer J Phys Anthrop 13(4):567-597.

Brothwell D. 1994. On the possibility of urban-rural contrasts in human population paleobiology. In: Hall A, and Kenward H, editors. Urban-rural Connexions: Perspectives from environmental Archaeology. Oxford: Oxbow Books. p 129-136.

Brown TA, Nelson DE, Vogel JS, and Southon JR. 1988. Improved collagen extraction by modified Longin method. Radiocarbon 30(2):171-177.

Bryant G. 2003. The Later History of Barton-on-Humber. Part One. The Church in Late Medieval Barton-on-Humber. Gainesborough: Workers' Educational Association.

Buckberry J. 2000. Missing, Presumed Buried? Bone Diagenesis and the Under-Representation of. Assemblage: University of Sheffield Graduate Student Journal of Archaeology.

Buikstra JE, Konigsberg LW, and Bullington J. 1986. Fertility and the development of agriculture in the prehistoric Midwest. Amer Antiq:528-546.

Buikstra JE, and Ubelaker DH. 1994. Standards for data collection from human skeletal remains. Fayetteville, Arkansas: Arkansas Archaeological Society.

Buikstra JE, and Beck LA. 2006. Bioarchaeology: The Contextual Analysis of Human Remains. Waltham, MA: Academic Press.

Bullock M, Márquez L, Hernández P, and Ruíz F. 2013. Paleodemographic age-at-death distributions of two Mexican skeletal collections: A comparison of transition analysis and traditional aging methods. Amer J Phys Anthrop 152(1):67-78.

Busvine JR. 1976. Insects, Hygiene and History. London: Athlone Press.

Calendar of Close Rolls -. 1892-1963. London.

Carlin M. 1998. Fast food and urban living standards in medieval England. In: Rosenthal JT, editor. Food and Eating in Medieval Europe. London: Hambledon Press. p 27-52.

Carlin M. 2008. "What say you to a piece of beef and mustard?": The Evolution of Public Dining in Medieval and Tudor London. Huntington Library Quarterly 71:199-217.

Castex D, and Kacki S. 2016. Demographic Patterns Distinctive of Epidemic Cemeteries in Archaeological Samples. Microbiology spectrum 4(4):1-9.

Chadwick P. 1991. Competitive tendering in archaeology: the curator's role. In: Swain H, editor. SCAUM/RESCUE. Hertford. p 7-11.

Chalke HD. 1962. The impact of tuberculosis on history, literature, and art. Medieval History 6:301-318.

Chew HM, and Kellaway W. 1973. London assize of nuisance, 1301-1431: a calendar: London Record Society.

Clapson R. 2005. The Later History of Barton-upon-Humber: Part Four. Barton and the River Humber 1086-1900. Belton, Gainesborough: Worker's Education Association.

Cohen MN. 1989. Health and the rise of civilization. New Haven, CT: Yale University Press.

Connell B, Gray Jones A, Redfern R, and Walker D. 2012. A bioarchaeological study of medieval burials on the site of St. Mary Spital: excavations at Spitalfields Market, London E1:1991-2007. London: Museum of London Archaeology.

Cowal L, Grainger I, Hawkins D, and Mikulski R. 2008. The Black Death Cemetery, East Smithfield. London.

Cox PR. 1972. Life Tables: John Wiley & Son Inc.

Craig OE, Biazzo M, O'Connell TC, Garnsey P, Martinez-Labarga C, Lelli R, Salvadei L, Tartaglia G, Nava A, and Renò L. 2009. Stable isotopic evidence for diet at the Imperial Roman coastal site of Velia (1st and 2nd Centuries AD) in Southern Italy. Amer J Phys Anthrop 139(4):572-583.

Cummins N, Kelly M, and Ó Gráda C. 2015. Living standards and plague in London, 1560-1665. The Econ Hist Rev 69:3-34.

Darvill T, and Russell B. 2002. Archaeology after PPG16: archaeological investigations in England 1990 - 1999. London: Bournemouth University School of Conservation Sciences.

Davenport R, Boulton J, and Schwarcz L. 2010. Infant and young adult mortality in London's West End, 1750-1824. Working Paper Newcastle University: The Pauper Lives Project.

Dawes JD. 1980. The human bones. In: Dawes JD, and Magilton J, editors. The cemetery of St Helen-on-the-walls, Aldwark: the archaeology of York. London: Council for British Archaeology. p 19-120.

De La Rúa C, Izagirre N, and Manzano C. 1995. Environmental stress in a Medieval population of the Basque Country. Homo 45(3):268-289.

Dean MC, and Scandrett AE. 1995. Rates of dentine mineralization in permanent human teeth. Intenat J Osteoarch 5:349-358.

DeNiro MJ. 1985. Postmortem preservation and alteration of in vivo bone collagen isotope ratios in relation to palaeodietary reconstruction.

DeNiro MJ. 1987. Stable isotopy and archaeology. American Scientist:182-191.

DeNiro MJ, and Epstein S. 1978. Influence of diet on the distribution of carbon isotopes in animals. Geochimica et cosmochimica acta 42(5):495-506.

DeNiro MJ, and Epstein S. 1981. Influence of diet on the distribution of nitrogen isotopes in animals. Geochimica et Cosmochimica Acta 45(3):341-351.

DeNiro MJ, and Schoeniger MJ. 1983. Stable carbon and nitrogen isotope ratios of bone collagen: variations within individuals, between sexes, and within populations raised on monotonous diets. J Archaeol Sci 10(3):199-203.

DeWitte S. 2009. The Effect of Sex on Risk of Mortality During the Black Death in London, A.D. 1349–1350. Am J Phys Anthropol 139:222-234.

DeWitte S. 2014. Mortality Risk and Survival in the Aftermath of the Medieval Black Death. PLoS ONE 9(5):e96513.

DeWitte S, and Stojanowski CM. 2015. The osteological paradox 20 years later: past perspectives, future directions. J Archaeol Res 23(4):397-450.

DeWitte S, and Yaussy S. 2015. Famine and Frailty: Crisis Mortality and Stature in Medieval London. American Association of Physical Anthropologists Meeting. Atlanta, GA.

DeWitte SN. 2010. Age patterns of mortality during the Black Death in London, AD 1349–1350. J Archaeol Sci 37(12):3394-3400.

DeWitte SN, and Wood JW. 2008. Selectivity of Black Death mortality with respect to preexisting health. PNAS 105(5):1436-1441.

DoE. 1990. Planning Policy Guidance 16: Archaeology and Planning. London, UK: Her Majesty's Stationary Office.

DoE. 2010. Planning Policy Statement 5: Planning for the Historic Environment. London, UK: Her Majesty's Stationary Office.

DoE. 2012. National Planning Policy Framework. London, UK: Her Majesty's Stationary Office.

Duby G, and Postan C. 1998. Rural economy and country life in the medieval West: University Of Pennsylvania Press.

Duncan S, Scott S, and Duncan C. 1993. The Dynamics of Smallpox Epidemics in Britain, 1550-1800. Demography 30(3):405-423.

Dupras T, Schwarcz H, Fairgrieve. 2001. Infant feeding and weaning pracrices in Roman Egypt. Amer J Phys Anthropol 115:204-212.

Dyer C. 1988. The consumption of freshwater fish in medieval England. In: Aston M, editor. Medieval Fish, Fisheries and Fishponds in England. Oxford: British Archaeological Reports p27-38.

Dyer C. 1989. Standards of Living in the later Middle Ages: Social change in England c. 1200-1520. Cambridge: Cambridge University Press.

Dyer C. 2002. Making a living in the Middle Ages: The people of Britain; 850-1520. New Haven, CT: Yale University Press.

Dyson T. 1991. On the Demography of South Asian Famines. Popul Stud 45:5-25.

Eerkens JW, Berget AG, and Bartelink EJ. 2011. Estimating weaning and early childhood diet from serial micro-samples of dentin collagen. J Archaeol Sci 38(11):3101-3111.

Ekwall E. 1947. Early London personal names: CWK Gleerup.

Evans J, Stoodley N, and Chenery C. 2006. A strontium and oxygen isotope assessment of a possible fourth century immigrant population in a Hampshire cemetery, southern England. J Archaeol Sci 33(2):265-272.

Everill P. 2007. British commercial archaeology: antiquarians and labourers; developers and diggers. Archaeology and capitalism. Walnut Creek, CA: Left Coast Press. p 119-136.

Farr W. 1846. The Influence of Scarcities and of the High Prices of Wheat on the Mortality of the People of England. Journal of the Statistical Society of London:158-174.

French G. 1979. Water in relation to human disease. In: Howe GM, and Loraine JA, editors. Environmental medicine. London: William Heinemann medical Books Ltd. p 47-69.

Fulford M, and Holbrook N. 2011. Assessing the contribution of commercial archaeology to the study of the Roman period in England, 1990–2004. The Antiquaries Journal 91:323-345.

Fuller BT, Fuller JL, Sage NE, Harris DA, O'Connell TC, and Hedges RE. 2004. Nitrogen balance and δ15N: why you're not what you eat during pregnancy. Rapid Communications in Mass Spectrometry 18(23):2889-2896.

Fuller BT, Fuller JL, Sage NE, Harris DA, O'Connell TC, and Hedges RE. 2005. Nitrogen balance and δ15N: why you're not what you eat during nutritional stress. Rapid Communications in Mass Spectrometry 19(18):2497-2506.

Fuller BT, Richards MP, and Mays S. 2003. Stable carbon and nitrogen isotope variations in tooth dentine serial sections from Wharram Percy. J Archaeol Sci 30(12):1673-1684.

Gage TB. 1985. Demographic estimation from anthropological data: New methods. Curr Anthrop:644-647.

Gage TB. 1988. Mathematical hazard models of mortality: an alternative to model life tables. Amer J Phys Anthrop 76(4):429-441.

Gage TB. 1989. Bio-mathematical approaches to the study of human variation in mortality. Amer J Phys Anthrop 32(S10):185-214.

Gage TB, DeWitte S, and Wood J. 2012. Demography Part 1: Mortality and Migration. In: Stinson S, Bogin B, and O'Rourke D, editors. Human Biology: An Evolutionary and Biocultural Perspective. Hoboken, NJ: Wiley-Blackwell. p 695-756.

Galloway JA, and Murphy M. 1991. Feeding the city: medieval London and its agrarian hinterland. The London Journal 16(1):3-14.

Galloway P. 1985. Annual variation in deaths by age, deaths by cause, prices, and weather in London 1670 to 1830. Popul Stud 39(3):487-505.

Galloway P. 1988. Basic patterns in annual variations in fertility, nupiality, mortality and prices, in pre-industrial Europe. Popul Stud 42(2):275-303.

Gilchrist R. 2012. Medieval life. Archaeology and the life course. Suffolk: The Boydell Press.

Goldberg P. 2001. Coventry's 'Lollard' Programme of 1492 and the Making of Utopia. In: Horrox R, and Rees-Jones S, editors. Pragmatic Utopias: Ideals and Communities. Cambridge: Cambridge University Press.

Goldberg P. 2004. Migration, youth and gender in later medieval England. In: Goldberg P, and Riddy F, editors. Youth in the Middle Ages. Rochester, NY: Boydell and Brewer. p 85-99.

Goodman A. 1991. Stress, adaptation, and enamel developmental defects. In: Ortner DJ, and Aufderheide A, editors. Human paleopathology: current syntheses and future options. Washington: Smithsonian Institution Press. p 280-287.

Goodman AH, Allen LH, Hernandez GP, Amador A, Arriola LV, Chávez A, and Pelto GH. 1987. Prevalence and age at development of enamel hypoplasias in Mexican children. Amer J Phys Anthrop 72(1):7-19.

Goodman AH, and Armelagos GJ. 1989. Infant and childhood morbidity and mortality risks in archaeological populations. Wld Archaeol 21(2):225-243.

Goodman AH, Brooke Thomas R, Swedlund AC, and Armelagos GJ. 1988. Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. Amer J Phys Anthrop 31(S9):169-202.

Gordon CC, and Buikstra JE. 1981. Soil pH, bone preservation, and sampling bias at mortuary sites. Amer Antiq:566-571.

Gowland RL, and Chamberlain A. 2005. Detecting plague: palaeodemographic characterisation of a catastrophic death assemblage. Antiquity 79:146-157.

Grauer AL. 1991. Patterns of life and death: the palaeodemography of medieval York. In: Bush H, and Zvelebil M, editors. Health in Past Societies. Oxford: British Archaeological Reports International Series. p 67-80.

Graves-Brown P. 1997. S/he who pays the piper... archaeology and the polluter pays principle. Assemblage 2.

Gummer B. 2009. The Scourginf Angel: The Black Death in the British Isles. London: Bodley Head.

Hall AR, and Kenward HK. 1994. Urban-rural Connexions: Perspectives from Environmental Archaeology. Oxford: Oxbow Books Ltd.

Hallam HE. 1981. Rural England, 1066-1348. San Francisco: Harper.

Hamilakis Y, and Duke P. 2007. Archaeology and capitalism: from ethics to politics. Walnut Creek, CA: Left Coast Press.

Hanawalt BA. 1995. Growing up in medieval London: the experience of childhood in history. Oxford: Oxford University Press.

Haydock H, Clarke L, Craig-Atkins E, Howcroft R, Buckberry J. 2013. Weaning at Anglo-Sexon Raunds: Implications for breastfeeding practice in Britain over two millennia. Amer J of Phys Anthropol 151:604-612.

Heaton TH, Vogel JC, von La Chevallerie G, and Collett G. 1986. Climatic influence on the isotopic composition of bone nitrogen. Nature 322:822-823.

Hedges R. 2004. Isotopes and red herrings: comments on Milner et al. and Lidén et al. Antiquity 78:34-37.

Hedges R, Clement JG, Thomas CDL, and O'Connell TC. 2007. Collagen turnover in the adult femoral mid-shaft: modeled from anthropogenic radiocarbon tracer measurements. Am J Phys Anthropol 133:808-816.

Hedges RE. 2002. Bone diagenesis: an overview of processes. Archaeometry 44(3):319-328.

Henderson RC, Lee-Thorp J, and Loe L. 2014. Early life histories of the London poor using δ13C and δ15N stable isotope incremental dentine sampling. Amer J Phys Anthrop 154(4):585-593.

Hill N, Fallowfield J, Price S, and Wilson D. 2011. Military nutrition: maintaining health and rebuilding injured tissue. Philosophical Transactions of the Royal Society B: Biological Sciences 366(1562):231-240.

Hillson S. 2005. Teeth. Cambridge: Cambridge University Press.

Hillson S and Bond S. 1997. Relationship of enamel hypoplasia to the pattern of tooth crown growth: a discussion. Amer j Phy Anthrop 104:89-103.

Hinde A. 2003. England's Population: A History Since the Domesday Survey. London: Hodder Arnold.

Hinton. 1992. Confidentiality and PPG16. Archaeological News 7(6):74.

Hobson KA, Alisauskas RT, and Clark RG. 1993. Stable-nitrogen isotope enrichment in avian tissues due to fasting and nutritional stress: implications for isotopic analyses of diet. Condor:388-394.

Holder S, Dupras T, Jankauskas R, Williams L, Schultz J. 2017. Reconstructing diet in Napoleon’s Grand Army using stab;e carbon and nitrogen isotope analysis. Amer J of Phys Anthropol 163:53-63.

Holt R, and Rosser G. 1990. The English Medieval Town: A reader in English urban history, 1200-1540: Addison-Wesley Longman Ltd.

Hoppa RD. 2002. Paleodemography: looking back and thinking ahead. In: Hoppa R, and Vaupel J, editors. Paleodemography: Age Distributions from Skeletal Samples. Cambridge: Cambridge University Pres. p 9-28.

Hoppa RD, and Vaupel J. 2002. The Rostock Manifesto for paleodemography: The way from stage to age. Paleodemography: Age distributions from skeletal samples. New York: Cambridge University Press. p 1-8.

Horden P. 2000. Ritual and Public Health in the Early Medieval City. In: Sheard S, and Powers H, editors. Body and City: Histories of Urban Public Health. Aldershot: Ashgate Publishing Ltd. p 17-40.

Howell N. 1976. Toward a uniformitarian theory of human paleodemography. In: Ward R, and Weiss K, editors. The Demographic Evolution of Human Populations. New York: Academic Press. p 25-40.

Huss-Ashmore R, Goodman AH, and Armelagos GJ. 1982. Nutritional inference from paleopathology. In: Schiffer M, editor. Advances in Archaeological Method and Theory. New York: Springer. p 395-474.

Johansson SR, and Horowitz S. 1986. Estimating mortality in skeletal populations: influence of the growth rate on the interpretation of levels and trends during the transition to agriculture. Amer J Phys Anthrop 71(2):233-250.

Jones R. 2011. Elemental Theory in Everyday Practice: Food Disposal in the Later Medieval English Countryside. In: Klápšte J, and Sommer P, editors. Processing, Storage, Distribution of Food Food in the Medieval Rural Environment, Ruralia VIII, Brepols: Brepois Publishers. p 145-154.

Jordan WC. 1997. The Great Famine: Northern Europe in the Early Fourteenth Century. Princeton, NJ: Princeton University Press.

Jørgensen D. 2013. Running Amuck? Urban Swine Management in Late Medieval England. Ag Hist 87(4):429-451.

Jørgensen D. 2014. Modernity and medieval muck. Nature and Culture 9(3):225-237.

Jørgensen DM. 2008a. Cooperative sanitation: managing streets and gutters in late medieval England and Scandinavia. Technology and Culture 49(3):547-567.

Jørgensen DM. 2008b. Private need, public order: urban sanitation in Late Medieval England and Scandinavia. PhD Dissertation: University of Virginia.

Katzenberg MA. 2007. Stable isotope analysis: a tool for studying past diet, demography, and life history. In: Katzenberg MA, and Saunders SR, editors. Biological Anthropology of the Human Skeleton. New York: Wiley-Blackwell. p 411-441.

Katzenberg MA, and Lovell NC. 1999. Stable isotope variation in pathological bone. Internat J Osteoarchaeol 9(5):316-324.

Keene D. 2001. Issues of water in medieval London to c. 1300. Urban History 28(2):161-179.

Kendall E, Montgomery J, Evans J, Stantis C, Mueller V. 2013. Mobility, mortliaty, and the middle Ages: Identification of migrant individuals in a 14th century Black Death cemetery population 150:210-222.

Kenward H, and Large F. 1998. Insect in urban waste pits in Viking York: Another kind of seasonality. Environ Archaeol 3:35-53.

Kenward HK. 1978. The analysis of archaeological insect assemblages: a new approach: York Archaeological Trust.

Kenward HK, and Allison E. 1994. Rural origins of the urban insect fauna. In: Hall A, and Kenward H, editors. Urban-rural connexions: Perspectives from environmental archaeology. Oxford: Oxbow Books Ltd. p 55-77.

Keys A, Brozek J, Henschel A, Michelsen O, and Taylor H. 1950. The biology of human starvation. Minneapolis, MN.

Klepinger LL. 1984. Nutritional assessment from bone. Ann Rev Anthropol 13:75-96.

Konigsberg LW, and Frankenberg SR. 1992. Estimation of age structure in anthropological demography. Amer J Phys Anthrop 89(2):235-256.

Konigsberg LW, and Frankenberg SR. 1994. Paleodemography:“Not quite dead”. Evol Anthropo 3(3):92-105.

Konigsberg LW, and Frankenberg SR. 2002. Deconstructing death in paleodemography. Amer J Phys Anthrop 117(4):297-309.

Kowaleski M. 2014. Medieval People in Town and Country: New Perspectives from Demography and Bioarchaeology. Speculum 89(3):573-600.

Kreger MMB. 2010. Urban Population Dynamics in a Preindustrial New World City: Morbidity, Mortality, and Immigration in Postclassic Cholula. PhD Dissertation: The Pennsylvania State University.

Kruskal WH, and Wallis WA. 1952. Use of ranks in one-criterion variance analysis. Journal of the Am Stat Assoc 47(260):583-621.

Kumar R, Singh MC, Ahlawat SK, Thakur JS, Srivastava A, Sharma MK, Malhotra P, Bali HK, and Kumari S. 2006. Urbanization and coronary heart disease: a study of urban-rural differences in northern India. Indian Heart Journal 58(2):126.

Lakin K. 2010. Diet and migration in medieval London: stable isotope analysis of human and faunal remains. PhD Dissertation. Reading, UK: University of Reading.

Landers J. 1993. Death and the Metropolis: Studies in the Demographic History of London, 1670-1830. Cambridge: Cambridge University Press.

Lee-Thorp JA, Sealy JC, and Van Der Merwe NJ. 1989. Stable carbon isotope ratio differences between bone collagen and bone apatite, and their relationship to diet. J Archaeol Sci 16(6):585-599.

Lewis M. 2002. Urbanisation and child health in medieval and post-medieval England: An assessment of the morbidity and mortality of non-adult skeletons from the cemeteries of two urban and two rural sites in England (AD 850-1859). Oxford: British Archaeological Reports.

Lewis M. 2016. Work and the Adolescent in Medieval England AD 900-1550: The Osteological Evidence. Med Archaeol 60(1):138-170.

Lewis ME. 2003. A comparison of health in past rural, urban and industrial environments. In: Murphy P, and Wiltshire P, editors. The Environmental Archaeology of Industry Symposia of the Association for Environmental Archaeology of Industry No 20. Oxford: Oxbow Books. p 154-161.

Lewis ME, and Gowland R. 2007. Brief and precarious lives: Infant mortality in contrasting sites from medieval and post-medieval England (AD 850–1859). Amer J Phys Anthrop 134(1):117-129.

Lewis ME, Roberts CA, and Manchester K. 1995. Comparative study of the prevalence of maxillary sinusitis in later Medieval urban and rural populations in northern England. Amer J Phys Anthrop 98(4):497-506.

Leyser H. 1995. Medieval Women: Social History of Women in England, 450-1500. Oxford: Oxford University Press.

Libby WF, Berger R, Mead JF, Alexander GV, and Ross JF. 1964. Replacement rates for human tissue from atmospheric radiocarbon. Science 146:1170-1172.

Lotka AJ. 1922. Contribution to the energetics of evolution. Proceedings of the National Academy of Sciences of the United States of America 8(6):147.

Lovejoy CO, Meindl RS, Pryzbeck TR, Barton TS, Heiple KG, and Kotting D. 1977. Paleodemography of the Libben site, Ottawa county, Ohio. Science 198(4314):291-293.

Lucy S. 1994. Children in early medieval cemeteries. Archaeol Rev Camb 13(2):21-34.

Lyman T. 2004. Barton-upon-Humber Conservation Area Appraisal Adopted Document. Council NL.

Magnusson RJ. 2013. Medieval Urban Environmental History. History Compass 11(3):189-200.

Manchester K. 1992. The palaeopathology of urban infections. In: Bassett S, editor. Death in Towns: Urban Responses to the Dying and the Dead 100-1600. Leicester: Leicester University Press. p 8-14.

Mann HB, and Whitney DR. 1947. On a test of whether one of two random variables is stochastically larger than the other. Ann Math Stats:50-60.

Martin DL, Harrod RP, and Pérez VR. 2013. Bioarchaeology: An Integrated Approach to Working with Human Remains. New York: Springer.

Massingberd W. 1906. Social and economic history. In: Page W, editor. The Victoria Histories of the Counties of England: a History of Lincolnshire. London: Archibald Constable and Co. p 292-380.

Mays S, Brickley M, and Ives R. 2006. Skeletal manifestations of rickets in infants and young children in a historic population from England. Amer J Phys Anthrop 129(3):362-374.

McEwan J, Mays S, and Blake G. 2005. The relationship of bone mineral density and other growth parameters to stress indicators in a medieval juvenile population. Int J of Osteoarch 15(3):155-163.

McKern T, and Stewart T. 1957. Skeletal Age Changes in Young American Males, Analyzed from the Standpoint of Age Identifcation. Technical Report EP-45. Natick, MA: Headquarters Quartermaster Research and Development Command.

McKinley JI. 2013. 'No Room at the Inn' ... Contract Archaeology and the Storage of Human Remains. In: Giesen M, editor. Curating Human Remains: Caring for the Dead in the United Kingdom. Suffolk, UK: The Boydell Press. p 135-143.

McNeill WH. 1976. Plagues and Peoples: Anchor.

McNeill WH. 1980. Migration patterns and infection in traditional societies. In: Stanley NF, and Joske RA, editors. Changing disease patterns and human behaviour. London: Academic Press. p 28-36.

Meindl RS, Lovejoy CO, Mensforth RP, and Carlos LD. 1985a. Accuracy and direction of error in the sexing of the skeleton: implications for paleodemography. Amer J Phys Anthrop 68(1):79-85.

Meindl RS, Lovejoy CO, Mensforth RP, and Walker RA. 1985b. A revised method of age determination using the os pubis, with a review and tests of accuracy of other current methods of pubic symphyseal aging. Amer J Phys Anthrop 68(1):29-45.

Mekota A-M, Grupe G, Ufer S, and Cuntz U. 2009. Identifying starvation episodes using stable isotopes in hair. Rechtsmedizin 19(6):431-440.

Mekota AM, Grupe G, Ufer S, and Cuntz U. 2006. Serial analysis of stable nitrogen and carbon isotopes in hair: monitoring starvation and recovery phases of patients suffering from anorexia nervosa. Rapid Communications in Mass Spectrometry 20(10):1604-1610.

Mellor J. 1992. PPG16 one year on. Rescue News 56:1.

Mielke J, Jorde L, Trapp P, Anderton D, Pitkanen K, and Eriksson A. 1984. Historical epidemiology of small pox in Aland, Finland 1751-1890. Demography 21:271-295.

Miller E, and Hatcher J. 2014. Medieval England: rural society and economic change 1086-1348 London: Routledge.

Milner GR. 1980. Epidemic disease in the postcontact Southeast: a reappraisal. Midcont J Archaeol:39-56.

Milner GR, and Boldsen JL. 2012. Transition analysis: A validation study with known-age modern American skeletons. Amer J Phys Anthrop 148(1):98-110.

Milner GR, Humpf DA, and Harpending HC. 1989. Pattern matching of age-at-death distributions in paleodemographic analysis. Amer J Phys Anthrop 80(1):49-58.

Milner GR, W. WJ, and Boldsen JL. 2007. Paleodemography. In: Katzenberg MA, and Saunders SR, editors. Biological Anthropology of the Human Skeleton. New York: Wiley-Liss. p 561-600.

Miyawaki T, Moriya N, Nagaoki T, and Taniguchi N. 1981. Maturation of B-Cell Differentiation Ability and T-Cell Regulatory Function in Infancy and Childhood. Immunological reviews 57(1):61-87.

Montgomery J, Evans J, Chenery S, Pashley V, and Killgrove K. 2010. 'Gleaming, white and deadly': using lead to track human exposure and geographic origins in the Roman period in Britain. Journal of Roman archaeology; supplementary series:199-226.

Montgomery J. 2010. Passports from the past: Investigating human dispersal using strontium isotope analysis of tooth enamel. Annals of Hum Bio 37(3):325-346.

Montgomery J, Evans JA, Powlesland D, and Roberts CA. 2005. Continuity or colonization in Anglo-Saxon England? Isotope evidence for mobility, subsistence practice, and status at West Heslerton. Amer J Phys Anthrop 126(2):123-138.

Moore M, Gould P, and Keary BS. 2003. Global urbanization and impact on health. International Journal of Hygiene and Environmental Health 206(4):269-278.

Morfin LM, McCaa R, Storey R, and Angel A. 2002. Health and nutrition in pre-Hispanic Mesoamerica. In: Steckel R, and Rose J, editors. The Backbone of History: Health and Nutrition in the Western Hemisphere, Cambridge University Press, Cambridge: Cambridge University Press. p 307-342.

Müldner G, Montgomery J, Cook G, Ellam R, Gledhill A, and Lowe C. 2009. Isotopes and individuals: diet and mobility among the medieval Bishops of Whithorn. Antiquity 83(322):1119-1133.

Müldner G, and Richards M. 2005. Fast or feast: reconstructing diet in later medieval England by stable isotope analysis. J Archaeol Sci 32:39-48.

Müldner G, and Richards M. 2007a. Diet and diversity at later medieval Fishergate: the isotopic evidence. J Am Anthropol 134:162-174.

Müldner G, and Richards MP. 2007b. Stable isotope evidence for 1500 years of human diet at the city of York, UK. Amer J Phys Anthrop 133(1):682-697.

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

Murphy E. 2011. Children's Burial Grounds in Ireland (*Cillini*) and Parental Emotions Toward Infant Death. Int J Histor Archaeol 15:409-428.

Nagaoka T, and Hirata K. 2007. Reconstruction of paleodemographic characteristics from skeletal age at death distributions: perspectives from Hitotsubashi, Japan. Amer J Phys Anthrop 134(3):301-311.

Neiburger E. 1990. Enamel hypoplasias: Poor indicators of dietary stress. Amer J of Phys Anthropol 82(2):231-232.

Nelson DR, Adger NW, and Brown K. 2007. Adaptation to Environmental Change: Contributions of a Resilience Framework. Annual Review of Environment and Resources 32:395-419.

Neuberger FM, Jopp E, Graw M, Püschel k, and Grupe G. 2013. Signs of malnutrition and starvation: Reconstruction of nutritional life histories by sereal isotopic analyses of hair. Forensic Sci Int 226:22-32.

Nicholas DM. 2014. The later medieval city: 1300-1500. New York: Routledge.

Nielsen-Marsh CM, and Hedges RE. 2000. Patterns of diagenesis in bone I: the effects of site environments. J Archaeol Sci 27(12):1139-1150.

O'Connor T. 1989. What shall we have for dinner? Food remains from urban sites. In: Serieantson D, and Waldron T, editors. Diet and Crafts in Towns: The Evidence of Animal Remains From the Roman to the Post-Medieval Periods. Oxford: British Archaeology Reports. p 12-23.

O'Leary MH. 1981. Carbon isotope fractionation in plants. Phytochemistry 20(4):553-567.

Olsen KC, White CD, Longstaffe FJ, Heyking K, McGlynn G, Grupe G, and Rühli FJ. 2014. Intraskeletal isotopic compositions (δ13C, δ15N) of bone collagen: nonpathological and pathological variation. Amer J Phys Anthrop 153(4):598-604.

Olsen K. 2013. A multi-purpose investigation of two medieval German populations: insights into the relationship among diet, disese, and tissue isotopic composition. London, Ontario: The University of Western Ontario.

Organization WH. 1984. Biomass fuel combustion and health. EEP/84.64. Geneva.

Organziation WH. 1968. Weanling diarrhea: A synergism of infection and nutrition. In: Organization WH, editor. Interactions of Nutrition and Infection. Geneva: World Health Organization. p 216-329.

Orten J, and Neuhaus O. 1982. Human biochemistry. The CV Mosby Company.

Ortner DJ. 2003. Identification of Pathological Conditions in Human Skeletal Remains. New York: Academic Press.

Orton C. 2000. Sampling in archaeology Cambridge: Cambridge University Press.

Paine RR. 2000. If a population crashes in prehistory, and there is no paleodemographer there to hear it, does it make a sound? Amer J Phys Anthrop 112(2):181-190.

Pate FD. 1994. Bone chemistry and paleodiet. J Archaeol Method Th 1(2):161-209.

Pearson SF, J LD, H GC, and del Rio CM. 2003. Effects of elemental composition on the incorporation of dietary nitrogen and carbon isotopic signatures in an omnivorous songbird. Oecologia 135:516-523.

Phenice TW. 1969. A newly developed visual method of sexing the os pubis. Amer J Phys Anthrop 30(2):297-301.

Platt C. 1976. English Medieval Town. London: Secker & Warburg.

Ponsard S, and Averbuch P. 1999. Should growing and adult animals fed on the same diet show different δ15N values? Rapid Communications in Mass Spectrometry 13:1305-1310.

Popkin BM. 1999. Urbanization, lifestyle changes and the nutrition transition. World Development 27(11):1905-1916.

Pounds N. 2005. The Medieval City. Westport, CT: Greenwood Publishing Group.

Preston S, Haines M, and Panuk E. 1981. Effects of industrialisation and urbanisation in developed countries. Proceedings World Population Conference. Manilla: International Union for the Scientfic Study of Population. p 233-253.

Privat KL, O'connell TC, and Richards MP. 2002. Stable isotope analysis of human and faunal remains from the Anglo-Saxon cemetery at Berinsfield, Oxfordshire: dietary and social implications. J Archaeol Sci 29(7):779-790.

Prothero RM. 1977. Disease and mobility: a neglected factor in epidemiology. International Journal of Epidemiology 6(3):259-267.

Morris, R. 1998. Riding down the road to regional chaos. British Archaeology 31(1):15.

Rawcliffe C. 2013. Urban Bodies: Communal Health in Late Medieval English Towns and Cities. London: Boydell & Brewer Ltd.

Rawcliffe C. 1995. Medicine and society in later medieval England. Phoenix Mill: Sutton.

Reid DJ, and Dean MC. 2000. Brief communication: the timing of linear hypoplasias on human anterior teeth. Amer J Phys Anthrop 113(1):135-139.

Reitsema LJ. 2013. Beyond diet reconstruction: Stable isotope applications to human physiology, health, and nutrition. Amer J Hum Biol 25(4):445-456.

Reitsema LJ, and Vercellotti G. 2012. Stable isotope evidence for sex-and status-based variations in diet and life history at medieval Trino Vercellese, Italy. Amer J Phys Anthrop 148(4):589-600.

Reitsema LJ, Vercellotti G, and Boano R. 2016. Subadult dietary variation at Trino Vercellese, Italy, and its relationship to adult diet and mortality. Amer J Phys Anthrop 160(4):653-664.

Richards M, and Hedges REM. 1999. Stable isotope evidence for similarities in the types of marine foods used by Late Mesolithic humans at sites along the Atlantic coast of Europe. J Archaeol Sci 26(6):717-722.

Richards M, and Montgomery J. 2012. Isotope analysis and paleopathology: a short review and future developments. In: Buikstra JE, and Roberts C, editors. The Global History of Paleopathology: Pioneers and Prospects. Oxford: Oxford University Press. p 718-731.

Richards MP, Mays S, and Fuller BT. 2002. Stable carbon and nitrogen isotope values of bone and teeth reflect weaning age at the Medieval Wharram Percy site, Yorkshire, UK. Amer J Phys Anthrop 119(3):205-210.

Roberts C. 2000. Infectious disease in biocultural perspective: past, present and future work in Britain. In: Cox M, and Mays S, editors. Human Osteology in Archaeology and Forensic Science. Cambridge: Cambridge University Press. p 145-162.

Roberts C, and Cox M. 2007. The impact of economic intensification and social complexity on human health in Britain from 4000 BC (Neolithic) and the introduction of farming to the mid 19th century AD and industrialisation (post-Medieval period). In: Cohen MN, and Crane-Kramer GMM, editors. Ancient health: skeletal indicators of agricultural economic intensification. Gainesville, FL: University Press of Florida. p 149-163.

Roberts CA. 2007. A bioarcheological study of maxillary sinusitis. Amer J Phys Anthrop 133(2):792-807.

Roberts CA. 2009. Health and welfare in medieval England: the human skeletal remains contextualised. In: Gilchrist R, and Reynolds A, editors. Reflections: 50 Years of Medieval Archaeology, 1957-2007. New York: Maney Publishing. p 307-325.

Roberts CA, and Cox M. 2003. Health and Disease in Britain: From Prehistory to the Present Day. Gloucestershire, UK: Sutton Publishing.

Roberts CA, Lewis ME, and Boocock P. 1998. Infectious disease, sex, and gender: The complexity of it all. In: Grauer AL, and Stuart-Macadam P, editors. Sex and gender in paleopathological perspective. Cambridge: Cambridge University Press. p 93-113.

Rodwell W, and Atkins C. 2011. St Peter's Barton-upon-Humber, Lincolnshire: A Parish Church and its Community. Oxbow: Oxbow Books.

Rosser AG. 2000. Urban Culture and the Church, 1300-1540. In: Palliser DM, editor. The Cambridge Urban History of Britain. Cambridge. p 600.

Rowsome P. 2000. Heart of the City: Roman, medieval and modern London revealed by archaeology at 1 Poultry. London: Museum of London Archaeology Service.

Sabine EL. 1934. Latrines and cesspools of mediaeval London. Speculum 9(3):303-321.

Sabine EL. 1937. City cleaning in mediaeval London. Speculum 12(1):19-43.

Sattenspiel L, and Harpending H. 1983. Stable populations and skeletal age. Amer Antiq:489-498.

Saunders SR, and Barrans L. 1999. What can be done about the infant category in skeletal samples? In: Hoppa RD, and Fitzgerald CM, editors. Human growth in the past: studies from bonesand teeth. Cambridge: Cambridge University Press. p 183-209.

Scheuer L, Black S, and Cunningham C. 2000. Developmental juvenile osteology. Cambridge: Academic Press.

Schoeller DA. 1999. Isotope fractionation: why aren't we what we eat? J Archaeol Sci 26(6):667-673.

Schoeninger MJ. 1995. Stable isotope studies in human evolution. Evolutionary Anthropology: Issues, News, and Reviews 4(3):83-98.

Schoeninger MJ, and DeNiro MJ. 1984. Nitrogen and carbon isotopic composition of bone collagen from marine and terrestrial animals. Geochimica et Cosmochimica Acta 48(4):625-639.

Schoeninger MJ, Moore KM, Murray ML, and Kingston JD. 1989. Detection of bone preservation in archaeological and fossil samples. Applied Geochemistry 4(3):281-292.

Schofield J. 1997. Excavations on the site of St Nicholas Shambles, Newgate Street, city of London, 1975-9. Transactions of the London and Middlesex Archaeology Society 48:77-136.

Schofield J. 2011. London, 1100-1600: The Archaeology of a Capital City. London: Equinox Publishing Limited.

Schofield J, and Vince AG. 2003. Medieval towns: the archaeology of British towns in their European setting. London: A&C Black.

Schurr MR. 1988. Using stable nitrogen-isotopes to study weaning behavior in past populations. Wld Archaeol 30:327-342.

Schwarcz HP, and Schoeninger MJ. 1991. Stable isotope analyses in human nutritional ecology. Amer J Phys Anthrop 34(S13):283-321.

Scrimshaw NS. 1987. The phenomenon of famine. Ann Rev Nutrit 7:1-21.

Scrimshaw NS, Taylor CE, and Gordon JE. 1968. Interactions of nutrition and infection. American J Med Sc 237(3):367-403.

Serjeantson D, and Waldron T. 1989. Diet and Crafts in Towns: The Evidence of Animal Remains From the Roman to the Post-Medieval Periods. British Archaeological Reports.

Shahar S. 1990. Childhood in the Middle Ages. London: Routledge.

Shapland F, Lewis M, Watts, R. 2015. The lives and deaths of young medieval women: the osteological evidence. Medieval Archaeology 59:272-289.

Sharpe RR. 1899. Calendar of Letter-Books of the City of London: A: 1275-1298. London: John Edward Francis.

Sharpe RR. 1905. Calendar of Letter-books of the City of London: G: 1352-1374. London: John Edward Francis.

Sidell J, Thomas C, and Bayliss A. 2007. Validating and improving archaeological phasing at St. Mary Spital, London. Radiocarbon 49(2):593-610.

Simoons FJ. 1994. Eat Not This Flesh: Food Avoidances From Prehistory to the Present. Wisconsin: University of Winconsin Press.

Singman JL. 1999. Daily life in medieval Europe. London: Greenwood Press.

Slavin P. 2012. The Great Bovine Pestilence and its economic and environmental consequences in England and Wales, 1318-50. Ec Hist Rev 65(4):1239-1266.

Sloane B. 2004. Archaeological Evidence for the Infrastructure of the Medieval City of London. In: Gläser M, editor. Lübecker Kolloquium zur Stadtarchäologie in Hanseraum IV: Die Infrastruktur: Lübek. p 90-91.

Smith BN, and Epstein S. 1971. Two categories of 13C/12C ratios for higher plants. Plant Physiology 47(3):380-384.

Smith, G. 2002. Commentary: Behind the Broad Street pump: Aetiology, epidemiology and prevention of cholera in mid-19th century Britain. Intl J of Epidem 31(5):920-932.

Smith DN. 2012. Insects in the City: An Archaeoentomological Perspective on London's Past. Oxford: Archaeopress.

Smith P, and Kahila G. 1992. Indentication of infancide in archaeological sites: a case study form the Late Roman-Early Bizantine Periods at Ashkelon, Israel. J Archaeol Sci 19:667-675.

Smith S. 1997. Geographical diversity in the resort to marriage in late medieval Europe: work, reputation, and unmarried females in the household formation systems of northern and southern Europe. In: Goldberg P, editor. Women in Medieval English society. Oxford: Sutton Publishing. p 16-59.

Sponheimer M, Robinson T, Ayliffe L, Roeder B, Hammer J, Passey B, West A, Cerling T, Dearing D, and Ehleringer J. 2003a. Nitrogren isotopes in mammalian herbivores: hair δ15N values from a controlled feeding study. Internat J Osteoarchaeol 13:80-87.

Sponheimer M, Robinson TF, Roeder Bl, Passey B, Ayliffe LK, Cerling T, Dearing MD, and Ehleringer JR. 2003b. An experimental study of nitrogen flux in llamas: is 14N preferentially excreted? J Archaeol Sci 20:1649-1655.

Steckel RH. 2005. Health and nutrition in pre-Columbian America: The skeletal evidence. Journal of interdisciplinary History 36(1):1-32.

Steckel RH, and Rose JC. 2002. The Backbone of History: Health and Nutrition in the Western Hemisphere. Cambridge: Cambridge University Press.

Stojanowski CM, Seidemann RM, and Doran GH. 2002. Differential skeletal preservation at Windover Pond: causes and consequences. Amer J Phys Anthrop 119(1):15-26.

Storey G. 2006. Urbanism in the preindustrial world: cross-cultural approaches. Tuscalosa, Alabama: University of Alabama Press.

Storey R. 1985. An estimate of mortality in a pre-Columbian urban population. Amer Anthrop 87(3):519-535.

Storey R. 1992. Preindustrial urbanlifestyle and health. In: Huss-Ashmore R, Schall J, and Hediger M, editors. Health and lifestyle change. Philadelphia: University Museum of Archaeology and Anthropology. p 33-42.

Sutherland LD, and Suchey JM. 1991. Use of the ventral arc in pubic sex determination. J Forensic Sci 36(2):501-511.

Sykes NJ. 2006. From cu and sceap to beffe and motton. In: Woolgar CM, Serjeantson D, and Waldron AH, editors. Food in Medieval England: Diet and Nutrition. Oxford: Oxford University Press. p 56-71.

Tames R. 2003. Feeding London: a taste of history. Phillimore: Historical Publictions.

Taylor CE. 1983. Synergy among mass infections, famines, and poverty. J Interdiscip Hist 14(2):483-501.

Thomas C. 2000. The Priory and Hospital of St. Mary Spital, London. Medieval Life(14):21-25.

Thomas C. 2002. The Archaeology of Medieval London. Stroud, UK: Sutton.

Thomas C, Sloane B, and Phillpotts C. 1997. Excavations at the Priory and Hospital of St Mary Spital, London. London: Museum of London Archaeology Service.

Thrupp LL. 1989. The Merchant Class of Medieval London, 1300-1500. Ann Arbor, MI: University of Michigan Press.

Todd TW. 1921. Age changes in the pubic bone. Amer J Phys Anthrop 4(1):1-70.

Trueman CN, McGill RA, and Guyard PH. 2005. The effect of growth rate on tissue-diet isotopic spacing in rapidly growing animals. An experimental study with Atlantic salmon (Salmo salar). Rapid Communications in Mass Spectrometry 19:3239-3248.

Tsutaya T, Nagaoka T, Sawada J, Hirata K, and Yoneda M. 2013. Stable isotopic reconstructions of adult diets and infant feeding practices during urbanization of the city of Edo in 17th century Japan. Amer J Phys Anthrop 153(4):559-569.

Tykot RH. 2006. Isotope analyses and the histories of maize. In: Staller J, Tykot RH, and Benz B, editors. Histories of Maize: Multidisciplinary Approaches to the Prehistory, Linguistics, Biogeography, Domestication, and Evolution of Maize. Burlington, MA: Leftcoast Press. p 131-142.

United Nations. 1996. United Nations Center for Human Settlements: An urbanizing world. London; Sterling, VA: Earthscan.

United Nations. 2001a. United Nations Center for Human Settlements: State of the World's Cities 2001. In: United Nations Center for Human Settlements (Habitat), editor. Nairobi.

United Nations. 2001b. United Nations Population Division: World urbanization prospects: the 1999 revision. Key Findings. In: United Nations Population Division, editor. New York.

Valentin J. 2003. Basic anatomical and physiological data for use in radiological protection: reference values: International Commission on Radiological Protection 89. Ann ICRP 32:1-277

Van Klinken GJ. 1999. Bone collagen quality indicators for paleodietary and radiocarbon measurements. J Archaeol Sci 26(687-695).

Van Klinken GJ, Richards MP, and Hedges BE. 2002. An overview of causes for stable isotopic variations in past European human populations: environmental, ecophysiological, and cultural effects. In: Ambrose SH, and Katzenberg MA, editors. Biogeochemical Approaches to Paleodietary Analysis. New York: Springer. p 39-63.

Vaupel JW, and Yashin AI. 1985. Heterogeneity's ruses: some surprising effects of selection on population dynamics. The American Statistician 39(3):176-185.

Velimirovic B. 1979. Forgotten people--health of the migrants. Bulletin of the Pan American Health Organization 13:66-85.

Vogel JC, and Van der Merwe NJ. 1977. Isotopic evidence for early maize cultivation in New York State. Amer Antiq 42(2):238-242.

Wainwright G. 2000. Time please. Antiquity 74(1):909-943.

Waldron T. 1989. The effects of urbanism on human health: the evidence from skeletal remains. In: Serieantson D, and Waldron T, editors. Diet and crafts in towns: The evidence of animal remains from Roman to the Post-Medieval periods. Oxford: British Archaeological Reports British Series, 199. p 55-73.

Waldron T. 2007. The St. Peter's Cemetery, Barton-upon-Humber, Lincolnshire - A Parish church and its Community. Oxford: Oxbow Books.

Walker PL. 1995. Problems of preservation and sexism in sexing. In: Saunders SR, and Herring A, editors. Grave Reflections: Portraying the Past through Cemetery Studies. Toronto: Canadian Scholars Press. p 31-47.

Walker PL. 2008. Sexing skulls using discriminant function analysis of visually assessed strait. American Journal of Physical Anthropology 136:39-50.

Walker PL, Bathurst RR, Richman R, Gjerdrum T, and Andrushko VA. 2009. The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis. Amer J Phys Anthrop 139(2):109-125.

Walker PL, Johnson JR, and Lambert PM. 1988. Age and sex biases in the preservation of human skeletal remains. Amer J Phys Anthrop 76(2):183-188.

Walrath DE, Turner P, and Bruzek J. 2004. Reliability test of the visual assessment of cranial traits for sex determination. Amer J Phys Anthrop 125(2):132-137.

Waters-Rist AL. 2010. The effect of growth on stable nitrogen isotope ratios in subadult bone collagen. Int J Osteoarch 20:172-191.

Watts R. 2013. Childhood development and adult longevity in an archaeological population from Barton-upon-Humber, Lincolnshire, England. Int J Paleopathol 3(2):95-104.

Walter BS and DeWitte SN. 2017 Urban and rural mortality and survival in medieval London. Ann Hum Biol. Available online: http://dx.doi.org/10.1080/03014460.2016.1275792

Way AB. 1976. Morbidity and postneonatal mortality. In: Baker PT, and Little MA, editors. Man in the Andes: a Multidisciplinary Study of High Altitude Quechua. Stroudsburg, PA: Dowden, Hutchinson, and Ross. p 147-160.

Weiss KM. 1972. On the systematic bias in skeletal sexing. Amer J Phys Anthrop 37(2):239-249.

Weiss KM. 1975. Demographic Disturbance and the Use of Life Tables in Anthropology. Memoirs of the Society for American Archaeology No 30, Population Studies in Archaeology and Biological Anthropology: A Symposium:46-56.

Weiss KM, and Wobst HM. 1973. Demographic models for anthropology. Memoirs of the Society for American Archaeology:i-186.

White CD, and Armelagos GJ. 1997. Osteopenia and stable isotope ratios in bone collagen of Nubian female mummies. Amer J Phys Anthrop 103(2):185-199.

White CD, and Schwarcz HP. 1994. Temporal trends in stable isotopes for Nubian mummy tissues. Am J Phys Anthropol 93:165-187.

Williams BA, and Rogers TL. 2006. Evaluating the accuracy and precision of cranial morphological traits for sex determination. J Forensic Sci 51(4):729-735.

Williams G. 1963. Medieval London: From Commune to Capital. London: Athline Press.

Wilson JJ. 2014. Paradox and promise: Research on the role of recent advances in paleodemography and paleoepidemiology to the study of “Health” in precolumbian societies. Amer J Phys Anthrop 155(2):268-280.

Wittwer-Backofen U, Buckberry J, Czarnetzki A, Doppler S, Grupe G, Hotz G, Kemkes A, Larsen CS, Prince D, and Wahl J. 2008. Basics in paleodemography: a comparison of age indicators applied to the early medieval skeletal sample of Lauchheim. Amer J Phys Anthrop 137(4):384-396.

Wood J, Milner G, Harpending H, and Weiss K. 1992. The osteological paradox: problems of inferring prehistoric health from skeletal samples. Curr Anthrop 33(4):343-370.

Wood JW, Holman DJ, O'Connor KA, and Ferrell RJ. 2002. Mortality models for paleodemography. In: Hoppa RD, and Vaupel JW, editors. Paleodemography: Age Distributions from Skeletal Samples. Cambridge: Cambridge University Press. p 129-168.

Woolgar CM. 2000. Take this penance now, and afterwards the fare will improve: Seafood and late medieval diet. In: Starkey DJ, Reid C, and Ashcroft N, editors. England's Sea Fisheries: The Commercial Sea Fisheries of England and Wales since 1300. London: Catham Publishing. p 36-45.

Woolgar CM. 2006. Meat and dairy products in late medieval England. In: Woolgar CM, Serjeantson D, and Waldron T, editors. Food in Medieval England: Diet and Nutrition. New York: Oxford University Press. p 88-101.

World Health Organziation. 1998. Life in the 21st Century: A vision for all. Geneva.

World Health Organziation. 2003. Gender and Tuperculosis. Geneva.

Wrigley EA. 1969. Population and History. London: Weidenfeld & Nicolson.

Yaussy S, Dewitte S, and Redfern R. 2016. Frailty and Famine: Patterns of Mortality and Physiological Stress Among Victims of Famine in Medieval London. American Journal of Physical Anthropology 160:272-283.

Zuckerman MK, Turner BL, and Armelagos GJ. 2012. Evolutionary thought in paleopathology and the rise of the biocultural approach. In: Grauer A, editor. A Companion to Paleopathology. Malden, MA: Wiley-Blackwell. p 34-57.

!LJLJŜyŘÅÈ !π tl·fŜ2ŘŜY23ℕl·LJKÅÕ 5l·ű· Ŧ2ℕ {ű al·ℕè {LJÅűl·t

Table F.1: Paleodemographic data for SPR98, including context number, temporal phase, burial type, sex, and point estimates of age-at-death

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	1031	17	A	M	76.0
SRP98	1055	15	D	M	15.0
SRP98	1851	15	D	U	17.5
SRP98	2304	17	A	U	13.5
SRP98	2481	17	D	F	32.1
SRP98	2487	17	D	F	31.6
SRP98	2626	15	B	M	39.2
SRP98	2679	17	A	M	49.8
SRP98	2687	15	A	F	23.6
SRP98	2694	16	D	M	32.4
SRP98	2843	16	D	F	26.1
SRP98	2893	17	A	F	ADULT
SRP98	2916	17	D	F	24.5
SRP98	2917	17	D	M	17.0
SRP98	3000	16	A	M	25.6
SRP98	3144	15	D	M	18.5
SRP98	3166	17	A	F	50.8
SRP98	3180	17	D	F	18.0
SRP98	3201	16	D	U	15.0
SRP98	3221	15	D	M	33.7
SRP98	3292	15	A	M	19.6
SRP98	3441	14	A	F	28.7
SRP98	3503	16	A	M	18.5
SRP98	3606	15	B	M	30.4
SRP98	3645	15	D	F	15.0
SRP98	3676	15	A	M	14.5
SRP98	3721	15	D	F	23.8
SRP98	3735	15	B	F	40.3
SRP98	3738	15	D	F	21.0
SRP98	3757	15	B	F	22.0

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	3775	17	A	F	42.0
SRP98	3805	15	D	F	76.2
SRP98	3806	15	D	M	38.4
SRP98	3825	17	D	M	35.1
SRP98	3845	15	D	F	9.5
SRP98	3849	15	D	F	37.5
SRP98	3852	17	D	M	33.4
SRP98	3868	14	A	F	15.0
SRP98	3934	15	D	F	37.1
SRP98	3975	15	B	M	18.5
SRP98	3975	15	B	M	18.5
SRP98	5018	14	A	M	42.5
SRP98	5258	14	A	M	33.5
SRP98	5287	14	A	M	63.8
SRP98	5420	14	A	M	36.8
SRP98	5561	14	C	M	37.7
SRP98	5638	16	A	M	32.8
SRP98	5670	16	A	F	18.0
SRP98	5674	16	B	U	15.0
SRP98	5677	14	A	M	31.3
SRP98	5895	14	A	M	75.5
SRP98	5907	15	D	U	14.5
SRP98	5997	14	A	M	36.2
SRP98	6427	17	A	M	26.8
SRP98	6535	14	C	F	31.4
SRP98	6571	14	A	M	18.9
SRP98	6616	17	B	F	26.3
SRP98	6620	14	A	M	18.0
SRP98	6924	17	A	M	18.5
SRP98	6928	14	D	F	34.6
SRP98	6948	16	C	M	77.7
SRP98	7036	17	A	M	57.6
SRP98	7064	17	A	M	42.2
SRP98	7104	14	D	M	16.0
SRP98	7124	17	A	M	ADULT
SRP98	7266	17	A	F	25.2
SRP98	7360	16	A	F	37.8
SRP98	7536	14	B	F	16.7
SRP98	7616	17	A	F	27.9
SRP98	7659	16	A	M	17.2
SRP98	7704	17	A	F	18.0

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	7906	17	A	F	20.6
SRP98	8194	17	A	M	22.4
SRP98	8279	15	B	F	19.0
SRP98	8343	16	A	M	18.5
SRP98	8372	14	A	M	42.2
SRP98	8425	16	A	F	31.5
SRP98	8539	17	B	M	27.3
SRP98	8650	17	A	F	70.4
SRP98	8781	17	A	F	18.0
SRP98	8849	17	C	M	ADULT
SRP98	8943	14	A	M	35.2
SRP98	9096	14	A	M	38.4
SRP98	9260	17	A	M	24.6
SRP98	9281	16	A	F	23.6
SRP98	9293	16	A	M	15.0
SRP98	9308	14	D	F	20.5
SRP98	9420	14	A	F	36.7
SRP98	9460	16	A	M	85.3
SRP98	9469	15	A	F	56.8
SRP98	9611	14	D	U	9.5
SRP98	9632	14	D	M	18.5
SRP98	9654	17	A	M	64.3
SRP98	9714	16	D	U	5.0
SRP98	9736	14	A	M	71.8
SRP98	9789	14	D	M	35.4
SRP98	9913	14	C	M	38.2
SRP98	9916	17	A	F	18.0
SRP98	10113	17	A	M	15.0
SRP98	10170	17	B	F	20.7
SRP98	10176	16	A	M	26.0
SRP98	10185	17	A	U	1.5
SRP98	10245	16	A	M	26.5
SRP98	10326	17	B	M	18.5
SRP98	10337	14	D	F	14.5
SRP98	10340	14	A	F	41.8
SRP98	10616	17	A	F	20.6
SRP98	10618	16	A	M	23.2
SRP98	10635	14	A	M	20.5
SRP98	10641	14	A	F	23.4
SRP98	10765	17	A	F	37.2
SRP98	10822	17	A	F	18.0

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	10837	16	A	F	ADULT
SRP98	11050	17	A	M	15.0
SRP98	11202	14	A	F	34.8
SRP98	11248	16	A	M	39.9
SRP98	11307	16	C	M	37.3
SRP98	11395	14	A	F	14.0
SRP98	11415	14	D	M	27.1
SRP98	11449	15	C	F	29.0
SRP98	11454	17	B	F	78.8
SRP98	11471	16	A	F	25.2
SRP98	11506	14	A	M	72.2
SRP98	11540	15	D	F	26.1
SRP98	11560	17	A	U	1.5
SRP98	11591	14	A	M	18.5
SRP98	11634	14	B	M	73.6
SRP98	11659	15	D	M	18.5
SRP98	11669	15	D	F	20.7
SRP98	11688	17	B	M	10.0
SRP98	11766	16	A	F	27.3
SRP98	11817	14	A	M	66.0
SRP98	11865	14	A	M	44.8
SRP98	11905	14	A	M	32.6
SRP98	11926	17	A	M	74.0
SRP98	11932	14	D	F	33.3
SRP98	11935	17	A	M	27.2
SRP98	11956	17	B	M	28.8
SRP98	11970	16	A	M	42.2
SRP98	11991	16	A	F	40.7
SRP98	12202	15	C	F	18.5
SRP98	12238	17	A	M	18.5
SRP98	12298	17	A	F	21.6
SRP98	12301	16	A	M	22.8
SRP98	12379	16	A	M	42.8
SRP98	12496	16	C	F	10.5
SRP98	12538	15	C	F	14.5
SRP98	12556	16	A	F	15.0
SRP98	12576	17	A	M	16.5
SRP98	12653	17	A	M	43.0
SRP98	12958	17	A	M	68.9
SRP98	13001	14	D	F	15.0
SRP98	13070	14	A	M	18.5

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	13232	17	A	F	18.0
SRP98	13366	15	D	F	26.8
SRP98	13384	16	D	M	35.3
SRP98	13467	17	A	M	31.1
SRP98	13614	14	A	F	69.2
SRP98	13635	16	A	F	19.5
SRP98	13661	14	D	F	18.8
SRP98	13870	14	A	F	35.0
SRP98	13880	16	A	F	70.9
SRP98	13909	17	A	F	27.0
SRP98	13982	17	A	U	0.0
SRP98	14004	14	A	F	20.5
SRP98	14067	17	A	F	77.3
SRP98	14096	14	C	F	29.4
SRP98	14109	17	A	F	18.0
SRP98	14276	14	A	F	32.5
SRP98	14379	14	A	F	30.6
SRP98	14381	15	D	M	15.0
SRP98	14567	14	B	M	19.5
SRP98	14574	15	B	F	32.3
SRP98	14600	14	A	F	13.5
SRP98	14694	15	C	M	23.3
SRP98	14882	14	D	M	31.4
SRP98	14934	15	C	F	37.3
SRP98	15277	15	B	M	9.0
SRP98	15337	14	A	U	10.0
SRP98	15573	15	D	F	9.0
SRP98	15841	15	D	U	5.0
SRP98	17539	16	A	M	81.0
SRP98	17649	14	A	F	15.0
SRP98	18138	16	A	F	18.0
SRP98	19016	15	B	M	24.9
SRP98	19034	17	D	M	56.1
SRP98	19058	17	A	F	18.5
SRP98	19124	14	A	M	31.9
SRP98	19147	17	D	M	16.5
SRP98	19150	17	A	M	40.0
SRP98	19164	17	D	M	26.3
SRP98	19173	17	D	M	18.5
SRP98	19260	17	D	U	8.0
SRP98	19261	17	D	U	5.0

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	19279	17	D	F	62.6
SRP98	19280	17	D	F	18.0
SRP98	19294	16	D	M	37.3
SRP98	19332	15	B	M	19.5
SRP98	19363	15	A	F	30.0
SRP98	19371	17	D	M	29.1
SRP98	19379	15	D	U	9.8
SRP98	19385	17	D	M	15.0
SRP98	19441	17	D	M	9.0
SRP98	19467	15	D	M	26.8
SRP98	19485	17	B	F	30.2
SRP98	19487	15	D	M	28.8
SRP98	19503	17	D	F	22.2
SRP98	19528	15	B	F	14.5
SRP98	19563	17	D	M	22.0
SRP98	19649	14	A	M	16.0
SRP98	19762	17	D	F	38.4
SRP98	19814	15	D	F	20.2
SRP98	19815	15	D	U	8.0
SRP98	19859	17	A	F	29.5
SRP98	19917	14	A	M	ADULT
SRP98	20160	14	B	M	20.5
SRP98	20161	14	B	U	12.0
SRP98	20187	14	B	F	38.3
SRP98	20350	15	D	M	37.1
SRP98	20435	15	D	M	19.5
SRP98	20462	17	A	M	22.9
SRP98	20463	17	A	U	10.0
SRP98	20481	14	B	F	25.0
SRP98	20545	16	D	U	12.0
SRP98	20559	17	A	U	15.0
SRP98	20563	15	D	M	18.0
SRP98	20590	17	A	F	31.9
SRP98	20592	15	D	M	39.4
SRP98	20634	17	A	F	16.0
SRP98	20637	15	D	M	6.0
SRP98	20682	15	D	M	34.9
SRP98	20764	17	A	M	16.5
SRP98	20807	16	D	M	28.8
SRP98	20932	15	B	M	77.7
SRP98	20940	17	A	M	45.1

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	21006	17	A	U	4.0
SRP98	21064	17	A	M	34.6
SRP98	21136	17	D	F	48.0
SRP98	21165	14	A	F	15.4
SRP98	21236	17	B	M	8.0
SRP98	21250	17	B	F	20.0
SRP98	21256	16	A	M	ADULT
SRP98	21273	14	A	F	24.9
SRP98	21298	17	A	M	21.5
SRP98	21308	16	D	U	6.0
SRP98	21371	17	A	F	19.2
SRP98	21402	14	A	M	14.5
SRP98	21412	15	C	M	18.5
SRP98	21431	17	D	M	ADULT
SRP98	21443	17	A	M	26.8
SRP98	21624	17	D	F	55.5
SRP98	21747	16	D	F	29.4
SRP98	21865	17	A	F	17.3
SRP98	21870	14	D	M	11.3
SRP98	21892	15	D	M	15.0
SRP98	21902	15	D	M	26.1
SRP98	21918	17	B	M	69.7
SRP98	21921	14	D	F	30.5
SRP98	22040	17	A	F	18.0
SRP98	22096	15	D	U	5.0
SRP98	22124	14	D	M	44.6
SRP98	22135	14	D	F	42.4
SRP98	22175	14	A	M	41.7
SRP98	22192	16	A	F	21.7
SRP98	22199	16	D	F	23.4
SRP98	22219	16	D	M	36.1
SRP98	22394	17	A	M	32.5
SRP98	22422	16	D	F	75.7
SRP98	22648	15	A	M	21.9
SRP98	22988	16	D	F	70.5
SRP98	23008	16	D	M	31.4
SRP98	23053	15	D	M	ADULT
SRP98	23090	15	A	U	14.5
SRP98	23131	14	B	F	12.0
SRP98	23163	17	A	F	40.7
SRP98	23276	14	D	U	10.0

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	23291	15	D	M	21.2
SRP98	23301	17	A	F	24.0
SRP98	23370	15	B	U	9.0
SRP98	23373	14	D	F	33.9
SRP98	23434	14	D	M	28.0
SRP98	23512	15	D	U	16.0
SRP98	23514	15	D	M	22.3
SRP98	23562	14	D	F	23.9
SRP98	23563	14	D	F	29.9
SRP98	23631	14	D	F	30.0
SRP98	23649	15	D	F	49.2
SRP98	23675	16	A	F	15.0
SRP98	23734	14	D	F	28.0
SRP98	23769	15	D	F	25.0
SRP98	24007	14	D	M	24.1
SRP98	24010	14	D	F	21.1
SRP98	24021	14	A	F	76.7
SRP98	24022	14	A	F	28.9
SRP98	24029	14	D	M	13.5
SRP98	24089	15	C	M	18.5
SRP98	24138	14	D	U	16.0
SRP98	24185	14	D	F	56.8
SRP98	24188	14	D	U	8.0
SRP98	25374	14	A	U	21.0
SRP98	25463	16	D	F	29.8
SRP98	25621	16	A	M	ADULT
SRP98	25999	14	A	M	26.8
SRP98	26079	16	B	F	30.4
SRP98	26163	15	D	U	15.0
SRP98	26179	14	D	M	74.9
SRP98	26182	14	D	M	25.8
SRP98	26296	14	A	F	ADULT
SRP98	26402	15	D	M	21.0
SRP98	26518	16	B	F	22.0
SRP98	26522	16	A	M	33.8
SRP98	26719	16	D	M	19.5
SRP98	27049	15	C	F	37.2
SRP98	27226	14	A	F	26.3
SRP98	27255	15	D	M	19.5
SRP98	27256	15	B	M	19.5
SRP98	27447	16	A	F	22.4

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	27463	14	B	F	29.6
SRP98	27476	14	A	M	30.6
SRP98	27544	15	A	F	18.2
SRP98	27593	14	A	F	15.5
SRP98	27608	16	C	M	71.8
SRP98	27659	14	A	M	33.5
SRP98	27674	14	D	F	24.3
SRP98	27690	16	D	F	23.8
SRP98	27777	14	A	F	18.5
SRP98	27799	16	D	F	19.5
SRP98	27815	16	D	F	24.9
SRP98	27824	14	D	F	36.2
SRP98	28107	14	A	M	15.0
SRP98	28110	16	D	M	65.2
SRP98	28161	16	D	M	73.5
SRP98	28197	15	C	U	9.0
SRP98	28199	15	C	M	18.5
SRP98	28298	16	A	U	5.0
SRP98	28438	15	C	F	20.2
SRP98	29050	17	C	F	20.9
SRP98	29058	16	D	M	21.3
SRP98	29062	16	D	F	15.0
SRP98	29188	16	D	F	79.8
SRP98	29698	15	A	F	22.7
SRP98	29857	16	B	M	15.0
SRP98	30039	15	A	F	19.5
SRP98	30135	15	D	U	14.5
SRP98	30165	16	A	M	ADULT
SRP98	30213	15	D	F	6.0
SRP98	30343	15	D	F	22.4
SRP98	30407	15	D	F	18.0
SRP98	30460	15	D	M	37.8
SRP98	30464	15	D	M	19.0
SRP98	30506	15	A	M	40.4
SRP98	30515	15	D	M	35.4
SRP98	30531	16	B	M	35.4
SRP98	30536	15	A	F	30.8
SRP98	30575	16	E	M	16.5
SRP98	30670	16	B	F	ADULT
SRP98	30829	16	D	M	76.1
SRP98	30838	15	D	U	5.0

Site Code	Context	Phase	Burial Type	Sex	Age
SRP98	30865	15	D	F	70.2
SRP98	30920	15	D	M	22.3
SRP98	30961	16	D	F	15.0
SRP98	30993	16	D	M	56.4
SRP98	31022	15	A	U	10.0
SRP98	31265	16	D	F	21.5
SRP98	31292	16	D	M	ADULT
SRP98	31315	16	D	U	10.5
SRP98	31320	16	D	F	35.1
SRP98	31328	16	D	M	30.3
SRP98	31341	15	B	F	22.3
SRP98	31358	16	D	F	48.6
SRP98	31389	16	D	F	16.2
SRP98	31390	16	D	U	9.8
SRP98	31416	16	D	M	35.2
SRP98	31426	16	D	M	29.3
SRP98	31430	16	D	F	33.5
SRP98	31431	16	A	U	7.5
SRP98	31437	16	D	U	7.0
SRP98	31439	16	D	F	18.0
SRP98	31472	15	D	U	11.8
SRP98	31572	15	D	F	24.9
SRP98	31687	16	D	M	32.5
SRP98	31706	15	D	M	15.3
SRP98	31753	16	D	M	35.8
SRP98	31849	16	B	F	40.9
SRP98	31878	16	D	M	23.3
SRP98	31892	16	A	M	76.1
SRP98	31910	16	D	U	9.5
SRP98	31925	16	D	F	18.0
SRP98	31956	16	A	F	20.8
SRP98	31962	15	D	F	21.9
SRP98	32091	15	A	F	44.0
SRP98	32217	16	D	F	21.2
SRP98	32222	16	D	F	67.9
SRP98	32251	16	D	F	16.0
SRP98	32279	16	D	M	18.5
SRP98	32293	15	A	M	36.2
SRP98	32302	15	D	F	19.5
SRP98	32303	15	D	M	19.5
SRP98	33235	15	D	M	18.5

!LJLJŜyŘʎÈ .π tI·fŜ2ŘŜY2ΞNI·LJKʎŎ 5I·ű·Ŧ2N {ű tŜűŜNΩă

Table G.1 Paleodemographic data for BOH, including context number, temporal phase, sex, and point estimates of age-at-death

Site Code	Context	Phase	Sex	Age
BOH	7	C	F	53.1
BOH	18	C	F	27.2
BOH	76	C	U	1.5
BOH	85	C	M	24.7
BOH	99	C	M	32.2
BOH	112	C	F	27.7
BOH	113	C	F	37.2
BOH	127	C	F	71.1
BOH	128	C	M	69.1
BOH	137	C	F	25.9
BOH	146	C	M	19.9
BOH	179	C	M	34.8
BOH	188	C	U	4.5
BOH	189	C	U	0.0
BOH	195	C	M	9.0
BOH	198	C	F	24.5
BOH	202	D	U	0.0
BOH	204	D	M	29.2
BOH	205	C	M	20.0
BOH	230	D	U	11.0
BOH	234	D	F	75.2
BOH	235	D	F	20.3
BOH	241	D	M	57.6
BOH	242	C	F	24.9
BOH	266	C	M	35.3
BOH	269	D	M	30.9
BOH	280	D	F	31.1
BOH	284	D	U	0.0
BOH	296	C	M	44.7
BOH	308	C	M	33.4
BOH	320	D	F	35.3

Site Code	Context	Phase	Sex	Age
BOH	326	D	F	28.8
BOH	338	D	U	0.0
BOH	339	C	M	16.0
BOH	340	C	M	12.0
BOH	360	D	U	6.0
BOH	363	D	M	58.9
BOH	365	D	U	0.0
BOH	366	D	U	10.0
BOH	371	D	U	0.2
BOH	379	D	F	37.6
BOH	380	D	M	22.2
BOH	429	D	M	10.0
BOH	431	C	F	61.4
BOH	437	C	U	5.0
BOH	446	C	M	45.1
BOH	448	D	U	4.0
BOH	467	D	F	20.2
BOH	468	C	M	56.2
BOH	469	D	F	24.2
BOH	471	C	F	18.2
BOH	475	C	U	10.5
BOH	483	D	U	5.0
BOH	484	C	M	ADULT
BOH	485	C	U	17.5
BOH	495	C	U	2.0
BOH	496	C	M	16.5
BOH	498	C	M	30.7
BOH	508	D	U	0.0
BOH	523	D	U	10.0
BOH	524	C	M	31.4
BOH	529	C	M	23.5
BOH	530	C	U	5.5
BOH	538	C	M	27.6
BOH	539	C	F	27.5
BOH	540	C	U	7.0
BOH	549	C	F	18.0
BOH	550	D	F	65.0
BOH	553	D	F	31.2
BOH	594	D	M	63.6
BOH	597	D	M	49.3
BOH	615	D	F	36.6
BOH	626	D	F	15.0

Site Code	Context	Phase	Sex	Age
BOH	641	C	M	18.5
BOH	649	C	M	31.8
BOH	650	C	M	75.8
BOH	652	C	F	37.8
BOH	653	C	M	75.3
BOH	660	C	M	31.7
BOH	667	C	F	68.8
BOH	668	D	F	30.6
BOH	671	D	M	40.2
BOH	675	C	F	28.2
BOH	681	C	U	3.0
BOH	684	C	M	26.9
BOH	685	C	F	43.9
BOH	687	C	F	34.0
BOH	688	C	F	52.6
BOH	691	C	F	41.0
BOH	697	C	F	24.9
BOH	700	C	U	8.0
BOH	705	C	F	40.3
BOH	707	C	U	11.5
BOH	710	D	M	26.6
BOH	715	C	U	0.0
BOH	717	D	F	37.1
BOH	725	D	U	5.0
BOH	729	D	U	8.0
BOH	730	C	F	24.8
BOH	731	C	F	24.1
BOH	732	C	M	29.3
BOH	737	C	M	43.4
BOH	738	C	F	41.8
BOH	742	D	M	19.0
BOH	744	D	U	8.0
BOH	748	C	F	30.5
BOH	749	C	F	25.2
BOH	754	C	F	44.1
BOH	755	C	F	35.9
BOH	756	D	U	5.0
BOH	757	D	U	2.0
BOH	759	D	M	29.9
BOH	765	C	M	35.6
BOH	766	D	M	16.5
BOH	767	D	M	44.6

Site Code	Context	Phase	Sex	Age
BOH	771	C	F	48.5
BOH	780	D	U	0.0
BOH	782	D	U	2.0
BOH	784	C	U	10.5
BOH	786	D	F	32.7
BOH	788	C	F	52.2
BOH	791	D	U	7.0
BOH	798	D	U	3.0
BOH	800	C	M	50.1
BOH	806	D	U	3.0
BOH	810	D	M	12.0
BOH	811	D	F	25.9
BOH	813	D	F	18.0
BOH	820	C	U	1.5
BOH	848	D	F	15.0
BOH	850	D	F	48.8
BOH	851	D	M	69.1
BOH	857	D	U	7.0
BOH	858	D	F	30.3
BOH	859	D	M	27.4
BOH	879	D	U	5.0
BOH	882	D	U	6.0
BOH	949	D	F	25.8
BOH	950	D	M	38.4
BOH	961	D	M	16.0
BOH	963	D	U	6.0
BOH	966	D	M	35.6
BOH	973	D	F	35.4
BOH	974	D	F	33.9
BOH	981	D	M	35.9
BOH	997	D	M	25.2
BOH	1022	D	M	57.6
BOH	1023	D	M	16.5
BOH	1025	D	M	29.6
BOH	1042	D	F	27.3
BOH	1046	D	F	33.2
BOH	1050	D	F	36.0
BOH	1051	C	M	74.2
BOH	1063	C	M	73.7
BOH	1095	D	M	16.0
BOH	1097	D	U	3.0
BOH	1133	D	F	66.5

Site Code	Context	Phase	Sex	Age
BOH	1134	D	M	33.5
BOH	1175	C	M	ADULT
BOH	1178	C	F	63.5
BOH	1184	C	F	18.0
BOH	1201	D	M	33.2
BOH	1217	D	M	25.5
BOH	1236	D	F	34.0
BOH	1246	D	F	11.5
BOH	1287	D	U	0.0
BOH	1320	D	F	81.8
BOH	1597	C	U	5.0
BOH	1638	C	U	4.0
BOH	1639	C	M	29.0
BOH	1640	C	F	23.7
BOH	1674	D	F	29.1
BOH	1676	D	M	11.0
BOH	1701	C	U	5.0
BOH	1707	D	M	26.2
BOH	1755	C	U	6.0
BOH	1773	D	M	17.5
BOH	1777	D	M	9.0
BOH	1805	D	M	54.4
BOH	1822	D	M	26.0
BOH	2038	D	F	24.5
BOH	2077	D	F	16.1
BOH	2171	D	M	7.0
BOH	2183	D	U	5.0
BOH	2187	D	F	76.8
BOH	2188	D	F	23.2
BOH	2219	D	M	41.1
BOH	2221	C	U	0.0
BOH	2269	C	U	9.0
BOH	2291	C	M	29.2
BOH	2294	C	M	43.9
BOH	2295	C	F	20.6
BOH	2299	D	U	0.0
BOH	2300	D	U	10.0
BOH	2301	C	M	70.9
BOH	2310	C	U	ADULT
BOH	2324	C	F	32.0
BOH	2327	D	M	31.8
BOH	2328	D	U	6.0

Site Code	Context	Phase	Sex	Age
BOH	2348	C	F	30.0
BOH	2359	C	M	60.7
BOH	2361	D	M	22.7
BOH	2375	C	M	12.0
BOH	2378	C	M	32.6
BOH	2380	D	U	0.0
BOH	2397	D	F	12.0
BOH	2420	D	U	2.0
BOH	2422	D	F	35.3
BOH	2424	D	U	9.0
BOH	2436	C	M	31.5
BOH	2456	D	U	7.0
BOH	2460	D	U	1.5
BOH	2503	D	F	23.0
BOH	2690	C	M	16.0
BOH	2701	C	F	40.9
BOH	2702	C	M	16.5
BOH	2706	C	M	ADULT
BOH	2708	C	F	ADULT
BOH	2714	C	M	63.7
BOH	2718	C	F	22.9
BOH	2720	C	M	45.2
BOH	2735	C	U	1.5
BOH	2737	D	U	0.0
BOH	2741	D	U	7.0
BOH	2746	D	F	30.3

Appendix C- Stable Isotope Data for Bone Collagen Samples

Table H.1 Stable isotope data of bone collagen samples from rib in St Mary Spital, including context number, $\delta^{15}N$ ‰, $\delta^{13}C$ ‰, nitrogen content, carbon content, carbon to nitrogen ratio, source of data (W = this study, L = Lakin (2010)), and sample viability

Site Code	Context	δ15N ‰	δ13C ‰	%N	%C	C:N	Source	Viable Sample
SRP98	1055	10.69	-19.38	15.20	41.65	3.20	W	Y
SRP98	2481	12.70	-18.75	14.81	40.57	3.20	W	Y
SRP98	2487	13.47	-18.54	14.67	40.47	3.22	W	Y
SRP98	2679	13.54	-18.88	15.26	41.56	3.18	W	Y
SRP98	2687	12.20	-19.40	15.31	42.46	3.24	L	Y
SRP98	2694	13.93	-18.43	14.75	40.35	3.19	W	Y
SRP98	3166	12.32	-19.12	15.28	41.49	3.17	W	Y
SRP98	3180	10.61	-19.99	15.18	41.31	3.18	W	Y
SRP98	3201	11.13	-19.49	14.69	40.20	3.19	W	Y
SRP98	3292	13.40	-18.89	13.86	38.75	3.26	W	Y
SRP98	3645	13.78	-18.71	14.86	40.70	3.20	W	Y
SRP98	3676	12.88	-19.07	14.84	40.74	3.20	W	Y
SRP98	3738	13.39	-18.82	15.29	41.71	3.18	W	Y
SRP98	3775	13.43	-19.11	14.45	39.92	3.22	W	Y
SRP98	3805	12.11	-19.75	15.76	42.72	3.16	W	Y
SRP98	3806	13.00	-18.80	15.97	42.87	3.13	L	Y

SRP98	3825	13.89	-18.10	13.89	38.27	3.21	W	Y
SRP98	3934	13.70	-18.86	15.10	41.09	3.17	W	Y
SRP98	3975	13.50	-18.89	14.49	39.67	3.19	W	Y
SRP98	5018	12.00	-19.36	15.31	41.95	3.20	W	Y
SRP98	5287	13.19	-19.09	14.64	40.14	3.20	W	Y
SRP98	5561	13.20	-19.17	14.75	40.19	3.18	W	Y
SRP98	5677	12.95	-19.64	15.47	41.93	3.16	W	Y
SRP98	5907	11.31	-20.02	15.24	41.31	3.16	W	Y
SRP98	6427	14.10	-18.24	14.83	40.81	3.21	W	Y
SRP98	6571	13.17	-19.56	14.95	40.85	3.19	W	Y
SRP98	6616	12.85	-19.50	15.63	42.56	3.18	W	Y
SRP98	6620	12.78	-19.45	14.57	40.03	3.20	W	Y
SRP98	6924	13.30	-19.38	14.86	40.54	3.18	W	Y
SRP98	7064	12.18	-19.91	14.94	40.81	3.19	W	Y
SRP98	7104	11.90	-19.13	15.58	41.99	3.14	W	Y
SRP98	7124	13.69	-19.48	13.93	40.33	3.38	W	Y
SRP98	7266	12.75	-19.71	15.16	41.29	3.18	W	Y
SRP98	7616	11.20	-20.04	15.04	40.84	3.17	W	Y
SRP98	8194	11.64	-19.52	14.72	40.07	3.18	W	Y
SRP98	8372	12.73	-19.16	14.65	40.15	3.20	W	Y
SRP98	8781	13.10	-18.79	13.43	36.68	3.19	W	Y
SRP98	8943	14.41	-18.71	14.65	40.08	3.19	W	Y
SRP98	9420	13.32	-18.57	14.55	39.74	3.19	W	Y
SRP98	9632	12.74	-19.36	15.20	41.21	3.16	W	Y
SRP98	9736	14.69	-18.57	15.24	41.54	3.18	W	Y
SRP98	9789	13.00	-18.77	14.67	40.54	3.23	W	Y
SRP98	9916	13.23	-18.79	14.56	39.99	3.20	W	Y
SRP98	10113	10.45	-20.48	13.28	36.26	3.18	W	Y

SRP98	10340	13.53	-19.08	15.00	40.51	3.15	W	Y
SRP98	10635	13.30	-18.59	11.91	33.18	3.25	W	Y
SRP98	10765	13.83	-19.46	13.10	36.20	3.22	W	Y
SRP98	10837	12.96	-19.31	12.41	34.10	3.21	W	Y
SRP98	11050	12.85	-19.41	13.90	38.17	3.20	W	Y
SRP98	11471	13.23	-18.67	13.24	36.57	3.22	W	Y
SRP98	11506	12.98	-18.83	14.08	38.62	3.20	W	Y
SRP98	11540	10.30	-19.20	14.49	38.83	3.13	L	Y
SRP98	11669	13.70	-18.80	15.43	42.68	3.23	L	Y
SRP98	11688	13.18	-19.57	12.57	34.75	3.23	W	Y
SRP98	11926	13.12	-18.76	14.95	40.59	3.17	W	Y
SRP98	12298	11.74	-19.44	14.66	39.64	3.15	W	Y
SRP98	12301	9.68	-20.47	11.62	32.10	3.22	W	Y
SRP98	12379	12.99	-19.10	15.11	41.10	3.17	W	Y
SRP98	12496	12.72	-19.21	14.90	40.53	3.17	W	Y
SRP98	12958	13.81	-18.84	14.62	39.95	3.19	W	Y
SRP98	13001	11.89	-19.38	13.74	37.55	3.19	W	Y
SRP98	13366	11.85	-19.35	15.21	41.32	3.17	W	Y
SRP98	13467	11.83	-19.69	14.80	40.77	3.21	W	Y
SRP98	13870	12.39	-19.21	15.13	40.91	3.16	W	Y
SRP98	13880	13.01	-18.58	15.14	41.22	3.18	W	Y
SRP98	13909	11.73	-19.57	15.01	41.19	3.20	W	Y
SRP98	14004	13.07	-19.14	14.46	39.55	3.19	W	Y
SRP98	14109	12.43	-19.86	15.05	41.16	3.19	W	Y
SRP98	14276	12.77	-18.94	15.29	41.89	3.20	W	Y
SRP98	14379	13.32	-18.90	15.13	41.11	3.17	W	Y
SRP98	14694	11.88	-19.46	14.24	39.58	3.24	W	Y
SRP98	14882	14.43	-18.20	14.96	40.98	3.20	W	Y

SRP98	14934	12.68	-19.19	13.89	38.66	3.25	W	Y
SRP98	17539	10.62	-19.72	14.83	40.83	3.21	W	Y
SRP98	18138	13.12	-18.98	15.15	40.89	3.15	W	Y
SRP98	19147	13.41	-19.24	14.89	40.67	3.19	W	Y
SRP98	19260	12.71	-19.16	15.03	41.20	3.20	W	Y
SRP98	19261	13.01	-19.33	14.58	40.07	3.21	W	Y
SRP98	19280	12.27	-19.15	14.99	41.18	3.21	W	Y
SRP98	19294	12.53	-19.03	14.76	40.49	3.20	W	Y
SRP98	19363	13.44	-18.91	14.64	40.31	3.21	W	Y
SRP98	19379	12.53	-18.59	15.37	41.53	3.15	W	Y
SRP98	19441	11.41	-19.75	15.53	42.30	3.18	W	Y
SRP98	19503	13.43	-18.94	14.79	40.81	3.22	W	Y
SRP98	19815	12.23	-18.69	15.45	41.90	3.16	W	Y
SRP98	20160	12.78	-19.40	15.22	41.16	3.16	W	Y
SRP98	20350	13.03	-18.51	15.06	41.43	3.21	W	Y
SRP98	20462	12.66	-18.79	13.44	36.81	3.19	W	Y
SRP98	20481	12.86	-18.85	15.07	41.04	3.18	W	Y
SRP98	20563	10.87	-20.15	14.61	40.02	3.20	W	Y
SRP98	20682	13.19	-18.31	14.72	39.92	3.16	W	Y
SRP98	20764	12.65	-19.33	14.49	39.47	3.18	W	Y
SRP98	20807	13.35	-19.95	14.99	40.94	3.19	W	Y
SRP98	20932	11.69	-19.37	14.85	40.25	3.16	W	Y
SRP98	21236	10.81	-10.01	14.04	38.40	3.19	W	Y
SRP98	21250	12.85	-18.77	14.70	39.78	3.16	W	Y
SRP98	21256	13.15	-19.30	14.63	40.09	3.20	W	Y
SRP98	21273	11.89	-19.23	14.02	37.29	3.10	W	Y
SRP98	21308	9.37	-20.59	14.44	38.74	3.13	W	Y
SRP98	21371	14.08	-18.85	14.44	39.34	3.18	W	Y

SRP98	21402	13.22	-19.04	14.56	38.82	3.11	W	Y
SRP98	21747	12.56	-18.86	15.24	40.93	3.13	W	Y
SRP98	21892	11.40	-19.20	15.79	43.46	3.21	L	Y
SRP98	21921	10.85	-20.07	14.43	39.60	3.20	W	Y
SRP98	22096	11.80	-19.00	15.02	41.35	3.21	L	Y
SRP98	22135	12.58	-18.92	15.21	40.45	3.10	W	Y
SRP98	22175	12.63	-19.59	14.70	40.04	3.18	W	Y
SRP98	22199	10.85	-19.42	15.04	40.64	3.15	W	Y
SRP98	22422	12.67	-18.56	15.09	40.97	3.17	W	Y
SRP98	22648	13.19	-18.92	14.87	40.88	3.21	W	Y
SRP98	22988	12.65	-18.84	13.30	36.39	3.19	W	Y
SRP98	23008	12.19	-19.53	14.01	38.05	3.17	W	Y
SRP98	23053	13.54	-18.19	14.79	40.22	3.17	W	Y
SRP98	23090	12.17	-18.99	13.52	36.82	3.18	W	Y
SRP98	23276	11.03	-18.98	14.14	38.09	3.15	W	Y
SRP98	23434	9.91	-20.12	12.11	32.84	3.16	W	Y
SRP98	23512	12.53	-19.13	12.06	32.97	3.19	W	Y
SRP98	23514	12.30	-18.80	15.92	43.38	3.18	L	Y
SRP98	23562	12.18	-19.59	15.39	41.84	3.17	W	Y
SRP98	23563	13.07	-18.94	9.65	26.28	3.18	W	Y
SRP98	23769	12.49	-18.91	8.89	24.43	3.21	W	Y
SRP98	24010	13.00	-19.13	19.40	52.37	3.15	W	N
SRP98	24021	11.43	-19.39	16.70	45.13	3.15	W	Y
SRP98	24022	13.12	-18.69	23.29	63.31	3.16	W	N
SRP98	24138	12.79	-18.25	15.47	42.01	3.17	W	Y
SRP98	24185	10.67	-19.41	13.75	37.21	3.16	W	Y
SRP98	25621	14.35	-18.02	12.35	33.65	3.18	W	Y
SRP98	26163	12.28	-18.74	14.67	39.78	3.16	W	Y

SRP98	26182	13.21	-18.54	13.57	36.52	3.14	W	Y
SRP98	26402	12.44	-19.02	13.15	35.75	3.17	W	Y
SRP98	26522	12.43	-19.12	13.12	37.52	3.34	W	Y
SRP98	27226	12.73	-18.79	11.56	31.80	3.21	W	Y
SRP98	27256	11.72	-19.19	11.72	33.16	3.19	W	Y
SRP98	27476	12.03	-19.03	12.03	33.72	3.19	W	Y
SRP98	27544	11.76	-19.77	13.17	36.14	3.20	W	Y
SRP98	27608	12.18	-22.07	11.36	44.79	3.51	W	Y
SRP98	27815	10.90	-19.86	14.84	40.44	3.18	W	Y
SRP98	29050	11.39	-19.31	5.41	15.30	3.30	W	Y
SRP98	29058	11.91	-18.97	9.27	25.48	3.21	W	Y
SRP98	29698	11.64	-19.53	15.51	42.05	3.16	W	Y
SRP98	29857	11.60	-19.54	14.17	38.53	3.17	W	Y
SRP98	30039	14.60	-17.70	16.10	44.11	3.20	L	Y
SRP98	30135	12.18	-26.37	1.60	18.37	2.98	W	N
SRP98	30343	12.72	-19.11	14.00	38.57	3.21	W	Y
SRP98	30407	10.80	-20.10	14.23	40.38	3.31	L	Y
SRP98	30464	10.33	-19.51	14.52	39.69	3.19	W	Y
SRP98	30515	No result	No result	No result	No result	No result	W	N
SRP98	30531	13.45	-18.81	14.86	41.22	3.24	W	Y
SRP98	30575	12.87	-18.65	14.31	38.80	3.58	W	Y
SRP98	30829	10.98	-19.77	14.74	40.20	3.18	W	Y
SRP98	30838	12.30	-18.60	16.86	45.00	3.12	L	Y
SRP98	30865	No result	No result	No result	No result	No result	W	N
SRP98	30920	11.55	-19.73	14.77	39.95	3.15	W	Y
SRP98	31022	8.50	-18.90	16.07	43.06	3.13	L	Y
SRP98	31320	13.60	-18.66	14.71	40.23	3.19	W	Y
SRP98	31328	10.79	-20.45	13.30	35.66	3.13	W	Y

SRP98	31341	10.87	-19.29	14.83	40.40	3.18	W	Y
SRP98	31358	11.09	-20.45	15.22	41.04	3.15	W	Y
SRP98	31416	12.94	-18.56	15.17	41.01	3.15	W	Y
SRP98	31472	11.30	-19.60	14.83	41.05	3.23	L	Y
SRP98	31687	11.95	-19.39	14.35	39.69	3.23	W	Y
SRP98	31706	13.31	-18.34	14.97	40.86	3.18	W	Y
SRP98	31753	12.84	-19.01	15.12	41.60	3.21	W	Y
SRP98	31910	12.36	-18.83	15.08	40.80	3.16	W	Y
SRP98	31925	12.73	-18.59	15.17	41.22	3.17	W	Y
SRP98	31956	11.58	-19.62	14.61	39.56	3.16	W	Y
SRP98	31962	10.13	-19.83	15.36	41.74	3.17	W	Y
SRP98	32091	12.90	-18.60	15.84	42.50	3.13	L	Y
SRP98	32222	13.80	-18.66	14.27	38.96	3.19	W	Y
SRP98	32251	12.01	-19.11	14.43	39.92	3.23	W	Y
SRP98	32293	12.81	-19.05	15.14	41.23	3.17	W	Y
SRP98	32302	11.10	-19.78	14.81	40.14	3.16	W	Y
SRP98	33235	12.63	-19.04	14.78	40.11	3.17	W	Y

Appendix D- Stable Isotope Data for Dentine Samples

Table D.1: Stable isotope data for incremental dentine samples for each dentine section from St Mary Spital, including SRP98-2487, -2679, -3934, -6571, -7266, -9420, -9632, -9789, and -10765.

	2487	**2679**	**3934**	**5287**	**6571**	**7266**	**9420**	**9632**	**9789**	**10765**
δ^{15}N 1	14.21	14.29	11.08	10.87	12.64	12.02	14.11	13.98	9.79	10.33
δ^{13}C 1	-18.56	-19.24	-20.49	-19.92	-20.84	-20.12	-18.98	-19.78	-19.91	-20.64
δ^{15}N 2	13.46	13.38	11.07	10.84	12.10	12.31	14.08	13.39	9.48	10.42
δ^{13}C 2	-18.38	-19.37	-20.18	-19.64	-20.62	-19.94	-18.73	-19.68	-20.23	-20.33
δ^{15}N 3	13.32	12.59	11.02	10.17	12.07	12.04	13.77	12.92	9.23	11.01
δ^{13}C 3	-18.31	-19.32	-19.96	-19.51	-20.39	-20.08	-18.81	-19.74	-20.49	-19.86
δ^{15}N 4	13.47	11.64	10.78	9.36	12.19	11.83	13.71	12.54	9.41	11.28
δ^{13}C 4	-18.13	-19.09	-20.48	-19.55	-20.25	-20.18	-18.97	-19.80	-20.60	-19.87
δ^{15}N 5	13.66	11.46	10.53	9.18	12.36	11.69	13.93	11.53	9.67	11.17
δ^{13}C 5	-18.00	-19.17	-20.21	-19.46	-19.93	-20.00	-19.42	-19.71	-20.93	-19.94
δ^{15}N 6	13.62	11.52	12.50	11.41	11.80	11.60	12.77	11.82	9.55	11.37
δ^{13}C 6	-17.94	-19.23	-19.08	-19.06	-19.71	-20.13	-19.82	-19.45	-20.82	-19.95
δ^{15}N 7	13.13	11.83	12.94	12.09	12.78	11.49	12.19	12.64	8.86	11.38
δ^{13}C 7	-18.18	-19.16	-19.13	-18.73	-19.62	-20.22	-19.69	-19.55	-20.53	-19.95
δ^{15}N 8	13.39	12.24	13.77	12.53	12.86	11.45	12.79	12.36	8.97	11.08
δ^{13}C 8	-18.16	-18.73	-18.82	-18.45	-19.92	-20.27	-19.58	-19.78	-20.22	-20.00
δ^{15}N 9	13.41	12.17	14.11	12.51	13.13	11.29	12.96	11.67	9.62	11.12

δ^{13}C 9	-18.06	-18.67	-18.12	-18.22	-19.73	-20.27	-19.53	-19.85	-19.83	-19.92
δ^{15}N 10	13.41	12.27	13.85	12.42	13.60	11.14	12.51	11.41	9.93	10.78
δ^{13}C 10	-18.12	-18.58	-18.38	-18.09	-19.54	-20.27	-19.59	-19.54	-19.58	-20.02
δ^{15}N 11	13.23	12.36	12.68	12.56	13.35	11.38	12.40	11.67	9.99	10.80
δ^{13}C 11	-18.23	-18.57	-19.96	-18.26	-19.50	-20.19	-19.26	-19.52	-19.64	-19.99
δ^{15}N 12	13.09	12.38	12.66	12.50	13.30	11.57	13.04	11.60	10.00	10.71
δ^{13}C 12	-18.24	-18.49	-19.02	-18.39	-19.50	-20.39	-18.87	-19.61	-19.74	-10.09
δ^{15}N 13	13.23	12.23	12.10	12.49	13.27	12.00	13.22	11.52	10.00	10.78
δ^{13}C 13	-18.19	-18.53	-19.19	-18.40	-19.52	-20.11	-18.69	-19.77	-19.69	-10.06
δ^{15}N 14	13.34	12.09	10.95	12.45	13.50	12.81	13.10	11.46	9.85	10.99
δ^{13}C 14	-18.19	-18.61	-19.48	-18.60	-19.65	-19.65	-18.85	-19.79	-20.04	-20.06
δ^{15}N 15	13.37	12.27	12.00	12.72	13.56	12.87	13.19	11.55	9.75	11.15
δ^{13}C 15	-18.12	-18.64	-19.83	-18.52	-19.67	-19.60	-19.04	-19.88	-20.11	-19.92
δ^{15}N 16	13.55	12.60		12.88	13.40	13.10	13.26	11.72	9.87	11.45
δ^{13}C 16	-18.02	-18.72		-18.63	-19.68	-19.44	-18.96	-19.70	-19.98	-19.89
δ^{15}N 17	13.47	12.58		13.43	13.68	13.59	13.60	11.98	10.29	12.21
δ^{13}C 17	-18.18	-18.94		-18.69	-19.50	-19.52	-18.89	-19.68	-19.67	-19.73
δ^{15}N 18	13.11				13.99		14.10	12.31	10.60	
δ^{13}C 18	-18.42				-19.40		-18.98	-19.71	-19.74	
δ^{15}N 19								12.92	11.03	
δ^{13}C 19								-19.40	-19.78	
δ^{15}N 20								13.37	11.25	
δ^{13}C 20								-19.25	-19.83	

Table D.2: Stable isotope data for incremental dentine samples for each dentine section from St Mary Spital, including SRP98-11471, -12379, -17539, -18138, -19147, -19503, -20462, -20682, and -21273.

	11471	**12379**	**17539**	**18138**	**19147**	**19294**	**19503**	**20462**	**20682**	**21273**
δ^{15}N 1	10.01	14.11	11.91	13.80	11.71	12.22	14.11	11.71	12.20	10.98
δ^{13}C 1	-19.71	-19.20	-20.24	-18.95	-19.60	-20.22	-19.09	-19.93	-19.90	-20.14
δ^{15}N 2	9.77	13.61	11.61	13.63	11.50	12.37	14.09	10.73	11.35	11.07
δ^{13}C 2	-19.83	-19.01	-20.05	-18.65	-19.62	-20.07	-18.97	-19.93	-20.01	-20.00
δ^{15}N 3	9.69	13.49	11.27	13.34	11.88	12.19	13.64	9.69	10.96	11.92
δ^{13}C 3	-19.67	-18.97	-20.29	-18.58	-19.69	-20.08	-19.22	-20.22	-19.65	-19.76
δ^{15}N 4	9.99	12.75	11.59	13.51	12.85	12.17	13.43	9.16	11.42	11.91
δ^{13}C 4	-19.41	-19.20	-19.89	-18.78	-19.57	-19.81	-19.19	-20.30	-19.17	-19.95
δ^{15}N 5	9.45	12.40	11.66	13.40	13.13	12.05	13.18	9.12	12.92	11.55
δ^{13}C 5	-19.69	-19.19	-19.89	-18.98	-19.28	-19.51	-19.26	-19.86	-19.12	-19.96
δ^{15}N 6	9.20	12.51	11.55	13.49	12.39	11.65	11.97	8.90	12.89	11.20
δ^{13}C 6	-19.68	-19.43	-20.02	-19.00	-19.22	-19.57	-19.37	-19.76	-19.43	-19.60
δ^{15}N 7	9.35	12.77	11.22	13.42	12.00	11.55	11.37	8.93	12.78	11.45
δ^{13}C 7	-19.76	-19.26	-19.91	-18.88	-19.25	-19.58	-19.56	-19.55	-19.47	-19.14
δ^{15}N 8	9.13	12.24	10.99	13.18	12.43	11.71	12.55	9.07	12.93	12.22
δ^{13}C 8	-19.97	-19.05	-20.23	-18.67	-19.31	-19.39	-19.34	-19.40	-19.38	-19.23
δ^{15}N 9	9.05	12.27	10.90	12.82	12.67	11.95	12.12	9.46	12.30	12.72
δ^{13}C 9	-19.96	-18.78	-19.94	-18.58	-18.95	-19.39	-19.07	-19.28	-19.33	-19.33
δ^{15}N 10	9.01	12.50	11.05	12.74	12.83	12.16	12.39	10.04	11.86	12.70
δ^{13}C 10	-19.85	-18.73	-19.67	-18.50	-18.94	-19.36	-19.08	-19.05	-19.38	-19.28
δ^{15}N 11	9.48	12.78	10.82	12.72	12.13	12.38	12.55	10.70	11.52	12.53
δ^{13}C 11	-19.50	-18.74	-19.54	-18.51	-19.26	-19.46	-18.92	-18.90	-19.41	-19.02
δ^{15}N 12	9.66	12.87	10.77	12.82	12.02	12.54	13.09	11.11	11.55	12.36

δ^{13}C 12	-19.36	-18.80	-19.56	-18.72	-19.18	-19.60	-18.65	-18.85	-19.39	-19.02
δ^{15}N 13	9.81	12.76	10.73	12.94	12.30	12.61	13.26	11.41	11.50	12.32
δ^{13}C 13	-19.36	-18.94	-19.66	-19.68	-19.14	-19.66	-18.28	-19.04	-19.26	-19.06
δ^{15}N 14	9.71	12.44	11.02		12.25	12.46	13.50	11.62	11.38	12.27
δ^{13}C 14	-19.29	-18.85	-19.78		-18.93	-19.69	-18.29	-19.07	-19.10	-19.10
δ^{15}N 15	9.98	12.50	11.34		12.09	12.06	13.52	11.71	11.24	12.16
δ^{13}C 15	-19.17	-18.83	-19.68		-18.92	-19.66	-18.39	-19.20	-19.02	-19.17
δ^{15}N 16	10.38	12.70	10.79		12.40	12.23	13.78	11.38	11.32	12.19
δ^{13}C 16	-19.15	-18.83	-20.07		-18.89	-19.53	-18.49	-19.36	-19.22	-19.28
δ^{15}N 17	10.39	12.68	9.99		12.73	12.47		10.46	11.07	12.34
δ^{13}C 17	-19.18	-19.01	-20.20		-18.82	-19.38		-19.36	-19.52	-19.26
δ^{15}N 18	10.39	13.14	9.97		12.89			11.31	11.45	12.43
δ^{13}C 18	-19.30	-19.14	-19.96		-18.66			-19.19	-19.55	-19.33
δ^{15}N 19	11.07	12.84	10.93		12.95			12.49	12.90	12.77
δ^{13}C 19	-19.31	-19.34	-19.47		-18.82			-19.01	-19.24	-19.25
δ^{15}N 20	11.26									
δ^{13}C 20	-19.38									

Table D.3: Stable isotope data for incremental dentine samples for each dentine section from St Mary Spital, including SRP98-22199, -24010, -24185, -26522, -27476, -29058, -29698, -30920, -32222, and -32302

	22199	**24010**	**24185**	**26522**	**27476**	**29058**	**29698**	**30920**	**32222**	**32302**
δ^{15}N 1	11.63	14.10	10.84	13.42	9.72	13.05	12.06	11.45	14.13	13.64
δ^{13}C 1	-19.99	-20.16	-19.97	-18.92	-20.51	-19.33	-20.08	-20.04	-18.60	-20.01
δ^{15}N 2	10.09	13.91	10.41	13.48	8.97	13.75	10.86	9.60	14.22	13.16
δ^{13}C 2	-20.03	-19.84	-19.80	-18.58	-20.40	-19.41	-20.00	-20.33	-18.56	-19.78
δ^{15}N 3	10.14	13.80	10.15	13.34	9.11	12.76	10.33	10.53	14.06	12.91
δ^{13}C 3	-20.21	-19.45	-19.77	-18.28	-20.09	-19.75	-20.02	-20.50	-18.53	-19.64
δ^{15}N 4	10.42	13.33	10.11	13.25	9.75	12.26	10.75	10.96	14.13	12.83
δ^{13}C 4	-20.01	-19.10	-19.95	-18.51	-19.93	-19.84	-19.63	-20.58	-18.57	-19.49
δ^{15}N 5	10.28	13.55	10.25	13.30	10.49	10.46	11.14	10.89	13.70	12.71
δ^{13}C 5	-19.83	-19.04	-19.91	-18.46	-20.05	-19.57	-19.65	-20.55	-18.71	-19.39
δ^{15}N 6	10.69	13.44	10.50	12.85	11.11	9.90	11.26	10.96	13.51	12.66
δ^{13}C 6	-19.45	-19.15	-19.84	-18.68	-19.55	-19.57	-19.68	-20.35	-18.82	-19.15
δ^{15}N 7	11.49	13.55	10.55	12.12	10.49	10.01	10.95	10.62	13.44	12.32
δ^{13}C 7	-19.28	-19.11	-19.51	-18.81	-19.63	-19.82	-19.67	-20.08	-18.83	-19.10
δ^{15}N 8	11.81	13.44	10.16	11.98	11.52	9.77	10.99	10.29	13.48	12.42
δ^{13}C 8	-19.05	-19.08	-19.69	-18.52	-19.30	-19.99	-19.43	-19.99	-18.74	-19.08
δ^{15}N 9	12.00	13.34	9.86	12.00	11.79	9.71	11.16	10.02	13.72	12.54
δ^{13}C 9	-19.08	-19.00	-19.39	-18.43	-19.17	-20.00	-19.39	-19.96	-18.65	-19.10
δ^{15}N 10	11.91	13.43	12.27	12.18	11.64	9.96	11.26	9.82	13.90	12.52
δ^{13}C 10	-19.04	-18.89	-18.58	-18.35	-19.13	-19.71	-19.55	-19.93	-18.71	-19.24
δ^{15}N 11	12.12	13.38	12.36	12.44	11.61	10.33	11.09	9.81	14.00	12.50
δ^{13}C 11	-19.27	-18.82	-18.57	-18.44	-18.90	-19.45	-19.57	-19.97	-18.69	-19.18
δ^{15}N 12	11.62	13.24	12.38	12.62	11.69	10.80	10.79	9.64	13.86	12.64
δ^{13}C 12	-19.52	-18.89	-18.49	-18.43	-19.08	-19.27	-19.61	-20.01	-18.80	-19.01

δ^{15}N 13	11.12	13.09	12.23	12.52	11.96	11.23	10.94	9.74	13.84	12.97
δ^{13}C 13	-19.32	-18.88	-18.53	-18.45	-19.24	-19.15	-19.69	-19.93	-18.44	-19.01
δ^{15}N 14	11.20	13.02	12.09	12.26	11.89	11.59	11.26	9.56	13.74	13.08
δ^{13}C 14	-19.41	-19.03	-18.61	-18.67	-19.62	-19.01	-19.63	-19.97	-18.41	-19.01
δ^{15}N 15	11.09	13.12	12.27	12.14	11.86	11.96	11.64	9.54	13.68	13.28
δ^{13}C 15	-19.29	-18.88	-18.64	-18.68	-19.40	-18.85	-19.63	-19.86	-18.50	-19.14
δ^{15}N 16	11.58	13.15	12.60	11.91	12.27	12.46	12.04	9.56	14.08	13.38
δ^{13}C 16	-19.52	-18.84	-18.72	-18.81	-19.31	-19.11	-19.29	-19.81	-18.43	-18.99
δ^{15}N 17	12.24	13.22	12.58	12.03		12.63		9.56	14.81	13.32
δ^{13}C 17	-19.44	-19.12	-18.94	-18.67		-19.11		-19.85	-19.29	-19.01
δ^{15}N 18	11.91	13.21		12.26		12.46		9.62		13.37
δ^{13}C 18	-19.60	-19.15		-18.54		-19.04		-20.02		-19.07
δ^{15}N 19	10.98			12.31		12.28		10.88		13.00
δ^{13}C 19	-20.08			-18.82		-19.11		-19.97		-19.30
δ^{15}N 20						12.16				
δ^{13}C 20						-19.13				
δ^{15}N 21						12.47				
δ^{13}C 21						-19.13				

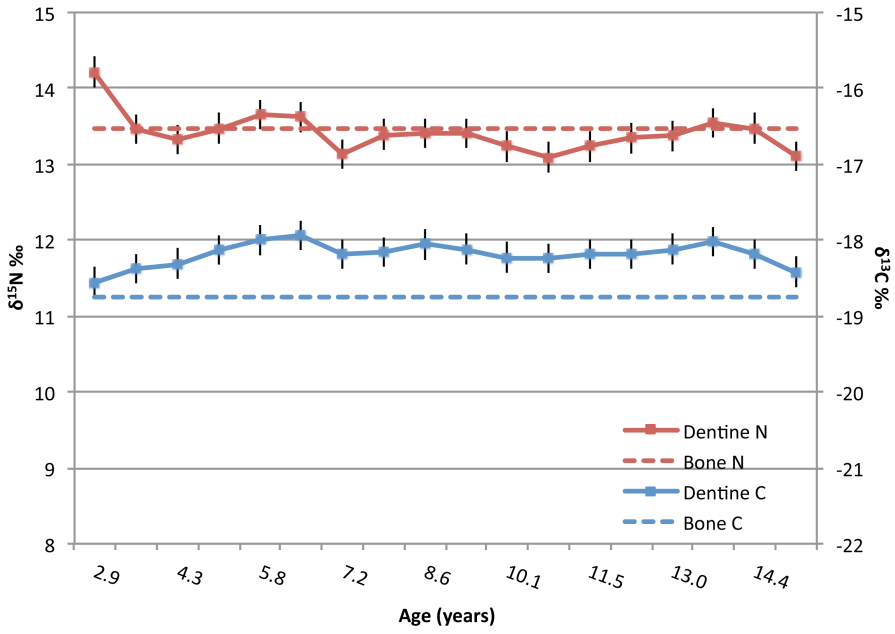


Figure E.1: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-2487

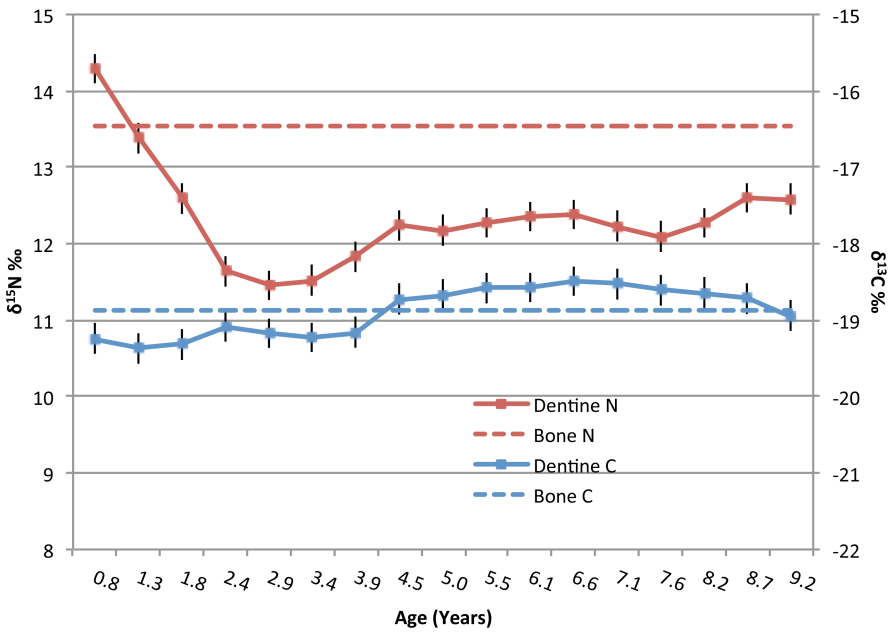


Figure E.2: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-2679

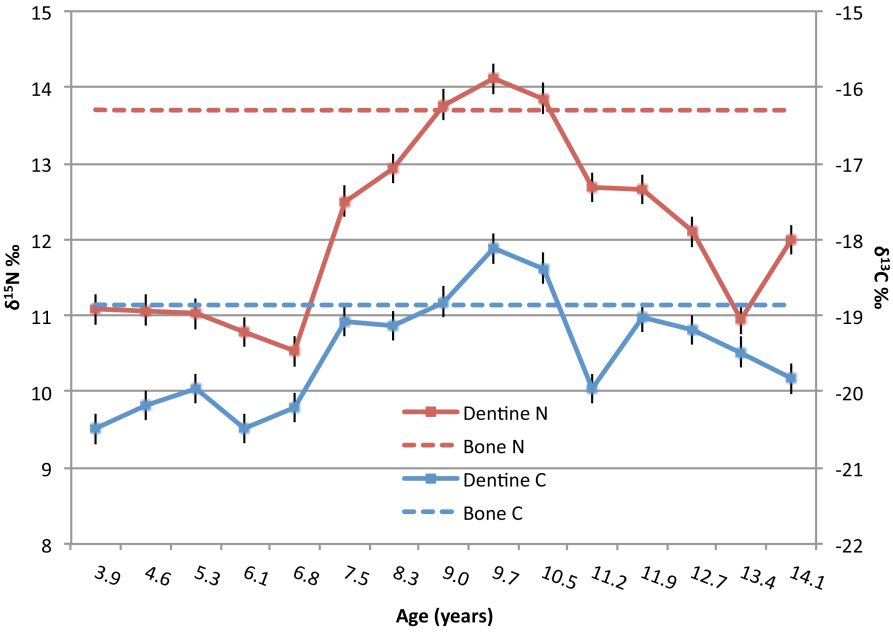


Figure E.3: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-3934

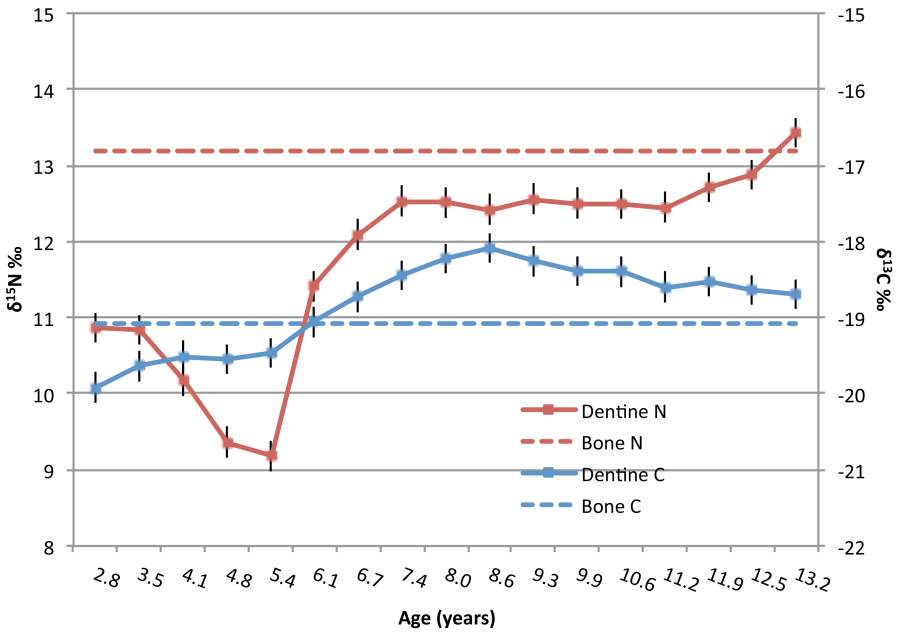


Figure E.4: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-5287

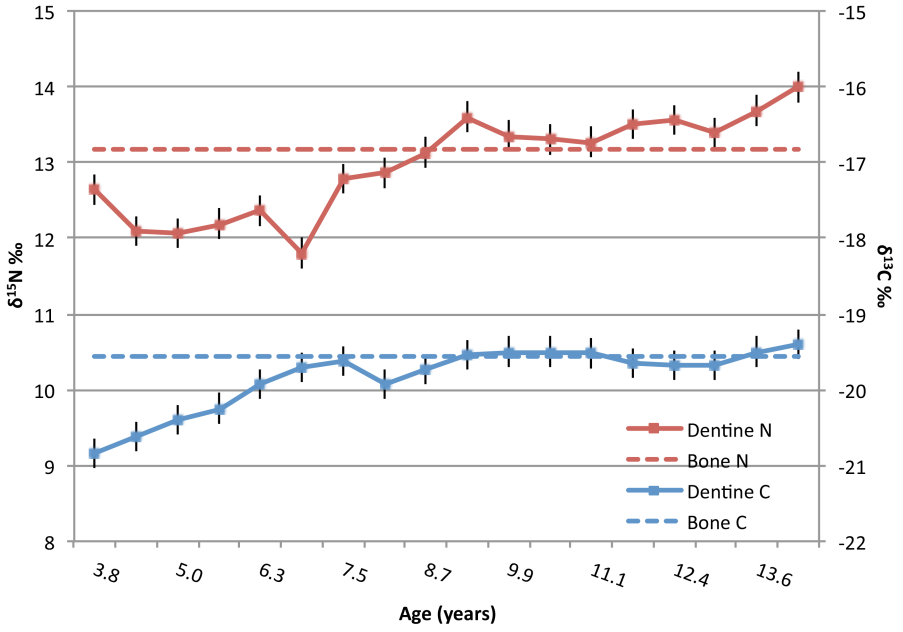


Figure E.5: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-6571

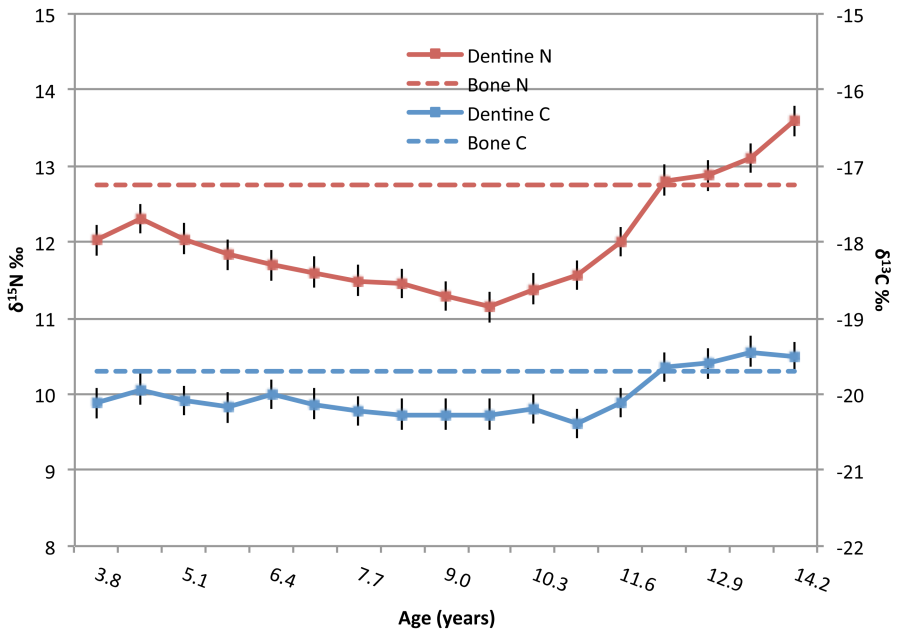


Figure E.6: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-7266

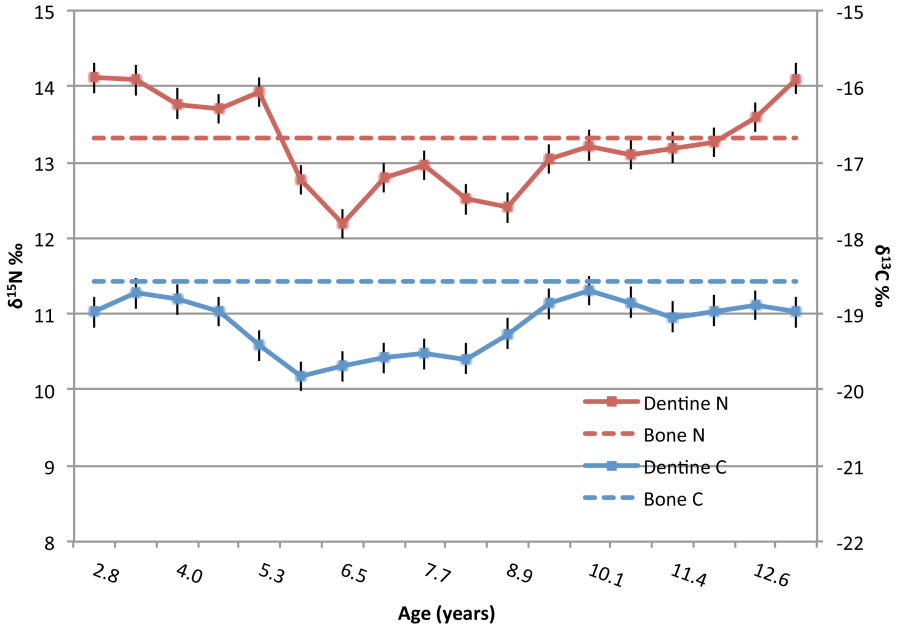


Figure E.7: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-9420

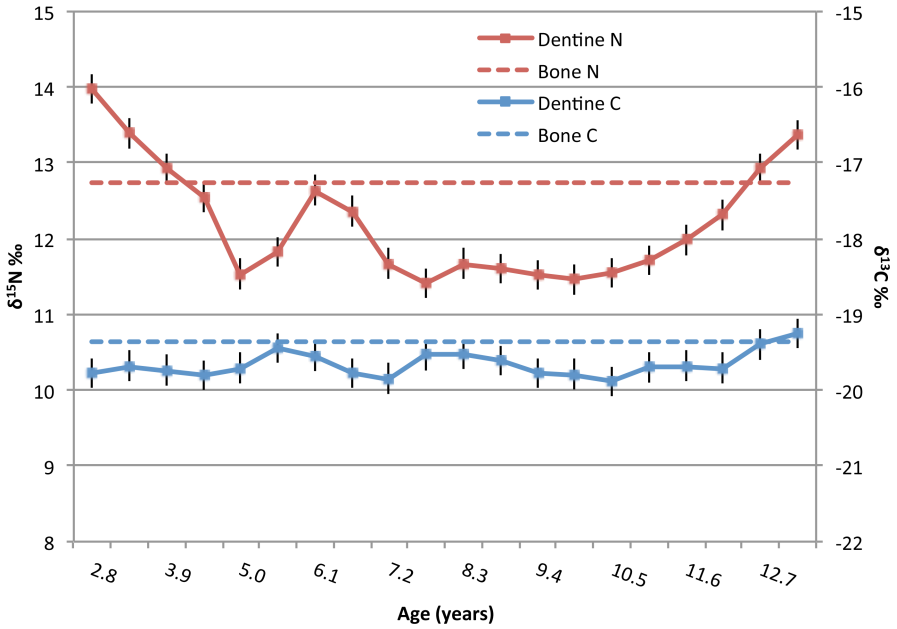


Figure E.8: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-9632

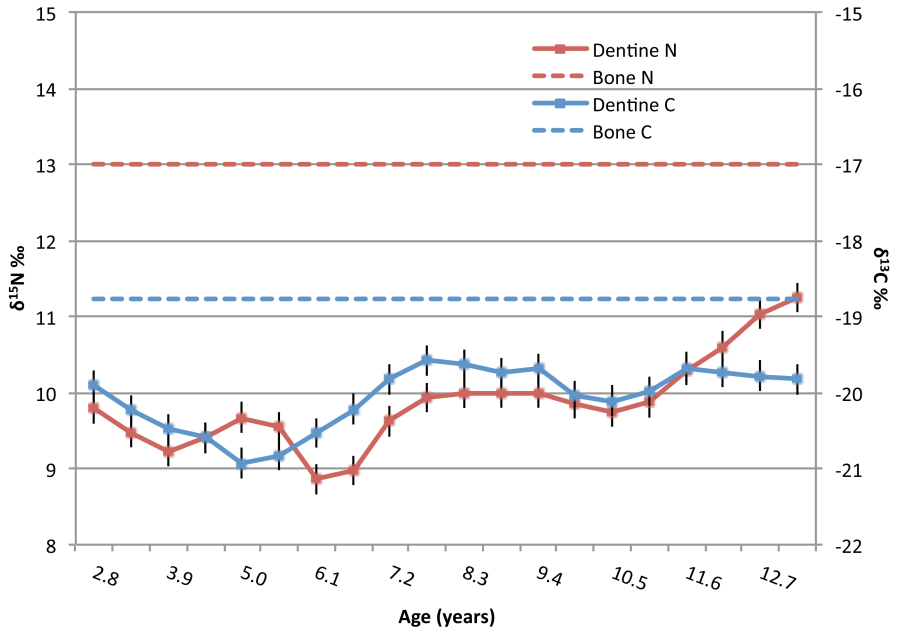


Figure E.9: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-9789

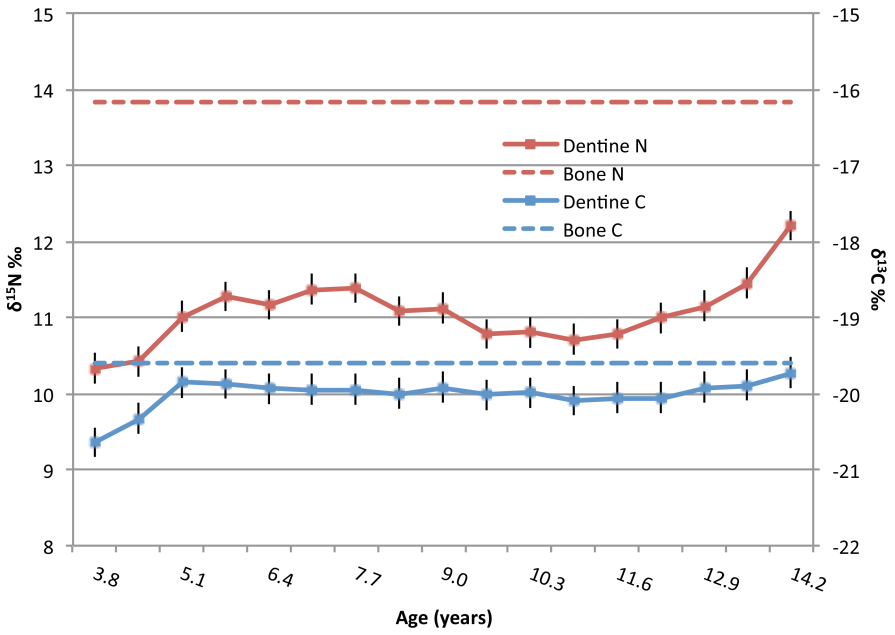


Figure E.10: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-10765

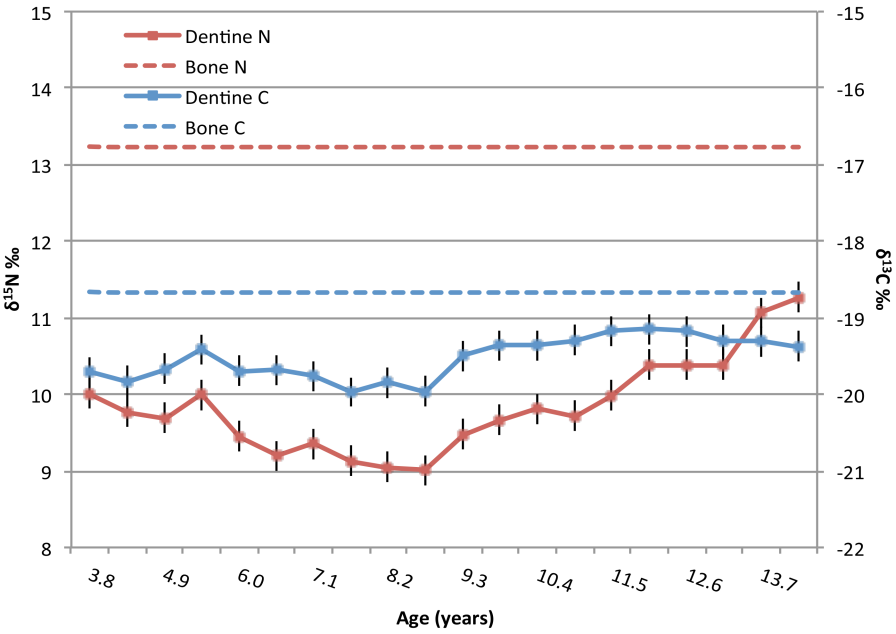


Figure E.11: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-11471

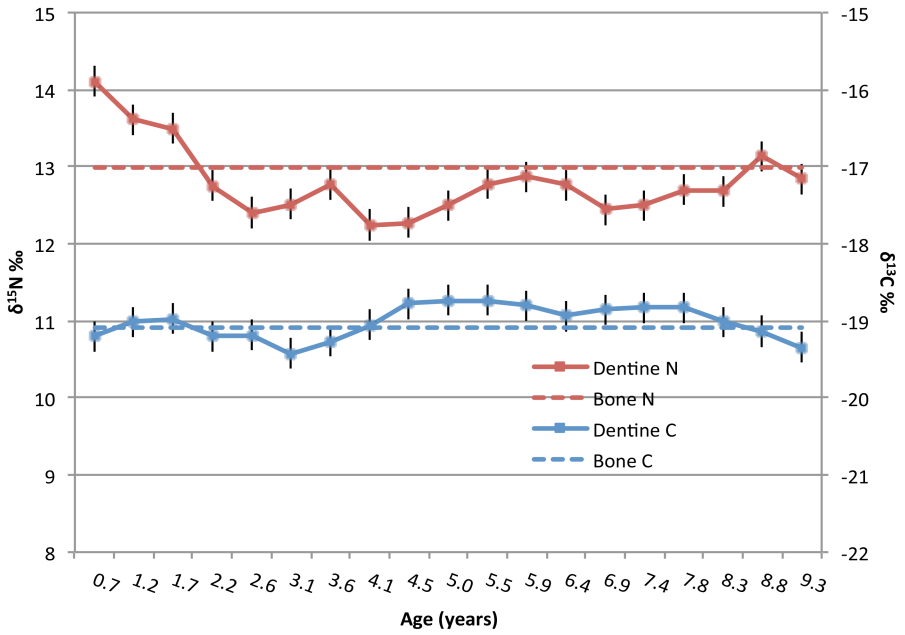


Figure E.12: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-12379

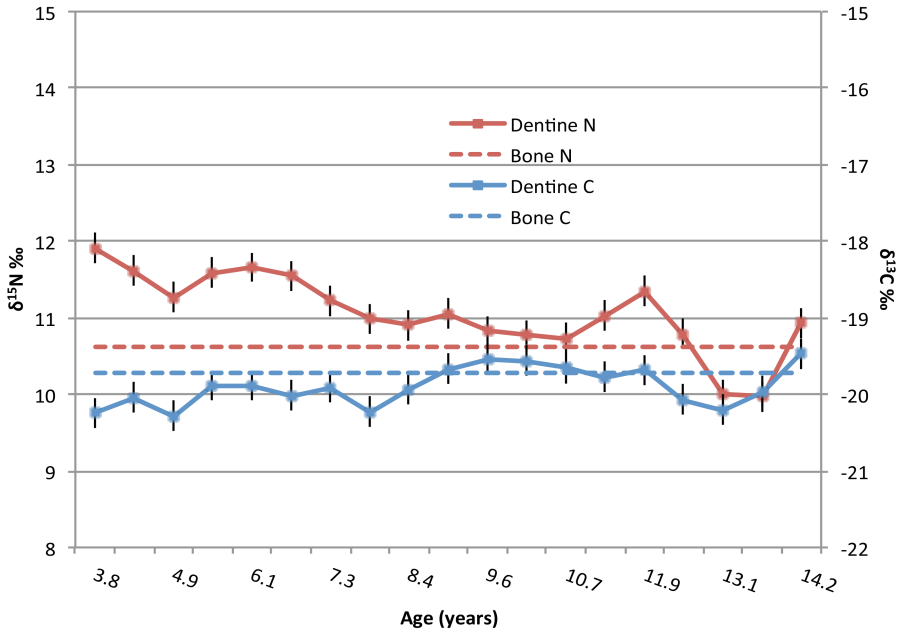


Figure E.13: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-17539

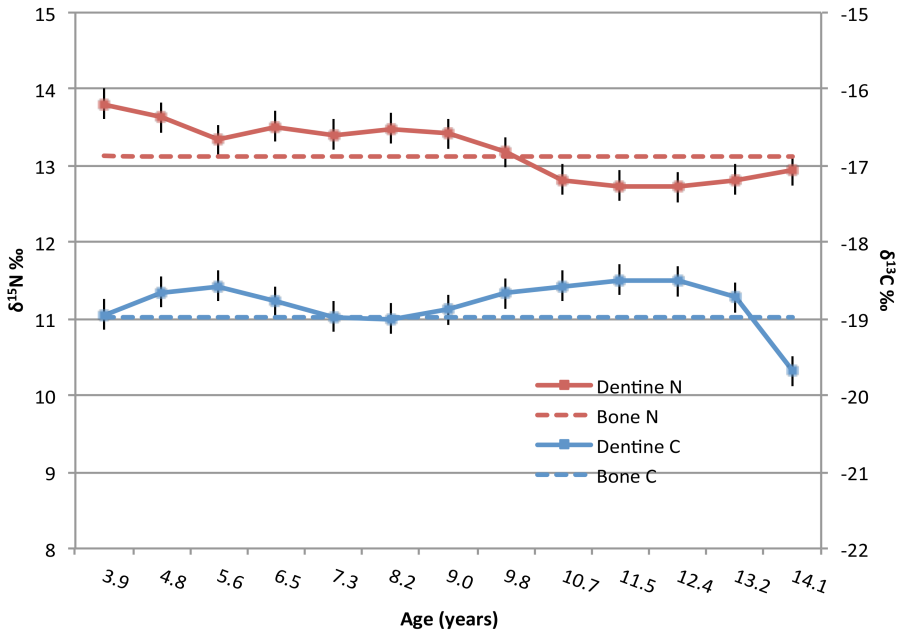


Figure E.14: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-18138

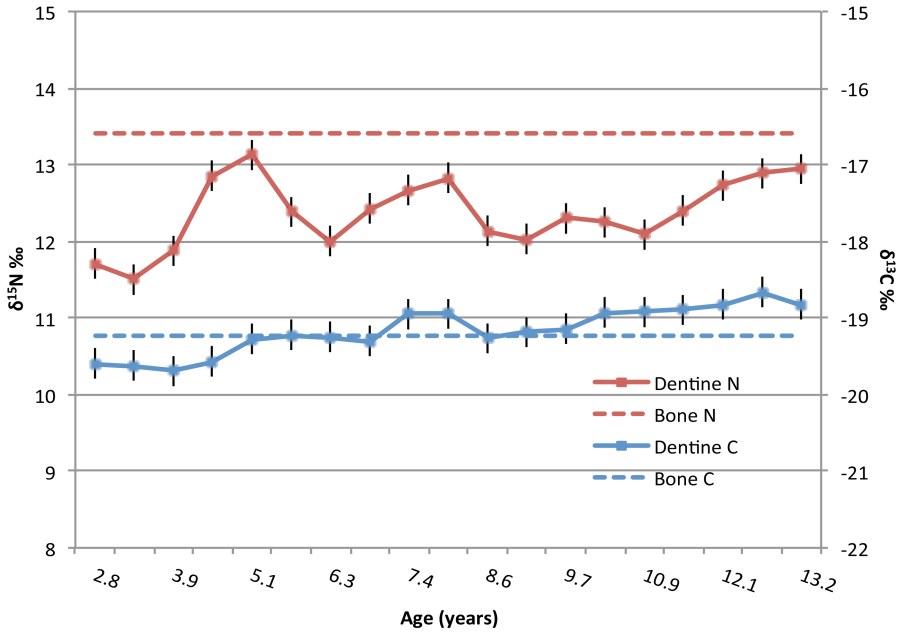


Figure E.15: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-19147

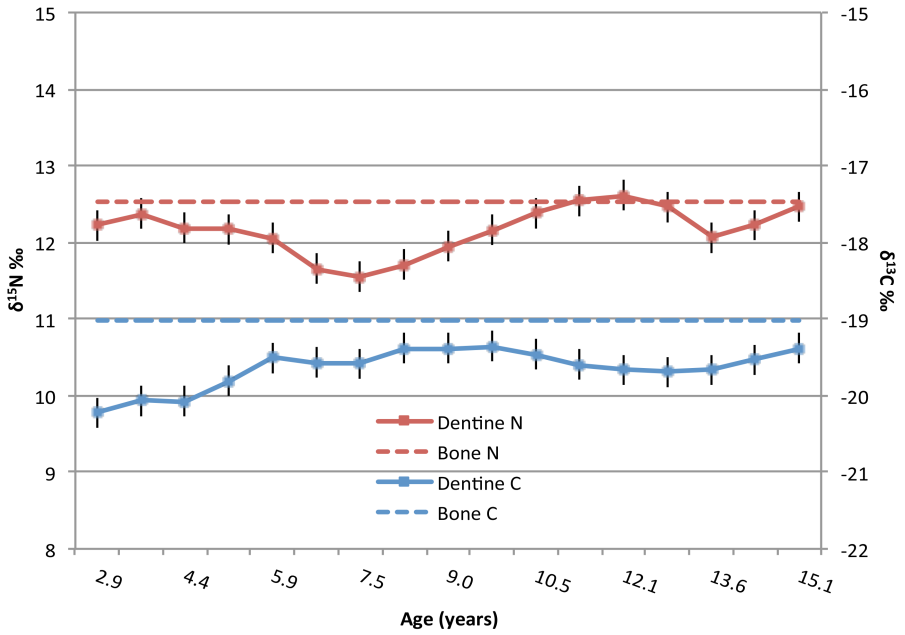


Figure E.16: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-19294

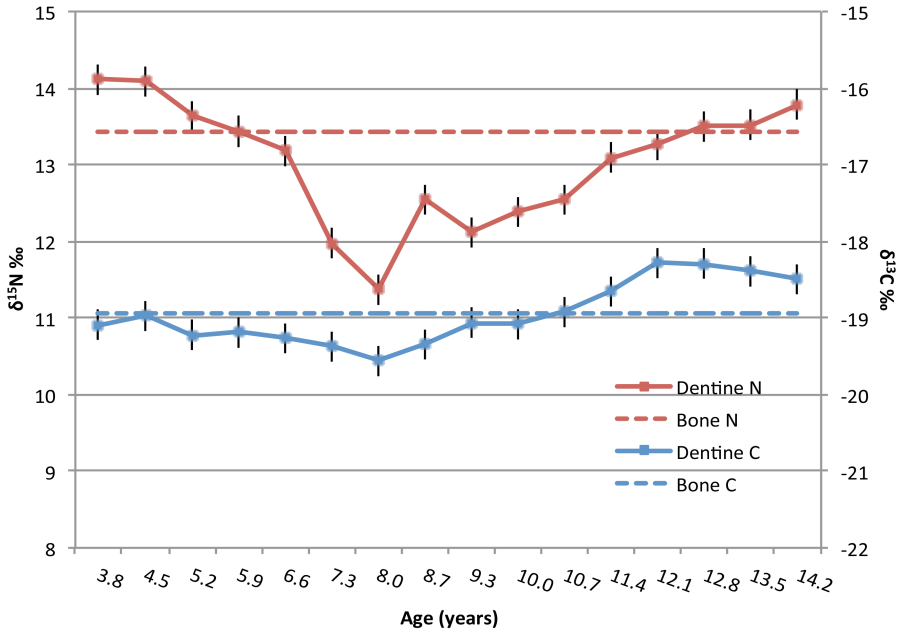


Figure E.17: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-19503

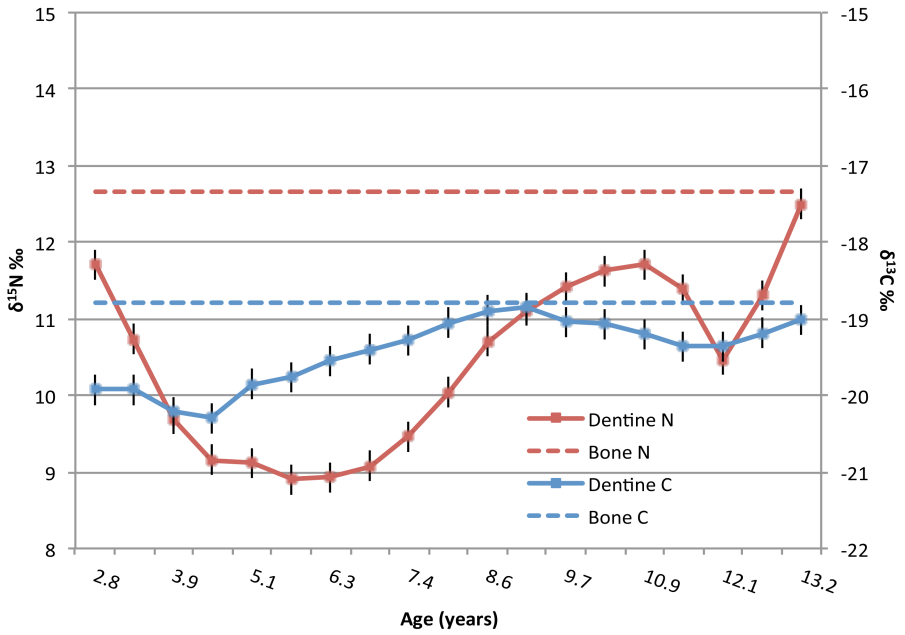


Figure E.18: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-20462

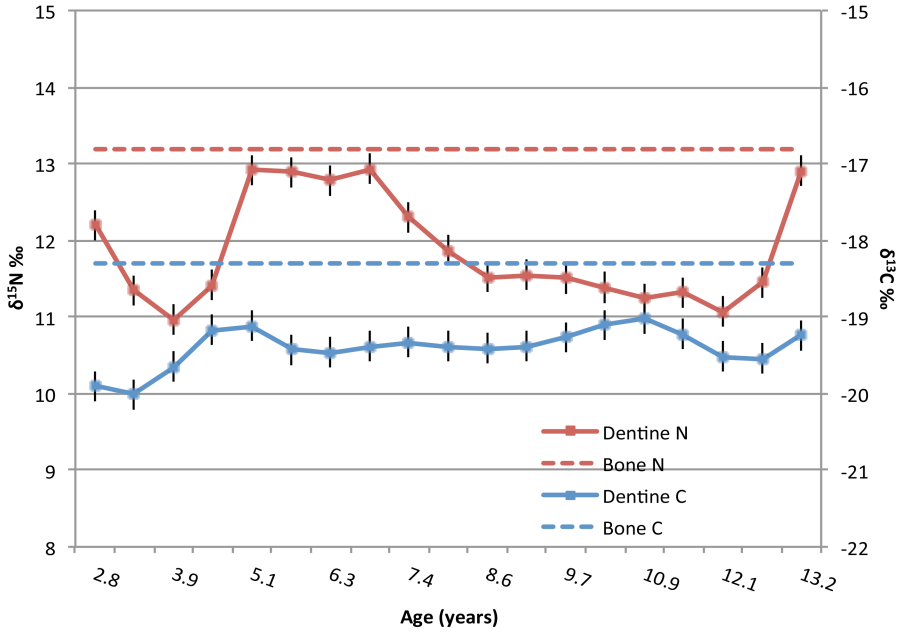


Figure E.19: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-20682

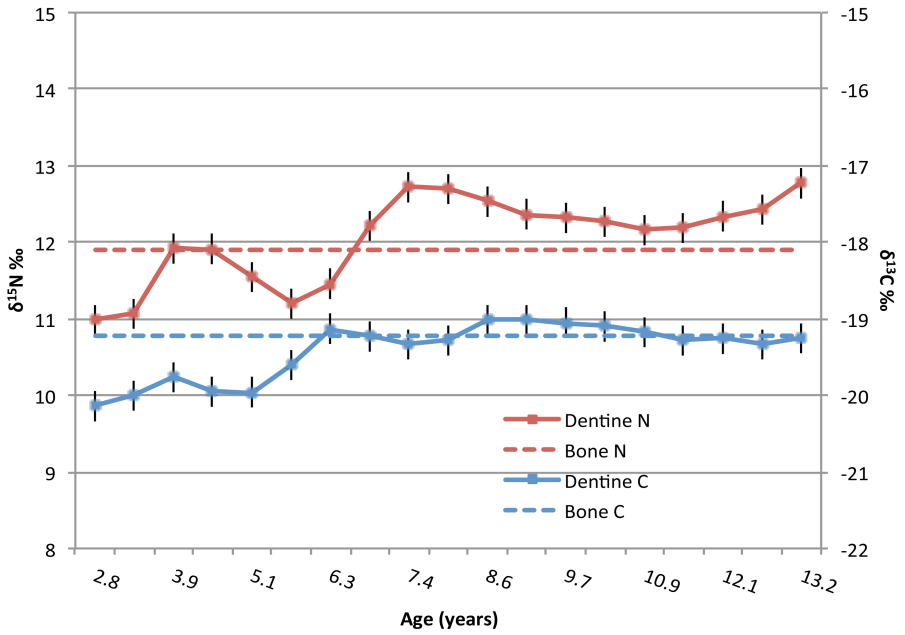


Figure E.20: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-21273

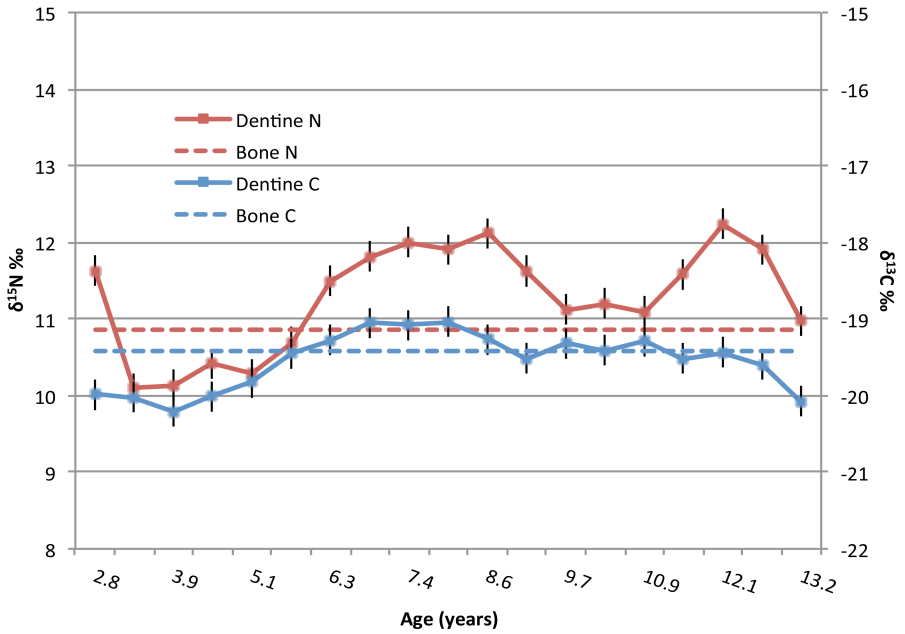


Figure E.21: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-22199

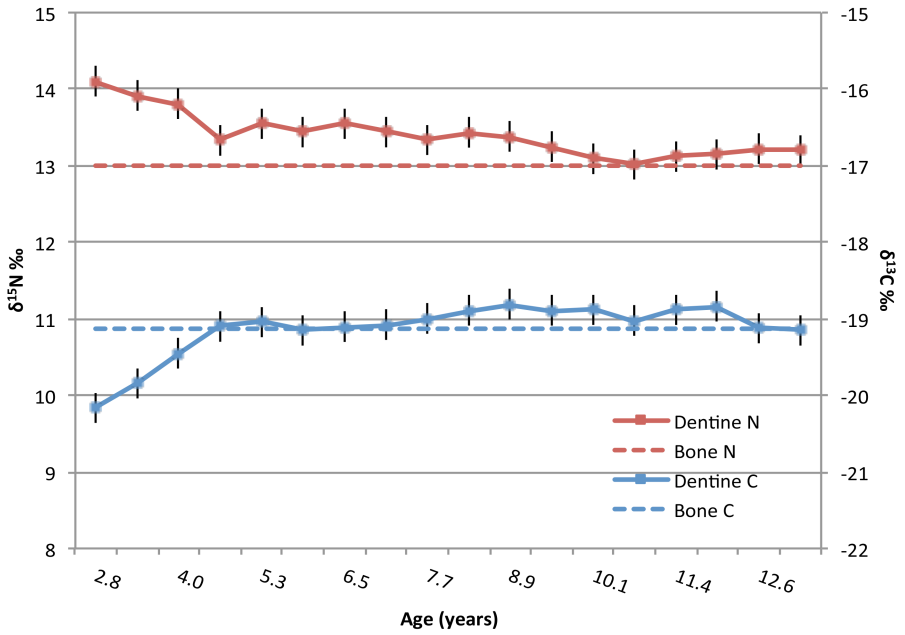


Figure E.22: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-24010

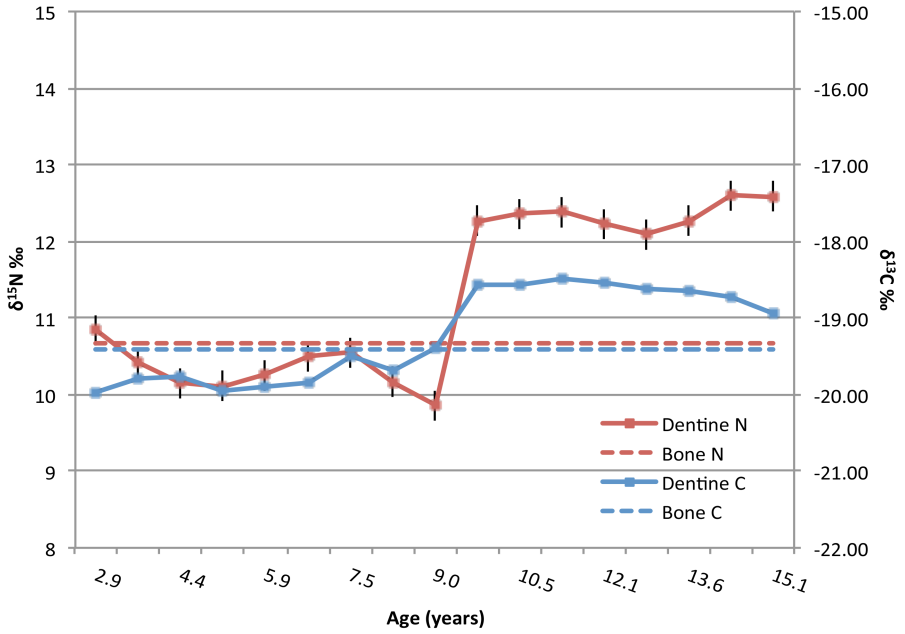


Figure E.23: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-24185

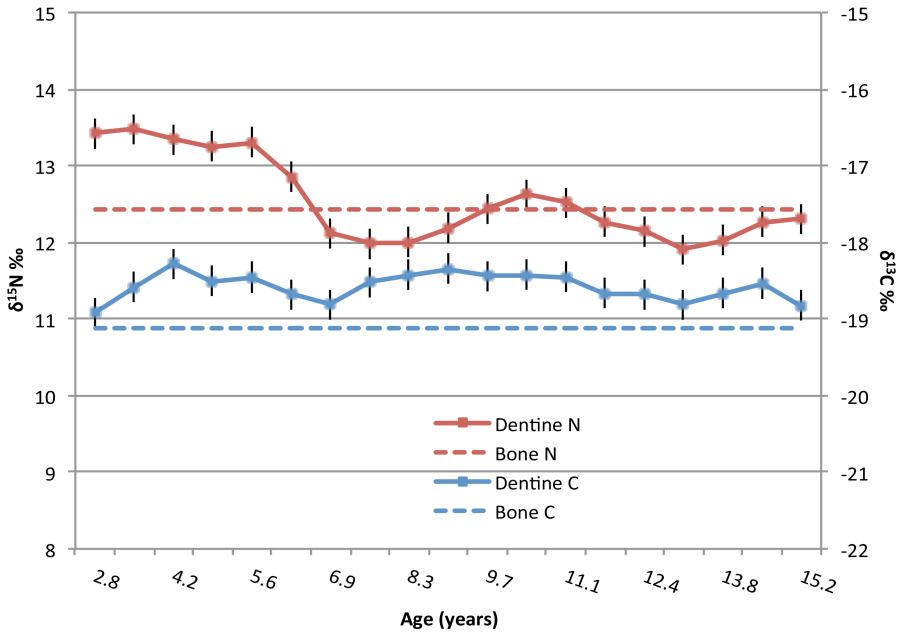


Figure E.24: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-26522

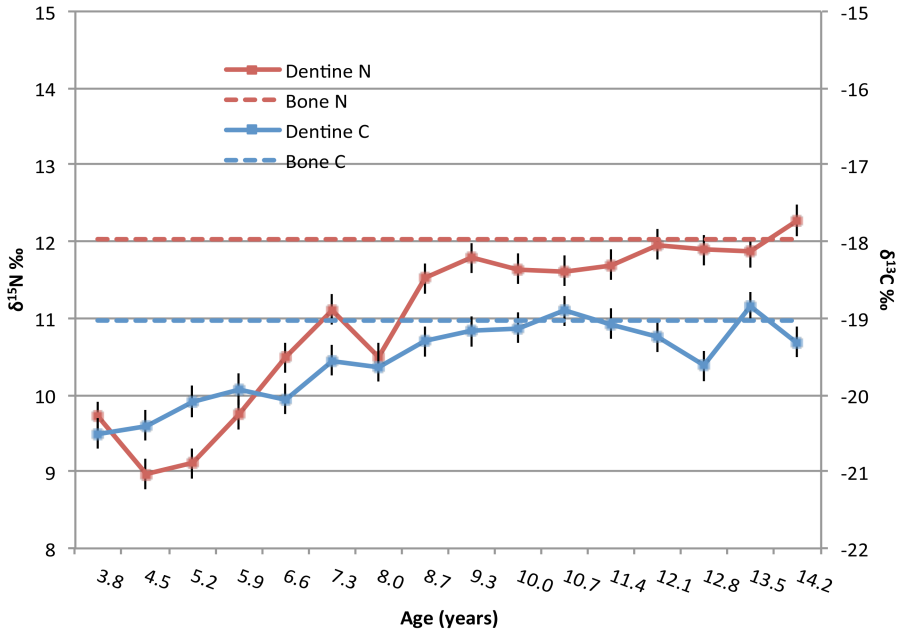


Figure E.25: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-27476

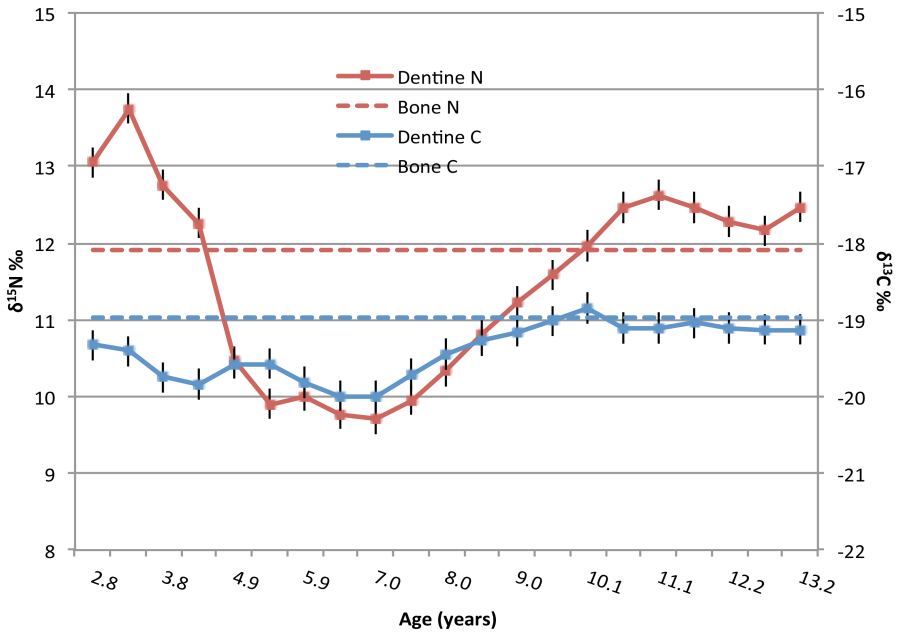


Figure E.26: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-29058

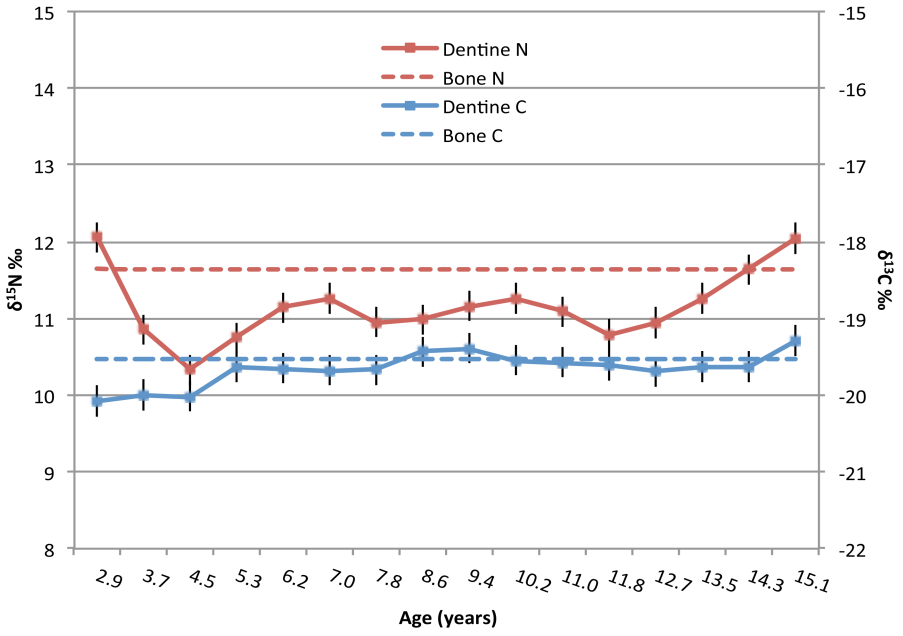


Figure E.27: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-29698

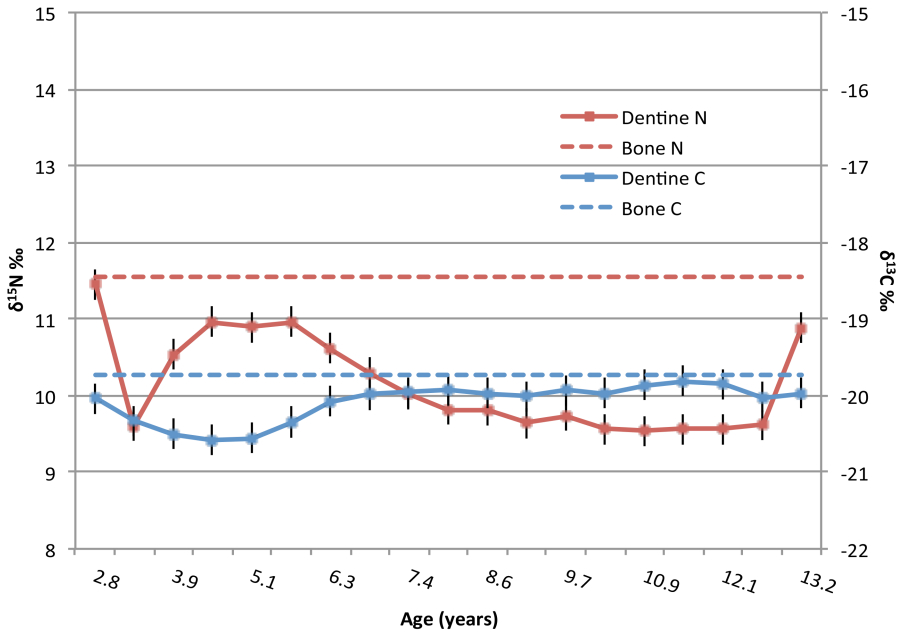


Figure E.28: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-30920

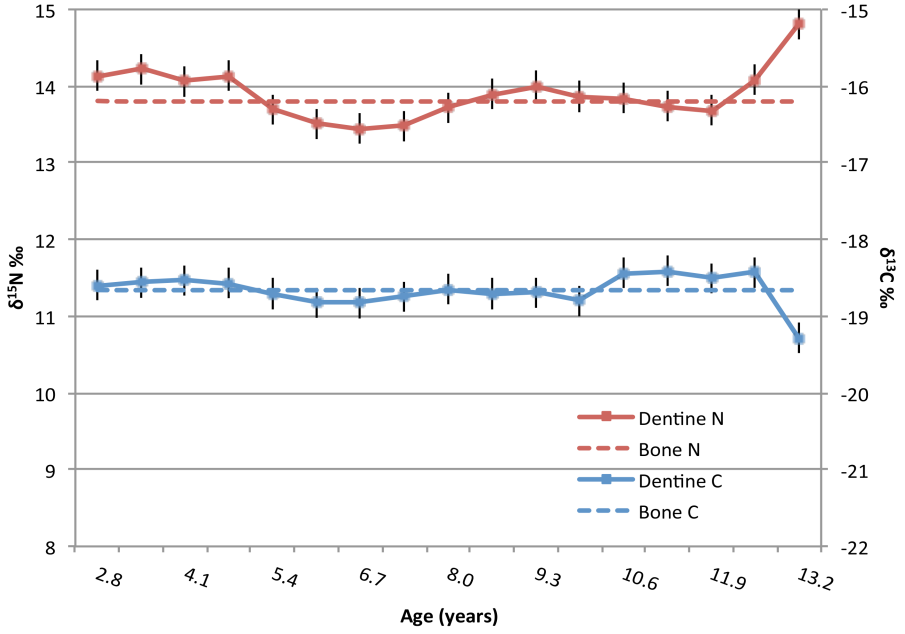


Figure E.29: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-32222

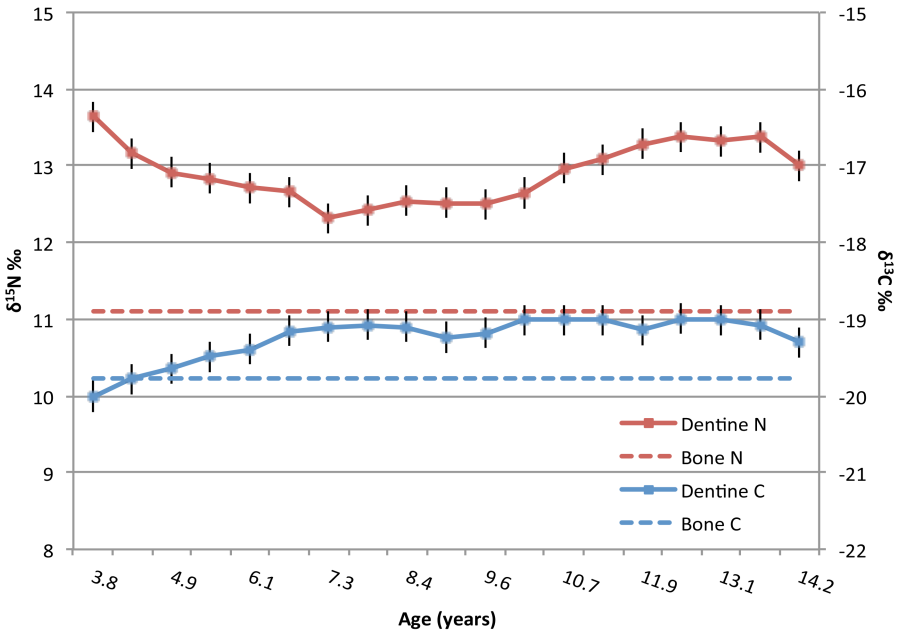
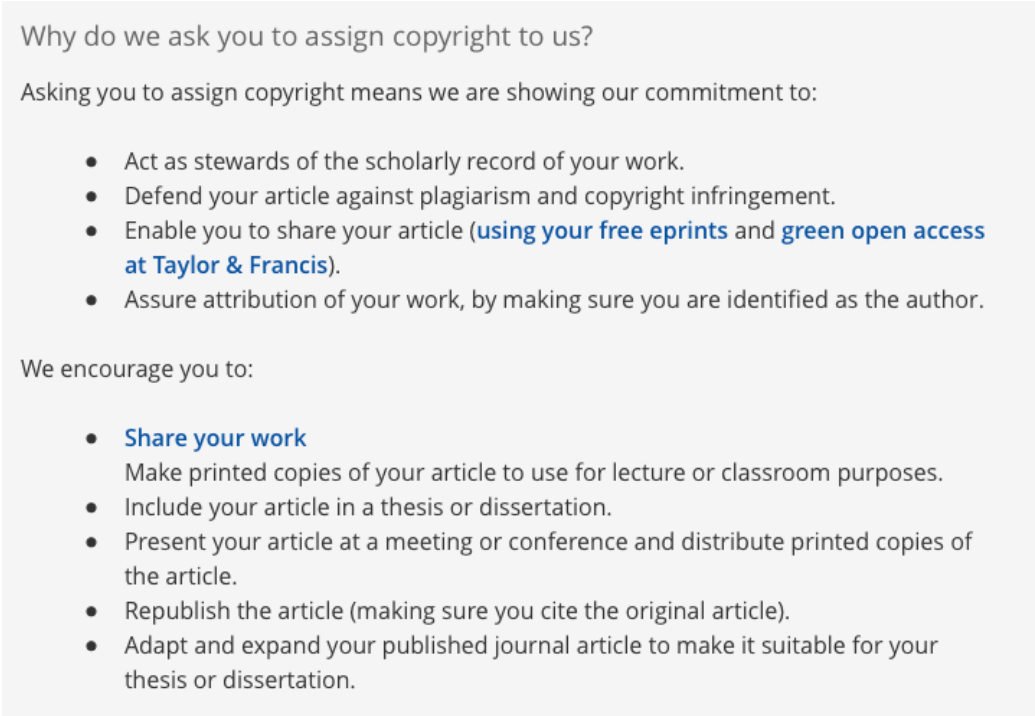


Figure E.30: Plotted bone and dentine collagen $\delta^{13}C$ and $\delta^{15}N$ values for SRP98-32302

Appendix F- Permission to Reprint

Research in Chapter 3 has been published in *Annals of Human Biology.*

Figure F.1 shows a screenshot from the publisher's website (http://authorservices.taylorandfrancis.com/copyright-and-you/) indicating permission to include published articles in the authors' dissertation.



Why do we ask you to assign copyright to us?

Asking you to assign copyright means we are showing our commitment to:

- Act as stewards of the scholarly record of your work.
- Defend your article against plagiarism and copyright infringement.
- Enable you to share your article (**using your free eprints** and **green open access at Taylor & Francis**).
- Assure attribution of your work, by making sure you are identified as the author.

We encourage you to:

- **Share your work**
  Make printed copies of your article to use for lecture or classroom purposes.
- Include your article in a thesis or dissertation.
- Present your article at a meeting or conference and distribute printed copies of the article.
- Republish the article (making sure you cite the original article).
- Adapt and expand your published journal article to make it suitable for your thesis or dissertation.

Figure F.1: Screenshot of Taylor & Francis website showing permission to include published articles in the authors' dissertation (http://authorservices.taylorandfrancis.com/copyright-and-you/ Accessed: April 26, 2017)