

July 2017

The effects of industrialization and urbanization on growth and development: A comparison of boys and girls from three Industrial European skeletal collections

Sarah Reedy
University of Massachusetts Amherst

Follow this and additional works at: https://scholarworks.umass.edu/dissertations_2



Part of the [Biological and Physical Anthropology Commons](#)

Recommended Citation

Reedy, Sarah, "The effects of industrialization and urbanization on growth and development: A comparison of boys and girls from three Industrial European skeletal collections" (2017). *Doctoral Dissertations*. 967.

<https://doi.org/10.7275/9999616.0> https://scholarworks.umass.edu/dissertations_2/967

This Open Access Dissertation is brought to you for free and open access by the Dissertations and Theses at ScholarWorks@UMass Amherst. It has been accepted for inclusion in Doctoral Dissertations by an authorized administrator of ScholarWorks@UMass Amherst. For more information, please contact scholarworks@library.umass.edu.

The effects of industrialization and urbanization on growth and development:
A comparison of boys and girls from three Industrial European skeletal collections

A Dissertation Presented

by

SARAH C REEDY

Submitted to the Graduate School of the
University of Massachusetts Amherst in partial fulfillment
of the requirements for the degree of

DOCTOR OF PHILOSOPHY

May 2017

Anthropology Department

© Copyright by Sarah C. Reedy 2017
All Rights Reserved

The effects of industrialization and urbanization on growth and development: A comparison of boys and girls from three Industrial European skeletal collections

A Dissertation Presented

By

SARAH C. REEDY

Approved as to style and content by:

Brigitte M Holt, Chair

Laurie R Godfrey, Member

Robert Schwartz, Member

Jacqueline L Urla, Department Head
Department of Anthropology

DEDICATION

To Mammaw, Grandmother, and Aunt Johanna.

ACKNOWLEDGMENTS

This dissertation has evolved in numerous ways from its original proposal. It has been a work in progress for a long time, and I love what it has become. I never would have had the opportunity to create this project, gather data or come to any of the conclusions I have if it weren't for the help and support of numerous people.

First and foremost, I would like to thank Dr. Brigitte Holt, my adviser. You have helped and supported me in so many ways. You have understood, without question, when I have had to put my family first. You have suggested new theories and methods, sometimes to my chagrin, but always for the better. I truly appreciate all of your help, guidance, and support.

Second, I would like to thank Dr. Laurie Godfrey, Dr. Robert Schwartz, Dr. Marjorie Coombs, Dr. Steve King, Dr. Thomas Leatherman, and Gina Agostini for reading numerous drafts of the proposal, dissertation chapters, and for additional guidance with the analysis and interpretation of the results. Your reviews have been thoughtful, interesting, and very helpful. I appreciate the time you have given me and the interest you've shown in my research.

Third, to Dr. Urla, the CHESS program, and my CHESS cohort for the funding, support, advice, and fun experiences during our three semesters together. Without the CHESS funding from NSF grant IIA-1261172, I could not have gathered my data. I am so grateful you chose to support me and my proposal. Because of CHESS I have made new friends and contacts from UMass to Europe, I've had the opportunity to present my

data internationally, and now have data to research and present for years to come.

Fourth, to the curators of the skeletal collections who allowed me generous access to study the remains of the children from these samples. Dr. Robert Kruszynski from the Natural History Museum from London, England for access to the Spitalfields Coffin Plate Sample. Dr. Garcia and Dr. Alves from the National Natural History Museum in Lisbon, Portugal for access to the Luis Lopez Skeletal Sample. Dr. Maria Belcastro from the University of Bologna in Bologna, Italy for access to the Bologna Skeletal Sample. All of you, your volunteers, and students were extremely helpful and supportive. You welcomed me, helped me when I was sick, provided assistance when my computer was broken, invited me to meals, took me on tours of your cities, and answered any questions I had about the samples. I am so grateful for your friendliness, help, and support!

Now, I'd like to thank all of my friends and family, from Tennessee to Becket, and UT to SUNY to UMass, for your support and encouragement, and to those who helped Kris and the boys when I left to gather data. I would especially like to thank Kris, Everett, and Gibby for supporting me. Thank you for being patient with me, helping me remember what is most important in life, and to find balance. Thank you for making me smile, laugh, and stay motivated. Thank you for putting up with me!

ABSTRACT

THE EFFECTS OF INDUSTRIALIZATION AND URBANIZATION ON GROWTH AND DEVELOPMENT: A COMPARISON OF BOYS AND GIRLS FROM THREE INDUSTRIAL EUROPEAN SKELETAL COLLECTIONS

MAY 2017

SARAH C. REEDY, B.A., UNIVERSITY OF TENNESSEE KNOXVILLE

M.A., STATE UNIVERSITY OF NEW YORK ALBANY

Ph.D., UNIVERSITY OF MASSACHUSETTS AMHERST

Directed by: Professor Brigitte Holt

Exposure to poor environments, malnutrition, and labor during childhood can lead to stunted height and increased mortality. Studies of skeletal samples from Industrial Era Europe show height is stunted when compared to Medieval samples, suggesting harsher conditions. While poor conditions can negatively impact all children, boys may be particularly disadvantaged, because girls can reserve nutritional components buffering them during times of stress. This study examines the environmental effects on growth in three Industrial European skeletal samples. Juveniles (0-18 years) from varied SES backgrounds were used to test three hypotheses.

H1) Industrial Era children will exhibit shorter femora relative to a healthy reference sample, those in the lower SES will be more stunted than those in higher SES groups, and boys will be more stunted than girls.

H2) There will be higher incidences of pathological stress markers (CO, PO, LEH, and periostitis), in lower SES than higher SES children, and boys will display more stress markers than girls, especially in the low SES sample.

H3) Lower SES children will exhibit higher mortality than higher SES children, boys will show higher risk of mortality than girls, and children with stress markers will have greater risk of mortality than those without.

Results show all Industrial Era children were shorter compared to the reference sample, with higher SES samples more stunted than low SES children. All adolescent girls (13-18 years) exhibit severe stunting, likely due to delayed pubertal growth reflecting cultural buffering of boys. Presence of LEHs are significantly higher in low SES children, and periostitis is significantly higher in lower SES groups. Boys display higher frequencies of pathologies than girls, with those in the higher SES exhibiting the most. Low SES children had the greatest survivability, children with CO and PO had a greater risk of mortality, and those with LEHs had greater survivability.

Industrial Era conditions did negatively impact children's ability to grow and survive. Less stunting, little variation in stress markers, and greater survivability within the low SES suggests environmental conditions were either improved compared to the other samples or status definitions of skeletal samples are problematic and require more consideration.

TABLE OF CONTENTS

	Page
ACKNOWLEDGMENTS.....	v
ABSTRACT.....	vii
LIST OF TABLES.....	xi
LIST OF FIGURES.....	xiii
CHAPTER	
1. INTRODUCTION.....	1
Goals and Hypotheses.....	6
Hypothesis 1.....	6
Hypothesis 2.....	7
Hypothesis 3	7
Background of this project and future goals	8
Structure of the Dissertation.....	10
Implications and Significance.....	10
2. INDUSTRIAL ERA.....	12
Industrial Era History.....	12
London.....	17
Bologna.....	21
Lisbon.....	24
3. GROWTH AND DEVELOPMENT, STRESS, AND VARIATION BETWEEN BOYS AND GIRLS.....	29
Growth.....	30
Growth Distinctions and Age Groups.....	31
Skeletal Sexual Dimorphism in Children	34
The Effects of Stress on Growth.....	37
Female Buffering.....	41
Evidence of Stress on Skeletal Remains.....	44
4. MATERIALS.....	49
The Spitalfields (London) sample (high SES).....	53
The Bologna (Italy) sample (middle SES).....	55
The Lisbon (Portugal) sample (low SES).....	56
The Denver Growth Study sample.....	57

5. METHODS.....	59
Hypothesis 1:.....	59
Stuntedness.....	60
Skeletal Growth Profiles.....	61
Comparing Sexes.....	61
Hypothesis 2:	63
Pathological Scores.....	63
Comparing pathologies among samples.....	64
Hypothesis 3:.....	64
Mortality analysis.....	65
6. RESULTS.....	66
Hypothesis 1:.....	66
Hypothesis 2:.....	76
Hypothesis 3.....	89
7. DISCUSSION.....	111
What does it all mean?.....	111
Why weren't the children in Lisbon more stunted?.....	114
Why were adolescent girls so severely stunted in all three samples?	116
Why did children in Lisbon survive better?.....	119
Why were LEHs associated with increased survivability?.....	121
Why did girls from Bologna display higher frequencies of stress?.....	123
Limitations.....	124
Future Directions.....	129
8. CONCLUSIONS.....	131
BIBLIOGRAPHY.....	133

LIST OF TABLES

	Page
Table 1: Samples utilized in this research.....	50
Table 2: Sample sizes separated by age-groups and sex.....	51
Table 3: Femur length means (mm) for boys and girls by sample.....	62
Table 4: Percent of those with normal and stunted diaphyseal femur lengths. Those from Bologna (middle SES) have the overall highest percentage of children stunted, while London (upper SES) and Lisbon (lower SES) have similar results.....	68
Table 5: Percent of stunted diaphyseal femur length by sample and sex. Lower and middle SES girls exhibit more stunting than any other group.....	69
Table 6: Z-scores based on mean diaphyseal and total femur length between boys and girls of each sample separated into age categories.	69
Table 7: Mann-Whitney U-test results between boys and girls, within samples and age-groups. Diaphyseal femur length regression residuals were used to test for significance in ages 0-12, and total femur length regression residuals were used for ages 13-18.....	75
Table 8: Chi-square results between samples for presence of pathology.....	77
Table 9: Chi-square results between sexes for presence of pathology.....	80
Table 10: Chi-square results for pathologies between sexes for the low SES sample.....	85
Table 11: Chi-square results for pathologies between sexes from the middle SES sample.....	85
Table 12: Chi-square results for pathologies between sexes from the high SES sample.....	86
Table 13: Kaplan-Meier survival analysis by SES samples.....	90
Table 14: Kaplan-Meier survival analysis results.....	91
Table 15: Kaplan-Meier survival analysis results between boys and girls within each sample.....	93

Table 16: Kaplan-Meier survival analysis results of those with or without CO.....	95
Table 17: Kaplan-Meier survival analysis results of those with or without PO.	96
Table 18: Kaplan-Meier survival analysis results of those with or without LEH.....	97
Table 19: Kaplan-Meier survival analysis results of those with three or more LEH compared to those with two or less.....	98
Table 20: Kaplan-Meier survival analysis results of those with or without periostitis.....	99
Table 21: Kaplan-Meier survival analysis results of those with or without CO within each sample.....	102
Table 22: Kaplan-Meier survival analysis results of those with or without PO within each sample.....	104
Table 23: Kaplan-Meier survival analysis results of those with or without LEH within each sample.....	107
Table 24: Kaplan-Meier survival analysis results of those with or without periostitis within each sample.....	110

LIST OF FIGURES

	Page
Figure 1: Number of individuals by Date of Birth (DOB) and sample.....	52
Figure 2: Number of individuals by Date of Death (DOD) and sample.	52
Figure 3: a, b, and c. Z-score results for boys and girls in each SES sample. Industrial Era children are shorter than DGS, stuntedness increases throughout growth, adolescent girls exhibit severe stuntedness.....	71
Figure 4: Graph showing the variation between samples and sexes using diaphyseal femur length (mm). Results suggest middle SES boys had the shortest femur length and low SES boys were closer aligned with the reference sample, and high SES boys exhibited an increase in growth between ages 7 and 10 years. Girls from the low and middle SES overlap quite a bit, but below the DGS, while high SES girls have the shortest femur lengths.	72
Figure 5: Graph showing the variation between samples and sexes using total femur length (mm). Results suggest middle and low SES boys had short, but variable femur lengths during this period. Girls from all three samples overlap quite a bit with the low SES sample showing the most variability.	73
Figure 6: Graph showing the variation between sexes in each sample using the percent of adult femur length achieved for diaphyseal femur length (mm). Results suggest very little variation between boys and girls in Lisbon and London, but boys in Bologna were quite shorter than girls of the same age.	74
Figure 7: Graph of total femur length (mm) of boys and girls from the DGS adolescents, who exhibit statistically significantly different results between sexes (p-value = .001)...	75
Figure 8: Frequency bar graph between samples with those from the lower SES sample (Lisbon) exhibiting statistically significantly (p=.0001) more presence of LEH.	77
Figure 9: Frequencies of periostitis between the samples showing the lower and middle SES samples having significantly (p=.002) highest rates.	78
Figure 10: Frequency bar graph of CO presence between the samples, Chi-Square results were not significant (p=.243).	78

Figure 11: Bar graph showing that the middle class sample (Bologna) has the highest frequency of PO, though not statistically significant ($p=.278$).	79
Figure 12: Frequencies expressing the overall presence of pathologies per individual, showing that individuals from the lower and middle SES samples exhibit more pathological conditions than those in the higher SES sample.	79
Figure 13: Frequencies of male and female percent presence of CO, Chi-square results were not significant ($p=.094$).	81
Figure 14: Frequencies of male and female percent presence of PO, Chi-square results were not significant ($p=.236$).	81
Figure 15: Frequencies of male and female percent presence of LEH, Chi-square results were not significant ($p=.633$).	82
Figure 16: Frequencies of male and female percent presence of periostitis, Chi-square results were not significant ($p=.444$).	82
Figure 17: Frequencies expressing the overall presence of pathologies of males and females, showing that males and females do not differ in the number of pathologies experienced.	83
Figure 18: Frequencies of male and female percent presence of CO within each SES sample, Chi-square results were significant ($p=.043$) in the low SES with males exhibiting more percentage, no significance in the middle SES ($p=.432$), and borderline significant in the high SES sample ($p=.079$).	86
Figure 19: Frequencies of male and female percent presence of PO within each SES sample, Chi-square results were significant ($p=.054$) in the low SES with males exhibiting more percentage, no significance in the middle SES ($p=.728$) and high SES samples ($p=.429$).	87
Figure 20: Frequencies of male and female percent presence of LEH within each SES sample, Chi-square results were not significant in any of the samples though males do exhibit a higher percentage in the low ($p=.739$), middle ($p=.485$), and high SES samples ($p=.821$).	88
Figure 21: Frequencies of male and female percent presence of periostitis within each SES sample, Chi-square results were not significant in the low SES ($p=.513$), middle SES ($p=.418$) though girls do exhibit a higher percentage, nor in the high SES ($p=.577$), though boys have a higher percentage of presence.....	88

Figure 22: Frequencies expressing the overall presence of pathologies of males and females within each SES sample, with low SES boys accumulating overall the most amount of pathological stress during life.....	89
Figure 23: Kaplan-Meier curves showing the lower SES sample exhibiting a statistically significant ($p=.0001$) greater survivability, and the upper SES sample with the lowest, especially early in life.	90
Figure 24: Kaplan-Meier curves between males and females (samples pooled), while not significant girls had a greater chance of survival than boys.....	91
Figure 25: Kaplan-Meier survival curves between males and females of the low SES sample (Lisbon), not statistically significant.....	92
Figure 26: Kaplan-Meier survival curves between males and females of the middle SES sample (Bologna), not statistically significant.....	92
Figure 27: Kaplan-Meier survival curves between males and females of the high SES sample (London), not statistically significant.....	93
Figure 28: When samples and sex are pooled, Kaplan-Meier curves show those without evidence of CO have statistically significantly ($p=.004$) greater survivorship.....	95
Figure 29: When samples and sex are pooled, Kaplan-Meier curves show those without evidence of PO have statistically significantly ($p=.002$) greater survivorship.....	96
Figure 30: Those with LEH have statistically significantly ($p=.0001$) greater chance of survival past the age of nine years than those without.	97
Figure 31: Those with three or more LEHs have statistically significantly ($p=.001$) greater chance of survival than those with two or less.	98
Figure 32: Kaplan-Meier curve shows that presence and absence of periostitis makes no difference ($p=.422$) in survivability.....	99
Figure 33: Kaplan-Meier curve shows the presence and absence of CO within the low SES exhibit a similar distribution of survivability.	100
Figure 34: Kaplan-Meier curve shows the presence and absence of CO within the middle SES exhibit a marked difference of those without having greater survivability.	101
Figure 35: Kaplan-Meier curve shows the presence and absence of CO within the high SES exhibit a similar distribution of survivability.....	101

Figure 36: Kaplan-Meier curves show the presence and absence of PO within the low SES exhibit a unique distribution with those having evidence of PO having greater survivability than those without.....	103
Figure 37: Kaplan-Meier curves show the presence and absence of PO within the middle SES showing those without evidence of PO having greater survivability than those with.....	103
Figure 38: Kaplan-Meier curves show the presence and absence of PO within the high SES showing those without evidence of PO having a slightly greater chance of survivability than those with PO.....	104
Figure 39: Kaplan-Meier curves show the presence and absence of LEH within the low SES showing those with LEH having a significantly ($p=.0001$) greater chance of survivability past the age of 5 years than those without.....	106
Figure 40: Kaplan-Meier curves show the presence and absence of LEH within the middle SES showing those with LEH having a significantly ($p=.0001$) greater chance of survivability past the age of 8 years than those without.....	106
Figure 41: Figure 41. Kaplan-Meier curves of presence and absence of LEH within the high SES showing those with having a significantly ($p=.0001$) greater chance of survivability past the age of 7 years than those without.....	107
Figure 42: Figure 42. Kaplan-Meier curves show the presence and absence of periostitis within the low SES showing no difference in survivability.....	108
Figure 43: Kaplan-Meier curves show the presence and absence of periostitis within the middle SES showing no difference in survivability.....	109
Figure 44: Kaplan-Meier curves show the presence and absence of periostitis within the high SES showing no difference in survivability.....	109

CHAPTER 1

INTRODUCTION

"In the little world in which children have their existence, whosoever brings them up, there is nothing so finely perceived and so finely felt, as injustice. It may be only small injustice that the child can be exposed to; but the child is small, and its world is small, and its rocking-horse stands as many hands high..."

From Great Expectations
By Charles Dickens, 1861

Children's growth is a reflection of the environment. When conditions are good, children grow and develop to their full genetic potential, yielding a population of healthy adults. In contrast, the impacts of poor environments on childhood growth and development can be devastating. Conditions of malnutrition, disease, starvation, overcrowding, and hard labor often lead to stunting, decreased muscle mass, failure to thrive and increased risk of death (Stini, 1969; Van Gerven and Armelagos, 1983; Tanner, 1989; Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002; Lewis, 2002; Cardoso, 2005; Cardoso and Garcia, 2009; Hughes-Morey, 2012).

There is a clear association between socioeconomic status (SES) and children's growth, with those in higher SES groups having more access to better nutrition, improved sanitary living conditions, and healthcare than those in lower SES groups. Children from higher SES households or populations are buffered from stressors such as stunted growth, disease, and increased risk of death (Stini, 1969; 1972; 1978; 1980; Tanner 1981; Tanner 1989; Komlos, 1994; Bogin and Loucky, 1997; Goodman and Leatherman, 1998;

Cardoso, 2005; Oyhenart, 2006; Schell and Magnus, 2007; Bateson et al., 2014).

When children grow under normal, healthy conditions, boys are, on average, larger than girls in height, weight, upper arm muscle mass, and head circumference, resulting in sexual dimorphism (i.e. differences between the sexes) (Tanner, 1989; Bogin, 1999; Cameron, 2002). While poor conditions negatively impact the growth of all children, boys may be at a particular disadvantage. Under conditions of stress, boys show more evidence of stunted growth, resulting in decreased dimorphism in height (Stini, 1969; Roede; 1978; Oyhenart, 2006). According to Buffa et al. (2001) girls are impacted less negatively because the evolution of hormonal mechanisms permits them to reserve nutritional components, like fat and protein, during growth (for use during pregnancy and nursing), buffering them during times of stress. Studies also suggest that height may be much more sensitive to poor living conditions than other variables such as head circumference and upper arm strength (Stini, 1972; Oyhenart, 2006). On the other hand, Hughes-Morey (2016) argues that lower SES females are in fact the most frail, and have a higher risk of mortality, especially as children, due to patriarchal cultural factors. For example, some historical references on the Industrial Era indicate preferential treatment towards boys and men with better access to nutritional foods within the household (Horrell and Oxley, 2012).

This study aims to understand better how the stressed environmental conditions of the Industrial Era (18th-20th Centuries) of Europe, such as high levels of pollution, infectious diseases, overcrowding, poor sanitation, malnutrition, and low socio-economic status impacted growth, survivability, and sexual dimorphism through comparisons of

three Industrial Era subadult samples with varying SES.

The Industrial Era is often credited as a time of development, innovation, and economic growth. While these may be true, it was also a time of vast urbanization, pollution, hard labor, distinct social classes, poor nutrition, and disease (Thompson, 1963; Hobbs et al., 1999; Cardoso and Garcia, 2009; DeWitte et al., 2015; Leatherman and Jernigan, 2015; Hughes-Morey, 2016). Skeletons from Industrial populations often express signs of stress like stunted height, pathologies, and increased risk of death to higher degrees than modern samples and even their Medieval counterparts (Cardoso and Garcia, 2009; Lewis, 2002; DeWitte et al., 2015). Consequently, many people, primarily those of the lower classes were exposed to harsh working conditions, poor nutrition, crowded living spaces, heavy pollution, lack of healthcare, and poor sanitation, putting them at risk of developing various infectious diseases (like tuberculosis, influenza, and cholera) and those of malnutrition (like anemia, rickets, and scurvy) (Thompson, 1963; Engels, 1958). In the urban centers, wages often declined due to new technical and machinery innovations (Hobbs et al., 1999). All members of families, were often forced to work long hours to provide enough income for household necessities. Children's small size was seen as an advantage to employers, who could send them under machines to retrieve or repair broken parts, despite the dangers, all while being paid significantly less than adults (Rahikainen, 2004). Managers often had reputations for brutality towards their workers, including children (Hobbs et al., 1999; Rahikainen, 2004).

As population size increased in urban settings, a growing reliance on grains increased as well, leading to an overall less nutritional diet (Horrell and Oxley, 2012) that

likely correlated with increasing incidents of malnutrition, especially amongst women and children. Historical height data reveal that urban women, who had less access to nutritious foods, were often shorter and exhibited higher mortality rates, compared to rural women who had better access to fresher and higher quality foods (Horrell and Oxley, 2012). Komlos (1993), found similar results amongst English and Irish men using military height data that shows declining height over the course of the 18th Century.

Historical data also suggests boys and men, in these patriarchal societies, likely had preferential treatment within the home, as well as better access to higher quality, nutritional food, since their earnings were more valued (Horrell and Oxley, 2012; Humphries, 2013). Within the home, women would often be forced to decide how to allocate food amongst her family, sometimes denying food from herself. This would not impact her height, but it could impact the growth of her children if some were given more than others. Horrell and Oxley (2012) tell us portions were dependent upon earnings, thus girls and younger children would be given less than older boys.

As evidence to support this historical data, Industrial Era skeletons from Europe often display stunted height when compared to Medieval samples (Lewis, 2002; Cardoso and Garcia, 2009), and high incidences of pathological stress markers and mortality rates all suggesting poor living conditions (Cardoso, 2005; DeWitte et al., 2015; Hughes-Morey, 2016). Many growth studies of juvenile skeletal remains offer valuable insight about the overall experiences of past populations (Lewis, 2002; Cardoso, 2005; Gültekin et al., 2006; Cowgill and Hager, 2007; Mays et al., 2008; Mays et al., 2009 & 2009b; Cardoso and Garcia, 2009; De Stefano and De Angelis, 2009; Guatelli-Steinberg, 2009;

Cowgill, 2010; Humphrey et al., 2011; Schillaci et al., 2013; Ruff et al., 2013). However, few focus on the impact of stress between boys and girls. In fact, many skeletal growth studies leave sex out of the analysis (Mays, 2009; Cowgill and Hager, 2007; Cardoso and Garcia, 2009; Lewis, 2002), disregarding how stress may impact boys' and girls' growth patterns differently. Analysis of variation between sexes in children can address issues of growth-related physiological trade-offs and help identify unequal access to nutrition, susceptibility to disease in childhood, and differential treatment of girls and boys (Sorenson, 2000; Sofaer, 2006).

This brief review highlights the complex interaction between gender, growth, and environmental conditions. Using three European Industrial Era juvenile skeletal samples of known age and sex, and varying SES, this study takes a biocultural approach to interpreting growth patterns, risk of mortality, and sexual dimorphism under conditions of stress. Taking such an approach is imperative in skeletal growth studies, since stress and stunting are directly related to socio-political, economic, social, and historical factors (Bogin and MacVean, 1978; Johnston and colleagues, 1984; Floud et al., 1990; Norgan, 2002; Schell and Knutsen, 2002). Children, generally the most fragile members of society (Tanner, 1982; Van Gerven and Armelagos, 1983; Goodman et al., 1984; Schell, 1989; Bogin, 1999; Cameron, 2002), are therefore key to understanding how these conditions influenced health differentially between those with better access to resources, i.e. higher status, and between the sexes, in the Industrial Era, a period of profound social change, distinct social classes, and poor environmental quality.

Goals and Hypotheses

This study aims to investigate the impact from and interaction between sex and status during the growth process when environmental conditions are poor. While the negative effects of low social status and poor conditions on growth is well understood, it is less understood how boys and girls respond to such conditions differently. Are girls more biologically buffered against harsh conditions compared to boys, therefore resulting in less stunting, less susceptibility of malnutrition and disease, and decreased risk of mortality? Or are cultural buffers (i.e. better access to higher quantity and quality food and healthcare), privileged to males in these patriarchal societies, giving them better growth and survivability than their female counterparts? These questions have led to the development of three distinct hypotheses made up of different predictions that are tested in this dissertation.

Hypothesis 1

Given that poor environmental conditions can lead to stunted height, I test the null hypothesis that there are no differences in femur length between the Industrial Era children compared to a healthy reference sample, between boys and girls, and between SES samples. Alternative hypotheses, if the null hypothesis is rejected include: first, that poor environmental conditions will lead to shortened femur lengths, with Industrial Era children exhibiting shorter femora relative to a healthy reference sample (*H1: Prediction 1*); second, that children from the lower SES will be more stunted than those from middle and high SES (*H1: Prediction 2*); and third, that boys will be more stunted than girls,

resulting in decreased sexual dimorphism in femur length (H1: Prediction 3), especially in the lower SES sample.

Hypothesis 2

Since poor environmental conditions correlate with indicators of stress in skeletal samples, this research tests the null hypothesis that there are no differences in the frequencies of stress markers between SES samples or between sexes. Alternative versions of this hypothesis are that we will find higher incidences of stress markers (such as cribra orbitalia (CO), porotic hyperostosis (PO), linear enamel hypoplasias (LEH), or periostitis), with increased evidence of stress in lower SES relative to either middle or high SES children (*H2: Prediction 1*); or that stress markers are more prevalent in boys than girls (*H2: Prediction 2*); or, finally, that stress markers are more prevalent in boys with low SES (*H2: Prediction 3*).

Hypothesis 3

Because poor environmental conditions also correlate with increased risk of mortality, I test the null hypothesis that there are no differences in mortality rates between SES samples, sex, or those with pre-existing signs of stress compared to those without. Alternative versions of this hypothesis are, first, that poor environmental conditions and sex differences correlate with increased risk of mortality, with lower SES children exhibiting higher risk of dying than middle and upper SES samples (*H3: Prediction 1*).

Second, under conditions of stress, boys are expected to show higher risk of early mortality than girls (*H3: Prediction 2*). Children with an existing pathological condition will exhibit a greater risk of mortality than those without (*H3: Prediction 3*).

Background of this project and future goals

This dissertation stems from an original goal of developing a technique for estimating sex in pre-pubertal skeletal remains. This goal, however, presents a number of challenges. First, by their very presence, children in skeletal samples are those who likely did not grow up under normal, healthy conditions. Second, given that growth can be impacted by environmental conditions, and boys and girls may be affected by such conditions differently, it is imperative to first determine those various effects before identifying sex. Third, small sample sizes, gaps in age distributions within samples, and discontinuity in time and space between samples add to the initial challenge of health impacts.

Given that boys should be larger than girls under healthy conditions, but may be at increased risk of stunted growth when environments are stressed, and that children in skeletal samples are inherently stressed given their presence in the sample, it is fair to say that research on juvenile skeletal sexual dimorphism has yielded little significant results. Research in juvenile sexual dimorphism has a long history (Weaver, 1980; Mittler and Sheridan, 1982; Schutkowski, 1987; 1993; Molleson and Cruse, 1998; Blake, 2005; Rissech and Malgosa, 2005; Cardoso, 2008; Vlak et al., 2008; Rogers, 2009). Most research, however, has been hindered by the lack of juvenile remains of known sex and

age, resulting in small sample sizes (Noren et al., 2005; Cardoso, 2008), the use of broad age categories that mask developmental differences between the sexes (Sutter, 2003; Noren, 2005), a focus on univariate analyses or multivariate analyses, limited to one region of the skeleton (Ridley, 2002; Cardoso, 2008; Gapert et al., 2009; Rogers, 2009; Veroni et al., 2010), and the use of secondary sex characteristics, like wider pelvic bones in females, that are not significantly dimorphic until puberty (Weaver, 1980; Mittler and Sheridan, 1992; Schutkowski, 1993). Since sex cannot be estimated in subadult remains, children are often left out of bioarchaeological analyses leaving out this key demographic in research from past populations. Given that children in these samples represent those who were likely stressed potentially decreasing their dimorphism, it is important to analyze areas of the skeleton that are less impacted by environmental conditions.

While this dissertation does not aim to discover pre-pubertal sexual dimorphism, it is important to acknowledge the distinctions between boys' and girls' growth trajectories and development to fully understand how biological and cultural buffers, as well as environmental constraints impact their growth, morbidity, and mortality differently. It will be a future goal from this study to analyze the remains of boys and girls for sexually dimorphic traits despite the effects of poor environmental conditions, so that further research of sexual dimorphism in subadult remains might be aimed at less environmentally controlled areas of the body therefore making such goals more attainable.

Structure of the Dissertation

In chapter two, I present a background of Industrial Era life, particularly in the cities of origins for the skeletal samples used in this research, i.e. London (England), Lisbon (Portugal), and Bologna (Italy). This chapter will explain the backgrounds of the samples, the political-economic climate the populations were facing, and a general review of social history surrounding social status, family life, access to food, living conditions, and overall life experiences, especially for children when the historical data is available.

Chapter three provides some background for childhood growth and development, outlining variation and dimorphism between boys and girls. Here, I explain how poor environmental conditions can affect growth and how boys and girls might respond to such conditions differently.

Chapter four outlines the specific samples used, their origins and location, and specific details for each sample. Chapter five is the Methods chapter discussing the statistical analyses used to test each hypothesis. Chapter six reports on the results of the analyses, and Chapters seven and eight provide Discussion and Conclusions, respectively.

Implications and Significance

Analysis of skeletal variation in growth by sex and SES bears on bioarchaeological and forensic contexts. Archaeological skeletal samples are often used to understand the relationship between child growth and health. However, without a clear understanding of how boys and girls responded to poor environments, it can impact the

ability to make interpretations about gender, SES, and access to resources. Not only do we need to fine-tune our understanding of distinct sex differences in growth, but also to provide better, more nuanced interpretations of boys and girls in past populations - something that has long been ignored. Stunting in children's remains can mislead us to believe we are looking at younger individuals (Hoppa, 1992; Lewis, 2002; Goode-Null et al., 2004; Cardoso, 2005; Pinhasi et al., 2005), or to misinterpret boys for girls.

Juvenile skeletal remains are often considered uninformative or only used for general growth profiles for pooled sexes (Formicola, 1988; Pettit and Bailey, 2000; Cowgill and Hager, 2007; Cardoso and Garcia, 2009; Gonzalez et al., 2010; Goikoetxea et al., 2012). This approach, however, disregards potentially important sex differences, especially under stressed environmental conditions that may result in sexual differences in morbidity and mortality levels, or affect the overall growth and health of one sex versus the other. The skeletal remains of past populations who lived under varying levels of environmental and social stress provide "natural experiments." The three samples of juvenile remains included in this study afford a unique opportunity to assess the impact of stress on growth. Results will 1) provide a better understanding of the way in which boys and girls grow and respond to various environmental stresses, 2) allow for broader perspectives and medical influence on future growth studies of living children, and 3) improve knowledge about sexual dimorphism among juvenile skeletal remains under both normal and stressed conditions, thus providing data crucial for future development of a way to identify the sex of these remains, a task that has generally proven impossible.

CHAPTER 2

INDUSTRIAL ERA

In this chapter, I will review a general history of Industrial Era Europe as it pertains specifically to living conditions, diet, and childhood since these address the hypotheses in this dissertation. Then, I focus on the Industrial histories of London (England), Bologna (Italy), and Lisbon (Portugal), since the skeletons analyzed in this research come from these cities.

Industrial Era History

The industrialization of Europe (18th –20th centuries) marks the point at which water and steam were widely used as a source of power. The typical perception of industrialization is of large factories using steam power for the large-scale production of goods. Most industries actually remained small with the manufacturing of artisanal, hand-crafted goods. Industrialization may be more accurately described as a time of rapid urbanization, migration, and economic growth in different sectors. Due to such rapid urbanization and unprecedented population growth, this period saw increases in inequality, rising costs, and extremely poor living conditions pushing humans into a new demographic transitional phase (Thompson, 1963; Webster, 1975; Szreter, 1988; 2004; Hobbs et al., 1999; More, 2000; Johnson, 2006; Cardoso and Garcia, 2009). Szreter (2004) discusses this as a period of disruption (from rising population and political instability), deprivation from a lack of quality food resources and healthcare, leading to

increased disease and death. Consequently, many people, primarily those of the lower classes, were exposed to harsh working conditions, poor nutrition, crowded living spaces, heavy pollution primarily due to coal burning fireplaces, lack of healthcare, and poor sanitation, putting them at risk for developing various diseases like tuberculosis, cholera, rickets, and typhoid (Thompson, 1963; Wohl, 1983).

In most European urban centers of the 18th and early 19th centuries, wage labor was the dominant work force, with wages oftentimes being pushed down by the development of new technologies (Webster, 1975; Hobbs et al., 1999). Entire families, including men, women, and children were often forced to work 10-15 hours per day to provide enough income for the household. Children in particular, were used in a variety of jobs because their small size made it easier for them to crawl under big machinery and they were paid significantly less than adults (Rahikainen, 2004). Work in factories was often brutal, both from the manual labor and the rough treatment from managers and parents themselves, who were often abusive and took advantage of children's earnings (Hobbs et al., 1999; Rahikainen, 2004).

The work of social historians describes a bleak picture for working class children of the Industrial Era in many parts of Europe and the U.K. In the 18th and early 19th centuries, children in parts of England, like Manchester, often started to work in mills and factories by age six (Thompson, 1963; Cox, 1996; Lewis, 2002), children in Portugal were often sent to factories to work by the age of 10 years or younger in the 19th and early 20th centuries (Goulart and Bedi, 2007; Cardoso and Garcia, 2009), while children in Italy were put to work in the textile and tobacco industries by age 12 in the 19th and early 20th

centuries (Kertzer and Hogan, 1989; Rahikainen, 2004). In Italy, children often earned higher wages than their mothers and, as such, their income was seen as family earnings. As long as they brought home money, children were sent to work (Cunningham and Viazzo, 1996).

In the 18th and early 19th century of England, women's and children's work and wages, which were often not recorded as part of household earning in census data, were usually necessary to family survival when the male head of the household was killed, injured, left, or did not earn enough (Humphries, 2010; Goose and Honeyman, 2013; Humphries, 2013). However, women and children's needs are often underrepresented in historical research and uncounted in living and household expenses (Humphries, 2013) thus often masking the day-to-day experiences of women and children. Though the Industrial Era women worked hard and often, their earnings were small, and they were still expected to complete household duties while raising children. Tanner (1982) describes that 19th century European women worked in laborious conditions in factories throughout their pregnancies.

Household income and food allocation was often left to the mother. Children's earnings were typically given to the mother to allocate towards supporting the whole family. Horrell and Oxley (2012) also suggest that within the home women would often “self-sacrifice” when there were food shortages. While this wouldn't directly affect a grown woman's height, it might affect the growth of her children depending on how she allocated food. A child's portion of food would often depend on how their contributions to the household were valued (Horrell and Oxley, 2012). Most women and young

children would receive the grain-based foods, some potatoes, and tea, while men and adolescent boys, who often earned higher wages, received most of the bread, in addition to meat, dairy, and beer, which had higher nutritional values (Humphries, 2013). This male preference is also accounted for in higher rates of female infanticide (Volland et al., 1997), and less time attributed to breastfeeding female infants compared to males, potentially contributing to higher female infant mortality from Christ Church Spitalfields (Humphrey et al., 2012).

Writings from a 19th century French medical professional documents the need of laws to provide social welfare for the public who were suffering from disease, famine, and short stature during the Napoleonic War (Tanner, 1982; La Berge, 2002). This work provided a framework for doctors in 1830s England to compare the growth of children working in factories to those not working, finding that children who labor had suffered growth insults (Tanner, 1982). According to Parkes (2012), the British Romantic view of childhood, as traced through children's literature from the 18th-19th centuries, initiated the removal of child labor in the course of the 19th century Britain. Factory Acts beginning in 1802 set age and hour limits for child labor and by 1851 most "brutal working conditions" were obsolete (Humphries, 2010). By 1901 it was largely illegal for British children under the age of 12 years to work in factories, a law that undoubtedly influenced other Industrial nations coming into their industrialism after this time, such as Italy and Portugal. These acts were motivated by a changing cultural belief that children should be separate from the market place and be given a proper childhood as opposed to the traditional early 18th century idea that children should not be sheltered, but raised as

“active participants,” thus eventually protecting children from labor (Parkes, 2012).

Living conditions in urban centers were often cramped and polluted as a result of the rapid urbanization (Johnson, 2006). In the mid-18th through 20th centuries, population size increased tremendously in many major urban centers like London and Lisbon, as families had more children and rural immigrants searched for work (Engels, 1958; Thompson, 1963). In other areas such as Bologna however, fertility rates actually declined, likely due to women's long history of work in the silk and weaving industries (Forgacs, 1990; Krause, 2012) that left them less time to raise children.

Many populations were exposed to societal stressors, such as poor nutrition, gender and age inequality, labor, disease, and poor environmental conditions, which are evident in height data and skeletal remains. For example, historians Horrell and Oxley (2012) found that, as a result of growing reliance on grains, especially those that became more processed, or milled down and pre-prepared for consumption, the nutritional quality of the English diet decreased. As nutritional components were less available, diseases like rickets, scurvy, and tuberculosis became more prevalent, especially amongst women and children. When Horrell and Oxley (2012) compared height data (between the sexes) with diet, they found that urban women, who worked away from home, had less access to nutritious foods and were often shorter and had higher mortality rates, compared to rural women who found domestic work and likely had better access to higher quality foods. Lower nutritional status during childhood likely led to overall stuntedness in both men and women (Humphries, 2013).

Skeletal research provides evidence of environmental stress and malnutrition on

the remains of people who lived during the Industrial Era, including children. Cardoso and Garcia (2009) found more evidence of stunted height in Industrial Era adults from Lisbon, Portugal than adults from the Medieval Period, indicating more environmental stress during adolescence of the Industrial Era individuals. Mary Lewis (2002) found that children from the Industrial Era of London, England compared to Medieval England had higher frequencies of linear enamel hypoplasias (LEHs,) dental caries and abscesses, cribra orbitalia, and stunted height, which are all indicative of stressed conditions. While the medieval period is often described as very bleak, in reality more of the population lived in rural areas with more agricultural produce available, making living conditions improved compared to the high population densities and poor sanitary conditions of the 18th and 19th centuries. Mariotti et al. (2015) discovered high rates of infectious diseases in individuals from a 19th century Bologna, Italy skeletal sample. Poor children from 18th-19th century England were more likely to die than those from higher status families (DeWitte et al., 2015). Hughes-Morey (2016) suggests that low status females were more stunted and had the highest risk of mortality during Industrial Era London given the preferential treatment for male children and the added stress of low social status.

London

This research focuses on a skeletal sample from the Spitalfields district from 1729-1859. London, overall had one of the largest populations in Europe from 18th -19th centuries largely due to migration and natural increases in population, as well as the development of the railway system allowing migration from rural areas into urban centers

particularly in the late 19th century (Pooley and Turnbull, 1996; More, 2000; Schwartz et al., 2011).

By the early to mid-18th century, London (in particular the Spitalfields district) experienced immigrants from France, fleeing religious persecution, bringing with them their silk and weaving manufacturing allowing some wealth and industry into the area (Molleson and Cox, 1993). This rise in industrial manufacturing also led to increased population and poor environmental conditions. The 1750s brought decreased wages and severe food shortages to London, especially for the working classes, until after the Napoleonic Wars when wages increased, at the same time the price of bread fell, improving conditions. Wages did not completely bounce back to the level of the 1740s until after the 1840s (Schwartz, 1985; Molleson and Cox, 1993). 1796 saw campaigns for health improvements after a typhus epidemic in Manchester, particularly among children who were exposed to such insults early in life (Douglas et al., 2002). Infant mortality rates were ~30% in the 18th century, with small-pox the major threat until the vaccination became available in 1800. After this, whooping cough took the leading role (Douglas et al., 2002). Records from the Marine Society and the Royal Marine recruitment data show those born before 1800 were severely stunted. For example the average height of a 13-year old boy born between 1753-1780 stood only 4'3" tall while 16-year old boys were 4'9", ~4 inches shorter than average, healthy 13 and 16-year old boys today (Molleson and Cox, 1993). In most industrializing populations in continental Europe urban children often grew taller than their rural counterparts (Tanner, 1982). The opposite pattern was true in England in the 19th century where rural children were taller than those in urban

centers, despite social status, suggesting English urban settings, like London, experienced more stressed conditions than most others (Tanner, 1982).

Early 19th century England saw rising mortality rates and decrease in height amongst the working classes, as well as increase in diseases, population, and pollution (Komlos, 1993; More, 2000; Mays et al., 2008; Bengtsson and van Poppel, 2011) all of which suggest overall poor living conditions. Thick layers of smoke and smog often sat atop London's industrial sectors, primarily northern England, like Manchester, due to the use of coal and steam powered factories and transportation (More, 2000). The smoke pollution in London from 1600-1900 is associated with increased cases of rickets (Molleson and Cox, 1993). Ever-growing buildings and narrow streets, likely caused an increase in nutritional deficiencies, further stunting the growth of the people in London (More, 2000).

Rising population growth in the 1820s and 30s led to a collapse in small, local governments causing even further decline in living conditions such as a lack of clean water and sewage facilities, decrease in available vaccinations for small-pox, and increase in over-crowded housing (More, 2000; Johnson, 2006). While Industrial Era London was experiencing major population and economic growth, agricultural output fell over the course of the 19th century making food, especially nutritious food, harder to come across (Pooley and Turnbull, 1996; More, 2000). The 1830s also saw the cholera outbreak, which marked the beginning of actions taken by British sanitary reformers to clean up housing, water, garbage, and to make better sewage removal (Douglas et al., 2002).

The 1840s and 50s brought increased mortality rates with tuberculosis, typhus, and typhoid outbreaks from overcrowding and unsanitary conditions (Woods and Woodward, 1984), as well as diarrheal diseases amongst infants due to contaminated milk (Douglas et al., 2002). Disease spread was the major cause for mortality in the 19th century, with social class making little difference, especially in highly populated urban areas (Cain and Hong, 2009; Bengtsson and van Poppel, 2011). With improper treatment of sewage came the "Great Stink" of 1851, when raw sewage flowed into the Thames River, which was unfortunately used for various purposes including drinking water. As a result of such unsanitary conditions, cholera epidemics broke out. It was not until John Snow's discovery of the contamination of the Broad Street water pump, which caused the cholera outbreak of 1854, that improving sanitary conditions became a priority (Johnson, 2006; Daunton, 2014; Frerichs, 2016). While attempts were made to improve most of these poor environmental conditions, it happened slowly (More, 2000).

Mortality rates, especially amongst infants, didn't truly begin to decline until the end of the 19th century with better public health policies, better nutrition, and better healthcare practices (Szreter, 1988). The poor environmental conditions of large urban areas, like Manchester, as well as marked class differences within those cities simply took a long time to improve (Douglas et al., 2002). In the 1860s, medical professionals noticed the poor died at younger ages amongst the poor compared to the wealthy. It was at this point that greater concern about the health and living conditions of the people were exhibited, real wages began to increase by two thirds for most of the working class (Bane, 1990; Bengtsson and van Poppel, 2011), and public health, sanitation, and housing

acts were passed (Szreter, 1988). In 1890 a waterborne sewage disposal system helped reduce instances of typhoid (Douglas et al., 2002). Unfortunately, most of these environmental improvements and reforms came after the skeletal sample's time period ended.

Bologna

The skeletal sample from industrializing Bologna, used in this project, dates from 1880-1935. During the 19th century, Italy was ruled by a parliamentary oligarchy, composed mainly by rural notables. These rural elites did not encourage revolutionary actions and industrialization, thus resulting in late Industrial growth in Italy (Webster, 1975; Cardoza, 1982). The ruling class was reluctant to promote steam-powered, factory-based production of goods for industry, since their income was primarily based on agriculture and the traditional patriarchal notions of family life in north Italian textile manufacturing, with women often weaving at home (Capecchi, 1997; Bosworth, 2013). Elites saw the potential for a big urban transition and a bigger middle-class disrupting their way of life (Webster, 1975; Cardoza, 1982).

In the late 1860s, Bologna saw small industries beginning with iron ore, steel, and chemical production, and by 1886 the ruling class discovered this industrial output would allow more political and economic independence thereby finally encouraging economic growth (Vaccaro, 1980; Capecchi, 1997). Italian Unification sought to gain notoriety among other European nations and be competitive as a primary political system (Vaccaro, 1980). Though, it was from 1883-1914, with the socialist mayor of Bologna who

provided public housing, public access to education and the university, rising incomes, and assistance programs improving social conditions, that industrial growth really started (Vaccaro, 1980; Capecchi, 1997).

Italy's industrialization officially began in 1883 with the first hydroelectric power station in Europe, in Milan (Forgacs, 1990) allowing better transportation of goods and services. Industrial growth was concentrated in northern Italian regions like Milan, Florence, and Bologna. The urban center of Bologna became well-known for their agricultural, engineering, silk, cotton, tobacco, steel, chemical, automotive, packaging, and meat industries (Vaccaro, 1980; Capecchi, 1997; Forgacs, 1990). Between 1900-1914, Italy was a leading competitor in the silk, cotton, and wool market, and saw a population increase by 30% with great prosperity and growth (Vaccaro, 1980).

Despite its seemingly "good" environment from socialist movements and leaders, the textile industries in Bologna employed 60-70% women and 24-32% children, compared to only 8% of men. In this patriarchal society women and children's work was often undervalued in the society and the home (Kertzer and Hogan, 1989; Krause, 1999; 2005; 2007). The disenfranchised women and children can be further understood by the infant mortality rate in Bologna, which was between 40-47% by 1910 (Klüsener et al., 2014).

Italy was ruled by the fascist leader Mussolini at the end of World War I in 1918 until the end of World War II in 1945 (Webster, 1975; Hobbs et al., 1999). Despite continued (yet slow) industrial growth, by 1921 half the country, including Bologna was still agricultural and very poor with rising unemployment and inflation compared to other

northern European standards due to war-time restrictions (Vaccaro, 1980; Capecchi, 1997; Bosworth, 2013). Most laborers of Bologna were required to work additional hours and days to simply earn a living wage (Capecchi, 1997). The Bolognese eventually led the market for hemp weaving, primarily by peasant women in the confines of their homes (Capecchi, 1997).

Men emigrated to find work leaving their families behind. Women were left to work in and out of the home, work on the land if they had it, handle the family finances, and educate their children on their own. Home-weaving systems worked well in the peasant society with women and children often left home to remain in their traditional roles as housewives and left out of industrialism and nationalism (Capecchi, 1997; Bosworth, 2013). Italy started experiencing a fertility decline over the course of the 20th century despite Mussolini's pro-natalist campaigns (Krause, 2005, 2007; Bosworth, 2013), perhaps a reflection of men emigrating out of the country and fighting in World War II, as well as the hard work women endured, and a lack of sufficient means to support a family.

Since women in Bologna were the driving force of industrial production primarily through weaving (Capecchi, 1997), it no doubt put pressure on family life. However, women and children were still considered inferior, submissive, and treated harshly (Krause, 1999; Rahikainen, 2004; Bosworth, 2013), which likely affected the health of both women and their infants if they were pregnant during times of hard work and stress (Tanner, 1982).

The 19th and 20th centuries saw the Spanish flu, tuberculosis, cholera epidemics,

starvation, poverty, and open sewage in the streets of some areas (Bosworth, 2013). By 1926, food staples, like vegetables, fruits, wine, and olive oil were largely unavailable forcing people to sell their land, homes, and go into extreme debt and eventually bankruptcy (Cardoza, 1982). The population became very unhappy with the fascist government, which was likely the cause of the overall poor living conditions including low wages and increased prices, and responded with labor strikes and unrest. As a result, in the 1930s Italy started to shift toward more modern standards of education with increasing numbers of children attending school (Forgacs, 1990). Eventually, leftist movements led to the end of the fascist regime at the end of World War II (1945) and Italy adopted a republic with a parliamentary democracy (Webster, 1975; Cardoza, 1982; Hobbs et al., 1999). Growth in industry and literacy reshaped the culture with workers beginning to receive higher wages and shorter working hours.

Lisbon

The time period of relevance for the skeletal sample from Lisbon is 1805-1975. During this time period, conditions for industrial development in Portugal were slow and living conditions for its populace were fairly poor. Historically, Portugal was ruled by a monarchy until the first Napoleonic invasions in 1807, lasting for six years leaving the country unstable (Cardoso, 2005; Oliveira and Pinho, 2010). This instability led to civil war between absolutists and liberals in the 1820s resulting in a monarchy led by King D. Pedro IV with parliamentary representation that allowed urban planning, industrial activity, and public works (Oliveira and Pinho, 2010). It was most likely this political

upheaval and turmoil of the first half of the 19th century, that caused the Portuguese population to fall from a decrease in natural growth, low immigration, and an increase in epidemics (Reis, 2009).

It was not until the 1850s that Lisbon experienced regrowth. Between 1864 (the first official census) and 1890, the number of inhabitants in Lisbon increased by 25% (Reis, 2009; Oliveira and Pinho, 2010; Da Silveira et al., 2013). By 1895, this increased migration resulted in increased industrial productivity. The main industries in Lisbon were textiles, metalworking, food, cork, fishing, ceramics, and tobacco (Reis, 2004). However, the international market was already established and competitive with lower priced foodstuffs, making Portuguese industrial growth slow and almost obsolete in all but cork, fishing, Port wine, olives, and the service sector, which were successful (Reis, 2004; 2009; Oliveira and Pinho, 2010). The labor force was very poor and comprised large numbers of women and children, with women earning the least (Reis, 2004).

Instability within the government led to the assassination of King Carlos I and his son in 1908 by anarchists. Portugal was established as a Republic state in 1910, and entered the 20th century carrying great political instability of the past, resulting in a weak capitalist system that had been geographically isolated from most other parts of Europe. At this point, Portugal was one of the least developed countries in Europe, with a strong, central Catholic influence, and fragile agricultural system with most farmers practicing subsistence farming through most of the 20th century (Giner, 1982; Reis, 2004; Cardoso, 2005; Reis, 2009; Oliveira and Pinho, 2010), and an infant mortality rate of 30-40% (Klüsener et al., 2014).

Most other European countries industrialized after increased productivity had expanded the food supply, while in Portugal such development was limited by the preponderance of small-scale subsistence farming mostly in the south where agriculture was under large land owners and population growth spurred out-migration into urban centers like Lisbon (Cardoso, 2005). Over the course of 1890 and 1960 the population of Lisbon tripled, mostly from rural migration of poor, young, illiterate, and unskilled men (Reis, 2004; 2009; Cardoso, 2005; Cardoso and Garcia, 2009), while most of the skilled workers and artisans migrated into more peripheral territories to follow new industrial enterprises (Reis, 2009). Within Lisbon, necessities (food, clean water, employment, and housing) could not keep up with demand causing increased prevalence of disease, mortality, and overall poorer living standards (Reis, 2009).

Portuguese industries were small often exploiting peasant labor, the illiterate, and women and children who had very few rights, earned the lowest wages, and worked under harsh and disciplined conditions (Giner, 1982; Reis, 2004; 2009; Cardoso, 2005). Boys started earning more money in factory and military positions by age 15 years, especially if they were skilled (Reis, 2004). Portuguese families were patriarchal with children and wives under the father's authority and boys were expected to inherit the land and property of the family. Efforts to prevent the division of property were great (Pina-Cabral, 1992).

Late 19th and early 20th century Lisbon was often described as a "nauseating place", full of disease and filth, overcrowded, and the working class inhabitants living in even worse conditions with inadequate foods mostly made up of cereal grains and

vegetables, with very little meat (Reis, 2009). The poor conditions, the effects of World War I, and the influenza epidemic of 1918 left the population faltering (da Silveira et al., 2011). Life expectancy in 1920 was at an all-time low, at 30-40 years, and half of all children died before they reached 15 years, primarily from infectious diseases like tuberculosis and influenza (Cardoso, 2005) reflecting the very poor societal conditions, especially amongst the underprivileged. Portugal was one of the shortest populations in Europe reflecting these conditions (Stolz et al., 2013). Portuguese height declined by 10% in the late 19th century. Compared to the UK, Portugal suffered more stuntedness and poorer living conditions, while those of the UK were steadily improving (Stolz et al., 2013).

After many years of governmental upheaval, Portugal fell under the establishment of the dictator Antonio de Oliveira Salazar in 1933. The Salazar dictatorship resulted in a fragile agricultural system and weak aristocracy. The average income of the people in Portugal became one of the lowest in all of Europe. After 1943, a rise in industry related to war efforts resulted in even poorer living conditions. Food was scarce and hard to come by, including staples like grains. Housing, sanitation, education, and healthcare were largely unavailable to most of the working class population. During World War II, food was even harder to get with fewer available resources, lower wages, and increased prices. Over half of the population lived in poor sanitary conditions and lacked power.

Eventually, strong social movements led to a military coup in 1974, the longest dictatorship of Europe ended, resulting in a democratic system. This is when conditions finally began to improve for the people of Portugal (Giner, 1982; Cardoso, 2005; Stolz et

al., 2013). Family incomes increased allowing them more access to nutritional food and healthcare, as well as better public health polices improving sanitary and living conditions throughout the city (Cardoso, 2005; 2006; 2008; Cardoso and Garcia, 2009). Military data show that since 1902, men's height has increased 8.93cm to 2000, with most improvement occurring after the 1970s.

It is important to note the changing social and political structures in each of these cities. In this research, the skeletal samples are defined by social status for comparative purposes. The sample from London is defined as high status, those from Bologna are middle class, while those from Lisbon represent the low social status. These status definitions are important for comparisons, are based on historical knowledge of the samples, and have been used to describe these individuals in past research. However, these samples span large periods of time, include numerous individuals who likely varied from each other in status, and represent changing social situations. While status is used as a variable in this project, it must be taken with caution.

CHAPTER 3
GROWTH AND DEVELOPMENT, STRESS, AND VARIATION
BETWEEN BOYS AND GIRLS

Typical growth in healthy populations follows a fairly specific pattern that has been mapped through decades of growth studies (Tanner, 1989; Bogin, 1999; Cameron, 2002). Boys and girls have similar, yet distinct, variation in their growth trajectories, with girls growing and maturing at a faster rate than boys. Boys, however, experience a longer period of overall growth, resulting in typically higher height and weight values than girls by the end of growth (Tanner, 1989; Bogin, 1999; Cameron, 2002). Children are some of the most vulnerable members within a population and their growth is often an indicator of the health status of a population (Tanner, 1982; Schell, 1989; Bogin, 1999; Cameron, 2002; Leatherman and Goodman, 2005). If environmental conditions interfere with their ability to grow and develop, they can suffer from stunting, increased morbidity through disease and malnutrition, and increased mortality (Stini, 1969; Van Gerven and Armelagos, 1983; Tanner, 1989; Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002; Cardoso, 2005; DeWitte et al., 2015; Hughes-Morey, 2016). While this is an issue for all children, boys may be at an increased risk compared to girls, who may have adapted a buffering response to stressed conditions through increased fat and protein reserves (Stini, 1969; Roede; 1978; Buffa et al., 2001; Oyhenart, 2006; DeWitte, 2010; Redfern and DeWitte, 2011). The result can be increased risk of stuntedness and morbidity in boys, thus reducing overall sexual dimorphism. Sex estimation in adult skeletal

assemblages is a relatively simple task through morphological and size variation assessment of the skull, pelvic bones, and most long bones. However, children's skeletons are more difficult to assess, especially if their growth was impacted by environmental stress. This chapter will review typical growth in healthy children, the variation of growth between boys and girls, the impacts of stress on their ability to grow and develop, and how growth, sexual dimorphism, and stress are analyzed on children's skeletal remains.

Growth

Throughout gestation and infancy, both males and females release testosterone and estradiol at rates equivalent to the pubertal growth spurt (Tanner, 1989; Quigley, 2002; Guatelli-Steinberg et al., 2008). A testosterone surge in infant males peaks around two months of age, reduces to pubertal levels by six months, and continues reducing to very low levels throughout childhood. Males estradiol levels peak from zero-two months, then reduce to pre-pubertal levels until the sixth month, in which it drops to very low levels (Guatelli-Steinberg et al., 2008; Winter et al., 1976; Gassler et al., 2000). Female infants have a testosterone surge that reaches pre-pubertal levels between the first week of life until the second month, while estradiol levels are above the pre-pubertal range from zero to six months (Gassler et al., 2000; Guatelli-Steinberg et al., 2008). These hormones drop from the sixth month until the first year in both sexes and will increase at a slow and steady rate until the onset of puberty (Gassler et al., 2000; Guatelli-Steinberg et al., 2008). These surges have a specific function in the development of male testes (Forest et al., 1973; Corbier et al., 1992; Dakin et al., 2008), but their effect on

differential skeletal growth of males and females is not understood.

As early as during fetal development, female skeletons can be up to three weeks more advanced than males, and at birth four to six weeks more advanced. Females reach around 50% of their adult height by age 1.75 years and boys at 2.0 years (Tanner, 1989). Males, on the other hand, appear to be more advanced in dental development and eruption (Tanner, 1989), and show more variation in skeletal maturation and growth when affected by factors of poor health (Tanner, 1989; Buffa et al., 2001). Therefore, girls continue to mature faster and enter pubertal growth around two years sooner than males and, as a result, reach terminal height sooner while boys continue growing for a longer period of time, ultimately allowing them to grow, on average, taller than their female counterparts.

Growth Distinctions and Age Groups

This project utilizes four age groups that reflect typical developmental markers like tooth eruption, growth spurts, and fusion of long bones. Skeletal biologists often use age categories in growth studies to compare variation in growth between sexes and samples in the most appropriate way. While researchers identify age groups in various ways, commonly used labels and age boundaries for groups are Infant (0-1 year), Young Child (2-5 years), Older Child (6-12 years), and Adolescent (13-18 years) (Baker et al., 2005; Roksandic and Armstrong, 2011). Utilizing age-groups can give researchers the opportunity to compare children according to growth and developmental phases.

However, age-groups can also create challenges, such as minimizing sample sizes and

comparing children on one end of the group to the other, for example comparing an 18 year-old boy to a 13 year-old girl. While these are serious challenges, they are to a large extent unavoidable in skeletal growth studies. The use of age groups can help narrow the age ranges being compared while attempting to maintain a minimum sample size for statistical analysis. To help understand the variation in growth in the skeletal samples, this project will use age groups in statistical comparisons and continual data in the form of growth curves when they are appropriate.

Infants, from birth to 1 year of age experience rapid brain and body growth. The skull bones are not fused together and there is no epiphyseal fusion of long bones (Black and Scheuer, 1996; Baker, 2005; White, 2005; Schwartz, 2007). Dentition typically starts erupting by around 6 months of age and continues in a fairly specific pattern (Buikstra and Ubelaker, 1994; White, 2005).

The Young Child age group represents individuals 2 to 5 years of age. Between the ages of 1 to 6 years, body growth slows to a steady rate, while brain growth continues to increase rapidly, reaching 91% adult size (Enlow, 1990; Eveleth and Tanner, 1990; Graw, 1999; Wahl and Graw, 2001; Kalmey and Rathbun, 2006; Gapert et al., 2009).

The Older Child group consists of those between 6 to 12 years of age. Children 7 to 9 years, typically experience a small “childhood” growth spurt (Tanner, 1989; Bogin, 1999; Cameron, 2002) and by 10-11 years, children enter a “pre-pubertal” period in which hormonal surges begin. Boys and girls alike experience most of their skeletal growth in their long bones during that time (Tanner, 1989; Eveleth and Tanner, 1990). By ages 11 and 12, girls' growth rates begin to accelerate and, as a result, they may exceed

boys of the same age in height and weight (Cameron, 2002). During this time the permanent canine typically erupts and many epiphyses, like the distal humerus, start to fuse.

The Adolescent age group contains individuals going through the pubertal growth spurt in which children grow a significant amount in short period of time (Tanner, 1989; Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002). This group is made up of 13-18 year olds. Puberty begins around 12-14 years of age in typical Western populations (Bogin, 1999; Cameron, 2002), resulting in distinct sexual differences in pelvic, cranial, and long bones (Buikstra and Ubelaker, 1994; LaVelle, 1995; White, 2005). Girls in Western cultures enter this growth spurt first, around 12 years of age and generally complete growth by 13-14 years. However, girls outside of Western culture, such as hunter-gatherers, or those from poor environments typically experience delayed pubertal growth with age of first menarche one or two years later than girls from wealthier populations (Ellison et al., 1986; Spielmann, 1989; Belachew et al., 2011). For example, as early as the 1600s Austrian physician Hippolyt Guarinoni, discovered that poor peasant girls began menstruation long after their urban, wealthier counterparts (Tanner, 1982), while even in our modern world Belachew and colleagues (2011) find that food insecure girls from Ethiopia experience first menarche one year behind food secure girls, and stunted girls are delayed an additional year. During this time girls experience rapid growth of the trunk and will start to experience increased fusion of epiphyses. During this period, boys are still undergoing growth of the long bones from their “pre-pubertal” growth. Their pubertal growth spurt begins roughly two years after girls, around 13-14

years of age. The male growth spurt primarily affects trunk growth and continues to peak until around age 16 (Tanner, 1989; Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002). They continue to grow slowly and steadily until all the epiphyses have fused. The last bones to fuse in both males and females are the clavicle and some aspects of the pelvis around age 25 years.

Skeletal Sexual Dimorphism in Children

Under normal, healthy conditions, boys generally grow larger than girls in height, weight, upper arm muscle mass, and head circumference, resulting in sexual dimorphism (Tanner, 1989; Bogin, 1999; Cameron, 2002). However, this is difficult to assess in children's skeletal remains. In adult remains, sex can be determined with high rates of probability. The morphology of pelvic bones often yields 90% or higher accuracy in sex estimation (Buikstra and Ubelaker, 1994; Bass, 1995; White, 2005). Cranial bones are often used to determine sex in adults, with male crania exhibiting larger and more robust features (Buikstra and Ubelaker, 1994; White, 2005; Williams and Rogers, 2006). Long bone lengths also provide a reliable means of determining sex (Buikstra and Ubelaker, 1994; Bass, 1995; White, 2005) because males continue to grow for a longer period of time than females (Tanner, 1989; Bogin, 1999; Cameron, 2002). These traits, however, do not exhibit statistically significant dimorphism in skeletal remains until after the pubertal growth spurt (Buikstra and Ubelaker, 1994; Scheuer and Black, 2000; Baker et al., 2005; White, 2005; Schwartz, 2007), suggesting that a shift to analyzing other areas of the skeleton is needed.

Research in juvenile skeletal sexual dimorphism has a long history (Weaver, 1980; Mittler and Sheridan, 1982; Schutkowski, 1993; Molleson and Cruse, 1998; Rissech and Malgosa, 2005; Cardoso, 2008; Vlcek et al., 2008; Rogers, 2009; Blake, 2011). Most of this research is hindered by various factors such as a lack of juvenile remains of known sex and age, small sample sizes yielding low statistical power (Noren et al., 2005; Cardoso, 2008; Blake, 2011), use of broad or no age categories, masking developmental differences between the sexes (Sutter, 2003; Noren et al., 2005) a focus on univariate analyses or multivariate analyses limited to one region of the skeleton (Ridley, 2002; Cardoso, 2008; Gapert et al., 2009; Rogers, 2009; Veroni et al., 2010), and use of secondary sex characteristics that do not become significantly dimorphic until the onset of puberty (Weaver, 1980; Mittler and Sheridan, 1992; Schutkowski, 1993).

A number of studies, however, suggest juvenile skeletons do exhibit sexual dimorphism in discrete morphological traits, such as pelvic and mandible shape, in infants and decreased dimorphism of these in childhood (DeVito and Saunders, 1990; Schutkowski, 1993; Ridley, 2002; Noren et al., 2005; Cardoso, 2008; Gapert et al., 2009; Veroni et al., 2010; Blake, 2011), possibly due to genetic and hormonal surge differences established during gestation and infancy (Tanner, 1989; Guatelli-Steinberg et al., 2008). Some studies aimed at evaluating sex differences in pre-pubertal remains, utilizing other aspects of the skeleton, have also yielded successful results ranging from 60-94% accuracy (DeVito and Saunders, 1990; Schutkowski, 1993; Noren et al., 2005; Cardoso, 2008; Gapert et al., 2009; Veroni et al., 2010; Gonzalez, 2011) showing that juvenile skeletons are, to some extent, sexually dimorphic (Tanner, 1989; Guatelli-Steinberg et al.,

2008). The cranial base, for instance (Wahl and Graw, 2001; Graw et al., 2005; Noren et al., 2005; Kalmey and Rathbun, 2006; Lieberman et al., 2006; Gapert et al., 2009), may become dimorphic in juveniles very early in development because the cranium reaches 91% of its adult size, along with brain growth, in the first five to six years of life (Enlow, 1990; Graw, 1999, 2005; Noren et al., 2005; Gonzalez, 2011). Some elements, like the petrous bone, reach adult size at two years (Noren et al., 2005). Size differences have been found in deciduous and permanent teeth, especially mesiodistal measurements of the canines, with males two to seven percent larger than females (Ditch and Rose, 1972; DeVito and Saunders, 1990; Iscan and Kedici, 2003; Kondo and Townsend, 2004; Holcomb et al., 2005; Kondo et al., 2005; Cardoso, 2008; Viciano et al., 2011). The Y-chromosome houses genes that may code for more dentine and enamel growth, allowing early increases in tooth size in males (Alvesalo, 1997; Guatelli-Steinberg et al., 2008). Finally, the morphology of the distal humerus, sometimes called the “carrying angle,” has been shown to be dimorphic once the distal epiphysis fuses, ~10 years of age. Rogers (2009) found males typically show 10-15° lateral angles of the radius and ulna relative to the humerus while females usually exhibit 20-25° angles.

This review of sexual dimorphism in juvenile remains highlights two points of particular significance for this project: 1) the effects of hormonal surges during fetal development and infancy should be reflected in early onset of sex differences in the entire skeleton, distinguishing boys from girls, and 2) the importance of realizing how some aspects of the skeleton may possess sexually distinct features despite environmental impacts during growth, a factor that most juvenile skeletal samples will face. However,

despite some evidence of sexual dimorphism in subadult remains, there is a lack of significant variation between sexes. One possible reason for the lack of significant dimorphism may lie with the poor health, malnutrition, and stunted growth in children who died early in these past populations.

The Effects of Stress on Growth

The long period of growth and plasticity characteristic of humans makes the environments under which child development takes place a particularly important determinant of health outcomes (Johnston, 2002). While boys and girls should exhibit dimorphism, research shows that those growing under conditions of stress exhibit significantly less dimorphism (Stini, 1969, 1972; Buffa et al., 2001). The rate of growth and overall adult height within a population reflects the environmental quality during childhood (Stini, 1969; Van Gerven and Armelagos, 1983; Tanner, 1989; Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002; Agarwal, 2016). The growth of children, therefore, is a measure of the overall health of a given population. Evidence of stunting, stress, and increased mortality in juvenile skeletal remains, reflects exposure to poor environmental conditions like malnutrition, illness, cramped and polluted living conditions, low SES, and hard labor, with those with reduced stature having greater incidences of evidence of other stressors (Stini, 1969; Van Gerven and Armelagos, 1983; Goodman et al., 1984; Martin et al., 1984; Tanner, 1989; Eveleth and Tanner, 1990; Bogin, 1995; 1999; Saunders, 2000; Stinson, 2000; Cameron, 2002; DeWitte et al., 2015; Agarwal, 2016).

Diet and nutrition are important factors in growth. Given that political and economic factors heavily influence access to proper nutrition, growth represents a potential measure of the economic contribution to societal needs (Norgan, 2002). Daily food intake is challenging to evaluate in living populations, and it is even harder to interpret food intake from past populations when relying on written records, many of which do not present accurate data on women versus men, children versus adults, girls versus boys, and between households. This challenge results in literature that is unclear about the day-to-day diets of people in the past, impacting interpretations of nutritional intake in skeletal research.

By the 19th century, medical doctors and scientists became aware of the negative impacts of poor nutrition during childhood (Norgan, 2002). Nutrition started being measured as a proxy to health and growth, as well as an understanding of growth cessation and catch-up when nutritional factors changed. A child is considered to have low body weight, be stunted in height, or wasted (weight for height) when they are ≤ 2 Z-scores below the mean of the population (Bogin, 1999; Cameron, 2002).

Malnourishment can begin in fetal development with lack of healthy foods available to the mother, oftentimes resulting in babies being born with low birthweight. According to Barker's fetal programming hypothesis and the Developmental Origins of Health and Disease (DOHaD), such early growth and developmental delays can result in long-term health consequences such as immune function impairments that can cause greater susceptibility to infectious diseases, decreased cognitive abilities and muscle mass, as well as a greater risk of morbidity and mortality throughout life (Barker and

Osmond, 1986; Barker et al., 1989; Stinson, 1992; Barker et al., 1993; Haas, 1998; Kuzawa, 1998, 2005; Barker, 2007; Armelagos et al., 2009; Wadhwa, 2009).

Maternal malnourishment can also impact the growth and health of the breastfeeding infant if breastmilk is insufficient in quality and quantity potentially decreasing the infant's immune system development (Allen, 1994; Haas, 1998; Bogin, 1999). At the point of weaning, the child becomes susceptible to stress from inadequate foods and diarrheal diseases through potential contamination of those foods (James et al., 1972; Rowland, 1983; Brown, 2003).

Aside from malnourishment, there are many other variables that can negatively impact growth. Populational growth stunting is often seen in times of famine, but also infections, disease outbreak, parasites, poverty, poor sanitation, pollution, war, and civil, political and economic unrest (Floud et al., 1990; Norgan, 2002; Schell and Knutsen, 2002). Low SES and poverty are likely the largest contributors to malnutrition through little access to high quality and quantity food, higher susceptibility to disease through poor living conditions, and higher risk of infectious diseases from a weakened immune system from such poor nutrition and living conditions. Malnutrition is a populational problem common in developing societies, like those undergoing industrialization.

If and when poor conditions improve, children may have the opportunity to "catch-up" to an expected height for age. Catch-up growth refers to a rapid growth velocity in height or weight in a short period of time (Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002). However, the timing, severity, and duration of the growth insult impact the ability for catch-up growth to be complete (Eveleth and Tanner, 1990; Bogin,

1999; Teranishi et al., 2001; Cameron, 2002; Agarwal, 2016). Continued exposure to stress may prevent catch-up growth. Some cultural factors have been shown to affect whether children have the ability to catch-up (Eveleth and Tanner, 1990; Bogin, 1999; Cameron, 2002; Norgan, 2002; Agarwal, 2016). For instance, having parents who are less likely to be sick, who have steady income, improved social status, and movement out of the stressed environment, as well as being the oldest child or having few siblings, which can result in more resources allocated toward the child, may all positively influence a child's growth (Norgan, 2002; Teranishi et al., 2001). When delayed growth occurs in early childhood, prior to three or four years of age, when growth should occur at a higher velocity, children with improved conditions are more likely to experience complete catch-up growth than those who become delayed in later childhood or adolescence (Martorell and Habicht, 1986; Eveleth and Tanner, 1990; Stinson, 1992; 2000; Bogin, 1999). Without the potential for catch-up growth, stunted children become shorter adults (Prader et al., 1963; Angel, 1972; Larsen, 1984).

Pollution, poverty, disease, unsanitary living conditions, malnutrition, hard labor, and war are all cultural factors negatively impacting growth in many of today's populations, as well as those from the past. These factors make up the overall environment of the child and interact with their genetic growth potential and result in the expression of such consequences. When conditions are good, children grow in regular intervals in height, weight, fat, and muscle mass. When conditions are stressed, children can experience slowed growth in all of these. In a study of 12-year old boys from Guatemala City, Bogin and MacVean (1978) and Johnston and colleagues (1984) found

that those from a low SES were shorter, lighter, and had less body fat and muscle mass compared to those from a middle SES.

Improvements to overall environmental conditions can improve the growth status of children and result in positive secular trends (Johnston, 2002). While most developed or industrial populations have experienced an increase in height over the last 50-100 years, many European populations exhibited a decline from the 11th - 19th centuries (Johnston, 2002), reflective of poor environmental conditions. Given the impacts of poor environments on growth and the stressed conditions of the Industrial Era in Europe, skeletal remains from that era afford an excellent opportunity to study how boys and girls respond to such conditions differently.

Female Buffering

While poor conditions negatively impact the growth of all children, boys may be at a particular disadvantage. Research since the 1960s suggests that girls are biologically buffered against environmental stress, morbidity, and mortality compared to boys perhaps reflecting evolved mechanisms tied to reproductive demands (Greulich, 1951; Stini, 1969, 1971, 1972, 1978, 1980; Roede and van't Hof, 1978; Brauer, 1982; Wolfe and Gray, 1982; Hiernaux, 1985; Stinson, 1985; 1992; 2000; Tanner, 1989; Eveleth and Tanner, 1990; Pucciarelli et al., 1993; Buffa et al., 2001; Oyhenart, 2006; DeWitte, 2010). This "female buffering" hypothesis states that girls' bodies are evolutionarily adapted to reserve nutritional components, like fat and protein, during growth (for use during pregnancy and nursing), buffering them during times of stress (Buffa et al., 2001). This

allows girls to continue growing while boys, suffering from the same environmental stresses, may experience more stunting (Stini, 1978, 1980; Wolfe and Gray, 1982; Buffa et al., 2001). In fact, females have enjoyed a longer life expectancy than males since the mid-19th century in most Industrial Era European populations (Preston, 1977). They also have more success surviving during times of stress and are more likely to experience catch-up growth in preadolescence (~8-13 years) than boys, who often remain stunted (Greulich, 1951; Stini, 1969, 1972; Tanner, 1989; Eveleth and Tanner, 1990), thereby decreasing sexual dimorphism in stature of adults (Stini, 1969, 1971; Roede and van't Hof, 1978; Brauer, 1982; Hiernaux, 1985; Pucciarelli et al., 1993; Oyhenart, 2006; Cardoso and Garcia, 2009).

Early growth studies such as Greulich's (1951) found that Guamanian boys and girls in 1947 experienced growth deficits compared to healthy U.S. children from Cleveland, OH. However, despite overall poor growth, boys' growth was slowed compared to that of girls, reducing overall sexual dimorphism. Buffa and colleagues (2001) also found that boys may endure more physiological stress throughout development. Oyhenart and colleagues (2006) examined living children in Argentina and found that overweight and healthy weight children show sexual dimorphism in height, weight, head circumference, and upper arm muscles at all ages. Underweight children exhibit dimorphism in weight, head circumference, and upper arm muscle, but not height. Malnourished and stunted children, on the other hand, only exhibit dimorphism in head circumference. Of the underweight and stunted children, males were more negatively affected than girls. Pucciarelli et al. (1993) found similar results in living children from

the outskirts of Buenos Aires. Their results indicate malnourished children do not show sexual dimorphism in standing height or upper arm muscle, but do exhibit significant dimorphism in head circumference.

Garvin and Ruff (2012) analyzed sexual dimorphism in adult skeletons from varied environmental conditions and found that individuals from lower SES backgrounds and growing under conditions of stress had decreased body size dimorphism. Cardoso and Garcia, 2009 found that adult dimorphism in height is decreased in an Industrial Portuguese sample that shows significant levels of environmental stress, while a Medieval Portuguese sample that exhibits evidence of catch-up growth and improved conditions, shows increased dimorphism in height. DeWitte (2010) discovered that men from Medieval London had an increased risk of mortality, especially when they exhibited signs of pathological stress, relative to women. Hughes-Morey (2012) found that low SES males had the greatest frailty compared to low status females, and high status males and females. Zakrzewski (2003) found that males exhibited more variability in stature, becoming more stunted in times of stress and increasing stature more than females when conditions were improved. All of this research suggests that while all individuals may suffer similarly in the same poor environmental conditions, females may be more resilient when exposed to stressful conditions.

On the other hand, males, especially from higher status, also tend to experience cultural buffering as a result of preferential treatment in patriarchal societies, potentially masking the positive effects of biological buffering in females (Stinson, 1985; Hughes-Morey, 2012). As discussed in Chapter 2, a number of Industrial Era social historians

mention the dietary preferences of higher quality and quantity of foods, such as meat, beer, and cheese given to boys and men (Horrell and Oxley, 2012; Humphries, 2013). Malnutrition increases stunting and vulnerability to other forms of morbidity, including infectious diseases, parasites, and frailty, and eventually mortality (Ortner, 1998). Such forms of stress impact frailty, mortality, and survivability in different ways depending on variables like genetics, sex-hormones, gender, environmental conditions, SES, and political-economic conditions of the population and potentially impact interpretations of skeletal research (Waldron, 1984; Wood et al., 1992; Klein, 2000; DeWitte and Stojanowski, 2015).

Evidence of Stress on Skeletal Remains

Defining the health status of a population is challenging. For example individuals who express evidence of disease or malnutrition may be defined as having "poor health," but if they are able to survive longer than those whose bodies exhibit no evidence of such stress, they may be better defined as having "good health" or better "survivability". A major aim of skeletal biology is to estimate the health conditions of past populations, but this task is rendered particularly difficult by the fact that, while any of the individuals who comprise skeletal samples may have experienced stressed conditions that impacted their overall health or survivability, only certain diseases or signs of stress may be visible on the skeleton, and only if the timing, severity, and even survival from such diseases are expressed in the first place (Ortner, 1991; Wood et al., 1992; Goodman, 1993; 1996; Goodman and Martin, 2002; Armelagos et al., 2009).

Since "health" can be problematic to define (DeWitte and Stojanowski, 2015), the goal instead is to identify levels of stress experienced by these individuals. This gives clues that allow interpretations about conditions of past populations. If the individual dies before the body has time to heal, evidence of stress may be left to identify. However, the lack of stress evidenced by the skeleton does not mean the individual did not suffer from health insults in life. The best ways to interpret stress is to analyze a range of indicators, such as stunted height (which can be interpreted through femur or tibia length), signs of infectious diseases, evidence of malnutrition, and mortality rates. These clues give a momentary glimpse into some of the conditions experienced by certain subgroups (those who were likely most vulnerable and frail) in the population, allowing the opportunity to make interpretations about stress within the overall population and between individuals and sub-groups (Wood et al., 1992; DeWitte and Stojanowski, 2015).

Evidence of disease and malnutrition are most commonly identified in skeletal remains from stunted femur length, linear enamel hypoplasias (LEHs) on the teeth, and non-specific indicators of stress like porotic hyperostosis (PO), cribra orbitalia (CO), and periostitis. These are often used as an assessment of stress and survivability within a population (Saunders and Hoppa, 1993; Goodman et al., 1998; Lewis, 2002; Hoppa and Fitzgerald, 2005; Cardoso and Garcia, 2009; Schillaci et al., 2012; DeWitte and Stojanowski, 2015; Larsen, 2015; Agarwal, 2016; Hughes-Morey, 2016).

Dental linear enamel hypoplasias (LEH) are widely used as an indicator of acute stress in early childhood (Goodman and Armelagos, 1985; Ogden, 2008). These can be formed when enamel is laid down over dentin during development. When children

experience systemic stress such as malnutrition, weaning-related malnutrition, and disease, enamel may not form properly or at all, leaving a permanent visible band of discoloring, pits, or furrows primarily on the anterior teeth such as the incisors and canines (Goodman et al., 1980; Goodman and Armelagos, 1985; Reid and Dean, 2000; Boldsen, 2007; Ogden et al., 2007; Ogden, 2008).

Periosteal new bone formation, or periostitis, can be a non-specific, generalized response to infection, malnutrition, or trauma visible as new woven bone is laid down through increased osteoblast activity on the periosteal layer of the long bones (Huss-Ashmore et al., 1982; Skerry, 1994; Larsen, 1997; Ortner, 2003; Roberts and Manchester, 2005; Geber and Murphy, 2012; Ragsdale and Lehmer, 2012; DeWitte and Stojanowski, 2015). Periostitis is linked with a higher risk of mortality (DeWitte and Wood, 2008; DeWitte and Stojanowski, 2015), making this a common indicator of stress in bioarchaeological research.

Porotic hyperostosis (PO) and cribra orbitalia (CO) are most likely indicators of malnutrition specific to hemolytic or megaloblastic anemias, possibly due to a lack of vitamin B-12 or iron (Ortner, 2003; 2008; Wapler et al., 2004; Walker et al., 2009; Oxenham and Cavill, 2010; McIlvaine, 2013). PO is recognized as diploic expansion on the frontal, parietals, and occipital bones. CO may be related to PO, but is visible as lesions similar to periostitis on the roof of the orbits rather than the cranial vault. Some research suggests this may be more indicative of scurvy (a vitamin C deficiency) (Walker et al., 2009) or rickets (a vitamin D deficiency) (Wapler et al., 2004) rather than anemia. Nonetheless these are both signs of malnutrition.

An extensive body of skeletal research on stress, from stuntedness to signs of malnutrition in juvenile remains, provides compelling data. Some researchers find that those who die at the youngest ages are more likely to be stunted and to have higher frequencies of skeletal lesions from infection and malnutrition, while those who survive to older ages are more likely to exhibit evidence of healing and recovery as a consequence of longer life (Littleton, 2011; Robbins Schug, 2011; Perry, 2014; Agarwal, 2016). However, others have found that those who survive to older ages are more likely to exhibit higher frequencies of lesions as a consequence of having more time to be exposed to stressors (Storey, 1997; Bennike et al., 2005; Watts, 2013). Some find that age at death is less impactful than other variables such as female biological buffering and survivability (Holland, 2013). Long bone growth and stress markers are often compared between samples as a means of assessing stress and health in past populations (Humphrey, 2000; Saunders, 2000; Kemkes-Grottenthaler, 2005; Mays et al., 2008; Cardoso and Garcia, 2009; Lewis, 2002; 2007; Schillaci et al., 2011; Agarwal, 2016). These suggest variation within and between populations among individuals and sub-groups (i.e. sex and age) for survivability, but comparing populations and sexes can allow more nuanced interpretations of stress in particular time periods or within similar populations such as European Industrial Era samples.

Skeletal research of sexual dimorphism and frailty often focus on adults, missing valuable information about growth variation, stress, and buffering in children. While sex assessment in juvenile remains is problematic, those from historic cemetery samples of known age and sex provide a unique opportunity to test these hypotheses. This project

aims to determine whether boys or girls from 18th-20th century European samples experienced differential growth and risk due to the poor environmental conditions and cultural constraints at the time.

CHAPTER 4

MATERIALS

The samples chosen for this research are 1) the Luis Lopez skeletal sample from Lisbon, Portugal, 2) the Spitalfields Coffin Plate Skeletal Collection from London, England, and 3) the Bologna Skeletal Collection from Bologna, Italy. These samples will be compared to a modern, healthy living reference sample, 4) the Denver Growth Study from Denver, CO. These samples provide a unique opportunity to compare children from similar genetic backgrounds (European), from the same point in history (Industrial Era, 18th - early 20th centuries), but from varied socio-economic (SES) backgrounds.

Each of these cemetery collections house large numbers of juvenile remains for which sex, age at birth and/or death, and cause of death are known. Many of the skeletal remains exhibit high levels of stress, while the children from the Luis Lopez and Spitalfields collections display stunted growth in height compared to medieval skeletal samples from similar geographic locations (Lewis, 2002; Cardoso and Garcia, 2009).

The samples vary from each other in SES levels. The Spitalfields sample (London, England) comprises individuals from primarily middle to upper class backgrounds (Molleson and Cox, 1993), the Bologna sample (Bologna, Italy) contains primarily middle to low SES individuals (Mariotti et al., 2015), while the Luis Lopez collection consists of those from primarily low SES (Cardoso, 2005). The backgrounds of these populations are ideal for testing hypotheses of differential growth of boys and girls under stressed conditions. Variation in SES among samples provides a means of comparison of

children from higher SES backgrounds, who likely had better access to nutrition and healthcare, than those of the lower SES samples who likely experienced more stressed conditions. Table 1 describes the samples, while Table 2 provides information on the sample sizes of each collection. Figures 1 and 2 show the distribution of the individuals utilized for this research by Date of Birth (DOB) and Date of Death (DOD) records.

Table 1: Samples utilized in this research.

Skeletal Samples	Location	Dates	SES
Spitalfields Coffin Plate	England	1729-1859	Middle to High
Bologna, part of Frassetto Collection	Italy	1880-1935	Middle to Low
Luis Lopez	Portugal	1805-1975	Low
Denver Growth Study Radiographs	Denver, CO	1927-1967	Upper-middle

Table 2: Sample sizes separated by age-groups and sex.

Sample	Age Groups								Total
	0-1 years		2-5 years		6-12years		13-18years		
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	
Luis Lopez	5	0	7	8	7	9	14	12	62
Spitalfields	15	12	13	7	2	2	2	7	60
Bologna	10	11	12	11	4	6	6	6	66
Total	30	23	32	26	13	17	22	25	188

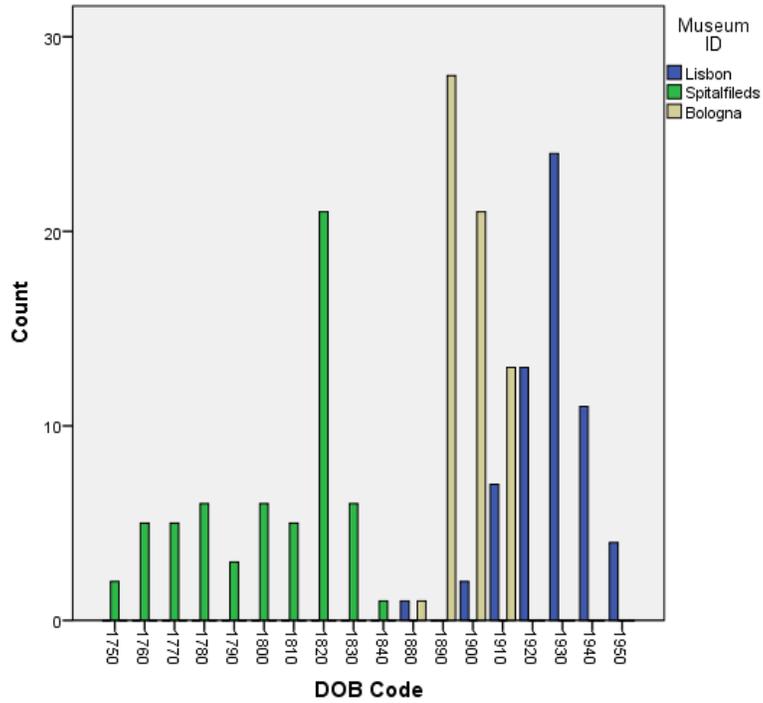


Figure 1: Number of individuals by Date of Birth (DOB) and sample.

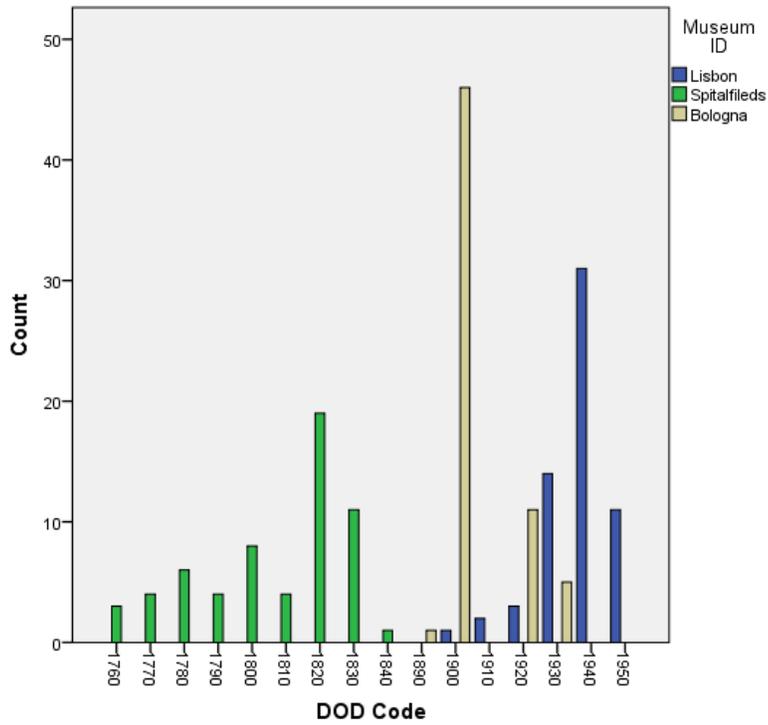


Figure 2: Number of individuals by Date of Death (DOD) and sample.

The Spitalfields (London) sample (high SES)

The London Spitalfields Coffin Plate Skeletal Collection dates to 1729-1859. This collection is comprised of congregation members of the Christ Church in Spitalfields who were buried in the private family vaults of the crypt. Records indicate that most individuals buried in the crypt were from middle to upper classes, with occupations such as craftsmen, surgeons, stockbrokers, and merchants, many of whom were estate owners (Cox, 1996; Lewis, 2002). They are described as having levels of wealth that likely helped buffer them from hardship, food shortages, and rising costs of living (Molleson and Cox, 1993; Nitsch et al., 2011; Hughes-Morey, 2016). According to the archaeological report, many families interred within the vaults of the crypt were relatively wealthy by London standards of the day, though fortunes of the quarter fell in the 19th century (Molleson and Cox, 1993).

Spitalfields was in the heart of industrial London, a dominant center of silk manufacturing and a neighborhood characterized by rapid urbanization and population growth that reached a prosperous peak with French Huguenot (protestant) refugees in the mid-18th century. The 17th and early 18th centuries saw increases in wages, economic growth, and relative wealth for those involved in the manufacturing and output of industrial goods, like those in the silk trade of Spitalfields (More, 2000). In 1773, the Spitalfields Acts, legislation allowing government officials to fix the prices for silk, the privatization and regulation of silk weavers, and the release of tariffs on foreign silk products, which eventually led to a decline in wages for the working class (Steinberg, 1999; Hupfel, 2012). By the early to mid-19th century, the silk industry in Spitalfields had

declined because of increased competition and the reorganization and decreased wages (Arch, 1822; Molleson and Cox, 1993; Cottereau, 1997; Steinberg, 1999; Hupfel, 2012). Conditions for the silk industry were so poor, especially amongst the laborers, that petitions for the repeal of the Spitalfields Acts were brought before Parliament in 1818 and 1823, met with political opposition (Hupfel, 2012). By the 1830s the silk trade of Spitalfields was economically disadvantaged and the district quickly fell to a predominant working-class district.

Because of the long time period from which this sample is comprised and the fluctuating economy under which they lived, these people are often referred to as the "middling sort" or those who "live well.. , labour hard but feel no want," who come from a solid middle to upper middle class standing (Molleson and Cox, 1993). Their SES has been well established based on their occupations and positions within their occupational structure, with 48% classified as artisans many of whom were known to inherit capital and/or land, 32% were Master craftsmen, 11% professionals, 7% were merchants and wholesalers, while 2% were independently wealthy. Wills, insurance policies, and estates provide insight into the acquisition of wealth (Molleson and Cox, 1993). Individuals in the sample from the earlier period are sometimes described as obese and gluttonous, with diets that were fashionably heavy in meat and fat, while those from the later part of the sample consumed more bread and tea, and at least some sources of protein and vegetables (Molleson and Cox, 1993).

Despite their more nutritional diet, there are high levels of stress markers associated with nutritional deficiencies such as stunted height, cribra orbitalia (CO) and

porotic hyperostosis (PO), as well as scurvy and rickets. Infants have a high frequency of CO and PO, perhaps indicative of mothers' stress during pregnancy and nursing, the use of wet-nurses, and newer hand feeding techniques that may have reduced availability of essential nutrients (Molleson and Cox, 1993). Vitamin C-rich fruits were likely unavailable to most of the population during this time, due to geographic, seasonal, and cost limitations.

While average income in London appears to have increased over the industrial period, food and rent costs increased as well (More, 2000). Those buffered by relative wealth may have been able to meet rising costs, but laborers and those dependent upon a declining industry like those in Spitalfields, likely could not. Despite the middle to upper class status of those interred in the crypt, they were still living without proper sewage systems, overcrowding, and air pollution. Drinking water was often polluted, and sewer systems open and exposed (Thompson, 1963).

The Bologna (Italy) sample (middle SES)

The Bologna Skeletal Collection is part of the Frassetto Skeletal Collections housed at the Museum of Anthropology, University of Bologna, Italy. The Frassetto Skeletal Collections are comprised of individuals from Sardinia, Bologna, Modena, and Parma and were assembled by Dr. Fabio Frassetto from the University of Bologna in 1908 (Belcastro et al., 2008). The Bologna sample contains 439 middle class individuals (adults and children) who lived in the urban center of Bologna from 1880 to 1944. These people were buried in the Certosa cemetery, and are all identified with known sex and

age, as well as adult occupation. The skeletons were exhumed and curated by Dr. Elsa Graffi Benassi during the 1950s (Mariotti et al., 2015). These individuals were removed from the Certosa cemetery to make room for more interments (Mariotti et al., 2015). They were primarily members of the middle to low classes (Mariotti et al., 2015). According to the inventory list of the individuals, about 30% of the adult men in this sample were factory workers, 20% worked in trade professions, while another 20% were police, military, businessmen, or retired professionals. Women, on the other hand, were overwhelmingly (70%) labeled as domestic workers and 20% as laborers. While this sample is a relatively newer collection accessible for research, research shows that over half the adults in this sample exhibit signs of stress such as tuberculosis (TB), non-TB pulmonary infections, and other bony lesions (Mariotti et al., 2015).

The Lisbon (Portugal) sample (low SES)

The Luis Lopez Skeletal Collection, housed at the National Natural History Museum in Lisbon, Portugal, was founded by Dr. Francisco Ferraz de Macedo in the 19th century. After a fire nearly destroyed an existing, very large collection in 1978, it was re-organized by Luis Lopes in the 1980s, then largely curated, documented, and published by Dr. Hugo Cardoso (Cardoso, 2005). This sample comprises skeletons from local cemeteries (Alto de São João, Benfica, and Prazeres), dating from 1805 to 1975. This is a documented sample with information including name, sex, age, and cause of death (Cardoso, 2005, 2006; Matos and Santos, 2006; Cardoso and Garcia, 2009). In Portugal, people are buried in graves for five years, then exhumed so the burial plot can be reused.

Family members must pay a fee to place their loved ones in more permanent graves or mausoleums. When the family cannot pay these fees, the skeletal remains are placed in communal graves, incinerated, or the National Natural History Museum collects them for research (Cardoso, 2005). Since the cemeteries used to comprise this sample are large, they likely include individuals from varied SES backgrounds.

This sample consists of those who lived in the densely urbanized capital city of Lisbon during a time of great political upheaval and turmoil between a falling monarchy to Napoleonic invasions (1807-1814), civil war leading to a monarchy with parliamentary control (1820-1908), to the establishment of a republic from 1910-1933, and eventually the longest European dictatorship of António de Oliveira Salazar (1933-1974). Portugal was considered one of the poorest European nations during this time with some of the highest mortality rates and shortest populations, suggesting overall poor conditions. Individuals from this sample lived during this period of political upheaval and the Salazar dictatorship, which is often described as a time of very poor living conditions with high infant mortality rates (Cardoso, 2005).

The Denver Growth Study sample

The children from the three skeletal collections will be compared to a contemporary sample of healthy, living children from the Denver Growth Study. This study was a longitudinal growth analysis of living children from Denver, Colorado, between 1927 and 1967. These children were from upper-middle class families of European ancestry. They represent children growing under healthy, modern conditions

(Maresh, 1955; McCammon, 1970; Ruff, 2003). Skeletal growth for this sample was obtained from databases with measurements of femur length taken from radiograph images taken every six months (Maresh, 1955; McCammon, 1970).

CHAPTER 5

METHODS

All statistical analyses were completed using Microsoft excel and SPSS statistical software, version 23 from IBM. Only well-preserved individuals with documented sex and age were utilized. Diaphyseal femur length, without epiphyses, was measured via osteometric board or spreading calipers for individuals between 0-12 years of age, and total femur length, with epiphyses, were measured with an osteometric board for all individuals with epiphyses from age 13-18 years (following Buikstra and Ubelaker, 1994). Left femora were used, except in cases where they were not available, then the right was used. This chapter outlines the methods for each hypothesis.

Hypothesis 1

Given that poor environmental conditions can lead to stunted height, I test the null hypothesis that there are no differences in femur length between the Industrial Era children compared to a healthy reference sample, between boys and girls, and between SES samples. Alternative hypotheses, if the null hypothesis is rejected include: first, that poor environmental conditions will lead to shortened femur lengths, with Industrial Era children exhibiting shorter femora relative to a healthy reference sample (H1: Prediction 1); second, that children from the lower SES will be more stunted than those from middle and high SES (H1: Prediction 2); and third, that boys will be more stunted than girls, resulting in decreased sexual dimorphism in femur length (H1: Prediction 3), especially

in the lower SES sample.

Stuntedness

Z-scores were calculated to determine if children from the Industrial Era skeletal samples were short and/or stunted compared to those from the reference sample. A Z-score is a measure of how many standard deviations an element is from the mean (StatTrek, 2016). In this case, a Z-score refers to how many standard deviations (SD) the individuals within a given sex and age group deviate from a reference sample from the Denver Growth Study (DGS). These are calculated using the following formula:

$$Z = (X - \mu) / \sigma$$

where X is the mean of the skeletal sample, μ is the mean of the DGS, and σ is the standard deviation from the DGS. Z-scores were calculated within sex and age groups, and for diaphyseal femur length in those 0-12 years and total femur length (with epiphyses) for those 13-18 years. If the z-scores are two SD below the reference sample mean, they are considered stunted, while three SD or more below the reference is considered severely stunted (World Health Organization Working Group, 1986; Leatherman and Goodman, 2005; Cardoso, 2005; Cardoso and Garcia, 2009; Leatherman, 2010).

Femur length means and standard deviations by sample, sex, and age group are listed in Table 3. Because the DGS dataset is based on radiographs, a correction for image parallax was made using a formula by Ruff (2007). The DGS data were recorded in centimeters, and were converted to millimeters for comparison.

Skeletal Growth Profiles

To determine if boys' femur lengths were more negatively impacted than girls, skeletal growth profiles (SGPs) within samples and sex were created by plotting age at death against the percent of adult femur length achieved (Lewis, 2002; Cardoso, 2005). Values for adult height achieved were calculated from adult femur measurements collected for different projects that used adults from these samples reflecting adult femur length for contemporary adults (Ruff et al., 2012; Agostini, 2015). This method was chosen to take into account the fact that all three samples span broad time periods characterized by changing political-economic situations. In addition, two of the samples include individuals who were born in the city and some who were immigrants, introducing potential variability from differing genetic backgrounds and environments. To ensure the differences seen between samples in the SGPs were not due to these secular changes and genetic variability within the adult population, femur length was plotted as the percent of adult femur length achieved. SGPs of femur length were also created between samples, including the DGS, within sexes to identify overall variation of femur length between samples.

Comparing Sexes

To test for statistically significant variation between boys and girls within each sample and age group, Mann-Whitney U-tests of femur length regression residuals were performed (Schillaci et al., 2011). These are non-parametric tests of means between two

independent populations or sub-groups, each with multiple individuals, with ordinal or continuous data that are not distributed normally or have small sample sizes, as is the case with these samples (Laerd Statistics, 2013). It is important to note that the boys and girls within each of these samples, especially those from London who come from the same church congregation, do not necessarily represent independent samples. However, this is the best test for comparing boys to girls within samples, so results must be taken with caution.

Table 3: Femur length means (mm) for boys and girls by sample.

		Low			Middle			High		
Age Groups	Sex	N	Mean	St. Dev.	N	Mean	St. Dev.	N	Mean	St. Dev.
Infants 0-1 year olds	Male	5	142.16	12.82	7	124.43	18.49	7	116.98	19.93
	Female	0	NA	NA	9	112.22	17.10	6	114.25	24.91
Young Children 2-5 year olds	Male	6	201.42	33.85	7	160.79	14.96	8	172.50	16.25
	Female	8	195.63	25.94	8	170.63	37.27	3	158.83	21.83
Older Children 6-12 year olds	Male	7	290.86	48.28	3	269.33	23.76	2	262.00	86.27
	Female	9	294.06	37.64	6	244.17	38.98	1	310.50	NA
Adolescents 13-18 year olds	Male	11	431.86	29.17	5	416.60	32.51	1	453.00	NA
	Female	11	398.00	36.46	6	403.50	8.55	7	398.00	22.07

Hypothesis 2

Since poor environmental conditions correlate with indicators of stress in skeletal samples, this research tests the null hypothesis that there are no differences in the frequencies of stress markers between SES samples or between sexes. Alternative versions of this hypothesis are that we will find higher incidences of stress markers (such as cribra orbitalia (CO), porotic hyperostosis (PO), linear enamel hypoplasias (LEH), or periostitis), with increased evidence of stress in lower SES relative to either middle or high SES children (H2: Prediction 1); or that stress markers are more prevalent in boys than girls (H2: Prediction 2); or, finally, that stress markers are more prevalent in boys with low SES (H2: Prediction 3).

Pathological Scores

Biological indicators of stress, such as CO, PO, LEH, and periostitis of the tibia were recorded as present or absent for all children in each sample (Goodman et al., 1980; Schultz, 1988; Buikstra and Ubelaker, 1994; Skerry, 1994; Larsen, 1997). In many cases preservation of the remains made it impossible to score, and those individuals were left out of the analysis. CO was identified as pitting on the roof of the orbits (Schultz, 1988; Stuart-Macadam, 1991; Ogden, 2008). PO was recognized as diploic expansion on the frontal, parietals, and occipital bones of the cranium (Ortner, 2003; 2008; Wapler et al., 2004; Walker et al., 2009; Oxenham and Cavill, 2010; McIlvaine, 2013). LEHs were identified as transverse furrows on the deciduous or permanent anterior (incisors and

canine) teeth. Individuals with one or two LEH per tooth were given a score of "1", and those with three or more LEH per tooth were given a score of "2". Periostitis was identified as new bone formation visible on the periosteal layer of the long bones (Huss-Ashmore et al., 1982; Skerry, 1994; Larsen, 1997; Ortner, 2003; Roberts and Manchester, 2005; Geber and Murphy, 2012; Ragsdale and Lehmer, 2012).

Comparing pathologies among samples

Frequencies of presence and absence of pathological conditions were compared using chi-square analysis to evaluate if statistically significant differences occurred between populations, or between sex, or age groups (StatTrek, 2016).

Hypothesis 3

Because poor environmental conditions also correlate with increased risk of mortality, I test the null hypothesis that there are no differences in mortality rates between SES samples, sex, or those with pre-existing signs of stress compared to those without. Alternative versions of this hypothesis are, first, that poor environmental conditions and sex differences correlate with increased risk of mortality, with lower SES children exhibiting higher risk of dying than middle and upper SES samples (H3: Prediction 1). Second, under conditions of stress, boys are expected to show higher risk of early mortality than girls (H3: Prediction 2). Children with an existing pathological condition will exhibit a greater risk of mortality than those without (H3: Prediction 3).

Mortality analysis

Differences in survivability of Industrial Era juveniles between SES samples and sex were assessed using the non-parametric Kaplan-Meier survival analysis with a Mantel-Cox log rank test to test for statistical significance. This model tests the proportion of survival distributions of two or more samples. This is often used for public health and medical research to estimate survivorship of various treatments for diseases, but it (and other survival models) has proven useful for bioarchaeological research to understand the difference in survivability between sub-groups, like sex, age, and status, in past populations (Boldsen et al., 2014; DeWitte, 2014, 2015; DeWitte et al., 2015; Hughes-Morey, 2016). Kaplan-Meier functions under the following assumptions: 1) the event status (dead or alive) should be mutually exclusive, 2) the timing of the event, or "death" should be clearly defined (age at death), 3) the starting point of participation should be clearly identifiable (birth), 4) there should be independence of censoring and the event, for those who no longer participate in the project (irrelevant here, since all individuals in the skeletal samples "participated"), 5) there should be no secular changes from the starting point to the end point (birth to age 18 years in this case) of participation (this is specific to changes occurring within the aging process of an individual), and 6) there should be a similar amount and pattern of "censoring" or survival time per group (all results from this research were equal at 0 or 0%, since no individuals survived).

CHAPTER 6

RESULTS

Hypothesis 1

Results of Z-scores for each SES sample can be seen in Table 4. Relative to the comparative Denver Growth sample (DGS), over half of the Industrial Era children exhibit stunted femur lengths, supporting the first prediction. Results by SES, however, shows that the middle SES sample from Bologna exhibits more stuntedness overall than the lower and upper SES samples (Table 4), thus not supporting the second prediction. When each SES sample is broken down by sex, boys in the upper SES sample do exhibit more stunting than girls overall, but girls from the lower and middle SES samples display a greater percentage of stuntedness than boys (Table 5).

Z-scores based on mean diaphyseal and total femur length for boys and girls of each SES group (Figures 3a, b, & c) reveal that Young and Older children are more likely to exhibit shorter femur length for their age compared to infants. Skeletal growth profiles (SGP) of diaphyseal and total femur length among the three skeletal samples compared to the DGS show this discrepancy between the reference sample and the stressed Industrial Era samples. Diaphyseal femur length of males shows that the middle SES group falters by age 4 and remains the shortest, while the low SES remains close in femur length to the DGS until ~ age 5, when they start to decline until age 9, followed by some evidence of recovery. The high SES sample stagnates in growth from 3 to 7 years and then appears to experience catch up growth. Girls from the high SES drop significantly from ages 2 to 3

years and remain quite short throughout their growth, while middle and low SES girls exhibiting less stunting than their high SES counterparts. These SGPs can be seen in Figure 4. Total femur length in low SES males exhibit a lot of variability, middle SES males show an increase in femur length between ages 15-17. Only one male represents the high SES group, which therefore is not represented by a line. Girls from all three samples are all similarly shorter than their DGS counterparts, except for one girl from the low SES sample who brings the mean down to age 16. SGPs for total femur length are in Figure 5. All children exhibit increased stunting throughout growth. All adolescent girls were severely stunted compared to boys. Those from the lower SES sample exhibit the least stuntedness, and those from the middle SES sample exhibit the most, while the upper SES children fall in the middle, supporting the third prediction in the lower and middle SES samples.

Because these skeletal samples are from the Industrial Era and likely exhibited stress, and are compared to a modern, healthy reference sample, they are likely not normally distributed and skewed to the left (negative). To account for this as a potential, a Skewness test (a measure for asymmetry of a distribution) was run for z-scores of both diaphyseal and total femur length. For diaphyseal femur length, Skewness statistic = .331, Std. error = .186, suggesting this variable is positively distributed. For total femur length, Skewness statistic = -1.099, Std. error = .258, suggesting adolescents of the skeletal samples are negatively distributed.

SGPs showing the percent of adult femur length achieved plotted against known age for each sample can be seen in Figure 6. These SGPs show very little variation by sex

in either low or high SES groups, but middle SES boys were shorter than girls of the same age, which is also reflected in Z-scores. Differences in residuals from regressions between femur length and age were evaluated between boys and girls of each age group with Mann-Whitney U-tests (p -value $<.05$, Table 7). Sexual dimorphism was found to be significant between boys and girls aged 13-18 years from the Denver Growth Study ($p = .001$, Figure 7), but was not significant in any age group from the Industrial Era samples. This suggests sexual dimorphism was reduced in the Industrial Era adolescents aged 13-18 years.

Table 4: Percent of those with normal and stunted diaphyseal femur lengths. Those from Bologna (middle SES) have the overall highest percentage of children stunted, while London (upper SES) and Lisbon (lower SES) have similar results.

Z-score	N	%
Lisbon (Low SES)		
Normal ($Z > -2.0$)	17	49
Stunted ($Z < -2.0$)	18	52
Bologna (Middle SES)		
Normal ($Z > -2.0$)	15	38
Stunted ($Z < -2.0$)	25	63
London (High SES)		
Normal ($Z > -2.0$)	13	48
Stunted ($Z < -2.0$)	14	52

Table 5: Percent of stunted diaphyseal femur length by sample and sex. Lower and middle SES girls exhibit more stunting than any other group.

Z-score	N	%	Z-score	N	%
Lisbon (Low SES) boys			Lisbon (Low SES) girls		
Normal (Z> -2.0)	11	61	Normal (Z> -2.0)	6	35
Stunted (Z< -2.0)	7	39	Stunted (Z< -2.0)	11	65
Bologna (Middle SES) boys			Bologna (Middle SES) girls		
Normal (Z> -2.0)	8	47	Normal (Z> -2.0)	7	30
Stunted (Z< -2.0)	9	53	Stunted (Z< -2.0)	16	69
London (High SES) boys			London (High SES) girls		
Normal (Z> -2.0)	7	41	Normal (Z> -2.0)	6	60
Stunted (Z< -2.0)	10	59	Stunted (Z< -2.0)	4	40

Table 6: Z-scores based on mean diaphyseal and total femur length between boys and girls of each sample separated into age categories.

Age-groups	Lisbon (Low SES)		Bologna (Middle SES)		London (High SES)	
	Boys	Girls	Boys	Girls	Boys	Girls
Infants (0-1 year)	.70	N/A	.00	-.38	-.32	-.39
Young Children (2-5 years)	-.31	-.60	-1.71	-1.03	-1.30	-1.69
Older Children (6-12 years)	-.85	-1.10	-2.06	-1.30	-2.01	-1.57
Adolescents (13-18 years)	-1.69	-6.76	-2.61	-6.94	-2.06	-6.83

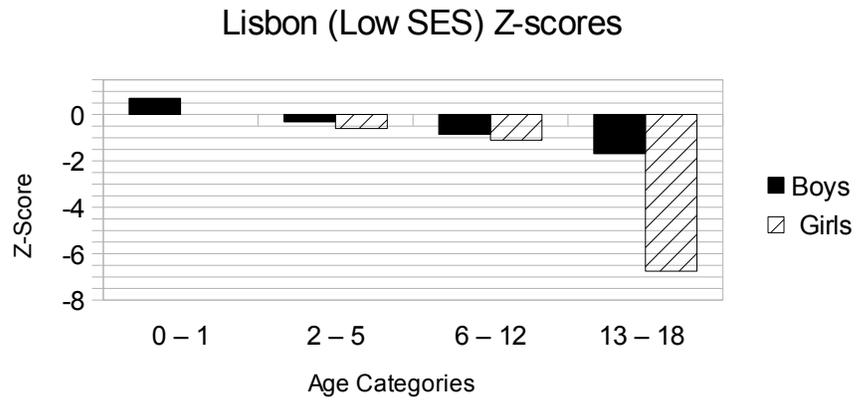


Figure 3a

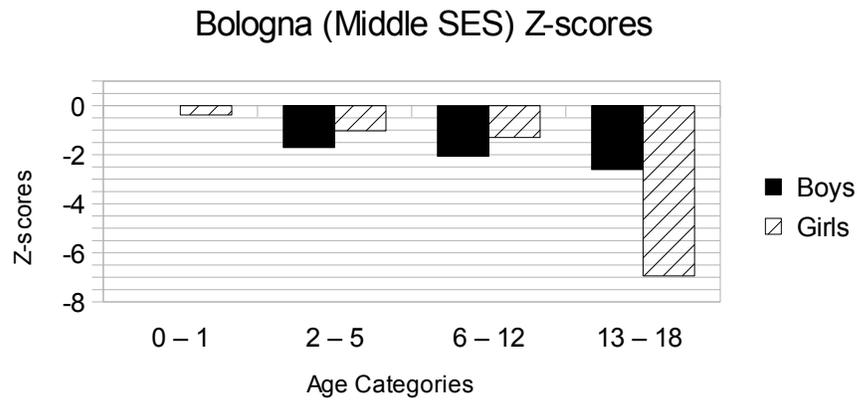


Figure 3b

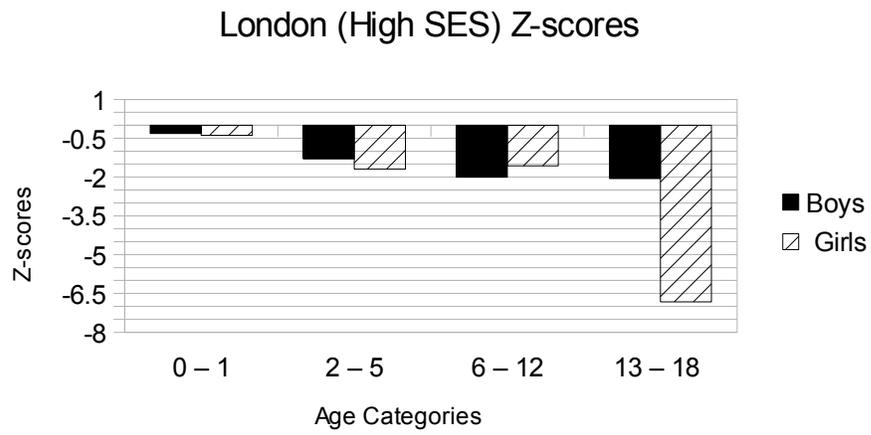


Figure 3c

Figure 3: a, b, and c. Z-score results for boys and girls in each SES sample. Industrial Era children are shorter than DGS, stuntedness increases throughout growth, adolescent girls exhibit severe stuntedness.

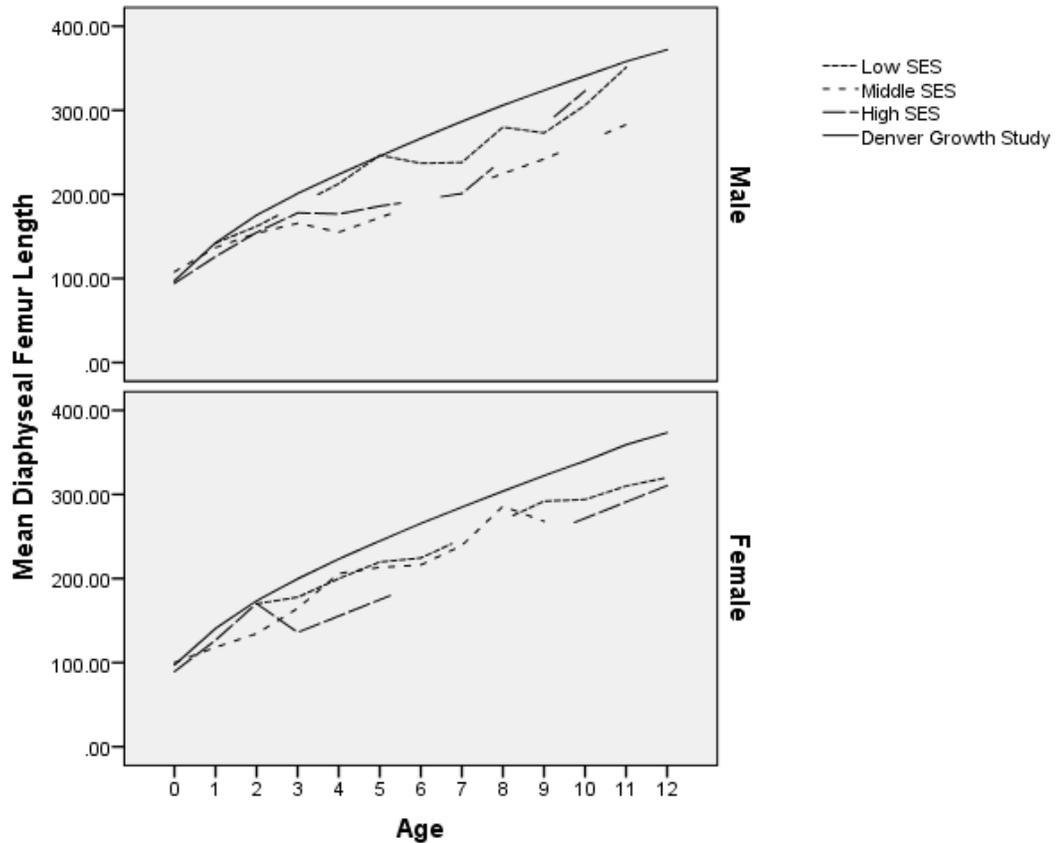


Figure 4: Graph showing the variation between samples and sexes using diaphyseal femur length (mm). Results suggest middle SES boys had the shortest femur length and low SES boys were closer aligned with the reference sample, and high SES boys exhibited an increase in growth between ages 7 and 10 years. Girls from the low and middle SES overlap quite a bit, but below the DGS, while high SES girls have the shortest femur lengths.

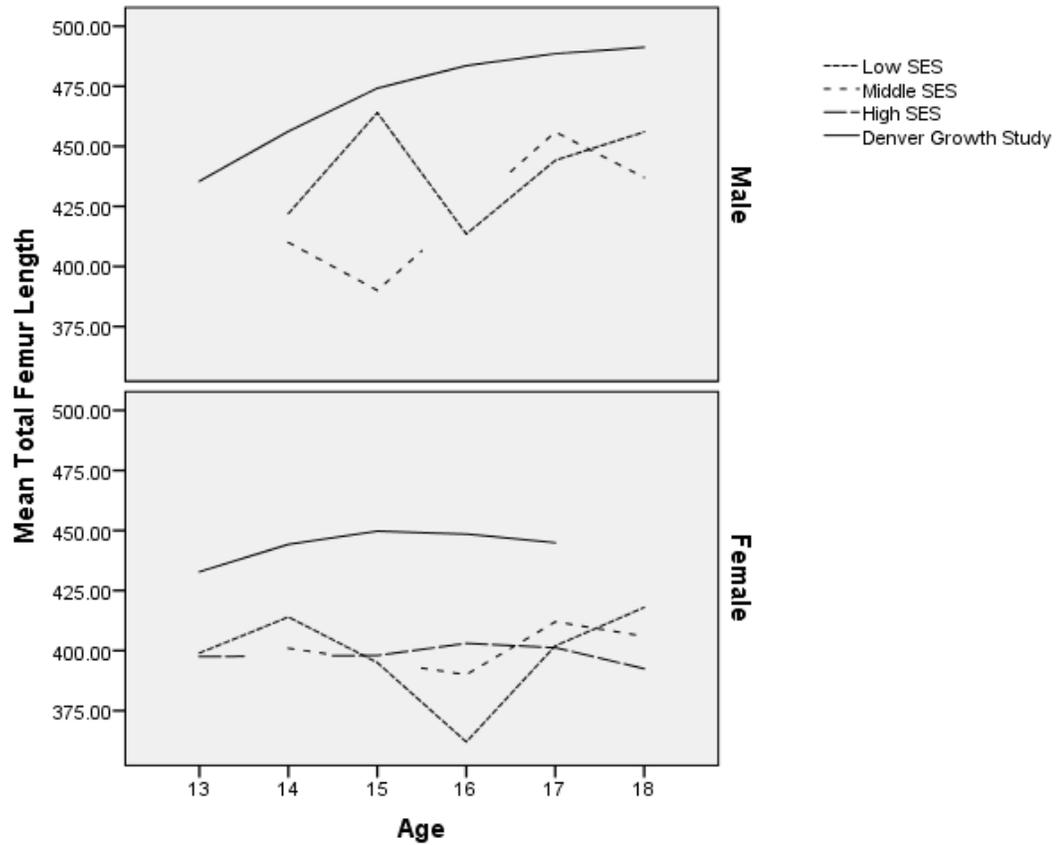


Figure 5: Graph showing the variation between samples and sexes using total femur length (mm). Results suggest middle and low SES boys had short, but variable femur lengths during this period. Girls from all three samples overlap quite a bit with the low SES sample showing the most variability.

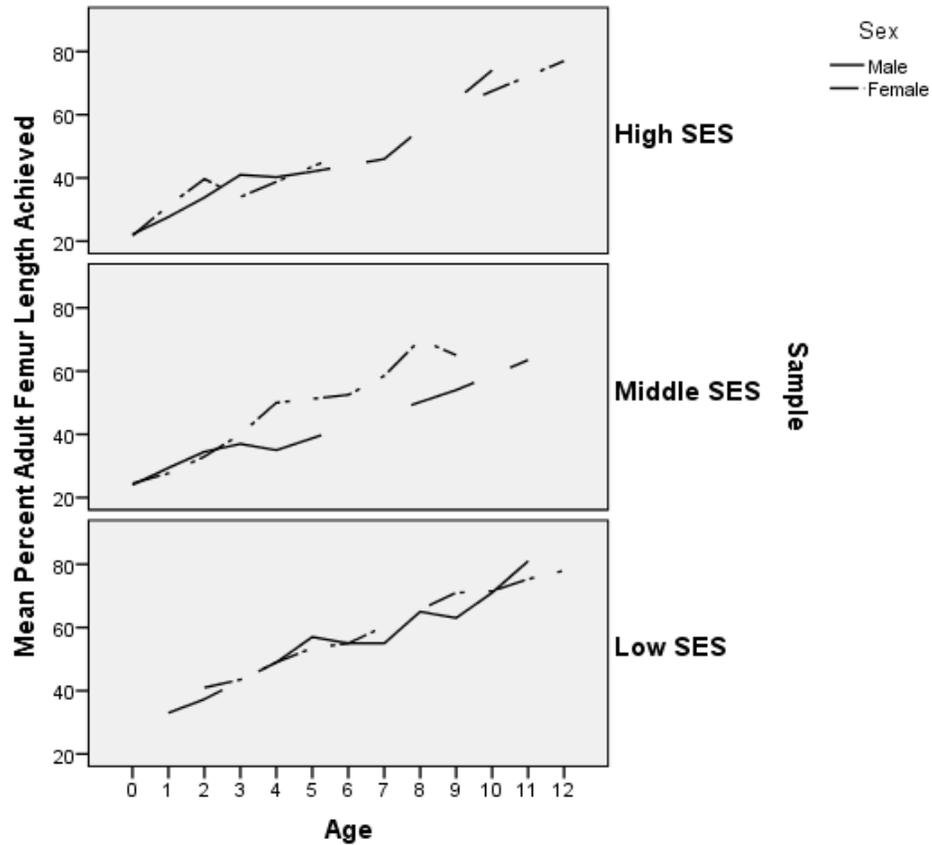


Figure 6: Graph showing the variation between sexes in each sample using the percent of adult femur length achieved for diaphyseal femur length (mm). Results suggest very little variation between boys and girls in Lisbon and London, but boys in Bologna were quite shorter than girls of the same age.

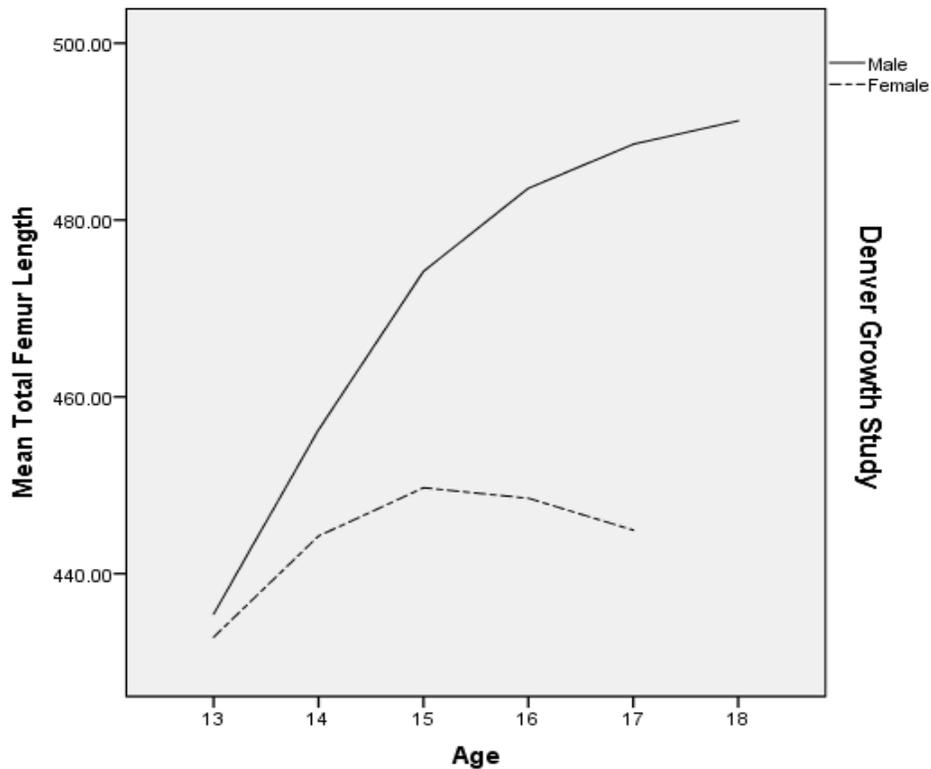


Figure 7: Graph of total femur length (mm) of boys and girls from the DGS adolescents, who exhibit statistically significantly different results between sexes (p-value = .001).

Table 7: Mann-Whitney U-test results between boys and girls, within samples and age-groups. Diaphyseal femur length regression residuals were used to test for significance in ages 0-12, and total femur length regression residuals were used for ages 13-18.

Age-groups	Low SES	Middle SES	High SES	DGS
0-1	-----	.142	.628	1.000
2-5	.282	.694	.630	.645
6-12	.174	.167	1.000	.390
13-18	.065	.126	.250	*.001

* statistical significance at the .05 level.

Hypothesis 2

Children from the low SES sample (Lisbon) have significantly higher rates of LEH ($p=.0001$) (Table 8, Figure 8), and those from low and middle SES both have significantly higher presence of general periostitis ($p=.002$) (Table 8, Figure 9). The low SES group also exhibits higher frequencies of CO (Figure 10) than the other two SES groups, but the difference did not reach statistical significance. The results for PO were also not significant, but this time Bologna (middle SES) has the highest frequency of presence (Figure 11). The low and middle SES groups both have overall more pathological indicators of stress per person (Figure 12) than the higher SES sample. Those with only one pathology present break down as follows: low SES 14%, middle SES 13%, and high SES 17%; those with two pathologies present: low SES 13%, middle SES 13%, and high SES 10%; three pathologies: low SES 10%, middle SES 8%, and high SES 2%; and finally those with four or more: low SES 2%, middle SES 1%, and high SES had none. The overall percentage of individuals with two or more pathologies present are: low SES 25%, middle SES 22%, and high SES 12%. These results show that low and middle SES children exhibit higher frequencies of pathologies than high SES ones, lending some support for the first prediction.

Table 8: Chi-square results between samples for presence of pathology.

Pathologies		Samples			Pearson X ²	df	p-value
		Low SES	Middle SES	High SES			
Count		62	66	60			
LEH	% Presence	55.8%	23.3%	20.9%	37.397	2	*.0001
Periostitis	% Presence	40.4%	45.6%	14%	12.115	2	*.002
CO	% Presence	39%	34.1%	26.8%	2.829	2	.243
PO	% Presence	24.5%	41.5%	34%	2.557	2	.278

* statistical significance at the .05 level.

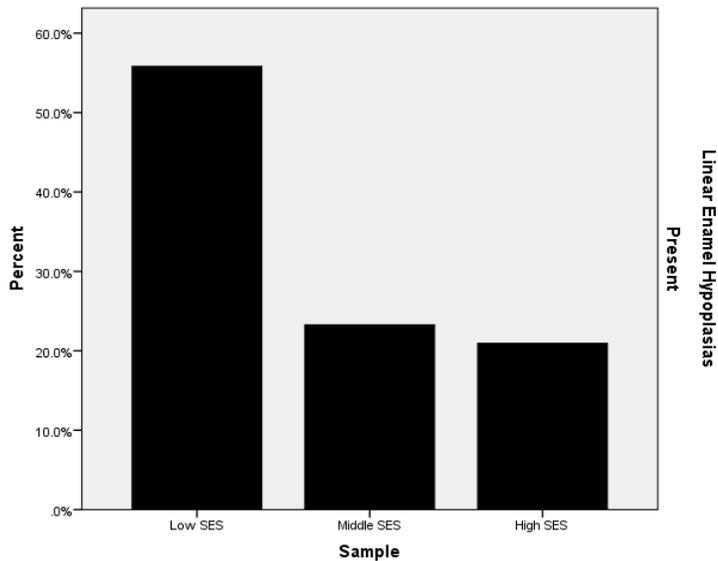


Figure 8: Frequency bar graph between samples with those from the lower SES sample (Lisbon) exhibiting statistically significantly (p=.0001) more presence of LEH.

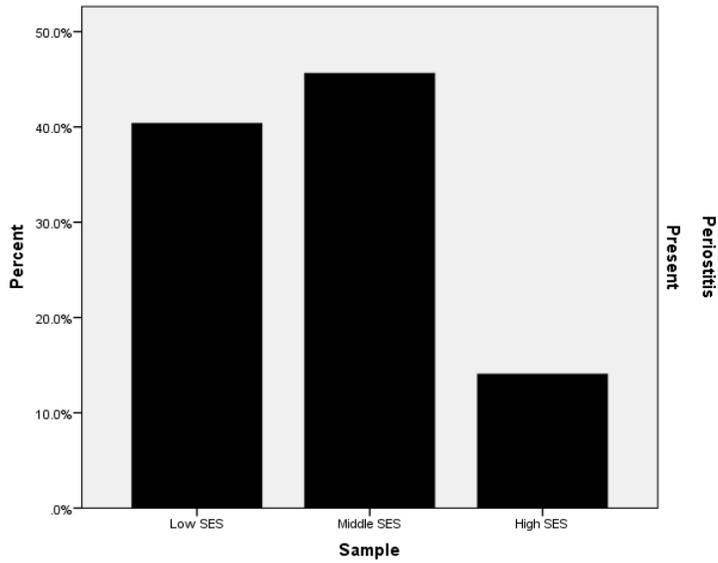


Figure 9: Frequencies of periostitis between the samples showing the lower and middle SES samples having significantly ($p=.002$) highest rates.

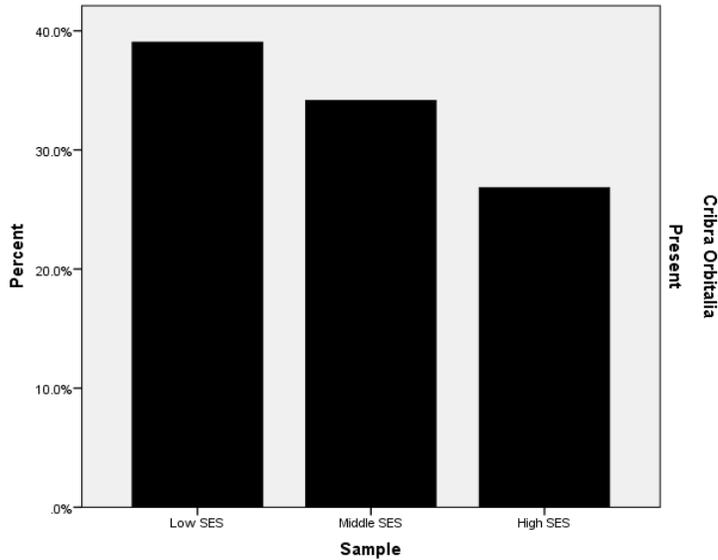


Figure 10: Frequency bar graph of CO presence between the samples, Chi-Square results were not significant ($p=.243$).

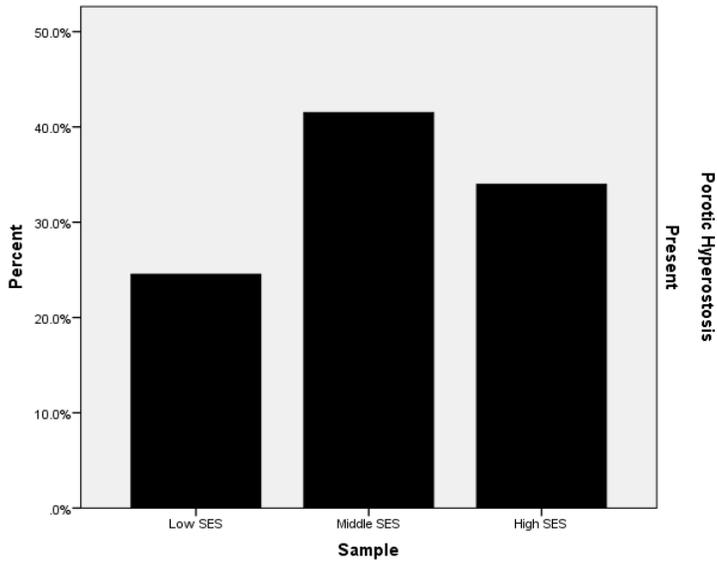


Figure 11: Bar graph showing that the middle class sample (Bologna) has the highest frequency of PO, though not statistically significant ($p=.278$).

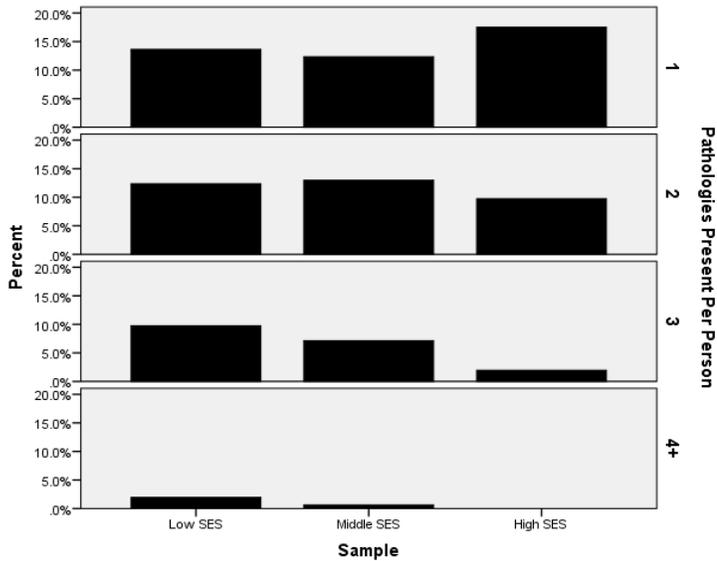


Figure 12: Frequencies expressing the overall presence of pathologies per individual, showing that individuals from the lower and middle SES samples exhibit more pathological conditions than those in the higher SES sample.

Results comparing boys to girls suggest that boys do exhibit higher frequencies of CO, PO, and LEH (Figures 13-15) although differences are not significant (Table 9). Boys and girls display no differences in presence of periostitis (Figure 16), or in the frequency of numbers of pathologies present per individual (Figure 17). There are 22% males with one pathology, 18% with two, 12% with three, and 3% with four or more, with a total of 33% having two or more. Females exhibit 23% with one pathology, 17% with two, 8% with three, and 1% with four or more, with a total of 26% with two or more. Overall, males and females tend to exhibit the same amount of pathologies. While boys do display more CO and PO, supporting the second prediction, they do not exhibit more evidence of stress in the form of periostitis or overall number of pathologies.

Table 9: Chi-square results between sexes for presence of pathology.

Pathologies		Sex		Pearson X ²	df	p-value
		Male	Female			
Count		97	91			
LEH	% Presence	53.5%	46.5%	.227	1	.633
Periostitis	% Presence	47.4%	52.6%	.585	1	.444
CO	% Presence	58.5%	41.5%	2.805	1	.094
PO	% Presence	58.5%	41.5%	1.405	1	.236

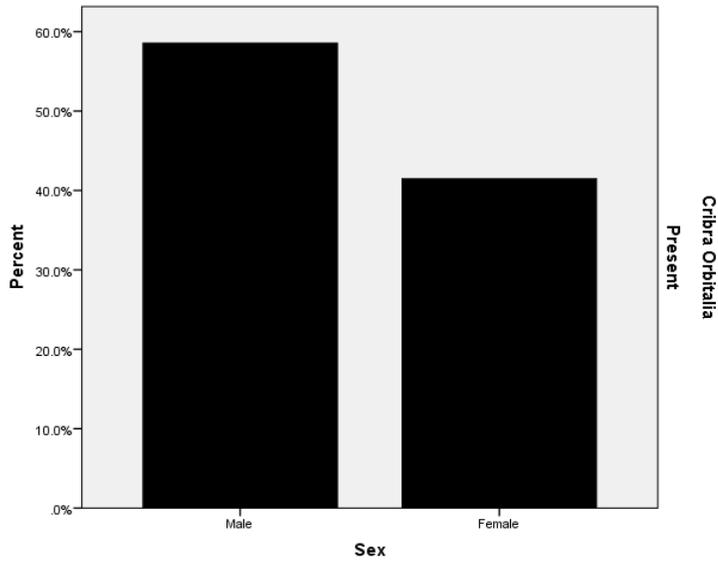


Figure 13: Frequencies of male and female percent presence of CO, Chi-square results were not significant ($p=.094$).

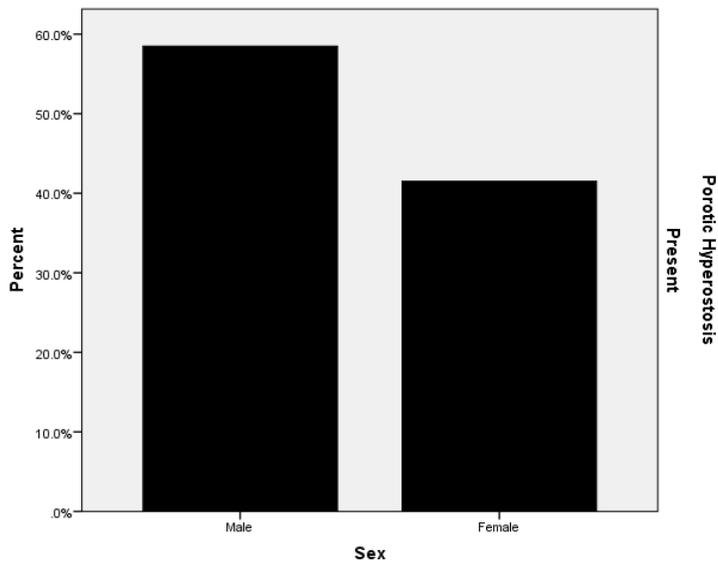


Figure 14: Frequencies of male and female percent presence of PO, Chi-square results were not significant ($p=.236$).

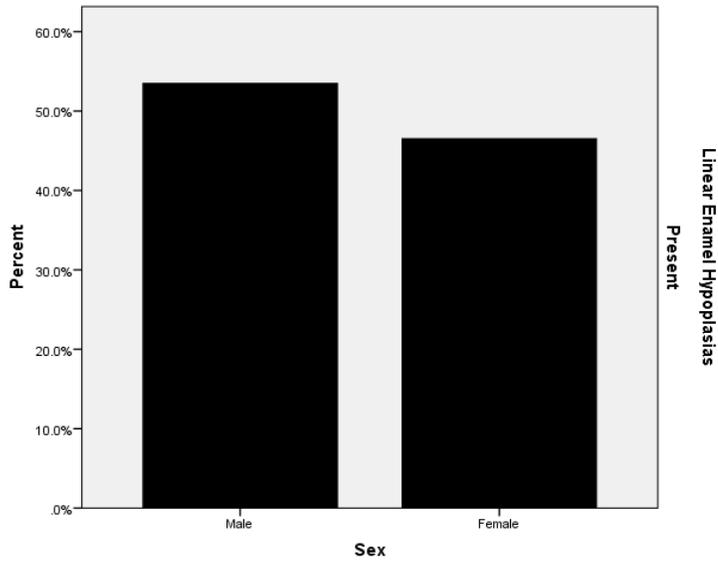


Figure 15: Frequencies of male and female percent presence of LEH, Chi-square results were not significant ($p=.633$).

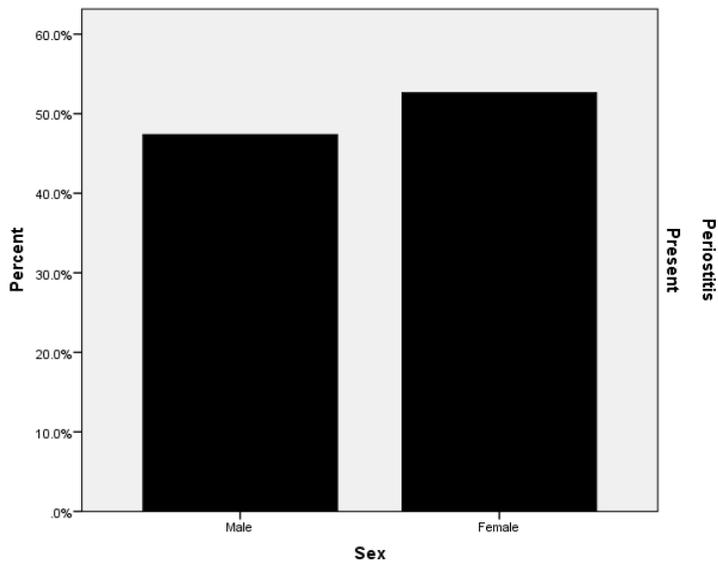


Figure 16: Frequencies of male and female percent presence of periostitis, Chi-square results were not significant ($p=.444$).

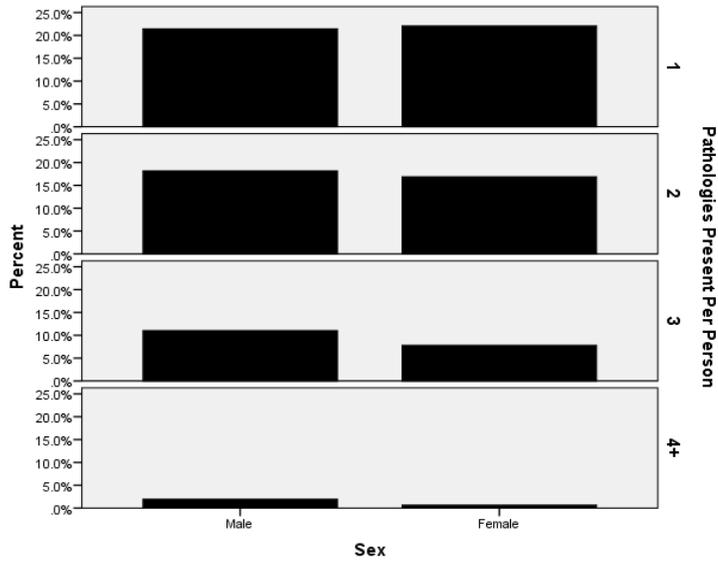


Figure 17: Frequencies expressing the overall presence of pathologies of males and females, showing that males and females do not differ in the number of pathologies experienced.

When differences between sex are compared within SES samples, a clearer picture of interaction between pathological condition, status, and sex emerges. Figures 18 through 22 show CO, PO, LEH, Periostitis, and the number of pathologies present per individual. In the lower SES sample, boys have a statistically significantly higher frequency of CO ($p=.043$) and borderline significantly higher frequency of PO ($p=.054$), while low SES boys and girls show no differences in the frequency of LEH and periostitis (Tables 10, 11, & 12). Middle SES Girls have higher frequencies of CO, PO, and periostitis, though none of these are statistically significant. Boys, however, do exhibit a slightly higher percentage of LEH. Boys from the high SES sample exhibit more presence of all pathological conditions, though none of these are statistically significant. Boys from the low and high SES samples exhibit a similar pattern of more

presence of stress than girls, while girls from the middle SES sample display more evidence of stress in all markers except LEH. In all three samples boys show slightly higher frequencies of LEHs than their female counterparts.

Analysis of the number of pathologies present per individual (Figure 22) reveals another pattern. 19% of the boys from the low SES sample had one pathological marker of stress, 12% exhibited two, 18% had three, and 5% had four or more with a total of 35% who displayed two or more signs of stress. Only one pathology is present in 18% of low SES girls, 20% had two, 8% exhibited three, and none had four or more, with a total of 28% who displayed two or three. Boys from the low SES sample exhibit more pathologies overall than girls.

Within the middle SES sample, 12% of boys had one pathology, 21% showed two, 9% displayed three, and none had four or more. Middle SES girls displayed one pathology in 25% of the sub-population, two markers of pathology within 18%, three within 10%, and four or more signs of stress found in 2%. Both boys and girls display 30% with two or more pathologies.

High SES boys displayed one pathology within 33% of the population, two were evident on 22%, three within 5%, and none at four or more. 27% of high SES girls had one pathology, 11% had two, 2% displayed three, and none had four or more. These total to 27% boys with two or more pathologies and 13% of girls with two or three, showing that boys in this sample have more pathologies than girls.

These results show that low SES boys display the highest percentage of pathologies overall, suggesting they had the highest risk of accumulating multiple

markers of stress, with low SES girls, middle SES boys, middle SES girls, and high SES boys exhibiting roughly the same percentage of accumulated stress. High SES girls, however, show the least percentage of accumulated pathologies overall, primarily exhibiting only one or two pathologies.

Table 10: Chi-square results for pathologies between sexes for the low SES sample.

Low SES Sample		Sex		Pearson X ²	df	p-value
Pathologies		Male	Female			
Count		33	29			
LEH	% Presence	52.1%	47.9%	.111	1	.739
Periostitis	% Presence	47.8%	52.2%	.428	1	.513
CO	% Presence	65.6%	34.4%	4.084	1	*.043
PO	% Presence	76.9%	23.1%	3.710	1	.054

* statistical significance at .05 level.

Table 11: Chi-square results for pathologies between sexes from the middle SES sample.

Middle SES Sample		Sex		Pearson X ²	df	p-value
Pathologies		Male	Female			
Count		32	34			
LEH	% Presence	55%	45%	.488	1	.485
Periostitis	% Presence	42.3%	57.7%	.655	1	.418
CO	% Presence	42.9%	57.1%	.617	1	.432
PO	% Presence	45.5%	54.5%	.121	1	.728

Table 12: Chi-square results for pathologies between sexes from the high SES sample.

High SES Sample		Sex		Pearson X^2	df	p-value
Pathologies		Male	Female			
Count		32	28			
LEH	% Presence	55.6%	44.4%	.051	1	.821
Periostitis	% Presence	62.5%	37.5%	.312	1	.577
CO	% Presence	68.2%	31.8%	3.077	1	.079
PO	% Presence	61.1%	38.9%	.625	1	.429

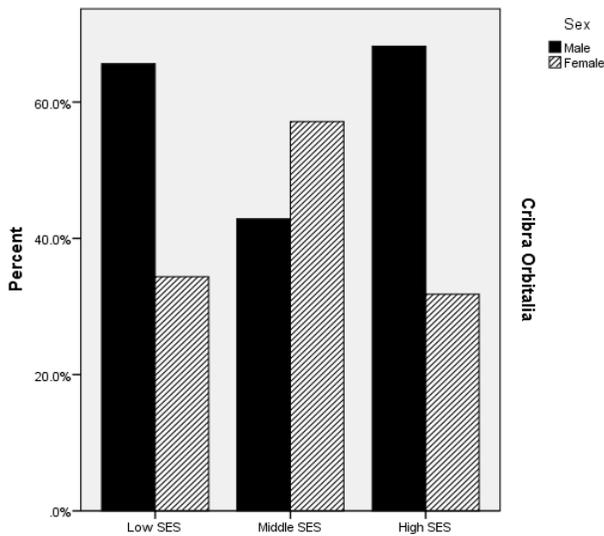


Figure 18: Frequencies of male and female percent presence of CO within each SES sample, Chi-square results were significant ($p=.043$) in the low SES with males exhibiting more percentage, no significance in the middle SES ($p=.432$), and borderline significant in the high SES sample ($p=.079$).

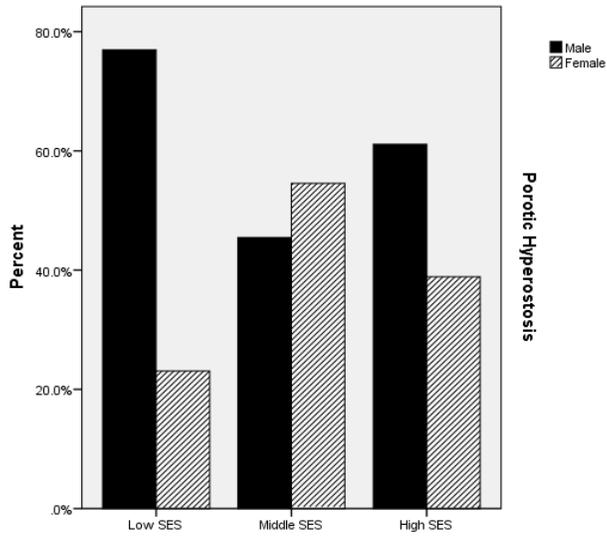


Figure 19: Frequencies of male and female percent presence of PO within each SES sample, Chi-square results were significant ($p=.054$) in the low SES with males exhibiting more percentage, no significance in the middle SES ($p=.728$) and high SES samples ($p=.429$).

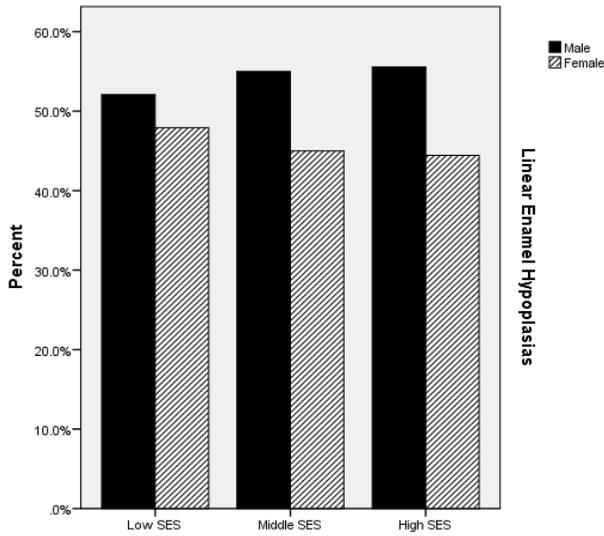


Figure 20: Frequencies of male and female percent presence of LEH within each SES sample, Chi-square results were not significant in any of the samples though males do exhibit a higher percentage in the low ($p=.739$), middle ($p=.485$), and high SES samples ($p=.821$).

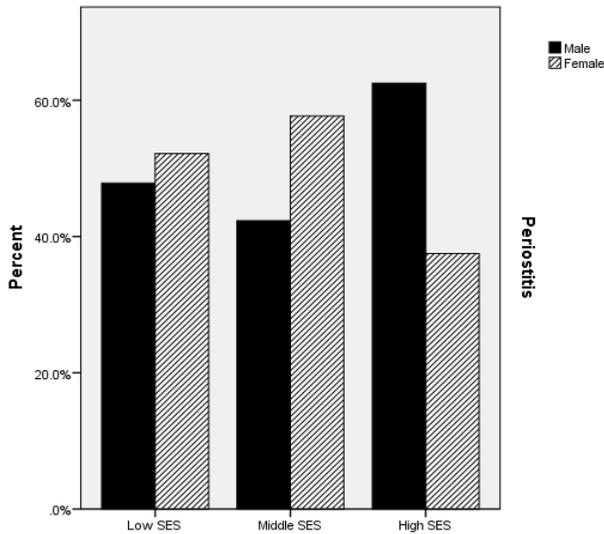


Figure 21: Frequencies of male and female percent presence of periostitis within each SES sample, Chi-square results were not significant in the low SES ($p=.513$), middle SES ($p=.418$) though girls do exhibit a higher percentage, nor in the high SES ($p=.577$), though boys have a higher percentage of presence.

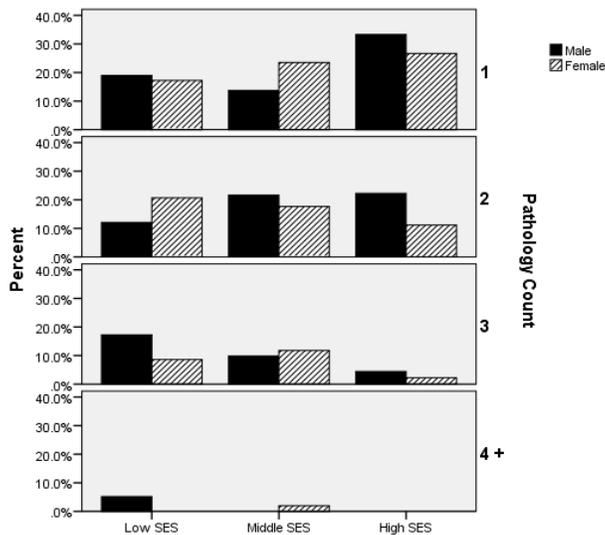


Figure 22: Frequencies expressing the overall presence of pathologies of males and females within each SES sample, with low SES boys accumulating overall the most amount of pathological stress during life.

Hypothesis 3

Kaplan-Meier survival curves show overall that juveniles from the low SES sample from Lisbon had a significantly greater chance of surviving from infancy to age 18 years ($p=.0001$), not supporting the first prediction (Figure 23 & Table 13). This SES sample displays a consistently better mortality pattern throughout growth than the two other samples. The high SES group had the greatest mortality risk from ages zero to four years, with the middle SES sample showing a similar, but slightly improved, condition. Both middle and low SES groups experienced a leveling in mortality risk from about age five until 15, where mortality increases again.

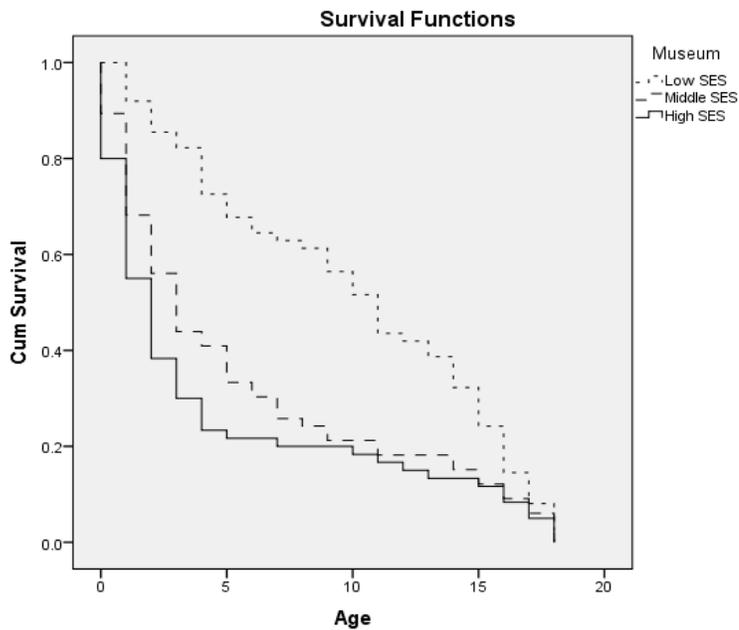


Figure 23: Kaplan-Meier curves showing the lower SES sample exhibiting a statistically significant ($p=.0001$) greater survivability, and the upper SES sample with the lowest, especially early in life.

Table 13: Kaplan-Meier survival analysis by SES samples.

Sample	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Low SES	10.00	8.56-11.44	17.17	*0.0001
Middle SES	5.52	4.12-6.91		
High SES	4.32	2.87-5.77		

*statistical significance at .05 level.

When survivability is tested by sex females show an improved condition over males (Figure 24, Table 14). Although the difference is not statistically significant, it lends some support for the second prediction. SES sample specific survivability curves (Table 15) show that, in the lower SES (Figure 25) and upper SES (Figure 27) samples,

girls survive better, though results are not significant, but again supporting the second prediction. The middle SES sample (Figure 26) exhibits no difference between sexes.

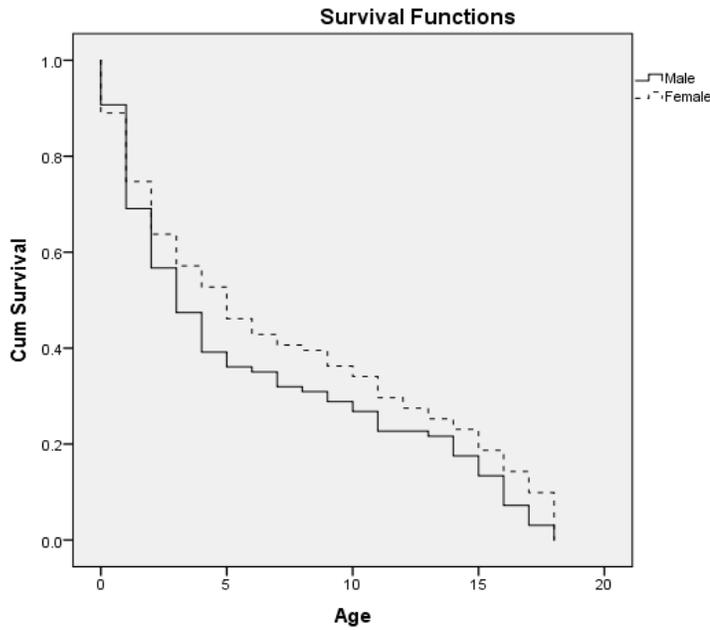


Figure 24: Kaplan-Meier curves between males and females (samples pooled), while not significant girls had a greater chance of survival than boys.

Table 14: Kaplan-Meier survival analysis results.

Sex	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Male	6.01	4.82-7.21	2.68	0.102
Female	7.25	5.93-8.58		

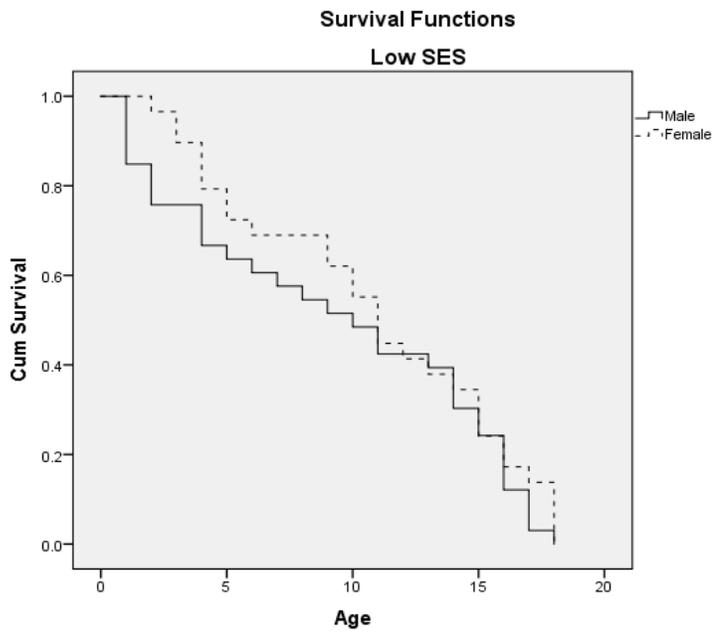


Figure 25: Kaplan-Meier survival curves between males and females of the low SES sample (Lisbon), not statistically significant.

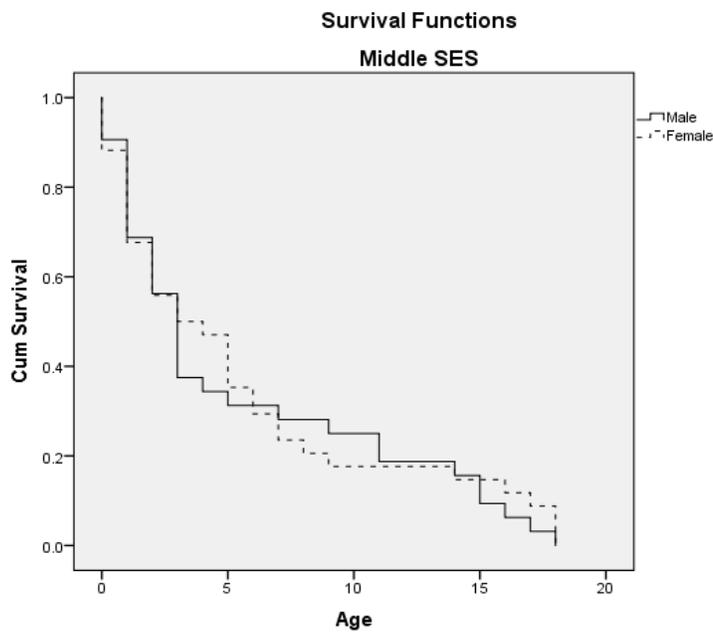


Figure 26: Kaplan-Meier survival curves between males and females of the middle SES sample (Bologna), not statistically significant.

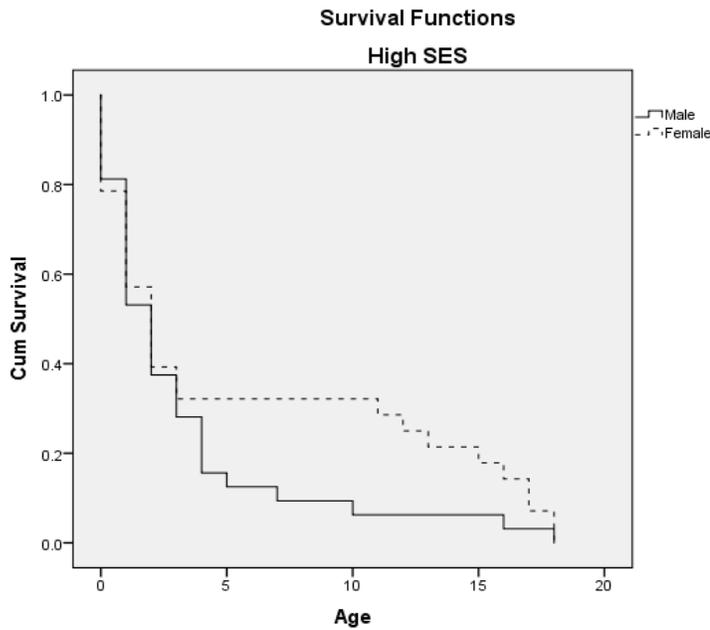


Figure 27: Kaplan-Meier survival curves between males and females of the high SES sample (London), not statistically significant.

Table 15: Kaplan-Meier survival analysis results between boys and girls within each sample.

Sample	Sex	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Low SES	Male	9.33	7.24-11.43	2.17	0.141
	Female	10.76	8.81-12.71		
Middle SES	Male	5.47	3.45-7.48		
	Female	5.56	3.59-7.53		
High SES	Male	3.13	1.65-4.60		
	Female	5.68	3.13-8.23		

When the presence of pathologies is analyzed as a covariate for survivability, there is a significant difference, with those without CO ($p=.004$) and PO ($p=.002$) having better survivability than those with the pathologies (Figures 28 and 29, Tables 16 & 17), supporting the third prediction. The opposite is true for LEH, with those exhibiting at least one LEH having significantly ($p=.0001$) better survivorship (Figure 30, Table 18) than those without one. Given this pattern and the high number of individuals with at least one LEH, survivorship patterns were also examined for those with more than one LEH. The results remained the same when analyzed for two LEHs, while those with three or more LEHs have significantly ($p=.001$) greater chance of survival than those with two or less (Figure 31, Table 19). Results for periostitis shows no significant difference between those who exhibited the stressor and those who did not (Figure 32, Table 20).

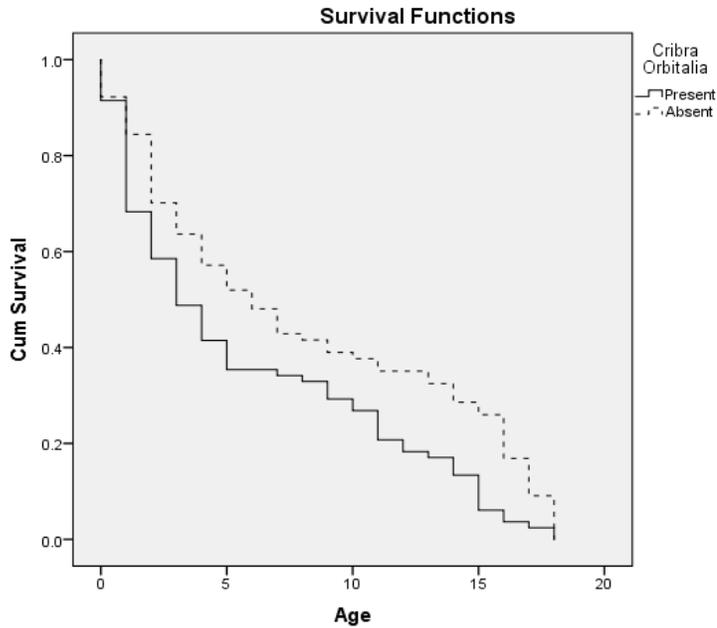


Figure 28: When samples and sex are pooled, Kaplan-Meier curves show those without evidence of CO have statistically significantly ($p=.004$) greater survivorship.

Table 16: Kaplan-Meier survival analysis results of those with or without CO.

Cribra Orbitalia	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Presence	5.84	4.63-7.06	8.27	*0.004
Absence	8.12	6.66-9.58		

* statistical significance at .05 level.

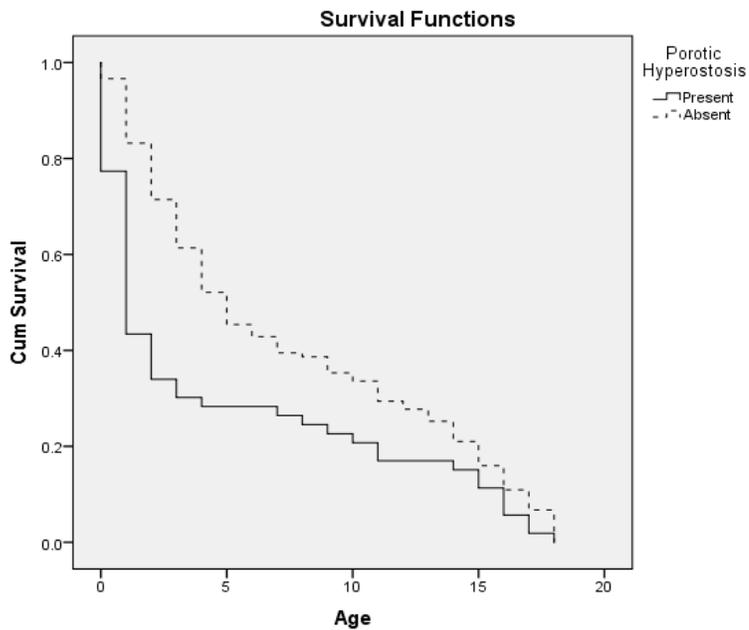


Figure 29: When samples and sex are pooled, Kaplan-Meier curves show those without evidence of PO have statistically significantly ($p=.002$) greater survivorship.

Table 17: Kaplan-Meier survival analysis results of those with or without PO.

Porotic Hyperostosis	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Presence	4.49	2.88-6.10	9.46	*0.002
Absence	7.37	6.29-8.45		

* statistical significance at .05 level.

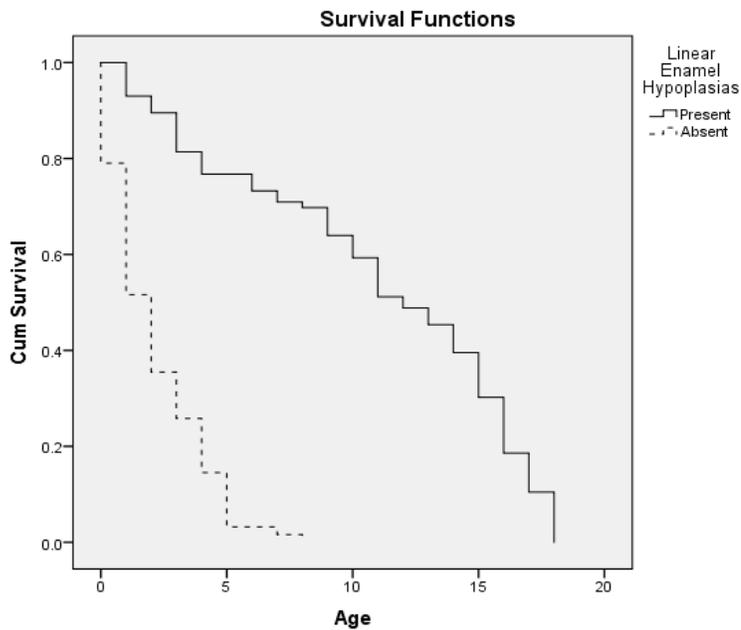


Figure 30: Those with LEH have statistically significantly ($p=.0001$) greater chance of survival past the age of nine years than those without.

Table 18: Kaplan-Meier survival analysis results of those with or without LEH.

LEH	Mean survival time (years)	95% CI	Mantel-Cox X2	p-value
Presence	10.99	9.79-12.19	105.60	*0.0001
Absence	2.15	1.67-2.62		

* statistical significance at .05 level.

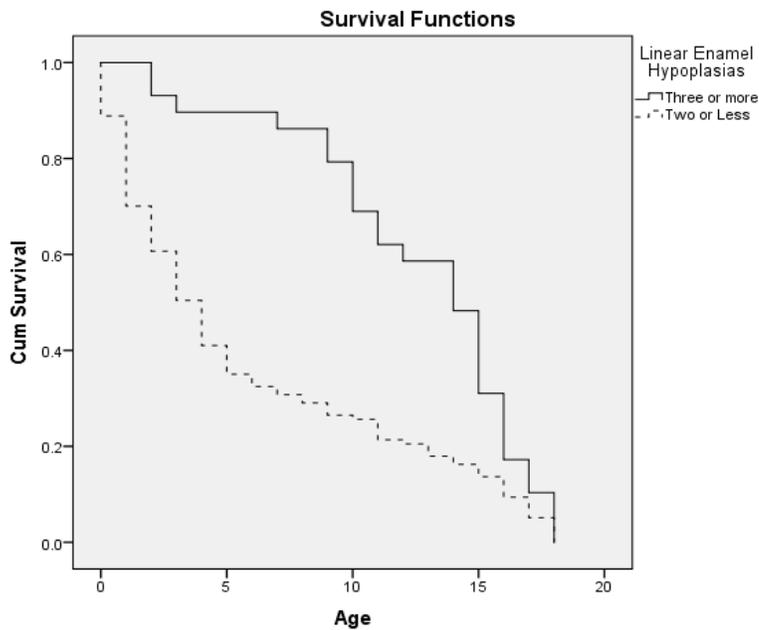


Figure 31: Those with three or more LEHs have statistically significantly ($p=.001$) greater chance of survival than those with two or less.

Table 19: Kaplan-Meier survival analysis results of those with three or more LEH compared to those with two or less.

LEH	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Presence of three or more	12.59	10.89-14.28	11.97	*0.001
Two or less	5.95	4.88-7.02		

* statistical significance at .05 level.

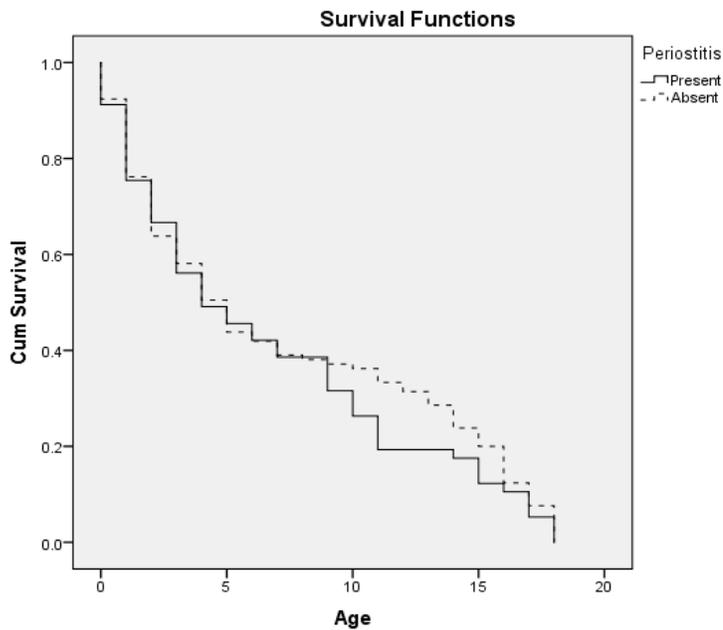


Figure 32: Kaplan-Meier curve shows that presence and absence of periostritis makes no difference ($p=.422$) in survivability.

Table 20: Kaplan-Meier survival analysis results of those with or without periostritis.

Periostritis	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Presence	6.65	5.13-8.17	.643	0.422
Absence	7.34	6.11-8.58		

When survivability with and without evidence for CO is examined for patterns within samples, results are statistically significant ($p=.002$, Table 21). In the low SES sample, those without CO exhibit a slight increase in survivability than those with the pathology, a pattern that is stronger in the middle than lower SES group (Figures 33 & 34). Children from the high SES group exhibit a similar pattern of mortality despite presence of absence of CO, though both have an overall increased risk of mortality from ages 0-5 years (Figure 35). These results find those without CO (in all samples) have increased survivability, though the difference is more marked in the middle SES sample.

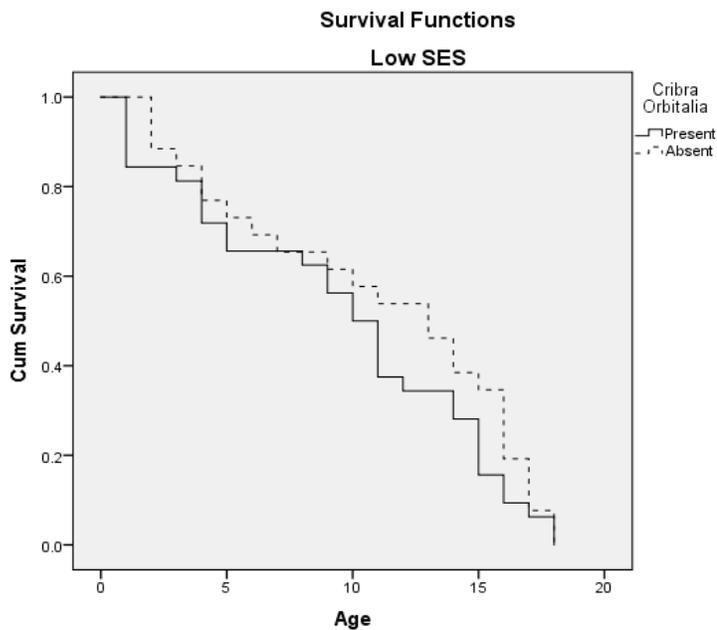


Figure 33: Kaplan-Meier curve shows the presence and absence of CO within the low SES exhibit a similar distribution of survivability.

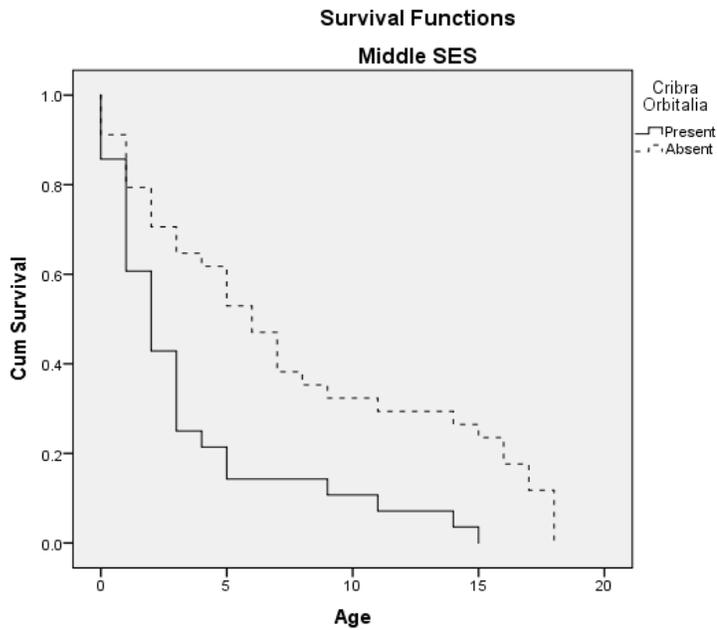


Figure 34: Kaplan-Meier curve shows the presence and absence of CO within the middle SES exhibit a marked difference of those without having greater survivability.

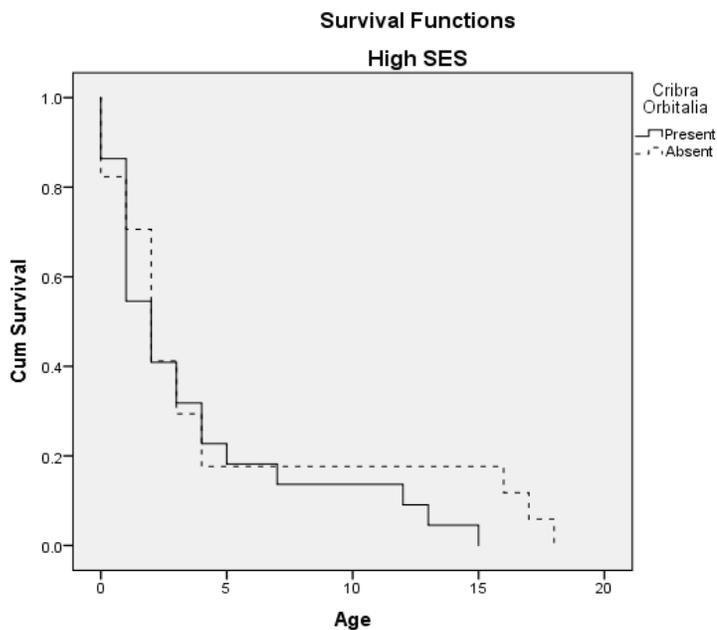


Figure 35: Kaplan-Meier curve shows the presence and absence of CO within the high SES exhibit a similar distribution of survivability.

Table 21: Kaplan-Meier survival analysis results of those with or without CO within each sample.

CO	Mean survival time (years)	95% CI	Mantel-Cox X ²	p-value
Low SES	Presence	7.56-11.50	9.74	*.002
	Absence	8.74-13.18		
Middle SES	Presence	1.90-4.89		
	Absence	5.56-9.91		
High SES	Presence	1.77-5.41		
	Absence	1.64-7.42		

* statistical significance at .05 level.

When survivability with and without evidence for PO is examined within populations, results are statistically significant ($p=.004$, Table 22). In the low SES sample, those with PO exhibit a slight increase in survivability over those without the pathology (Figure 36). Individuals in the middle SES without PO have greater survivability, while those with the pathology have a greater risk mortality before the age of 5 years (Figure 37). Children from the high SES exhibit a similar pattern of mortality despite presence of absence of PO, though those with PO have a slight increased risk of mortality from ages 0-5 years (Figure 38). These results find those without PO in the middle and high SES have increased survivability, while those in the low SES exhibit greater survivability if they do show evidence of PO.

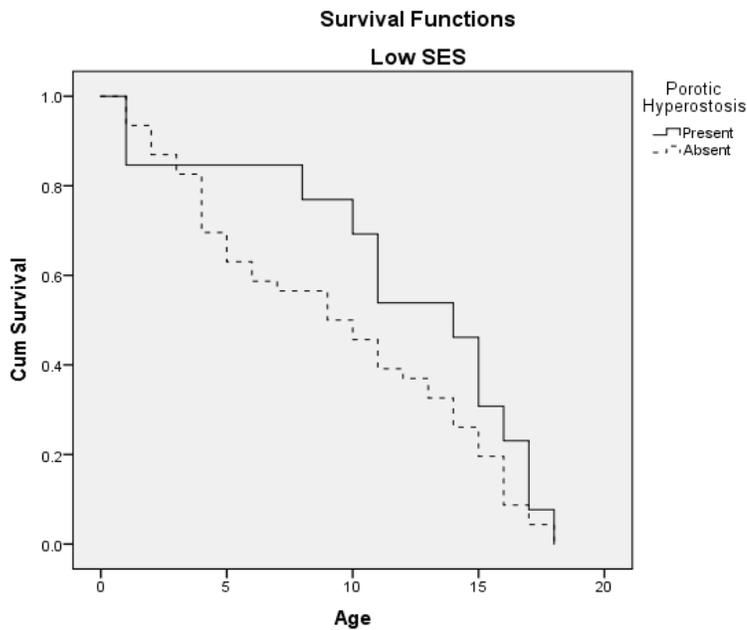


Figure 36: Kaplan-Meier curves show the presence and absence of PO within the low SES exhibit a unique distribution with those having evidence of PO having greater survivability than those without.

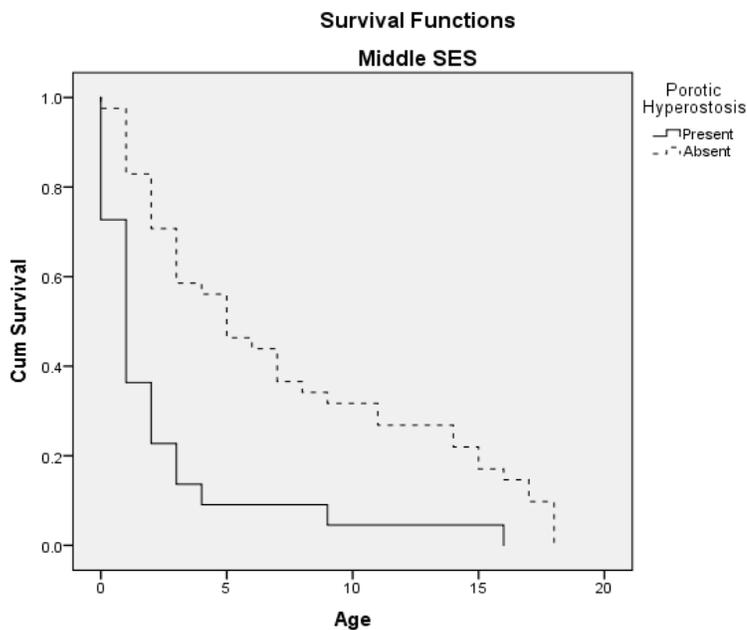


Figure 37: Kaplan-Meier curves show the presence and absence of PO within the middle SES showing those without evidence of PO having greater survivability than those with.

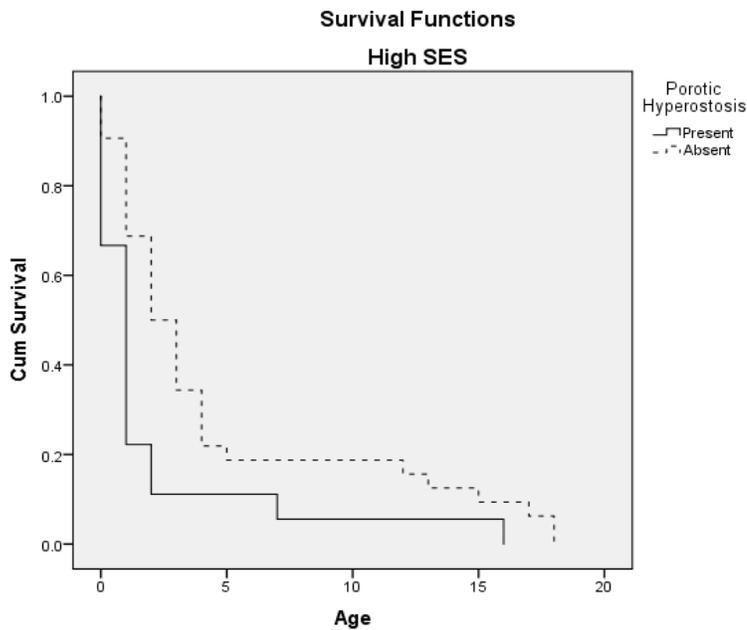


Figure 38: Kaplan-Meier curves show the presence and absence of PO within the high SES showing those without evidence of PO having a slightly greater chance of survivability than those with PO.

Table 22: Kaplan-Meier survival analysis results of those with or without PO within each sample.

PO	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Low SES	Presence	8.76-14.94	8.25	*.004
	Absence	7.69-10.92		
Middle SES	Presence	.70-3.76		
	Absence	5.46-9.23		
High SES	Presence	.16-3.73		
	Absence	2.70-6.55		

* statistical significance at .05 level.

When survivability with and without evidence for LEH is examined within populations, results are statistically significant ($p=.0001$, Table 23). In the low SES sample, individuals with LEH exhibit a much greater chance of survivability past the age of five years than those who do not have an LEH (Figure 39). Those in the middle SES exhibit the same pattern as the low SES, with those having LEH exhibiting significantly ($p=.0001$) greater survivability past the age of eight years (Figure 40). Similar to the other two samples, children from the high SES have a significantly higher ($p=.0001$) chance of survivability when they exhibit an LEH (Figure 41), while those without an LEH are not likely to survive past the age of seven years. These results find those with an LEH (in all samples) have increased survivability, while those who do not exhibit an LEH are likely to die prior to the age of 10 years.

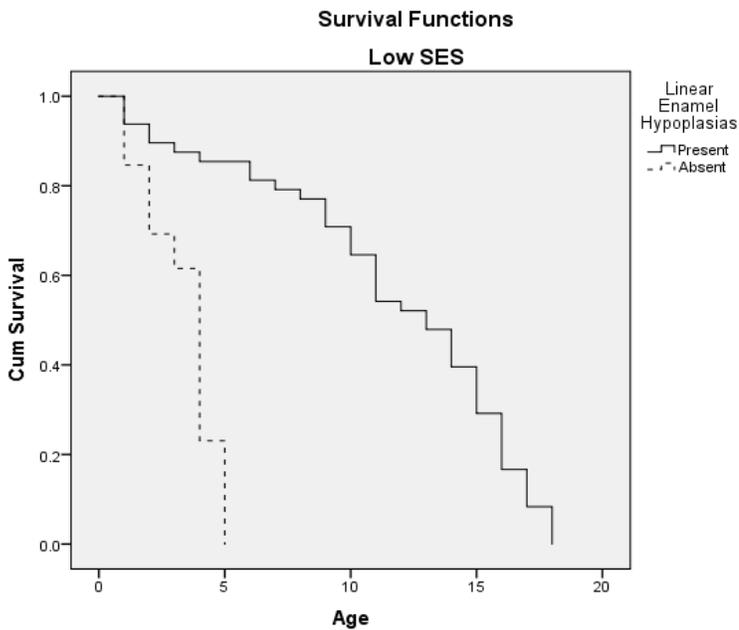


Figure 39: Kaplan-Meier curves show the presence and absence of LEH within the low SES showing those with LEH having a significantly ($p=.0001$) greater chance of survivability past the age of 5 years than those without.

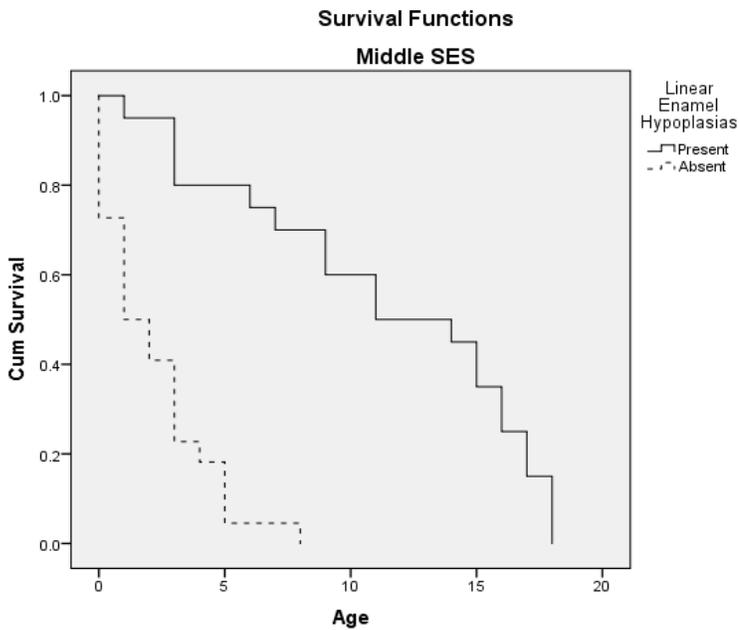


Figure 40: Kaplan-Meier curves show the presence and absence of LEH within the middle SES showing those with LEH having a significantly ($p=.0001$) greater chance of survivability past the age of 8 years than those without.

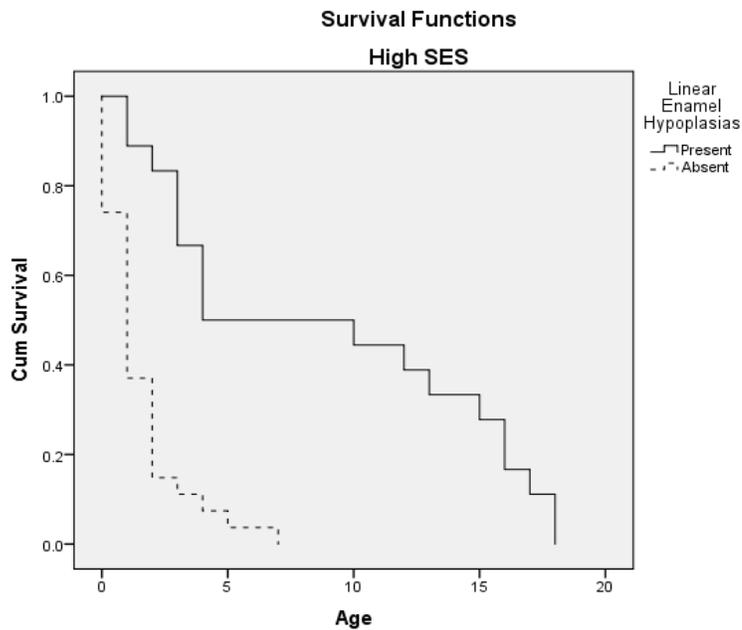


Figure 41: Figure 41. Kaplan-Meier curves of presence and absence of LEH within the high SES showing those with having a significantly ($p=.0001$) greater chance of survivability past the age of 7 years than those without.

Table 23: Kaplan-Meier survival analysis results of those with or without LEH within each sample.

LEH	Mean survival time (years)	95% CI	Mantel-Cox X^2	p-value
Low SES	Presence	10.16-13.10	82.95	*.0001
	Absence	2.60-4.17		
Middle SES	Presence	8.79-13.91		
	Absence	1.27-3.09		
High SES	Presence	5.83-11.95		
	Absence	.90-2.14		

* statistical significance at .05 level.

When survivability with and without evidence of periostitis are examined within populations, results are no longer statistically significant ($p=.287$, Table 24). Presence of periostitis has no effect on survivability. The results for the low SES sample can be seen in Figure 42, the middle SES in Figure 43, and the high SES in Figure 44.

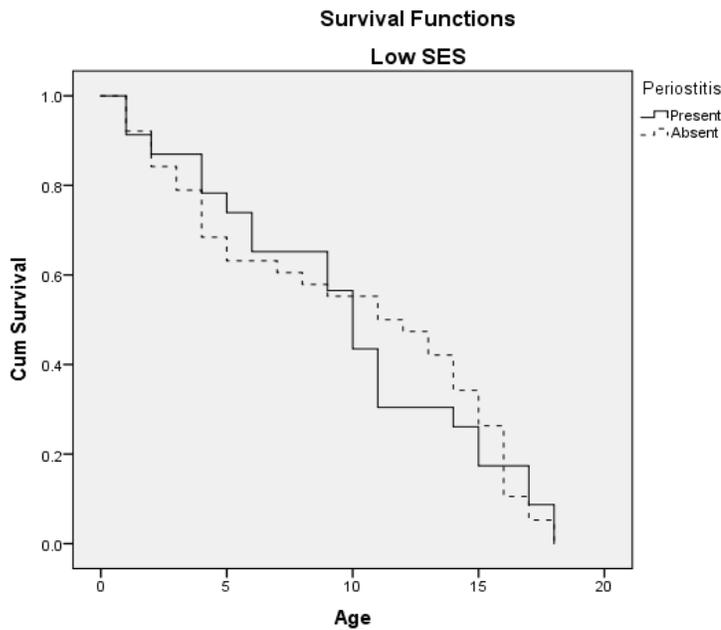


Figure 42: Figure 42. Kaplan-Meier curves show the presence and absence of periostitis within the low SES showing no difference in survivability.

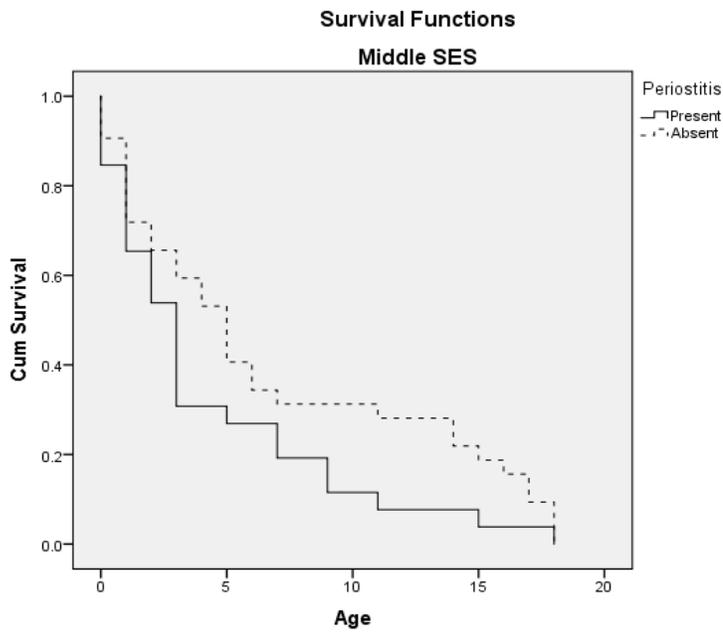


Figure 43: Kaplan-Meier curves show the presence and absence of periostitis within the middle SES showing no difference in survivability.

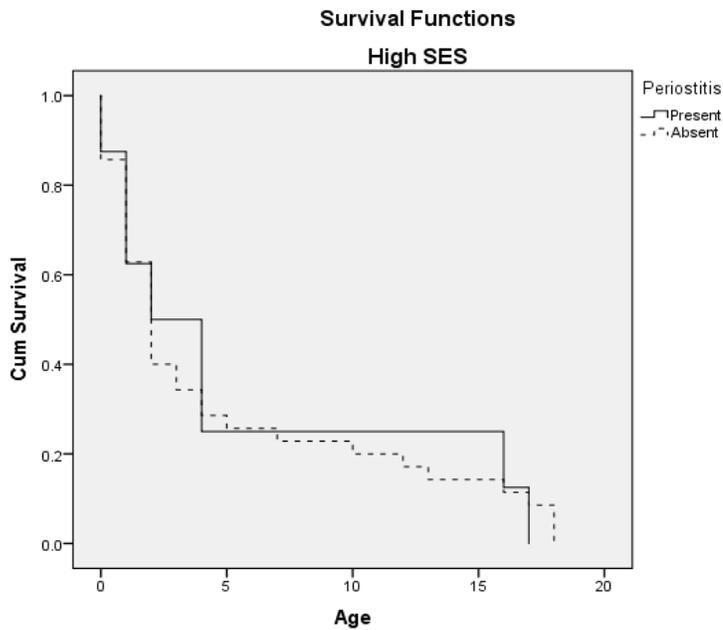


Figure 44: Kaplan-Meier curves show the presence and absence of periostitis within the high SES showing no difference in survivability.

Table 24: Kaplan-Meier survival analysis results of those with or without periostitis within each sample.

LEH	Mean survival time (years)	95% CI	Mantel-Cox X ²	p-value
Low SES	Presence	7.52-11.96	1.13	.287
	Absence	8.04-11.85		
Middle SES	Presence	2.41-6.05		
	Absence	4.66-9.15		
High SES	Presence	.87-10.38		
	Absence	2.91-6.92		

CHAPTER 7

DISCUSSION

What does it all mean?

The first prediction of Hypothesis one stated that the Industrial Era children would exhibit stunted femur length compared to a healthy, modern reference sample (the Denver Growth Study). Results do show that over half the children had stunted femora, suggesting this time period was indeed stressful and did impact children's growth regardless of their social status. The longer children lived, the more likely they were to display stunted femur length. These results support those found by Lewis (2002) and Cardoso and Garcia (2009) and confirm that these samples were stressed.

Part of the third prediction of the first hypothesis was that sexual dimorphism of femur length would be reduced in the Industrial Era samples, particularly the low SES, given evidence of biological female buffering often resulting in more stunted males and less dimorphism (Pucciarelli et al., 1993; Oyhenart et al., 2006; Garvin and Ruff, 2012). Mann-Whitney U tests confirmed this hypothesis prediction, since none of the Industrial Era adolescents proved dimorphic compared to the Denver adolescents, who were statistically significantly dimorphic.

The presence of lesions (CO and PO) associated with nutritional deficiencies in almost one third of individuals suggests that the overall diet during the Industrial Era was deficient in vitamin B12, iron, and vitamin C. Since all three samples showed similar distributions of these, we cannot claim that SES was directly associated with the

likelihood of experiencing these deficiencies. While it was expected that the London sample would have fewer frequencies of these pathologies, the fact that they don't seems to suggest nutrient-rich food was not always accessible during this time (Molleson and Cox, 1993; Horrell and Oxley, 2012; Humphries, 2013). It appears that no matter the social standing, healthy food was hard to come across. The significant association of LEHs in the Lisbon sample however, suggests that children in the lower class sample were at increased risk of experiencing times of acute stress than the children in middle and upper class samples. In this case, SES does play a direct role in the development of LEHs. The low and middle SES samples display significantly more presence of periostitis, leaving the high SES expressing the fewest signs of pathology, overall, thus supporting the first prediction of the second hypothesis.

While results are not statistically significant, boys do display a pattern of greater frequency of CO, PO, and LEH. There are no differences in periostitis. These somewhat support the second prediction that boys, being more vulnerable and frail, are more susceptible to the accumulation of pathologies. Within each sample, boys from the lower and upper SES groups exhibit a pattern of greater frequency of pathology presence, supporting the third prediction of the second hypothesis.

The third hypothesis predicted that girls would have greater survivability than boys. With samples pooled, the survivability curve for girls is consistently above the curve for boys, suggesting girls did survive better, overall. However, differences were not statistically significant. These results somewhat support this prediction and the female buffering hypothesis. When adjusted for samples, we see that girls in Lisbon had greater

survivability until the age of 12, at which point they are even with boys. Middle SES (Bologna) boys and girls overlap in their risk of mortality throughout the age ranges, so neither sex appears more frail than the other. The high SES sample from London shows that boys and girls have a similar risk of mortality until the age of five years, when boys risk of death increases and girls' survivability increases.

When samples are pooled, the presence of CO and PO are both associated with a significantly greater risk of death, while absence of these comes with greater survivability. These results support the third prediction of the third hypothesis. The presence of periostitis did not affect survivability. Interestingly, though, the presence of at least one LEH, and (even the presence of) three or more, was associated with a significantly greater chance of survivability. These results were unexpected since LEHs are often correlated with increased risk of death (Goodman, 1989; Steckel, 2005; Boldsen, 2007; Hughes-Morey, 2012).

Separating the samples did not change the results for survivorship or significance, though some pattern does emerge for CO, with the middle SES sample exhibiting variation between those with CO having greater mortality than those without. Presence of PO surprisingly provides greater survivability in the juveniles of the low SES sample, but absence of PO is significantly associated with greater survival in the middle and high SES samples, reflecting the pattern for pooled sample and samples pooled and with the overall prediction that presence of pathologies should be associated with lower survivorship. Survivability of those with and without periostitis exhibits more overlap in the low and high SES samples, while the middle SES displays better survivability in

those without.

Why weren't the children in Lisbon more stunted?

The first hypothesis predicted that children from the lower SES sample from Lisbon would exhibit the most stunting compared to the middle SES from Bologna and the high SES from London. The results did not support this prediction. The children from Bologna and London showed more stunting in every age category, while children from the middle SES had an overall higher percentage of stunted femur length when sexes and ages were pooled.

When looking at sex within SES groups, results show that boys aged 0-12 years from the middle and high SES groups are shorter and have lower z-scores than those in the lower SES group. Girls have z-scores more similar to each other, though the high SES sample tends to be shorter than the other two.

There are two possible interpretations for these results. First, children in Lisbon may have simply died at the time of stress, before having the opportunity to become stunted. If the children from the middle and high SES samples had the means to survive through stress, their bodies may have been more likely to exhibit stunting than their lower SES counterparts, thus this could be a reflection of survivability of the middle and upper class children (Wood et al., 1992; DeWitte et al., 2015; Hughes-Morey, 2016). This would suggest the children from the lower SES sample may have been more frail and likely to die prior to accumulating stuntedness. If this is the case, it is strong evidence that social class had a strong influence on a child's growth and survivability, perhaps yielding

more stunting in middle and upper SES samples of children.

Second, this could reflect the fact that conditions were poor for everyone, despite SES. According to Tanner (1981), urban English children experienced more stress than their rural counterparts, a trend that was quite different than other parts of Europe where urban children grew and survived better, such as in Portugal where rural inhabitants were shorter than those in Lisbon (Reis, 2009). These results support those of the z-scores showing that low SES children were not as stunted as the middle and high SES groups. Individuals from the high SES sample lived 70 years earlier than the low SES sample and 150 years earlier than the middle SES sample. While the high SES group may have experienced greater wealth relative to their populations than the other two groups, the time period in which they lived and their location may have placed them in a more strenuous environment. Urban industrial London in the 18th century saw a host of stressful events, like cholera outbreaks, influenza, food shortages (particularly fruits and vegetables in the winter months), as well as air, water, and ground pollution due to industrial waste and lack of proper sewerage (Thompson, 1963).

While higher SES is often attributed to higher standards of living and increased survivability, we must always consider populational context. In 1748, William Cadogan, a British physician, realized the lack of care given to infants from wealthy families. He wrote:

The mother who has only a few rags to cover her child loosely and little more than her own breast to feed it sees it healthy and strong, and very soon able to shift for itself; while the puny insect, the heir and hope of a rich family lies languishing under a load of finery that

overpowers his limb, abhorring and rejecting the dainties he is crammed with, till he dies a victim of the mistaken care and tenderness of his fond mother.

Another possibility to consider is that, despite their predominantly lower SES status and the poor conditions throughout Lisbon during this time, these children may have experienced better overall environmental conditions than those from the other two samples. For example, Cardoso and Garcia (2009) find evidence of catch-up growth reflected in adult stature of this sample, suggesting the Portuguese environment was good enough to allow improved growth. Lisbon is a port city, with a rich fishing history, sunlight, and naturally growing citrus-rich fruits. The strong, Catholic, traditional family unit was considered the "primary institution of society" (Reis, 2004; 2009; Cardoso, 2005; Oliveira and Pinho, 2010; Klüsener et al., 2014). Perhaps these strong familial ties and better environment kept conditions improved, relative to the other samples.

Why were adolescent girls so severely stunted in all three samples?

The third prediction of the first hypothesis suggested boys would exhibit more stunting than girls, as predicted by the buffering hypothesis that females have an adaptive evolutionary mechanism allowing them to retain protein and fat (Stini, 1969, 1971, 1972, 1978, 1980; Wolfe and Gray, 1982; Hiernaux, 1985; Roede and van't Hof, 1978; Brauer, 1982; Tanner, 1989; Eveleth and Tanner, 1990; Pucciarelli et al., 1993; Buffa et al., 2001; Oyhenart, 2006; DeWitte, 2010). Results show that the lower and middle SES girls express an overall higher percentage of stunting than boys, while the higher SES boys

have a higher percent of stuntedness than girls. When broken down into age groups, girls in the low SES sample always express a lower z-score than boys. The middle SES boys have lower z-scores than girls in early and middle childhood (ages 2-12 years), but then girls become more stunted in adolescence. In the high SES, girls are more stunted in infancy and early childhood (ages 2-5 years), boys are more stunted in ages 6-12 years, then girls become more stunted in adolescence. All three samples express a similarity in both male and female general stunting patterns, with stunting increasing with age. SGPs of each sample (Figure 6) show variation in these z-scores between males and females, with the middle SES expressing the most marked differences, with boys being shorter than girls from ages 3-10 years. Overall the results vary between samples in the ages of 0-12 years. The low SES does not support the prediction that boys would be more stunted, since girls' z-scores fall below boys'. The middle SES results do support this prediction, with boys' z-scores falling below girls and even reaching -2 z-scores below the reference. The high SES varies between boys and girls during these ages, not supporting or refuting the prediction, rather suggesting there is no difference in girls and boys pattern of stuntedness.

However, one clear pattern emerges in all three samples. All girls exhibit severe levels of stunting (-6 to -7 z-scores) in adolescence, while boys remain at 2 z-scores or less below the reference. If we assume that female buffering exists, this severe stuntedness could be a consequence of girls surviving early childhood stressors better than boys and thus accumulating severe stunting over time. Perhaps boys were dying before having the opportunity to express such stunting, similarly predicted to higher SES

children displaying more stunting than the lower SES children. However, Kaplan-Meier results of survivability between boys and girls does not agree with this interpretation, showing that girls survive better than boys.

Instead, this distinct adolescent girl response may be due to delayed puberty in the Industrial Era girls relative to the modern, healthy reference sample from Denver, thus exaggerating the severity of the z-scores. There is evidence to suggest that, when girls are stunted in height, their pubertal growth can be delayed one to two years behind those from more modern or healthy populations (Tanner, 1982; Belachew et al., 2011). Belachew et al., 2011 found that food insecure girls in undeveloped Ethiopia started menarche, on average, one year after food secure girls, who started on average at 14 years. Those who were stunted began menarche even later (Belachew et al., 2011). The results suggest Industrial Era girls were delayed in growth compared to boys who appear to have a similar trajectory (albeit below) to the Denver boys, supporting conclusions by Cardoso (2005) who found that girls in the Lisbon sample exhibited greater delay than boys. It seems more likely that female buffering was overridden by cultural buffering of boys in these patriarchal societies. Indeed, a potential, social interpretation arises about preferential treatment of boys, especially at the point of pubertal growth. If males were favored, and historical reference suggests that they were given more access to nutritional food and healthcare at the time of adolescence when they were working away from home and bringing earnings to the household (Stinson, 1985; Horrell and Oxley, 2012; Humphries, 2013), allowing them to continue on their expected growth trajectory in adolescence, while girls' growth took a negative hit and became delayed. Even with boys,

potentially working in more stressed conditions outside of the home, girls appear to be under greater stress.

Why did children in Lisbon survive better?

The survivability results are unexpected and do not support the first prediction of the third hypothesis, with the high and middle SES samples displaying a greater risk of mortality than the low SES. The first notable difference is the higher infant mortality rates in the high and middle SES samples compared to the low SES sample. This could be due to a few possibilities.

The unsanitary conditions and widespread infectious diseases in London during the 18th and 19th centuries led to very high infant mortality rates (Woods and Woodward, 1984), an ill-effect that does not care about social status. The mortality profile for urban Bologna suggests similar conditions there. Breastfeeding practices in 18th and 19th century England may have led to higher infant mortality because colostrum was considered harmful. The newborn infant would be fed butter, sugar, or even wine in those first days until breastmilk was available. Infants were also weaned from breastmilk entirely between 7-18 months. This lack of colostrum and prolonged breastfeeding, in conjunction with contamination of foods and utensils, would often cause fatal, gastrointestinal illnesses in infants, as well as leaving them vulnerable to infectious diseases (Fildes, 1986). Breastfeeding practices may also contribute to the age of growth deficit seen in each of the samples, with most faltering taking place in the 2-5 year age-group. If we assume most infants were no longer breastfed sometime around the age of

one year, as is suggested by English and Portuguese historical reference (Fildes, 1986; Cardoso, 2005), growth delay at this point makes sense.

However, we cannot believe infant mortality was less dramatic in Portugal. Cardoso (2005) tells us half of all children died prior to age 15 years in 1900, and infant mortality was at a rate of 200 deaths per 1000 births, and remained one of the highest in comparison to other industrial European countries until the 1960s when public health programs increased (Cardoso, 2005; Klüsener et al., 2014). Death rates amongst older children up to age 14 years only started to decline in the 1940-50s, with most deaths attributed to nutritional deficiencies and infectious diseases (Cardoso, 2005). So, the improved mortality profile for the Lisbon sample observed in the present study, particularly amongst infants, must be explained another way.

Lisbon's burial practices during the 19th and 20th centuries are well documented (Cardoso, 2005). All individuals were placed in a temporary grave for five years, at which point they were exhumed by cemetery workers. The bodies were either placed in a more permanent burial if the family paid a fee. If they were unclaimed or the family could not pay the fee, they were placed in a communal grave or incinerated. The Bocage Museum began taking individuals that were unclaimed and destined for communal burial or incineration for this skeletal collection (Cardoso, 2005; 2006). Many stillbirths and infants were often buried in communal graves rather than interred in their own burials (Cardoso, 2005). Missing infants from this Lisbon collection could be due to untrained graveyard workers who exhumed, and likely missed, tiny infant bones (Cardoso, 2005). It is also worth mentioning that infants and children not placed in this sample could have

been placed in a more permanent burial if their families paid the interment fee, since those present are most likely there because their family could not pay that fee, thus a reflection of the lower status of this sample. It is also important to keep in mind there is a higher rate of older children present in the Lisbon sample, compared to the others, lending some support for the historical data regarding child death up to age 15 years (Cardoso, 2005). Perhaps this is actually a reflection of the frailty within the older children of the Lisbon sample.

LEHs are often more observable in older individuals with more permanent teeth, given the fact that they form in early childhood. This is not to suggest LEH cannot be present on deciduous teeth as indicators of maternal stress. Results do show LEHs were overwhelmingly skewed toward the Lisbon sample, perhaps a reflection of the greater number of adolescents.

Why were LEHs associated with increased survivability?

While the results for survivability in those with and without presence of cribra orbitalia, porotic hyperostosis, and periostitis were expected, those with LEH display an unexpected pattern of greater survivability. This pattern persists across samples with those exhibiting at least one having greater survivability than those without an LEH. Presence of an LEH is associated with children surviving past the ages of 5-8 years.

It seems clear that metabolic stress associated with poor nutrition was detrimental to children's survival in Industrial Era cities, regardless of their SES. Children who experienced such accumulation of metabolic stressors were likely more frail than those

who did not. The fact that most children past the ages of 5-8 exhibit at least one LEH indicates Industrial Era European environments were stressful for children and inhibited childhood growth. If a child was able to survive the acute moments of stress leading to LEHs, they were more likely to survive to older ages. These results are similar to others who find a positive association between LEH and longevity (Lewis, 2002; Bennike et al., 2005). A higher prevalence of LEH may be associated with increased survivability over than those with only one LEH (Amoroso et al., 2014).

This research suggests LEHs are not always a good measure of frailty or heightened risk of mortality. Rather, it is important to analyze samples as context-specific units. According to Amoroso et al. (2014), exposure to stressors throughout life is a stronger factor for predicting longevity than any one non-specific indicator of stress, like CO, PO, or LEH. Cain and Hong (2009) also suggest overall life conditions are a better predictor of survivability than early life stressors. They found that military recruits in London were more likely to die during wartime exposure to disease, famine, and stress if they were born in rural areas than if they were born in urban areas and already exposed to such stressful conditions in early life. They argue that rural born recruits, though healthier, were less able to survive the insults experienced during war than were urban-born recruits who had already experienced stress. This goes against DOHaD arguments that early life stressors make an individual more frail and susceptible to death, and rather supports the argument that overall life experiences are a stronger predictor for survivability. Ultimately, the children in these samples did not survive into adulthood, thus taking these LEH laden individuals out of the adult, living population.

Lower SES kids show increased risk of mortality as they age compared to the other two samples, as well as a significantly greater frequency of LEHs. This is not necessarily surprising, given that an older aged sample would be more likely to exhibit higher rates of LEHs compared to younger children who have not had as much time to accumulate pathologies. It may be that low SES older children and adolescents were more frail than those in the middle and high SES samples given their increased mortality past the ages of 5-8 years. Though their z-scores do not suggest increased stress during early growth periods, higher frequencies of LEH do suggest early life stress. These subadults may have experienced selective mortality and thus higher frailty at these particular ages than their middle and high SES counterparts and simply died before having chance to accumulate other stressors at higher rates, such as stunting (Wood et al., 1992). While it appears the middle and high SES samples experienced increased infant mortality, this pattern likely reflects non-inclusion of infant and young child remains in the low SES sample.

Why did girls from Bologna display higher frequencies of stress?

The middle SES girls from Bologna have a higher frequency of all pathologies, except LEHs, compared to middle SES boys, suggesting girls from this sample experienced greater cultural stress than boys. Italy was certainly a traditionally Catholic and patriarchal society that has traditionally viewed women as domestic workers, devaluing their place outside of the home (Buscemi, 2014) and often treating them harshly by pushing against women's rights, as well as liberal and social policies (Krause,

1999; Rahikainen, 2004; Bosworth, 2013; Dawes, 2014). Though male migration to find work outside of Italy and the loss of adult male life after two World Wars resulted in women and younger boys taking on more responsibilities outside of the home and women as heads of households (Friedman-Kasaba, 1996). Fascist dictatorships, like Mussolini's, often involved oppression of working women (Capecchi, 1997; Chang, 2015). These historical factors of labor and treatment of women and children may help explain why young Bologna boys experienced more stunted femur lengths than girls through hard labor outside the home, but also why girls may have been more susceptible to poor health from lack of healthy food and medical care inside the home.

Limitations

Skeletal growth research is difficult due to a lack of large samples with known sex and age. Sample sizes are often small, but when they are further divided by sex and age, they become even smaller. Many of them also display uneven distributions of ages, as in this study, making some comparisons difficult. In some categories, like the upper class sample with only one girl in the 6-12 years old age group and one boy in the adolescent group, sample sizes are very small. This undoubtedly impacts the results of the Mann-Whitney U-tests between sexes.

It is important to keep in mind that these results are based on samples that comprise some of the most vulnerable and frail individuals within the general living population. These indicators of stress (stunted femur, pathology, and mortality) do not necessarily reflect those of the entire population (Wood et al., 1992). There may also be

hidden heterogeneity, given that these are large assemblages that span long periods of time, the prevalence of stuntedness, pathology, and mortality could be due to multiple factors. These include secular changes, genetic differences that cause particular individuals to be more susceptible to certain diseases, socio-economic variations within the samples targeting the lowest within each sample, and gender or sibling preference within families causing others to be more vulnerable to stress and death despite genetic potential or SES (Wood et al., 1992).

Skeletal collections can seem biased with regards to linear growth in height, arguing that those who died and are included within the samples do not represent the overall population that continued to live (Buikstra and Cook, 1980; Wood et al., 1992). This mortality bias can lead to overestimation of stuntedness and pathologies within a population if it is assumed those within the skeletal sample represent the living population. If stunted growth and susceptibility to disease increase one's risk of death, then those within a skeletal sample will likely exhibit those signs of stress (Haas, 1990; Saunders and Hoppa, 1993; Cardoso 2005). In addition to this mortality bias, these skeletal samples are cross-sections of populations that spanned large periods of time, with individuals living at different points in their cities' histories. It is important to keep in mind that these samples were compared to a modern, healthy reference sample, which could further exaggerate the stuntedness of the past populations. The mortality bias is confounded by changing environmental, political, and economic factors.

Also, in general, pathologies are difficult to interpret in skeletal remains. Aside from the fact that they are hard to identify and their origins often unknown, their

relationship to social status is not clear. Stress markers by themselves do not necessarily indicate poor health. Not all diseases leave skeletal evidence and a lack of skeletal stress does not directly indicate good health or the ability to live and function any better than those who display signs of stress. Stress at older ages, such as the stunting (likely caused from delayed growth) seen in the older girls, could simply be a consequence of living longer in stressed conditions. Within any given population these factors must be considered when attempting to interpret stress, however the patterns seen across all three of these samples, such as boys having a higher frequency of CO, PO, and LEH, and the high SES sample expressing the lowest frequencies of pathologies, make general interpretations of stress more feasible.

Another confounding issue is the definition of status of bioarchaeological samples. Much like Temple and Goodman (2014) suggested that "Bioarchaeology has a health problem," this research brings up the question "does bioarchaeology have a status problem?" Many bioarchaeological researchers analyze the remains of those from past populations with status being a variable used for comparisons (Lewis, 2002; Cardoso, 2005; Redfern and DeWitte, 2011; DeWitte et al., 2015; Redfern et al., 2015; Hughes-Morey, 2016), suggesting "status" is important in bioarchaeological investigations. While research skeletal samples are often placed into low, middle, and upper SES categories based on grave goods, historical records of occupation, and neighborhood of residence, it is likely that individuals within the samples vary from each other, especially over the course of time. It is not likely that all individuals fit into that particular status definition.

The length of time in which the individuals in the samples were living, likely

allowed the fluctuation and variation of secular changes, political-economic shifts, environmental conditions, and status. The individuals from Lisbon, for example, lived through a very tumultuous political economy, but their femur lengths were less stunted than those from London and Bologna. While this result could be a reflection of higher SES children surviving through stressed conditions long enough to accumulate stunted femur length, it could mean that the lower SES sample from Lisbon (despite lower status) had better access to food and healthcare than the middle and upper SES samples in Bologna and London, or it could suggest that SES is not clearly defined or understood in these samples.

Status is multi-dimensional and includes many factors. For example, those buried in the crypt from the Spitalfields sample enjoyed relative wealth during most of the 18th century, but as the silk industry declined, so did the economic prosperity of that district, perhaps decreasing the "high" SES status of the sample over time. Cardoso (2005) discovered that, for the children in the Lisbon sample, many of the father's occupations did not correlate with the neighborhood the family lived in and depending on how SES was determined (father's occupation or neighborhood) results for his dental development data could change. In Lisbon and Bologna, most women were defined as "doméstica", a term that could mean stay-at-home wives and mothers to varied forms of domestic workers, that all have varied levels of "status" meanings.

Defining samples by status for comparisons can become problematic in research design and interpretations if the status definition is not entirely accurate or if these definitions are contradictory. Variation between samples may indeed be a reflection of

status, but may also be a reflection of urban versus rural living conditions, gender variation, overall environmental factors that could include epidemics, political situations, famine, population increases, pollution, or populational variation due to genetics. It is also important to remember that those within skeletal samples are those who did not survive the stresses of life. If the children represented in these samples could not survive through these acute or chronic stresses, their father's occupation and their neighborhood of residence did not make a difference, given their place within the skeletal sample in the first place.

While there are limitations in sample sizes, status definitions, and secular changes, large cemeteries do represent a large demographic (Van Gerven and Armelagos, 1983; Buikstra and Konigsberg, 1985; Saunders, 1993; Larson, 1997), making reconstructions and interpretations about the overall conditions and growth patterns of the population possible. This project aimed to understand how environmental stress from industrialization and female buffering impacted growth, morbidity, and mortality patterns. To do this, large cemetery samples comprised of known age and sex, with children aged 0-18 years, and relatively similar numbers of males and females were found. Though these three samples vary in geographic region and time periods, they were all undergoing their society's industrialization period within densely populated, urban environments. While the social status of each sample could be debated as mostly middle SES, these were chosen to represent three varied SES groups (low, middle, and high), given the historical context of each sample.

This research supports the findings of others that the environmental insults of the

Industrial Era impacted children's growth and survivability, that females experienced delayed growth compared to boys most likely due to the preferential treatment of males, though males more often exhibited evidence of pathology, and that presence of chronic stress from malnutrition lead to a higher incidence of death (Lewis, 2002; Cardoso and Garcia, 2009; DeWitte et al., 2015; Hughes-Morey, 2016).

Future Directions

This research has raised interesting patterns that lead to further questions. Given the higher survivability of those with LEH in all samples, it would be interesting to assess if having multiple indicators of stress (both chronic and acute) including LEH in addition to CO, PO, and periostitis impact survivability and growth. I anticipate that those who died with evidence of chronic stress will exhibit more stunted femur length than those with only LEHs.

While the samples used in this research were designated low, middle, and high SES, it is likely they are all more indicative of middle SES samples given their widespread time frames, large numbers of interred individuals within the cemeteries, and populational changes in political and economic climates their populations experienced over time. It is necessary to perform a broad analysis of how skeletal samples have been and still are defined by status and to begin a broader conversation of how to interpret status within these large cemetery samples.

While pooling skeletons from different collections for larger sample sizes can create challenges such as populational, SES, urban/rural variation, it would be interesting

to analyze larger samples from each of these geographic locations and to analyze the effects of industrialization between countries. Larger samples from the same geographic locations can also provide more information about secular changes by analyzing growth, stress, and mortality between sexes within date-of-birth and date-of-death cohorts.

Unfortunately, sample sizes are too small to analyze this data now, but with increased sample sizes this could be done. Lastly, analyzing adult stature in addition to childhood growth, as a proxy of "catch-up" growth or further stress to assess at what point in time were conditions improved or worsened.

CHAPTER 8

CONCLUSIONS

This study assesses the growth of boys and girls from low, middle, and upper class skeletal samples from the Industrial Era of Europe. Results show that children from this time period did experience stunted growth, regardless of their SES or sex. This suggests that all children exhibited stress during this time. However, unexpectedly, high and middle SES children expressed more stuntedness than children from the lower SES sample. Adolescent girls aged 13-18 years from all three samples expressed severe stunting likely due to delayed pubertal growth. While girls experience this delay in growth, boys do not. This is likely the effect of cultural preference of adolescent boys in these patriarchal societies. Despite girls' delay in pubertal growth, there is a lack of sexual dimorphism in femur length in all of the samples, indicative of adolescent male stress.

The presence of pathological conditions associated with nutritional deficiencies, like cribra orbitalia and porotic hyperostosis, was discovered in over one third of the entire sample. These conditions did not prove to be directly associated with status, but presence of LEHs did, with the low SES sample exhibiting statistically significantly more presence, and periostitis in the middle and low SES. This suggests that while most children lacked adequate nutrition, those from lower classes were at an increased risk of experiencing acute stress during childhood.

Though not statistically significant, boys did exhibit higher frequencies of presence of LEH, PO, and CO, with no distinction between sexes in periostitis. Boys

within the middle and high SES do exhibit a greater frequency of all pathological conditions. Mortality patterns reveal that the low SES sample displays the greatest survivability compared to the other samples, though this result could be biased due to fewer infant/young child remains included in the sample. Boys and girls exhibit no differences in survivability. It was discovered that those with presence of CO and PO have higher risk of mortality compared to those with presence of three or more LEH, who have a higher survivability.

These results suggest Industrial Era conditions were indeed stressful and inhibited children's ability to grow and survive. Less stunting, no significant variation in metabolic stressors, and a greater survivability within the low SES may be a result of environmental conditions being improved in the low SES sample compared to the middle and high SES samples, or even variation within samples in terms of status and secular changes.

“When my mother died I was very young,
And my father sold me while yet my tongue
Could scarcely cry “Weep! Weep! Weep! Weep!”
So your chimneys I sweep, and in soot I sleep.”
From The Chimney Sweeper
By William Blake, London Poet (1757-1827)

BIBLIOGRAPHY

- Agarwal SC. 2016. Bone morphologies and histories: Life course approaches in bioarchaeology. *Yearbook of Physical Anthropology* 159: 130-149.
- Agostini G. 2017. Can long bone structural variability detect among-population relationships? Ph.D. Dissertation. University of Massachusetts, Amherst.
- Albanese J, Cardoso H, Saunders SR. 2005. Universal methodology for developing univariate sample-specific sex determination methods: An example using the epicondylar breadth of the humerus. *Journal of Archaeological Science* 32:143-152.
- Allen LH. 1994. Nutritional influences on linear growth: a general review. *European Journal of Clinical Nutrition* 48: S75-89.
- Alvesalo L. 1997. Sex chromosomes and human growth: a dental approach. *Human Genetics* 101:1-5.
- Amoroso A, Garcia SJ, Cardoso H. 2014. Age at death and linear enamel hypoplasias: Testing the effects of childhood stress and adult socioeconomic circumstances in premature mortality. *American Journal of Human Biology* 26: 461-468.
- Angel JL. 1972. Biological relations of Egyptian and Eastern Mediterranean populations during Pre-dynastic and Dynastic times. *Journal of Human Evolution* 1:307-313.
- Arch J, Arch A. 1822. Observations on the ruinous tendency of the Spitalfields Act to the silk manufacture of London. London: Printed for John and Arthur Arch, Cornhill.
- Aries E. 1962. Centuries of childhood: A social history of family life, trans. In: Robert B, editor. *Everyman in Europe: Essays in social history. V1 The preindustrial millennia*. New York: Alfred A. Knopf, Inc. pp. 390-404.
- Armstrong GJ, Goodman AH, Harper KN, Blakey ML. 2009. Enamel hypoplasias and early mortality: bioarchaeological support for the Barker hypothesis. *Evolutionary Anthropology* 18:261-271.
- Baker JB, Dupras TL, Tocheri MW. 2005. The osteology of infants and children. Texas A&M University Press.
- Bane D. 1990. Real wages, the economic cycle, and mortality in England and Wales 1870-1914. *International Journal of Health Services* 20:43-52.

- Barker DJ, Osmond C. 1986. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* 1(8489) 1077-1081.
- Barker DJ, Winter PD, Osmond C, Margetts B, Simmonds SJ. 1989. Weight in infancy and death from ischaemic heart disease. *Lancet* 2 (8663) 577-580.
- Barker DJ, Gluckman PD, Godfrey KM, Harding JE, Owens JA, Robinson JS. 1993. Fetal nutrition and cardiovascular disease in adult life. *Lancet* 341 (8850) 938-941.
- Barker DJ. 2007. The origins of the developmental origins theory. *Journal of Internal Medicine* 261(5): 412-417.
- Barrier IO. 2007. Sex determination from the bones of the forearm in a modern South African sample. Dissertation. University of Pretoria.
- Bass, W. 1995. *Human osteology: A laboratory and field manual*. Missouri Archaeological Society, Inc.
- Bastir M, Rosas A, Kuroe K. 2004. Petrosal orientation and mandibular ramus breadth: Evidence for an integrated petroso-mandibular developmental unit. *American Journal of Physical Anthropology* 123: 340-350.
- Bateson P, Gluckman P, Hanson M. 2014. The biology of developmental plasticity and the Predictive Adaptive Response hypothesis. *Journal of Physiology* 592(11): 2357-2368.
- Belachew T, Hadley C, Lindstrom D, Getachew Y, Duchateau L, Kolsteren P. Food insecurity and age at menarche among adolescent girls in Jimma Zone Southwest Ethiopia: A longitudinal study. *Reproductive Biology and Endocrinology* 9:125.
- Belcastro MG, Rastelli E, Mariotti V. 2008. Variation of the degree of sacral vertebral body fusion in adulthood in two European modern skeletal collections. *American Journal of Physical Anthropology* 135:149-160.
- Bengtsson T, van Poppel F. 2011. Socioeconomic inequalities in death from past to present: An introduction. *Explorations in Economic History* 48: 343-356.
- Bennike P, Lewis ME, Schutkowski H, Valentin F. 2005. Comparison of child morbidity in two contrasting medieval cemeteries from Denmark. *American Journal of Physical Anthropology* 128: 734-746.
- Black S, Scheuer L. 1996. Age changes in the clavicle: From the early neonatal period to skeletal maturity. *International Journal of Osteoarchaeology* 6:425-434.

- Blake K. 2011. An investigation of sex determination from the subadult pelvis: A morphometric analysis. Ph.D. Dissertation. Department of Anthropology, University of Pittsburgh.
- Blankenship JA, Mincer HH, Anderson KM, Woods MA, Burton EL. 2007. Third molar development in the estimation of chronologic age in American blacks as compared with whites. *Journal of Forensic Science* 52:428-433.
- Bogin B, Loucky J. 1997. Plasticity, political economy, and physical growth status of Guatemala Maya children living in the United States. *American Journal of Physical Anthropology* 102:17-32.
- Bogin B, MacVean RB. 1978. Growth in height of urban Guatemalan primary school children of high and low socioeconomic class. *Human Biology* 50: 477-488.
- Bogin B. 1995. Plasticity in the growth of Mayan refugee children living in the United States. In: Bogin B, Mascie-Taylor CG, editors. *Human Variability and Plasticity*. Cambridge University Press.
- Bogin B. 1999. *Patterns of human growth*, 2nd ed. Cambridge: Cambridge University Press.
- Boldsen JL. 2007. Early childhood stress and adult age mortality: A study of dental enamel hypoplasia in the medieval Danish village of Tirup. *American Journal of Physical Anthropology* 132:59-66.
- Boldsen JL, Milner GR, Weise S. 2014. Cranial vault trauma and selective mortality in medieval to early modern Denmark. *PNAS* 112(6): 1721-6.
- Boocock P, Roberts C, Manchester K. 1995. Maxillary sinusitis in medieval Chichester, England. *American Journal of Physical Anthropology* 98:483-496.
- Bosworth RJB. 2013. *Italy and the wider world: 1860-1960*. Routledge.
- Brauer GW. 1982. Size, sexual dimorphism, and secular trend: Indicators of subclinal malnutrition? Sexual dimorphism in *Homo sapiens* 245-259.
- Brown K. 1991. The importance of dietary quality versus quantity for weanlings in less developed countries: A framework for discussion. *Food and Nutrition Bulletin* 13: 86-94.
- Brown K. 2003. Diarrhea and Malnutrition. *Journal of Nutrition* 133: 328S-332S.

- Buffa R, Marini E, Floris G. 2001. Variation in sexual dimorphism in relation to physical activity. *American Journal of Physical Anthropology* 13: 341-348.
- Buikstra JE, Cook DC. 1980. Paleopathology: An American account. *Annual Review of Anthropology* 9: 433-470.
- Buikstra JE, Konigsberg LW. 1985. Paleopathology: Critiques and controversies. *American Anthropologist* 87(2): 316-334.
- Buikstra J, Ubelaker D. 1994. Standards for data collection from human skeletal remains. *Arkansas Archaeological Survey*.
- Buscemi F. 2014. Television as a trattoria: Constructing the woman in the kitchen on Italian food shows. *Journal of Communications* 29(3): 304-318.
- Cain L, Hong SC. 2009. Survival in 19th century cities: The larger the city, the smaller your chances. *Explorations in Economic History* 46: 450-463.
- Cameron N. 2002. *Human growth and development*. Elsevier Science.
- Capecchi V. 1997. In search of flexibility: The Bologna metalworking industry, 1900-1992. In: Sabel CF, Zeitlin J, editors. *World of possibilities: Flexibility and mass production in Western Industrialization*. Cambridge University Press.
- Cardoso H. 2005. Patterns of growth and development of the human skeleton and dentition in relation to environmental quality: A biocultural analysis of a sample of 20th Century Portuguese subadult documented skeletons. Ph.D. Dissertation. McMaster University.
- Cardoso H. 2006. Brief communication: The collection of identified human skeletons housed at the Bocage Museum (National Museum of Natural History), Lisbon, Portugal. *American Journal of Physical Anthropology* 129:173-176.
- Cardoso H. 2008. Sample-specific (universal) metric approaches for determining sex of immature human skeletal remains using permanent tooth dimensions. *Journal of Archaeological Science* 35(1): 158-68.
- Cardoso H, Garcia S. 2009. The not-so-dark ages: Ecology for human growth in medieval and early twentieth century Portugal as inferred from skeletal growth profiles. *American Journal of Physical Anthropology* 138: 136-147.
- Cardoza AL. 1982. *Agrarian elites and Italian fascism: The province of Bologna, 1901-1926*. Princeton University Press.

- Chang NV. 2015. *The crisis-woman: Body politics and the modern woman in fascist Italy*. University of Toronto Press.
- Corbier P, Edwards DA, Roffi J. 1992. The neonatal testosterone surge: A comparative study. *Archives internationales de physiologie, de biochimie, et de biophysique*. 100:127.
- Cottureau A. 1997. The fate of collective manufactures in the industrial world: The silk industries of Lyons and London, 1800-1850. In: Sabel CF, Zeitlin J, editors. *World of possibilities: Flexibility and mass production in Western industrialization*. Cambridge University Press.
- Cowgill L, Hager L. 2007. Variation in the development of postcranial robusticity: An example from Çatalhöyük, Turkey. *International Journal of Osteoarchaeology* 17: 235-252.
- Cowgill L. 2010. The ontogeny of Holocene and Late Pleistocene human postcranial strength. *American Journal of Physical Anthropology* 141: 16-37.
- Cowgill L, Eleacer C, Auerbach B, Temple D, Okazaki K. 2012. Developmental variation in ecogeographic body proportions. *American Journal of Physical Anthropology* 148:557-570.
- Cox M. 1996. *Life and death in Spitalfields 1700 to 1850*. Council for British Archaeology.
- Cucina A, Cantillo CP, Sosa TS, Tiesler V. 2011. Carious lesions and maize consumption among the Prehispanic Maya: An analysis of a coastal community in northern Yucatan. *American Journal of Physical Anthropology* 145: 560-567.
- Cunningham H, Viazzo PP. 1996. *Child labour in historical perspective: 1800-1985: Case studies from Europe, Japan, and Colombia*. UNICEF, United Nations Children's Fund, International Child Development Centre. Florence, Italy.
- Dakin CL, Wilson CA, Kallo I, Coen CW, Davies DC. 2008. Neonatal stimulation of 5-HT₂ receptors reduces androgen receptor expression in the rat anteroventral periventricular nucleus and sexually dimorphic preoptic area. *European Journal of Neuroscience* 27: 2473-2480.
- Daunton M. 2014. London's "Great Stink" and Victorian urban planning. *History Trails Victorian Britain*. bbc.co.uk/history.
- Da Silveira LE, Alves D, Lima NM, Alcântara A, Puig J. 2011. Population and railways in Portugal, 1801-1930. *Journal of Interdisciplinary History* XLII:1 29-52.

- Da Silveira LE, Alves D, Pinho M, Costa AC, Alcântara A. 2013. The evolution of population distribution on the Iberian Peninsula. *Scholarly Incursion* 46(3):157-174.
- Dawes H. 2014. *Catholic women's movements in liberal and fascist Italy*. Palgrave Macmillan.
- De Stefano GF, De Angelis F. 2009. Anthropometric growth pattern in Ethiopian infants and children: An evaluation based on different international growth references. *Collegium Antropologicum*. 33(3): 729-34.
- De Vito C, Saunders SR. 1990. A discriminant function analysis of deciduous teeth to determine sex. *Journal of Forensic Science* 35:845-858.
- DeWitte S, Wood J. 2008. Selectivity of Black Death mortality with respect to preexisting health. *Proceedings of the National Academy of Sciences, USA* .105: 1436-1441.
- DeWitte S. 2010. Sex differentials in frailty in Medieval England. *American Journal of Physical Anthropology* 143:285-297.
- DeWitte S. 2014. Differential survival among individuals with active and healed periosteal new bone formation. *International Journal of Paleopathology* 7: 38-44.
- DeWitte S. 2014. Health in post-Black Death London (1350-1538): Age patterns of periosteal new bone formation in a post-epidemic population. *American Journal of Physical Anthropology* 155: 260-267.
- DeWitte S. 2015 Setting the stage for Medieval plague: Pre-Black Death trends in survival and mortality. *American Journal of Physical Anthropology* 158: 441-451.
- DeWitte S, Hughes-Morey G, Bekvalac J, Karsten J. 2015. Wealth, health, and frailty in industrial-era London. *Annals of Human Biology* DOI: 10.3109/03014460.2015.1020873.
- DeWitte S, Stojanowski C. 2015. The osteological paradox 20 years later: Past perspectives, future directions. *Journal of Archaeological Research* 23:397-450.
- Ditch L, Rose J. 1972. A multivariate dental sexing technique. *American Journal of Physical Anthropology* 37:61-64.
- Douglas I, Hodgson R, Lawson N. 2002. Special section: European environmental history and ecological economics. *Ecological Economics* 41: 235-255.

- Ellison P, Peacock N, Lager C. 1986. Salivary progesterone and luteal function in two low-fertility populations in northeast Zaire. *Human Biology* 58:473-483.
- Engels F. 1958. *The condition of the working class in England*. Stanford University Press.
- Enlow DH. 1990. *Facial growth*, Third edition. Philadelphia; Saunders.
- Eveleth PB, Tanner JM. 1990. *Worldwide variation in human growth*. Cambridge University Press.
- Facchini F, Veschi S. 2004. Age determination on long bones in a skeletal subadults sample (b-12 years). *Collegium Antropologicum* 28(1): 89-98.
- Fildes VA. 1986. *Breasts, bottles, and babies: A history of infant feeding*. Edinburgh: Edinburgh University Press.
- Floud R, Wachter KW, Gregory A. 1990. *Health and history: Nutritional status in the United Kingdom 1750-1980*. Cambridge University Press.
- Forest MG, Cathiard AM, Bertrand JA. 1973. Evidence of testicular activity in early infancy. *Journal of Clinical Endocrinology & Metabolism* 37: 148.
- Forgacs D. 1990. *Italian culture in the Industrial Era: 1880-1980*. Manchester University Press.
- Formicola V. 1988. The male and the female in the Upper Paleolithic burials from the Grimaldi Caves (Liguria, Italy). *Bulletin du Musee d'Anthropologie prehistorique de Monaco* 31: 41-48.
- Frerichs R. 2016. *Deadly river: Cholera and cover-up in post-earthquake Haiti*. Cornell University Press.
- Friedman-Kasaba K. 1996. *Memories of migration 1870-1902*. Albany: State University of New York.
- Gapert R, Black S, Last J. 2009. Sex determination from the occipital condyle: Discriminant function analysis in an eighteenth and nineteenth century British sample. *American Journal of Physical Anthropology* 138: 384-394.
- Garvin HM, Ruff CB. 2012. Sexual dimorphism in skeletal browridge and chin morphologies determined using a new quantitative method. *American Journal of Physical Anthropology* 147:661-70.

- Gassler N, Peuschel T, Pankau R. 2000. Pediatric reference values of estradiol, testosterone, lutropin, follitropin, and prolactin. *Journal of Clinical Laboratory Analysis* 46: 553-560.
- Geber J, Murphy E. 2012. Scurvy in the Great Irish Famine: Evidence of vitamin C deficiency from a mid-19th century skeletal population. *American Journal of Physical Anthropology* 148: 512-524.
- Giner S. 1982. Political economy, legitimization and the state in Southern Europe. *British Journal of Sociology* 33(2): 172-199.
- Goikoetxea I, Leonard W, Mateos A, Martin-Gonzalez J, Rodriguez-Gomez G, Rodriguez J. 2012. Energy demands of growth in Neanderthal children. *American Journal of Physical Anthropology* 147:152.
- Gonzalez PN, Bernal V, Perez SI, Barrientos G. 2007. Analysis of dimorphic structures of the human pelvis: its implications for sex estimation in samples without reference collections. *Journal of Archaeological Science* 34:1720-1730.
- Gonzalez P, Perez S, Bernal V. 2010. Ontogeny of robusticity of craniofacial traits of modern humans: A study of South American populations. *American Journal of Physical Anthropology* 142:367-379.
- Gonzalez RA. 2011. Determination of sex from juvenile crania by means of discriminant function analysis. *Journal of Forensic Sciences* 57: 24-34.
- Goode-Null SK, Shujaa K, Rankin-Hill LM. 2004. Subadult growth and development. In: Blakey ML, Rankin-Hill LM, editors. *The New York African burial ground. Skeletal biology final report. Volume 1. The African Burial Ground Project.* Washington, D.C. Howard University.
- Goodman AH, Armelagos GJ, Rose JC. 1980. Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Human Biology* 52: 515-528.
- Goodman AH, Armelagos GJ. 1985. Factors affecting the distribution of enamel hypoplasias within the human permanent dentition. *American Journal of Physical Anthropology* 68:479-493.
- Goodman AH. 1989. Dental enamel hypoplasia in prehistoric populations. *Advances in Dental Research* 3: 265-271.
- Goodman AH. 1993. On the interpretation of health from skeletal remains. *Current Anthropology* 34: 281-288.

- Goodman AH. 1996. Early life stresses and adult health: Insights from dental enamel development. In: Henry CJ, Ulijaszek SJ, editors. *The backbone of history: Health and nutrition in the western hemisphere*. Cambridge University Press, Cambridge, pp. 163-182.
- Goodman AH, Lallo J, Armelagos GJ, Rose JC. 1984. Health changes at the Dickson Mounds, Illinois (AD 950-1300). In: Cohen MN, Armelagos GJ, editors, *Paleopathology at the origins of agriculture*. Academic Press, pp. 271-305.
- Goodman A, Leatherman T. 1998. *Building a new biocultural synthesis*. The University of Michigan Press.
- Goodman AH, Martin DL. 2002. Reconstructing health profiles from skeletal remains. In: Henry CJ, Ulijaszek SJ, editors. *The backbone of history: Health and nutrition in the western hemisphere*. Cambridge University Press, Cambridge, pp 11-60.
- Goose N, Honeyman K. 2013. *Childhood and child labour in Industrial England: Diversity and agency, 1750-1914*. Ashgate Publishing Company.
- Goulart P, Bedi AS. 2007. *A history of child labour in Portugal*. Working Paper No. 448. Institute of Social Studies.
- Grabiner MD. 1989. The elbow and radioulnar joints. In: Rasch PJ., editor. *Kinesiology and applied anatomy*. London: Lea and Febiger, pp 136-150.
- Graw M. 1999. Metric sex determination of the skull base. *Homo* 50(2): 101-106.
- Graw M, Wahl J, Ahlbrecht M. 2005. Course of the meatus acusticus internus as criterion for sex differentiation. *Forensic Sciences International* 147: 113-117.
- Greulich WW. 1951. The growth and developmental status of Guamanian school children in 1947. *American Journal of Physical Anthropology* 9:55-70.
- Guatelli-Steinberg D, Sciulli PW, Betsinger TK. 2008. Dental crown size and sex hormone concentrations: Another look at the development of sexual dimorphism. *American Journal of Physical Anthropology* 137:324-333.
- Guatelli-Steinberg D. 2009. Recent studies of dental development in neanderthals: implications for neanderthal life histories. *Evolutionary Anthropology* 18:9-20.

Gültekin T, Özer BK, Katayama K, Akin G. 2006. Age-related patterns of upper arm muscle and fat area in Turkish children and assessment of nutritional status. *International Journal of Anthropology* 21:231-239.

Haas JD. 1990. Mortality and morbidity consequences of variation in early child growth. In: Swedlund AC, Armelagos GJ., editors. *Disease in populations in transition: Anthropological and epidemiological perspectives*. New York: Bergen & Garvey, pp. 223-247.

Haas J. 1998. Fetal growth retardation. In: Ulijaszek SJ, Johnston FE, Preece MA, editors. *The Cambridge encyclopedia of human growth and development*. Cambridge University Press.

Hauspie C. 1994. Genetic variance in the pattern of the growth curve for height: a longitudinal analysis of male twins (Polish data). *Annals of Human Biology* 21:347-62.

Hens S, Rasseli E, Belcastro G. 2008. Age estimation from the human os coxa: A test on a documented Italian collection. *Journal of Forensic Sciences*. 53: 1040-1043.

Hierneaux J. 1985. Comparison of the shoulder-hip-width sexual dimorphism in sub-Saharan Africa and Europe. *Human Sexual Dimorphism* 191-206.

Hobbs S, McKechnie T, Lavalette M. 1999. *Child labor: A world history companion*. ABC-CLIO.

Holcomb S, Konigsberg L, Flin H. 2005. Alternative dental measurements: proposals and relationships with other measurements. *American Journal of Physical Anthropology* 97: 113-125.

Holland EJ. 2013. *Bring childhood health into focus: Incorporating survivors into standard methods of investigation*. PhD dissertation, Department of Anthropology, University of Toronto, Toronto.

Hoppa RD. 1992. Evaluating human skeletal growth: An Anglo-Saxon example. *International Journal of Osteoarchaeology* 2:275-288.

Hoppa RD. 2000. What to do with long bones: Toward a progressive palaeoauxology. *Anthropologie* 38:23-32.

Hoppa RD, Fitzgerald CM. 2005. *Human growth in the past: Studies from bones and teeth*. Cambridge University Press.

- Horrell S, Oxley D. 2012. Bringing home the bacon? Regional nutrition, stature, and gender in the industrial revolution. *Economic History Review* 65(4): 1354-1379.
- Hughes-Morey G. 2012. Body size and mortality in post-Medieval England. Ph.D. Dissertation. State University of New York, Albany. ProQuest LLC Publishing UMI 3516348.
- Hughes-Morey G. 2016. Interpreting adult stature in Industrial London. *American Journal of Physical Anthropology* 159:126-134.
- Humphrey L. 2000. Growth studies of past populations: An overview and an example. In: Cox M, Mays, editors. *Human osteology in archaeology and forensic science*. Cambridge University Press.
- Humphrey L, Bello S, Rousham E. 2012. Sex differences in infant mortality in Spitalfields, London, 1750-1839. *Journal of Biosocial Science* 44: 95-119.
- Humphries J. 2010. *Childhood and child labour in the British Industrial Revolution*. Cambridge University Press.
- Humphries J. 2013. The lure of aggregates and the pitfalls of the patriarchal perspective: A critique of the high wage economy interpretation of the British industrial revolution. *Economic History Review* 66(3): 693-714.
- Hupfel S. 2012. The Spitalfields Acts and the classics: Ricardo, J.S. Mill, Bowring, and Senior on the London silk industry (1823-1841). *European Journal of History of Economic Thought* 19:2 165-195.
- Husmann P, Samson D. 2011. In the eye of the beholder: Sex and race estimation using the human orbital aperture. *Journal of Forensic Sciences* 56: 1424-1429.
- Huss-Ashmore R, Goodman AH, and Armelagos GJ. 1982. Nutritional inference from paleopathology. *Advances in Archaeological Method and Theory* 5:395-474.
- Iscan M, Kedici P. 2003 Sexual variation in bucco-lingual dimensions in Turkish dentition. *Forensic Science International* 137:160-164.
- James W, Drasar B, Miller C. 1972. Physiological mechanism and pathogenesis of weanling diarrhea. *American Journal of Clinical Nutrition* 25: 564-571.
- Johnson S. 2006. *The ghost map: The story of London's most terrifying epidemic and how it changed science, cities, and the modern world*. Riverhead Books, New York.

- Johnston F. 2002. Social and economic influences on growth and secular trends. In: Noel Cameron, editor. *Human growth and development*. Academic Press.
- Johnston F, Bogin B, MacVean RB. 1984. A comparison of international standards versus local reference data for the triceps and subscapular skinfolds of Guatemalan children and youth. *Human Biology* 56: 157-171.
- Kalmey J, Rathbun T. 2006. Sex determination by discriminant function analysis of the petrous portion of the temporal bone. *Journal of Forensic Sciences* 41: 865-867.
- Kemkes-Grottenthaler A. 2005. The short die young: The inter-relationship between stature and longevity - evidence from skeletal remains. *American Journal of Physical Anthropology* 128: 340-347.
- Kertzer DI, Hogan DP. 1989. *Family, political economy, and demographic change: The transformation of life in Caslecchio, Italy, 1861-1921*. The University of Wisconsin Press.
- Klein SL. 2000. The effects of hormones on sex differences in infection: From genes to behavior. *Neuroscience Biobehavioral Review* 24: 627-638.
- Klüsener S, Ekamper P, Gruber S, van Poppel F, Solli A, Devos I, Gregory IN, Martí-Henneberg J, da Silveira LE. 2014. Spatial inequalities in infant survival at an early stage of the longevity revolution: A pan-European view across 5000+ regions and localities in 1910. *Demographic Research* 30(68): 1849-1864.
- Komlos J. 1993. The secular trend in the biological standard of living in the United Kingdom, 1730-1860. *The Economic History Review, New Series* 46(1): 115-144.
- Kondo S, Townsend GC. 2004. Sexual dimorphism in crown units of mandibular deciduous and permanent molars in Australian aborigines. *Homo* 55:53-64.
- Kondo S, Townsend GC, Yamada H. 2005. Sexual dimorphism of cusp dimensions in human maxillary molars. *American Journal of Physical Anthropology* 128:870-877.
- Krause E. 1999. *Natalism and nationalism: The political economy of love, labor, and low fertility in central Italy*. Ph.D. Dissertation. University of Arizona.
- Krause E. 2005. Toys and perfumes: Imploding Italy's population paradox and motherly myths. In: Douglas C., editor. *Barren states*. Oxford. 159-182.
- Krause E. 2007. Memory and meaning: Genealogy of a fertile protest. *Journal of Modern Italian Studies* 12(4): 406-416.

- Kuzawa C. 1998. Adipose tissue in human infancy and childhood: An evolutionary perspective. *Yearbook of Physical Anthropology* 41: 177-209.
- Kuzawa C. 2005. Fetal origins of developmental plasticity: Are fetal cues reliable predictors of future nutritional environments? *American Journal of Human Biology* 17:5-21.
- La Berge AEF. 2002. *Mission and method: The early nineteenth-century French public health movement*. Cambridge University Press.
- Laerd Statistics, Lund Research Ltd. 2013. statistics.laerd.com.
- Larsen CS. 1984. Health and disease in prehistoric Georgia: The transition to agriculture. In: Cohen MN., Armelagos GJ., editors. *Paleopathology at the origins of agriculture*. Academic Press, pp 367-392.
- Larsen CS. 1997. *Bioarchaeology: Interpreting behavior from the human skeleton*. Cambridge University Press.
- La Velle M. 1995. Natural selection and developmental sexual variation in the human pelvis. *American Journal of Physical Anthropology* 98: 59-72.
- Leatherman T, Goodman A. 2005. Coca-colonization of diets in the Yucatan. *Social Sciences & Medicine* 61: 833-846.
- Leatherman T, Goodman A, Stillman T. 2010. Changes in stature, weight, and nutritional status with tourism-based economic development in the Yucatan. *Economics and Human Biology* 8:153-158.
- Leatherman T, Jernigan K. 2015. Introduction: Biocultural contributions to the study of health disparities. *Annals of Anthropological Practice* 38(2): 171-186 ISSN: 2153-957X.
- Lewis M. 2002. Impact of industrialization: Comparative study of child health in four sites from medieval and postmedieval England (A.D. 850-1859). *American Journal of Physical Anthropology* 119:211-223.
- Lewis M. 2007. *The bioarchaeology of children: Perspectives from biological and forensic anthropology*. Cambridge University Press.
- Lieberman D, McBratney B, Krovitz G. 2006. The evolution and development of cranial form in *Homo Sapiens*. *Proceedings of the National Academy of Sciences of the United States of America*, 99: 1134-1139.

- Listi GA. 2010. The impact of racial metric variation in the os coxae on the morphological assessment of sex. *Journal of Forensic Sciences* 55: 1157-1161.
- Littleton J. 2011. Moving from the canary in the coalmine: Modeling childhood in Britain. In: Agarwal SC, Glencross BA, editors. *Social biology*. Wiley-Blackwell, Malden, MA, pp. 361-389.
- Loth SR, Henneberg M. 1996. Mandibular ramus flexure: A new morphologic indicator of sexual dimorphism in the human skeleton. *American Journal of Physical Anthropology* 99:473-485.
- Loth S, Henneberg M. 2001. Sexually dimorphic mandibular morphology in the first few years of life. *American Journal of Physical Anthropology* 115: 179-186.
- Maresh MM. 1955. Linear growth of long bones of extremities from infancy through adolescence. *American Journal of Diseases of Children* 89:725-742.
- Mariotti V, Zuppell M, Pedrosi ME, Bettuzzi M, Brancaccio R, Peccenini E, Morigi MP, Belcastro MG. 2015. Skeletal evidence of tuberculosis in a modern identified human skeletal collection (Certosa Cemetery, Bologna, Italy). *American Journal of Physical Anthropology* 00:00-00.
- Martin DL, Armelagos GJ, Goodman AH, Van Gerven DP. 1984. The effects of socioeconomic change in prehistoric Africa: Sudanese Nubia as a case study. In: Cohen MN, Armelagos GJ, editors. *Paleopathology at the origins of agriculture*. Academic Press, pp 193-214.
- Martorell R, Habicht J. 1986. Growth in early childhood in developing countries. In: Falkner F, Tanner JM, editors. *Human growth: A comprehensive treatise* 2nd edition. New York: Plenum Press.
- Matos V, Santos AL. 2006. On the trail of pulmonary tuberculosis based on rib lesions; Results from the human identified skeletal collection from the Museo Bocage (Lisbon, Portugal). *American Journal of Physical Anthropology* 130: 190-200.
- Mays S, Brickley M, Ives R. 2006. Skeletal manifestations of rickets in infants and young children in a historic population from England. *American Journal of Physical Anthropology* 129:362-374.
- Mays S, Brickley M, Ives R. 2008. Growth in an English population from the Industrial Revolution. *American Journal of Physical Anthropology* 136:85-92.

- Mays S, Brickley M, Ives R. 2009. Growth and vitamin D deficiency in a population from 19th century Birmingham, England. *Journal of Osteoarchaeology* 19: 406-415.
- Mays S, Ives R, Brickley M. 2009b. The effects of socio-economic status on endochondral and appositional bone growth, and acquisition of cortical bone in children from 19th century Birmingham, England. *American Journal of Physical Anthropology* 140:410-416.
- McCummon RW. 1970. Human growth and development. Charles C. Thomas, pp 155-200.
- McIlvaine BK. 2013. Implications of reappraising the iron-deficiency anemia hypothesis. *International Journal of Osteoarchaeology* 25: 997-1000.
- Mensforth RP, Lovejoy OC, Lallo JW, Armelagos GJ. 1978. The role of constitutional factors, diet and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. *Medical Anthropology* 2:1-59.
- Milella M, Belcastro MG, Zollikofer C, Mariotti V. 2012. The effect of age, sex, and physical activity on enthesal morphology in a contemporary Italian skeletal collection. *American Journal of Physical Anthropology* 148: 379-388.
- Mittler D, Sheridan S. 1992. Sex determination in subadults using auricular surface morphology: a forensic perspective. *Journal of Forensic Sciences* 37: 1068-1075.
- Molleson T, Cox M. 1993. The Spitalfields project: Volume 2: The anthropology. CBA Research Report 86 Council for British Archaeology.
- Molleson T, Cruse K. 1998. Some sexually dimorphic features of the human juvenile skull and their value in sex determination in immature skeletal remains. *Journal of Archaeological Sciences* 25:719-728.
- More C. 2000. *Understanding the Industrial Revolution*. London. New York. Routledge.
- Murail P, Bruzek F, Cunha H. 2005. DSP: A tool for probabilistic sex diagnosis using worldwide variability in hip-bone measurements. *Bulletins et mémoires de la société d'Anthropologie de Paris* 17: 3-4.
- Murphy A. 1994. Sex determination of prehistoric New Zealand Polynesian clavicles. *New Zealand Journal of Archaeology* 16: 85-91.

- Nitsch EK, Humphrey LT, Hedges REM. 2011. Using stable isotope analysis to examine the effect of economic change on breastfeeding practices in Spitalfields, London, UK. *American Journal of Physical Anthropology* 146:619-628.
- Noren A, Lynnerup N, Czarnetzki A, Graw M. 2005. Lateral angle: A method for sexing using the petrous bone. *American Journal of Physical Anthropology* 128:318-323.
- Norgan NG. 2002. Nutrition and growth. In: Cameron, N , editor. *Human growth and development*. Academic Press.
- Norušis MJ. 1994. *SPSS Professional statistics 6.1*. SPSS Inc.
- Ogden AR, Pinhasi R, White WJ. 2007. Gross enamel hypoplasias in molars from subadults in a 16th – 18th Century London graveyard. *American Journal of Physical Anthropology* 133: 957-966.
- Ogden AR. 2008. Advances in the paleopathology of teeth and jaws. In: Pinhasi R, Mays S, editors. *Advances in Human Paleopathology*. John Wiley & Sons.
- Oliveira V, Pinho P. 2010. City profile: Lisbon. *Cities* 27:405-419.
- Ortner DJ. 1998. Male-female immune reactivity and its implications for interpreting evidence in human skeletal paleopathology. In: Grauer AL, Stuart-Macadam P, editors. *Sex and gender in paleopathological perspective*. Cambridge University Press, pp 79-92.
- Ortner DJ. 2003. *Identification of pathological conditions in human skeletal remains*. Academic Press. San Diego, CA.
- Ortner DJ. 2009. Issues in paleopathology and possible strategies for dealing with them. *Anthropologischer Anzeiger* 67:323-340.
- Ortner DJ, Kimmerle EH, Diez M. 1999. Probable evidence of scurvy in subadults from archaeological sites in Peru. *American Journal of Physical Anthropology* 108:321-331.
- Oxenham MF, Cavill I. 2010. Porotic hyperostosis and cribra orbitalia: The erythropoietic response to iron-deficiency anaemia. *Anthropological Sciences* 118: 199-200.
- Oyhenart E. 2006. Sexual dimorphism in schoolchildren and its relation with nutritional status. *Anthropologie*. 44:263-268.
- Paraskevas G, Papadopoulos A, Papaziogas B, Spanidou S, Argiriadou H, Gigis J. 2004. Study of the carrying angle of the human elbow joint in full extension: A morphometric analysis. *Surgical & Radiological Anatomy* 26: 19-23.

- Parkes C. 2012. *Children's literature and capitalism: Fictions of social mobility in Britain, 1850-1914*. New York: Palgrave Macmillan.
- Patriquin ML, Loth SR, Steyn M. 2003. Sexually dimorphic pelvic morphology in South African whites and blacks. *Homo* 53:255-62.
- Perry MA. 2014. Tracking the second epidemiological transition using bioarchaeological data on infant morbidity and mortality. In: Zuckerman MK, editor. *Modern environments and human health: Revisiting the second epidemiological transition*. Wiley Blackwell, Hoboken, NJ, pp. 225-241.
- Pettit P, Bailey R. 2000. *Neanderthals on the edge*. Oxbow Books.
- Pina-Cabral J. 1992. Family and neighborhood in Portugal today. In: Herr R, editor. *The new Portugal: Democracy and Europe*. University of California at Berkeley.
- Pinhasi R, Teschler-Nicola M, Knaus A, Shaw P. 2005. Cross-population analysis of the growth of long bones and the ox coxae of three early medieval Austrian populations. *American Journal of Human Biology* 17:470-480.
- Pooley CG, Turnbull J. 1996. Migration and mobility in Britain from the eighteenth to the twentieth centuries. *Local Population Studies* 50-71.
- Prader A, Tanner JM, Von Harnack G. 1963. Catch-up growth following illness or starvation. *Journal of Paediatrics* 62:645-659.
- Preston SH. 1977. Mortality trends. *Annual Review of Sociology* 3:163-178.
- Pucciarelli HM, Carnese FR, Pinotti LV, Guimarey LM, Goicoechea AS. 1993. Sexual dimorphism in schoolchildren of the Villa IAPI neighborhood (Quilmes, Buenos Aires, Argentina). *American Journal of Physical Anthropology* 92: 165-172.
- Quigley C. 2002. The postnatal gonadotropin and sex steroid surge – insights from the androgen insensitivity syndrome. *Journal of Clinical Endocrinology & Metabolism* 87: 24-28.
- Rahikainen M. 2004. *Centuries of child labor: European experiences from the seventeenth to the twentieth century*. Economic History Association.
- Redfern R, DeWitte S. 2011. Status and health in Roman Dorset: The effect of status on risk of mortality in post-conquest populations. *American Journal of Physical Anthropology* 146: 197-208.

- Reid DJ, Dean MC. 2000. Brief communication: The timing of linear hypoplasias on human anterior teeth. *American Journal of Physical Anthropology* 113: 135-139.
- Reis J. 2004. Human capital and industrialization: The case of a latecomer - Portugal, 1890. In: Ljungberg J, Smits JP, editors. *Technology and human capital in historical perspective*. Palgrave Macmillan.
- Reis J. 2009. Urban premium or urban penalty? The case of Lisbon, 1840-1912. *Historia Agraria* 47:69-94.
- Relethfod JH, Hodges DC. 1985. A statistical test for differences in sexual dimorphism between populations. *American Journal of Physical Anthropology* 66:55-61.
- Ridley J. 2002. Sex estimation of fetal and infant remains based on metric and morphognostic analyses. Louisiana State University and Agricultural and Mechanical College, Master's Thesis.
- Rissech C, Malgosa A. 2005. Ilium growth study: Applicability in sex and age diagnosis. *Forensic Sciences International* 147: 165-174.
- Roberts CA, Manchester K. 2005. *The archaeology of disease*. Cornell University Press.
- Robbins Schug G. 2011. *Bioarchaeology and climate change: A view from South Asian prehistory*, University Press of FA, Gainesville.
- Roede J, van't Hof MA. 1978. Errors associated with the estimation of intersection of male and female growth curves. *Human Biology* 50:411-23.
- Rogers T. 2009. Sex determination of adolescent skeletons. *American Journal of Physical Anthropology* 140:143-148.
- Roksandic M, Armstrong S. 2011. Using the life history model to set the stages of growth and senescence in bioarchaeology and paleodemography. *American Journal of Physical Anthropology* 145: 337-347.
- Rowland M. 1983. Epidemiology of childhood diarrhea in the Gambia. In: Scrimshaw NS, Vallerstein MB, editors. *Nutrition policy implementation: Issues and experience*. New York: Plenum Press.
- Ruff C, Walker A, Trinkaus E. 1994. Postcranial robusticity in *Homo*. III: Ontogeny. *American Journal of Physical Anthropology* 93:35-54.

- Ruff C. 2003. Growth in bone strength, body size, and muscle size in a juvenile longitudinal sample. *Bone* 33:317-329.
- Ruff C. 2007. Body size prediction from juvenile skeletal remains. *American Journal of Physical Anthropology* 133:698-716.
- Ruff CB, Garofalo E, Holmes MA. 2013. Interpreting skeletal growth in the past from a functional and physiological perspective. *American Journal of Physical Anthropology* 150: 29-37.
- Ruff C, Holt BM, Niskanen M, Sldek V, Berner M, Grofalo E, Garvin HM, Hora M, Maijanen H, Niinimaki S, Salo K, Schuplerova E, Tompkins D. 2012. Stature and body mass estimation from skeletal remains in the European Holocene. *American Journal of Physical Anthropology* 148:601-617.
- Saunders SR, Hoppa R. 1993. Growth deficit in survivors and non-survivors: Biological mortality bias in subadult skeletal samples. *Yearbook of Physical Anthropology* 36: 127-51.
- Saunders SR. 2000. Subadult skeletons and growth-related studies. In: Katzenberg MA and Saunders SR., editors. *Biological anthropology of the human skeleton*. New York: Wiley-Liss.
- Schell L. 1989. Community health assessment through physical anthropology: Auological epidemiology. *Human Origins* 45: 321-327.
- Schell L. 1997. Culture as a stressor: A revised model of biocultural interaction. *American Journal of Physical Anthropology* 102:67-77.
- Schell L, Knutsen K. 2002. Environmental effects on growth. In: Cameron, N., editor. *Human growth and development*. Academic Press.
- Schell L, Magnus PD. 2007. Is there an elephant in the room? Addressing rival approaches to the interpretation of growth perturbations and small size. *American Journal of Human Biology* 19:606-614.
- Scheuer L, Black S. 2000. *Developmental juvenile osteology*. Elsevier Ltd.
- Schillaci MA, Nikitovic D, Akins NJ, Tripp L, Palkovich AM. 2011. Infant and juvenile growth in ancestral Pueblo Indians. *American Journal of Physical Anthropology* 145:318-326.

- Schillaci MA, Nikitovic D, Akins NJ, Tripp L, Palkovich AM. 2011. Infant and juvenile growth in ancestral Pueblo Indians. *American Journal of Physical Anthropology* 145:318-326.
- Schutkowski H. 1987. Sex determination of fetal and neonate skeletons by means of discriminant analysis. *International Journal of Anthropology* 4: 347-352.
- Schutkowski H. 1993. Sex determination of infant and juvenile skeletons: I. morphognostic features. *American Journal of Physical Anthropology* 90: 199-205.
- Schwartz J. 2007. *Skeleton keys: An introduction to human skeletal morphology, development, and analysis* 2nd Ed. Oxford University Press.
- Schwartz LD. 1985. The standard of living in the long run: London 1700-1860. *Economic History Review* 38: 24-41.
- Schwartz R, Gregory I, Thevenin T. 2011. Spatial history: Railways, uneven development, and population change in France and Great Britain, 1850-1914. *Journal of Interdisciplinary History* 42(1): 53-88.
- Skerry TM. 1994. The effects of the inflammatory response on bone growth. *European Journal of Clinical Nutrition* 48:190-197.
- Sofaer J. 2006. *The body as material culture: A theoretical osteoarchaeology*. Cambridge University Press.
- Sorenson M. 2000. *Gender archaeology*. Wiley & Sons, Inc.
- Spielmann K. 1989. A review: Dietary restrictions on hunter-gatherer women and the implications for fertility and infant mortality. *Human Ecology* 17: 321-345.
- Statistics and probability dictionary. 2016. StatTrek.com.
- Steckel RH. 2005. Young adult mortality following severe physiological stress in childhood: Skeletal evidence. *Economic Human Biology* 3: 314-328.
- Steinberg MW. 1999. *Fighting words: Working-class formation, collective action, and discourse in early nineteenth-century England*. Cornell University Press.
- Steyn M, Pretorius E, Hutten L. 2004. Geometric morphometric analysis of the greater sciatic notch in South Africans. *Homo* 54:197-206.

- Stini A. 1969. Nutritional stress and growth: Sex difference in adaptive response (Colombia). *American Journal of Physical Anthropology* 31: 417-26.
- Stini A. 1972. Reduced sexual dimorphism in upper arm muscle circumference associated with protein-deficient diet in a south American population. *American Journal of Physical Anthropology* 36:341-51.
- Stini WA. 1978. Human growth as an adaptive strategy. *Colloquia in Anthropology* 2:47-62.
- Stini WA. 1980. Human adaptability to nutritional stress. *Nutrition, food, and man* 124-140.
- Stinson S. 1985. Sex differences in environmental sensitivity during growth and development. *American Journal of Physical Anthropology* 28:123-147.
- Stinson S. 1992. Nutritional Adaptation. *Annual Review of Anthropology* 21: 143-170.
- Stinson S. 2000. Growth variation: Biological and cultural factors. In: Stinson S, Bogin B, Huss-Ashmore R, O'Rourke D, editors. *Human Biology: An evolutionary and biocultural perspective*. Wiley-Liss.
- Stolz Y, Baten J, Reis J. 2013. Portuguese living standards, 1720-1980, in European comparison: Heights, income, and human capital. *Economic History Review* 66(2): 545-578.
- Storey R. 1997. Individual frailty, children of privilege, and stress in Late Classic Copán. In: Whittington S, Reed D, editors. *Bones of the Maya*. University of Alabama Press, Tuscaloosa, pp. 116-126.
- Stuart-Macadam PL. 1991. Anemia in Roman Britain: Poundbury Camp. In: Bush H, Zvelebil M, editors. *Health in past societies: Biocultural interpretations of human skeletal remains in archaeological contexts*. Oxford: British Archaeological Research International Series. p. 101-113.
- Sutter R. 2003. Nonmetric subadult skeletal sexing traits. I. A blind test of the accuracy of eight previously proposed methods using prehistoric known-sex mummies from northern Chile. *Journal of Forensic Sciences* 48: 927-935.
- Szreter S. 1988. The importance of social intervention in Britain's mortality decline c. 1850-1914: A re-interpretation of the role of public health. *Social History of Medicine* 1:1-37.

- Szreter S. 2004. Industrialization and health. *British Medical Bulletin* 69:75-86.
- Tanner J. 1982. The potential of auxological data for monitoring economic and social well-being. *Social Science History* 6(4): 571-581.
- Tanner J. 1989. *Foetus into man*, 2nd edition. Castlemead Publications.
- Teranishi H, Nakagawa H, Marmot M. 2001. Social class difference in catch up growth in a national British cohort. *Archives of Disease in Childhood*. 84: 218.
- Thompson EP. 1963. *Making of the English working class*. Pantheon Books.
- Vaccaro R. 1980. Industrialization in Spain and Italy (1860-1914). *Journal of European Economic History* 9(3):709-751.
- VanGerven DP, Armelagos GJ. 1983. "Farewell to Palaeodemography?" Rumours of its death have been greatly exaggerated. *Journal of Human Evolution* 12:353-360.
- Veroni A, Nikitovic D, Schillaci M. 2010. Brief communication: Sexual dimorphism of the juvenile basicranium. *American Journal of Physical Anthropology* 141: 147-151.
- Viciano J, Aleman I, D'Anastasio R, Capasso L, Botella MC. 2011. Odontometric sex discrimination in the Herculaneum sample (79 AD, Naples, Italy), with application to juveniles. *American Journal of Physical Anthropology* 145: 97-106.
- Vlak D, Roksandic M, Schillaci MA. 2008. Greater sciatic notch as a sex indicator in juveniles. *American Journal of Physical Anthropology* 137: 309-315.
- Voland E, Dunbar RIM, Engel C, Stephan P. 1997. Population increase and sex-biased parental investment in humans: evidence from 18th and 19th century Germany. *Current Anthropology* 38: 129-135.
- Wadhwah PD, Buss C, Entringer S, Swanson JM. 2009. Developmental origins of health and disease: Brief history of the approach and current focus on epigenetic mechanisms. *NIH Public Access* 27: 358-368.
- Wahl J, Graw M. 2001. Metric sex differentiation of the pars petrosa ossis temporalis. *Intl Journal of Legal Medicine* 114: 215-23.
- Waldron I. 1984. The role of genetic and biological factors in sex differences in mortality. In: Lopez AD, Ruzicka LT, editors. *Sex differentials in mortality: Trends, determinants, and consequences*. Canberra: Department of Demography, Australian National University, pp 141-164.

- Walker PL, Bathurst RR, Richman R, Gjerdrum T, Andrushko VA. 2009. The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis. *American Journal of Physical Anthropology* 139: 109-125.
- Wapler U, Crubézy E, and Schultz M. 2004. Is cribra orbitalia synonymous with anemia? Analysis and interpretation of cranial pathology in Sudan. *American Journal of Physical Anthropology* 123: 333-339.
- Watts R. 2013. Childhood development and adult longevity in an archaeological population from Barton-upon-Humber, Lincolnshire, England. *International Journal of Paleopathology* 3:95-104.
- Weaver D. 1980. Sex differences in the ilia of a known sex and age sample of fetal and infant skeletons. *American Journal of Physical Anthropology* 52: 191-195.
- Webster RA. 1975. *Industrial Imperialism in Italy 1908-1915*. University of California Press.
- White T. 2005. *The human bone manual*. Elsevier Science.
- Williams B, Rogers T. 2006. Evaluating the accuracy and precision of cranial morphological traits for sex determination. *Journal of Forensic Sciences* 51: 729-735.
- Winter J, Hughes I, Reyes F, Faiman C. 1976. Pituitary-gonadal relations in infancy, Part 2: Patterns of serum gonadal steroid concentrations in many from birth to two years of age. *Journal of Clinical Endocrinology & Metabolism* 42: 679-686.
- Wohl AS. 1983. *Endangered lives: Public health in Victorian Britain*. Littlehampton Book Services Ltd. 1st UK Edition.
- Wolfe LD, Gray JP. 1982. Subsistence practices and human sexual dimorphism of stature. *Journal of Human Evolution* 11:575-580.
- Wood J, Milner G, Harpending H, Weiss K, Cohen M, Eisenberg L, Hutchinson D, Jankauskas R, Cesnys G, Katzenberg A, Lukacs J, McGrath J, Roth E, Ubelaker D, Wilkinson R. 1992. The osteological paradox: Problems of inferring prehistoric health from skeletal samples. *Current Anthropology* 33:343-370.
- Woods R, Woodward J. 1984. *Urban disease and mortality in nineteenth-century England*. London: Batsford Academic and Educational.

World Health Organization Working Group. 1986. Use and interpretation of anthropometric indicators of nutritional status. WHO Working Group. Bulletin World Health Organization 64(6): 929-41.

Zakrzewski SR. 2003. Variation in ancient Egyptian stature and body proportions. American Journal of Physical Anthropology 121: 219-222.