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Physiological Stress, Bone Growth and Development in Imperial Rome

By

Patrick Denis Beauchesne

A dissertation submitted in partial satisfaction of the  
requirements for the degree of  
Doctor of Philosophy

in

Anthropology

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

Associate Professor Sabrina Agarwal, Chair

Professor Laurie Wilkie

Professor Sharmila Majumdar

Spring 2012





## **Abstract**

Physiological Stress, Bone Growth and Development in Imperial Rome

by

Patrick Denis Beauchesne

Doctor of Philosophy in Anthropology

University of California, Berkeley

Associate Professor Sabrina Agarwal, Chair

This study investigates bone maintenance and loss within a life course perspective in the Imperial Roman population of Velia. The use of biocultural factors, such as diet, reproductive history and physical activity are emphasized in the interpretive process. A total of 135 individuals were examined. Bone loss is assessed using three methods: radiogrammetry of the 2<sup>nd</sup> metacarpal, rib cortical histomorphometry, and the analysis of trabecular architecture in L4 vertebrae. Physiological stress in adults is explored using porotic hyperostosis, cribra orbitalia and periostitis. Stress during growth and development is investigated through dental enamel hypoplasias, vertebral neural canal sizes and skeletal growth profiles of juvenile skeletons. Analyses of bone of maintenance and loss reveal that bone loss in the Velia population did not follow similar patterns to modern communities. The most meaningful difference was that no sex differences were observed between males and females for all three measures of bone maintenance and loss. In addition, the analyses of radiogrammetry, histomorphometry and trabecular architecture all support a hypothesis that strenuous physical activity over the life course helped mediate bone loss with age, particularly in females. The reproductive history of females in the Roman period is also hypothesized to have played a protective role in mediating bone loss. Physiological stress was common in the juvenile and adult stages of life, but this stress did not seemingly affect bone remodeling and loss with age or between the sexes. The approaches used in this dissertation advance bioarchaeological theory by implementing a life course perspective that emphasize developmental plasticity to investigating bone loss. This study also contributes methodologically by demonstrating the importance of using multiple lines of evidence when exploring bone loss in past populations.

## **Dedication**

To my family and my wife Trisha. Without their love, patience and support this work would not have been possible.

*The purpose of anthropology is to make the world safe for human differences.*

- Ruth Benedict

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## List of Abbreviations

### Radiogrammetry

TW – Total Width

MW – Medullary Width

CT – Cortical Thickness

CI – Cortical Index

### Rib Cortical Histomorphometry

On.Ar – Mean Osteon Area

OPD – Osteon Population Density

$\bar{U}_{RC}$  – Mean Annual Activation Frequency

$V_{f,r,t}$  – Mean Annual Bone Formation Rate

$_{net}V_{f,r,t}$  – Net Osteonal Remodeling

MWT – Mean Wall Thickness

Tt.Ar – Total Area

Ct.Ar – Cortical Area

En.Ar – Endosteal Area

Ct.Ar/Tt.Ar – Percent (relative) Cortical Area

### Trabecular Architecture

BV/TV – Bone Volume

Tb.N – Trabecular Number

Tb.Sp – Trabecular Spacing

Tb.Th - Trabecular Thickness

ConnD – Connective Density

DA – Degree of Anisotropy

SMI – Structural Model Index

*Stress Markers*

VNC – Vertebral Neural Canal

AP – Anterior-Posterior distance of VNC

ML – Medial-Lateral distance of VNC

ABH - Anterior body height

PBH - Posterior body height

DEH – Dental Enamel Hypoplasia

SGP – Skeletal Growth Profile

## Acknowledgements

I would not have been able to complete this dissertation without the help of numerous academic and personal mentors. I must first thank Dr. Sabrina Agarwal, whose friendship, leadership and guidance has been invaluable. The time and effort she devotes to her students is exemplary. I can think of no better graduate supervisor. I look forward to years of collaborative work with her. I must thank my mother next, who has supported me in my academic endeavors from the beginning without hesitation. Thank you for believing in me. I need to also thank my grandmother and aunt, whose support has also been equally inspiring. My wife, Trisha, has also been extraordinarily understanding and supportive. There are no words for what her support has meant to me.

I would also like to thank the faculty in the Department of Anthropology at UC Berkeley. My graduate career in the department has been enriched by the rewarding classes, lectures, and discussions I've had over the years. My dissertation committee deserves special mention. The last few months of writing were quite hectic and their patience and understanding was greatly appreciated. Their comments proved invaluable and made this work better.

None of this research would have been possible without the collaboration and assistance of Dr. Luca Bondioli and Dr. Alessandra Sperduti at the Pigorini Museum in Rome. They kindly allowed me to work with the Velia material and were tremendously supportive throughout the whole process. They have been wonderful colleagues and I enjoy the prospect of continuing our work on the Velia material in the coming months and years.

My cohort was wonderful as well; I will always look back on "Beer Nights" with great fondness. I would also like to thank the friends I've had over the years who have cheered me on and helped me personally or academically. In particular, I'd like to thank Al and Mira Cross, Sandra Wheeler, and Lana Williams.

Finally, I'd like to thank Liam Reidy and Dr. Roger Byrne for helping with the radiogrammetry. In addition, Andrew Burghardt was indispensable in helping me with the pQCT and Dr. Sam Stout was very patient and helpful with my histomorphometry questions. I would also like to thank those who have funded my work during my dissertation. In particular, the Social Sciences and Humanities Research Council of Canada was tremendously supportive. The Archaeological Research Facility Stahl grant was also invaluable, as was my summer research grant from the Graduate Division. Finally, to everyone who has supported and helped me over the years, thank you.

# Curriculum Vitae

## Education

**PhD - UC Berkeley, Anthropology – May 2012**

*Thesis:* A Life Course Approach to Measuring Bone Health in Archaeological Populations

*Committee:* Dr. Sabrina Agarwal (chair); Dr. Laurie Wilkie, Dr. Sharmila Majumdar

**M.A - University of Western Ontario, Anthropology — 2003 - 2005**

*Thesis:* Protein Consumption and Bone Health in Ancient Peru: A Histomorphometric Perspective

*Committee:* Dr. Andrew Nelson (Chair); Dr. Christine White; Dr. Michael Spence

**B.A - McMaster University, Anthropology — 2000 - 2003**

## Research Interests

Bioarchaeology; Biocultural Theory/Methodology; Skeletal Biology; Growth and Development; Bone Histology; Bone Imaging (pQCT and  $\mu$ CT); Bone Metabolic Disorders; Gender and Health

## Publications

### Peer Reviewed Articles/Book Chapters

Beauchesne, P., Agarwal, SC. 2011. Age-related cortical bone maintenance and loss in an Imperial Roman population. *International Journal of Osteoarchaeology* July 2011. In press.

Agarwal SC, Beauchesne P. 2011. It is Not Carved in Bone: Development and Plasticity of the Aged Skeleton. In *Social Bioarchaeology*, Agarwal SC and Glencross B (eds.). Wiley-Blackwell: New York; 312-332.

Agarwal, Sabrina; Glencross, Bonnie; & Beauchesne, Patrick. (2011). Bone Growth, Maintenance and Loss in the Neolithic Community of Çatalhöyük, Turkey: Preliminary Results. UC Berkeley: Archaeological Research Facility. Retrieved from <http://escholarship.org/uc/item/9m13784c>

Agarwal, S.C. Glencross, B., and Beauchesne, P. “Bone maintenance and fragility” (Human Remains: Health and Diet in Neolithic Çatalhöyük) *Submitted to:* Hodder, I (ed). Çatalhöyük, *to be published* with Cotsen Institute of Archaeology, Cotsen Institute Press, LA., pp xx

Beauchesne, P., Colquhoun, I., Cross, A., Longstaffe, F., Marciano, L., Metcalfe, J., Nelson, A.J., Pawlowski, A., Wheeler, S., White, C and L. Williams. 2008. The Lady Hudson Project. *Proceedings of the World Congress on Mummy Studies* 637- 640.

Beauchesne, P. and Saunders, S. 2006. A test of the revised Frost's rapid manual method for the preparation of bone thin sections. *International Journal of Osteoarchaeology* 16 (1): 82-87.

### **Conference Presentations**

Beauchesne, P. Agarwal, S.C. and Bondioli, L. 2011. Bone growth and loss in a Roman population using a multi-method approach. Conference abstracts, *American Journal of Physical Anthropology* 144 (S52): 84-85.

Beauchesne, P. Agarwal, S.C. and Bondioli, L. 2010. A Developmental Perspective on Metacarpal Radiogrammetry and Trabecular Architecture from an Imperial Roman Skeletal Population. Poster Presentation, American Society for Bone and Mineral Research annual meetings, Toronto, On, Canada.

Beauchesne, P., Agarwal, S.C., and Bondioli, L. 2009. Metacarpal Radiogrammetry at Velia: A Growth and Development Perspective. Podium Presentation, Canadian Association of Physical Anthropology annual meetings, Vancouver, BC, Canada.

Agarwal, S.C and Beauchesne, P. 2008. Age and sex-related changes in trabecular architecture over the life cycle in past populations. Conference Abstracts, *American Journal of Physical Anthropology* 135 (s46): 57-58.

Lipps, A.N., Agarwal, S.C., Beauchesne, P., and Hamada, Y. 2008. Cortical bone remodeling and trabecular architecture in Japanese macaques with degenerative joint disease. Conference Abstracts, *American Journal of Physical Anthropology* 135: (s46): 141.

Agarwal, S.C., Beauchesne, P., Burghart, A., Hamada, Y., Majumdar, S. 2007. Age-Related Changes in Vertebral Trabecular Architecture in Female Japanese Macaques. Poster Presentation, American Association of Physical Anthropologists annual meetings; Philadelphia, Pennsylvania.

Wheeler, S., Beauchesne, P., and Molto, E. 2005. Broken Bones: A possible case of child abuse from ancient Egypt. Podium Presentation, Paleopathology Association annual meetings, Milwaukee, Wisconsin.

Gardner, J., Beauchesne, P., Spence, M. 2005. The Identification of Paget's Disease in a Prehistoric Specimen from Ontario, Canada. Poster Presentation, Paleopathology Association annual meetings, Milwaukee, Wisconsin.



## **Teaching Experience**

### **STANFORD UNIVERSITY - Department of Anthropology**

*Social Bioarchaeology and the Reconstruction of Life from the Skeleton* (Bioarchaeological Method and Theory; upper division course) Winter 2012

### **UC BERKELEY - Department of Anthropology**

#### ***Instructor***

*Introduction to Skeletal Biology and Bioarchaeology* (upper division course), Summer 2011

*Reconstruction of Life from the Skeleton* (Bioarchaeological Method and Theory; upper division course) Summer 2010

#### ***Teaching Assistant/Graduate Student Instructor***

*The Archaeology of Health and Disease* - Spring 2011

*Introduction to Biological Anthropology* - Spring 2007, 2010

*Introduction to Skeletal Biology and Bioarchaeology* - Spring 2006 and 2008

*Primate Anatomy* (reader) - Fall 2007

### **UNIVERSITY OF WESTERN ONTARIO - Department of Anthropology**

#### ***Teaching Assistant/Graduate Student Instructor***

*Introduction to Archaeology and Biological Anthropology* - Winter 2004 and 2005

*Principles of Archaeology* - Fall 2004

*Biological Anthropology* (2nd year lab course) - Fall 2003

## **Invited Lectures**

**UC San Francisco** - Musculoskeletal Quantitative Imaging Research Group, Department of Radiology and Biomedical Imaging. *Guest lecture topic* "A Developmental Perspective on Metacarpal Radiogrammetry and Trabecular Architecture from an Imperial Roman Skeletal Population". 2010.

**UC Berkeley** - Course: The Archaeology of Sex and Gender with Prof. Rosemary Joyce - *Guest lecture topic* "Bioarchaeology: Sex and Gender Determination from Archaeological Skeletons". 2007.

## **Undergraduate Outreach**

**UC Berkeley.** 2006-2009. Directly trained and mentored 7 undergraduate students under the university's Undergraduate Research Assistant Program (URAP) directive. The URAP students were given extensive training in various laboratory procedures related to paleo-histological and computed imaging analysis and then given real responsibilities in assisting with three major ongoing research projects in the department of anthropology's Skeletal Biology Laboratory.

## **Awards/Fellowships**

### **Outstanding Graduate Student Instructor Award - 2011**

**Social Science and Humanities Research Council of Canada (SSHRC)**  
Doctoral Fellowship, 2005 – 2009

**UC Berkeley Archaeological Research Facility Stahl Grant**  
Graduate Research Assistance Grant, 2007-2008

**UC Berkeley Graduate Division Summer Research Grant**  
Graduate Research Grant, 2011

**Lowie-Olson Fellowship**  
Graduate Research Assistance Fellowship 2005-2009

**Ontario Graduate Scholarship**  
Doctoral Fellowship, 2005 (declined for SSHRC)

**University of Western Ontario Special University Scholarship**  
**M.A. fellowship, 2003 - 2004**

## **Field/Laboratory Experience**

*Manager, Skeletal Biology Laboratory, Department of Anthropology, UC Berkeley 2006-present*  
Management duties include: purchasing, supervising undergraduate student assistants, equipment upkeep, organization of lab materials, safety protocols, and training graduate and undergraduate students on lab equipment.

*UC, San Francisco, Department of Radiology*  
2006 to 2010  
Gained experience with high resolution peripheral computed tomography (HR-pQCT).

*Luigi Pigorini National Museum of Prehistory and Ethnography in Rome, Italy*  
2006 to 2011  
Obtained experience working with numerous colleagues on museum collections to develop and advance a large research project.

*Archaeological Site of Çatalhöyük, Turkey*

2007 and 2008

Worked with leading scholars on a multinational excavation and research project. Participated in both excavation and laboratory based research.

*Archaeological Site of Farfan, North Coast, Peru*

2004

Participated heavily in burial excavations and laboratory work that involved cleaning and inventorying of skeletal material, as well as multiple methods of skeletal analysis.

*Skeletal Histology Lab, Department of Anthropology, McMaster University*

2003

Developed an improved method for processing archaeological and forensic samples for histological analysis, leading to a publication in the International Journal of Osteoarchaeology. Also assisted doctoral candidates in producing histological slides for their research projects.

## Chapter 1 - Introduction

Bone loss and osteoporosis is a growing medical and social problem in Western societies, with 44 million people affected in the United States alone, and over 200 million affected worldwide (Reginster and Burlet, 2006). With increasingly aging populations in the West, the prevalence of osteoporosis is estimated to increase dramatically, placing mounting financial and social burdens on society (Becker et al., 2010). The National Osteoporosis Foundation estimates that over half of people over the age of 50 will develop osteoporosis, and that 80 percent of those developing osteoporosis will be female (NOF, 2010)

It is now well established that osteoporosis is a heterogeneous disorder and that a suite of factors influence its manifestation. For example, physical activity, proper nutrition, hormonal status, and reproductive history have all been shown to be strongly associated with osteoporosis (Christodoulou and Cooper, 2003). In addition, lifestyle factors such as smoking and alcohol consumption can also have an effect (Cummings et al., 1995; Seeman, 1996). While all of these factors play contributing roles in the etiology of the disease, influences are modulated and change over the life course. Physical activity is a perfect example. The adolescent bone growth period is particularly sensitive to the influence of physical activity (Rauch et al., 2004), and during this highly responsive period, physical activity allows bone mass to increase and accumulate into young adulthood. While mechanical strain and activity continue to influence bone formation in adult and older age (Ruff et al., 2006), the greatest impact of activity is considered to be limited to earlier periods in life (Pearson and Lieberman, 2004). Initial “peak” quantity of bone accretion is known to be directly related to how much bone is taken into adulthood and able to buffer subsequent bone loss in senescence. (Bonjour et al., 2007).

The heterogeneity and complex nature of bone loss and osteoporosis is undoubtedly why many biological anthropologists have studied the disease in past populations. The modern causes and prevalence of the disease are situated in a particular social and temporal context. Our modern lifestyle is arguably dramatically different than what most humans have experienced throughout of human history. Human societies have changed dramatically through time, and past populations provide a window into the unique biosocial and environmental contexts with which to interpret patterns of bone loss. Osteoporosis is then a perfect subject to explore the interplay between culture, biology and health. Furthermore, this cross-cultural and diachronic perspective stands to make significant contributions to modern interpretations of osteoporosis, in addition to broadening our knowledge of health in the past.

What bioarchaeological studies have shown to date is that past populations did not gain and lose bone throughout life in the same way that we see in modern groups (Agarwal, 2011). While bone loss in adulthood is universal and has been shown in every study (Agarwal, 2008), typical patterns of postmenopausal bone loss seen in modern Western women are often not seen in past populations (Agarwal and Grynepas, 1996, 2009; Grynepas, 2003; Robling and Stout, 2003; Nelson et al., 2003; Glencross and Agarwal, 2011). In modern populations, women are roughly twice as likely to suffer from osteoporosis than men, once menopause has completed (NOF, 2011). One of the more striking findings in several archaeological samples has been the noted lack of sex differences in bone loss, particularly in the critical period after menopause (Agarwal, 2008). In addition, the prevalence of fragility fractures is often much lower than we would

expect, if basing our assumptions on modern trends (Agarwal 2008). Fragility fractures are fractures that occur under forces that would not break normal, healthy bone, and are thus an important indicator of overall bone health. Although a lack of clear sex differences in age-related bone loss has been found in many archaeological samples, some studies do demonstrate patterns of bone loss that more closely follow those we see in modern populations (Kneissel et al., 1994; Mays, 1998; McEwan et al., 2005). There may be a number of reasons for this. First, the use of different methods on varying parts of the skeleton may be partially responsible for these conflicting characterizations of bone loss in the past (Peck and Stout, 2007; Agarwal and Grynepas, 2009). Second, cross-cultural factors that specifically affect growth and development may also be important to bone maintenance later in adult life (Cooper et al., 2006; Agarwal and Beauchesne, 2011). There are also a number of inherent biases in skeletal samples (Wood et al., 1992; Jackes, 2011) that can confound results and makes inter-study comparisons challenging. Even so, the weight of the evidence suggests that the temporal and geographic variation in age- and sex-related patterns of bone loss in past populations is real, and is intimately tied to divergent biocultural contexts. All of the current research to date strongly supports continued research into the complex etiology of bone loss and fragility, as well as what bone loss can reveal about past lifeways.

### **Life Course Approaches to Understanding Bone Loss**

Previous studies of bone loss and osteoporosis in past populations have contextualized bone loss as a product of several key biocultural influences, particularly diet, physical activity and reproductive history (Mays, 1996; 2006; Agarwal et al. 2004; Agarwal, 2008; Brickley and Ives, 2008; Agarwal and Grynepas, 2009; Cho and Stout, 2011). One area that has not been explored more carefully is how these factors affected the skeleton over the life course (Agarwal, 2008). Life course approaches, which come from many disciplines (Bengston and Allen, 1993; Elder et al., 2003; Knudson and Stojanowski, 2008), offer at their core a developmental and historical framework to studying the lives of individuals or groups. The individual is seen as an active agent, both influencing and being influenced by social contexts and structures. For bioarchaeology, this has important repercussions; most importantly, we can move beyond attributing particular morphologies in skeletons to environmental change, and rather consider an individual's life long connection to changing social and historical contexts (Knudson and Stojanowski, 2008). The life course approach then offers a holistic and anthropological approach to studying biocultural effects, such as diet and reproductive history, over the life cycle. The life course approach to the study of bone loss is an important step forward. Clinical work has shown that adult skeletal health has its roots in early development through adolescence (Javaid and Cooper, 2002; Cooper et al., 2006). For example, an infant's birth weight is correlated with adult bone mass (Cooper et al., 2006). Proper nutrition and adequate physical exercise during childhood and adolescence are seemingly critical in reducing fragility fracture risk in adulthood (Rauch et al., 2004; Cooper et al., 2006). Life course approaches in bioarchaeology have the potential to compliment these clinical observations by provide information on patterns of bone loss across the life cycle from greatly varying biocultural contexts. This can produce useful ways to test hypotheses about the relative influence of the biocultural factors that can mediate bone loss.

The life course approach in bioarchaeology has its root in past studies (e.g. Armelagos et al., 1972), but it is only now gaining prominence, with only a handful of studies expressly linking early life experiences with measures of bone loss in adulthood (Rewekant, 2001; McEwan et al., 2005), although others have linked stress during development with increased risk of death later in life (Clark et al., 1986; Boldsen, 2007). The lifecourse perspective used here borrows from clinical work on developmental plasticity (Cooper et al., 2006) and developmental systems theory (Griffiths and Gray, 1994; Oyama, 2000a, 2000b; Gray, 2001), and from anthropological work on lifecourse theory (Ben-Shlomo and Kuh, 2002, Elder et al., 2003; Fausto-Sterling, 2005).

The life course approach also stands to make important contributions to investigations of social identity, (cultural) aging, social agency and gender in bioarchaeology (Glencross, 2011; Hollimon, 2011; Sofaer, 2011). For example, Glencross (2011) has reexamined skeletal trauma in the Indian Knoll population as accumulated pathology over the life course, rather than single unrelated events. In this way, variable risk across the life cycle can be estimated and then interpreted through questions of cultural aging and social agency (Glencross, 2011). Sofaer (2011) challenges bioarchaeologists to take a more sophisticated view of age and aging by not relying on grave goods, and to examine 'age' as both a category and a process. A lifecourse approach to age and aging might consider social age as a new variable, in order to reinterpret what health may have been like in socially relevant population-specific categories (Sofaer, 2011). Most of these studies utilizing a life course approach emphasize that the body is truly a meeting point between biology and culture (Sofaer, 2006), and this remains central to the research in this study as well.

## **Research Objectives**

The principal goal of this dissertation is to contribute to ongoing research as to why patterns of bone loss seem to differ across cultures, and in particular, between modern and past populations (e.g. Nelson et al., 2003; Agarwal, 2008; Agarwal and Grynepas, 2009). A lifecourse theory approach is used to help answer this question. The lifecourse approach was chosen to specifically challenge the assumption that bone maintenance and bone loss is tied entirely to menopause and senescence and that women will inevitably have poorer bone health than men in old age. The substantial clinical evidence that suggests that poor childhood growth is associated with higher risk of adult fracture (Cooper et al. 2006), has important repercussions for the study of bone health in past populations as growth in historical populations was often stunted (Humphrey, 2003). Bioarchaeological research has only just begun to explore this link between the juvenile period and bone loss later in life. This dissertation takes the position that cross-cultural comparisons of archaeological populations are vital to improving our understanding of human aging in anthropology, and to the broader fields of science that study aging as both biological and cultural processes.

The lifecourse perspective was implemented in this dissertation in two ways. The first was to examine indicators of childhood stress in adults to see if those indicators of developmental stress translated into advanced bone loss in adulthood. While many studies have examined various aspects of growth and development, what is unclear in existing research is what those effects are on the health of the adult skeleton. Only a few studies have emphasized

the role of developmental stress in the formation of adult skeletal morphology and bone maintenance (how well bone is retained with age), and how differences in early metabolic stress could explain phenotypic differences between the adults of those populations (Kneissel et al., 1997; Rewekant, 2001; McEwan et al. 2005; Gosman and Ketcham, 2009). Analytically this project builds on these previous studies but contributes in a number of novel ways. First, there is a strong emphasis on examining the relationship between early life stress and bone maintenance later in life. While this has been attempted before (Rewekant, 2001; McEwan et al., 2005), this research provides a much more comprehensive investigation using multiple indicators of juvenile and adult stress. Second, age related bone loss is also contextualized within the specific Roman biocultural experience, including skeletal evidence for nutritional and environmental stress. One of the most significant aspects of this work is that for a first time, bone maintenance and loss is assessed with three analytical methods in a single archaeological population.

The use of multiple methods is an important step forward in studies of bone loss as the approach recognizes that the human skeleton is not homogeneous structure and that there can be great variability throughout the body. For example, cortical bone, which is typically very thick and dense, changes slowly over time in response to metabolic demands, or in response to physical exercise (Martin and Burr, 1989). In contrast, trabecular bone is “spongy” in structure and has a much larger surface area and often responds to these same demands more rapidly. Independently, both types of bone are useful indicators of bone loss (Brickley and Agarwal, 2003), but examined together they provide a much more complete picture of changes occurring throughout the skeleton. What is unique about this multi-method approach is that it uses the differences in environmental sensitivity of cortical and trabecular bone to explore changes over the life course. For example, changes in cortical bone take many years with changing influences on the inner and outer cortical envelopes at different points of the life cycle (Ruff et al., 2006), whereas changes in trabecular can happen on a much shorter time scale (Barak et al., 2011). Consequently, the use of a combination of methods has the potential to reveal skeletal changes that are “recorded” at different points throughout the life course and these can be interpreted in light of biocultural contexts to explore how Roman daily life may have affected bone health. The previous study of the archaeological medieval sample Wharram Percy provides an excellent example of why multi-method approaches are needed. Mays (1998) found significant sex differences in femoral bone mineral density in adults over the age of 50. However, Agarwal et al. (2004) found no sex differences in trabecular bone volume or measures of trabecular architecture in the spine. These conflicting age-related patterns of bone loss highlight the heterogeneity of bone and the need to uncover a more complete description of changes occurring throughout the whole body.

This complex nature of the human skeleton can thus be used to our advantage to arrive at a life course perspective. Using a multi-method approach reveals differential timing of bone loss. However, it the investigation of biocultural context, such as diet/nutrition and occupations (physical activity) may help explain the timing of the bone loss event. This captures a more complete description of bone health than looking at any singular skeletal element with a single method of measurement. Consequently, the central working hypothesis of this project is that the skeletal maintenance and fragility of adult skeletons is a product of life course influences. An important secondary hypothesis of this project is that environmental disturbances during skeletal growth and development have a negative effect on bone maintenance throughout the life course.

Together, the developmental stress and multi-method approaches recognize that adult bone morphology is a product of previous life experiences.

Subsequently, the dissertation has four primary goals: 1) to refine knowledge of how bone remodels and maintains itself at the tissue level over the biological life course; 2) to examine how stress during childhood or early life effects bone maintenance and loss across the lifecourse; 3) to contribute to our understanding of why patterns of bone maintenance loss vary through time and space; 4) to advance bioarchaeological interpretive frameworks through the combined use of multiple indicators of bone loss and of physiological stress.

## **Methods and Materials**

The three primary methods used in this dissertation to evaluate bone growth and loss are radiogrammetry of the second metacarpal, trabecular architecture of the 4th lumbar vertebra, and cortical histomorphometry of the rib. The use of multiple methods accounts for the fact that bones behave differently according to their metabolic and/or biomechanical roles; this results in different remodeling histories over the lifecourse that would otherwise go unobserved. These multiple remodeling histories can then be contrasted, highlighting lifecourse “pathways” and can help evaluate important factors such as nutritional, reproductive, or biomechanical history. Another advantage is that a multi-method approach can help elucidate timing of bone loss throughout lifecourse.

The three methods used in this dissertation were chosen to reflect changes in bone quantity (the amount of bone) and quality (aspects such as bone microstructure or bone material properties) as well as difference in cortical and trabecular bone tissues. The first, rib histomorphometry, has been extensively studied in anthropology (Stout and Teitelbaum, 1976; Stout and Lueck, 1995; Mulhern, 2000; Robling and Stout, 2000; Schultz, 2001; Cho and Stout, 2003). Ribs are often used in histomorphometric studies of archaeological populations because they are easily accessible for invasive sampling and are typically well preserved. Most importantly, ribs are less mechanically active than other skeletal areas, such as the femur, and thus potentially offer a more neutral view on baseline metabolic activity (Robling and Stout, 2003).

The second method, trabecular architecture, is valuable because vertebrae are one of the primary skeletal regions affected by bone loss due to higher average metabolic activity, and as a result, they are a sensitive marker for remodeling changes that might not be seen in ribs or metacarpals as those regions are composed primarily of thick, less metabolically active cortical bone (Compston, 1999; Brickley and Agarwal, 2003). Vertebrae were also selected as they can be studied across the life course (Kneissel et al., 1997), providing valuable information on the growth and development of the sample. This will be informative in exploring how developmental stress affect trabecular architecture in juveniles, as well as adults. This study uses only the fourth lumbar vertebra as it has been the most extensively studied and is more sensitive to structural changes during growth and development (Roschger et al., 2001).

The third and final method, metacarpal radiogrammetry, was developed by clinicians around 50 years ago as a safe and quick way to gauge fracture risk in patients (Barnett and



Nordin, 1960). Shortly after, the method became instrumental in a number of studies that tracked bone growth and development and sex and age-related patterns of aging (Virtama and Helelä, 1969; Garn, 1970). One clear benefit is that radiogrammetry is a sensitive marker to changes in bone quantity that can be used to track longitudinal changes quite easily (Nielsen, 2001). Metacarpal radiogrammetry also has long-standing value in bioarchaeology and has been shown to be informative about sex and age-related patterns of bone growth and loss (Mays, 1996, 2000, 2001; 2006; Lazenby, 2002; Ives and Brickley, 2004). Given that the majority of the skeleton is comprised of cortical bone, the study of cortical bone is vital to our understanding of age and sex-related patterns of bone loss in the past and present (Mays, 2006).

## **The Port Town of Velia, Italy**

The port city of Velia began as a Greek colony in 540 BC. The context of the necropolis burials place the sample of this study to the Imperial Roman period, between the 1st and 2nd centuries A.D. (Crowe et al., 2010). Velia was a port city, with its industry revolving around fishing, including all related occupations, from the manufacture of boats, to the preservation and distribution of fish to other areas of the Roman world. Archaeological reconstructions suggest that most of the people from the necropolis represent those from middle or lower classes of Roman society (Fiammenghi, 2003). As such, this population represents an exciting opportunity to study the non-elite, who are often forgotten in studies of the Roman world (Toner, 2002; 2009). In addition, there are many well-preserved children in this sample, which facilitates explorations of health for the whole population. Velia is also similar and contemporaneous to the well-researched Isola Sacra archaeological population (Bondioli and Macchiarelli, 1999; Cho and Stout, 2003; Prowse et al., 2005; Fitzgerald et al., 2006), although with some important socioeconomic differences. For example, the Isola Sacra population represented an urban, middle-class sub-group of the Roman population (Garnsey, 1999), while Velians came from a more lower class background with ties to agriculture (Craig et al., 2009). As Paine et al. (2009) noted, the bioarchaeology of Imperial Roman skeletons is sorely lacking compared to the amount of historical and archaeological information available. This project stands to make an important contribution to studies of bone loss, but also to Roman bioarchaeology in general.

## **Chapter Summaries**

In Chapter 2, important background information on the biology of bone growth, maintenance and loss is provided. First, the fundamentals of bone biology are reviewed to better inform the reader. Second, the nature of osteoporosis is reviewed, including both modern and archaeological manifestations and understandings of the disease. This chapter concludes with an argument about why the developmental stress and multi-method approaches are used in this dissertation to assess bone maintenance and loss in the past and how they form an improved model over most existing studies in the examination of bone health.

Chapter 3 is a review of the literature on lifecourse theory in bioarchaeology. The development of lifecourse theory in bioarchaeology is traced back to the emergence of related ideas in the clinical world, such as developmental systems theory and developmental plasticity.

The history of biocultural theory and life history models in bioarchaeology are discussed as they also form an important aspect of lifecourse theory.

Chapter 4 provides background information on the site of Velia. The sample composition, origin, cultural and archaeological contexts are discussed. Important biocultural contexts are provided about daily life, living in (port) cities, nutrition and diet, and gender roles and experiences.

Chapter 5 provides detailed descriptions of all methods used in this dissertation. The chapter outlines the three measures used to assess bone loss (radiogrammetry, analysis of trabecular architecture, and histomorphometry), as well as methods used to determine age and sex in the archaeological skeletal sample. All measures of physiological stress are discussed as well. The chapter concludes with a discussion of the statistical analyses used in the dissertation.

Chapter 6 lists the complete results for all measures in this project. This is followed by a discussion of the results in Chapter 7. Chapter 8 concludes the dissertation with a summary of the findings from Chapter 7, as well as implications for future work on bone maintenance and loss in bioarchaeological contexts.

## Chapter 2 - Bone Loss and Maintenance in the Past and Present

As noted in the Introduction, Osteoporosis is an increasingly serious medical, social and economic problem in aging Western populations, given that 44 million people affected in the United States alone, and over 200 million affected worldwide (Reginster and Burlet, 2006). Estimates from The National Osteoporosis Foundation place half of all people over the age of 50 as osteoporotic. Even more concerning for women, females comprise 80 percent of those affected (NOF, 2010). The high prevalence of osteoporosis for women today is clearly associated with menopause, in addition to general senescence, but osteoporosis has a multi-factorial etiology and the risk of developing osteoporosis is greatly mediated by factors that are independent of the menopause-induced drop in estrogen. Some of the extrinsic factors that mediate the risk of fracture and advanced bone loss include diet, hormonal status, physical activity, and reproductive history (Stevenson et al., 1989; Ward et al., 1995; Worthman, 1995; Agarwal and Stuart-Macadam, 2003; Rauch et al., 2004). Osteoporosis is then clearly a heterogeneous disorder, and as such, has received steady interest by biological anthropologists. Bioarchaeological studies have attempted to investigate not only the natural history of the disease, but also contribute to our understanding of the complex influences on bone maintenance and loss. Past populations, each with their own unique social and ecological environments, present unique insights into human skeletal variation, thereby allowing us to place modern observations of bone health into more diachronic and nuanced contexts.

The focus of this chapter is to examine patterns of bone loss in modern populations and to then contrast the epidemiology with what has been found in bioarchaeological studies. Reviews of key concepts in bone biology are first provided. The chapter concludes with a discussion of how seemingly purely biological risk factors for osteoporosis are in fact biocultural constructions that can vary between cultures to a large extent.

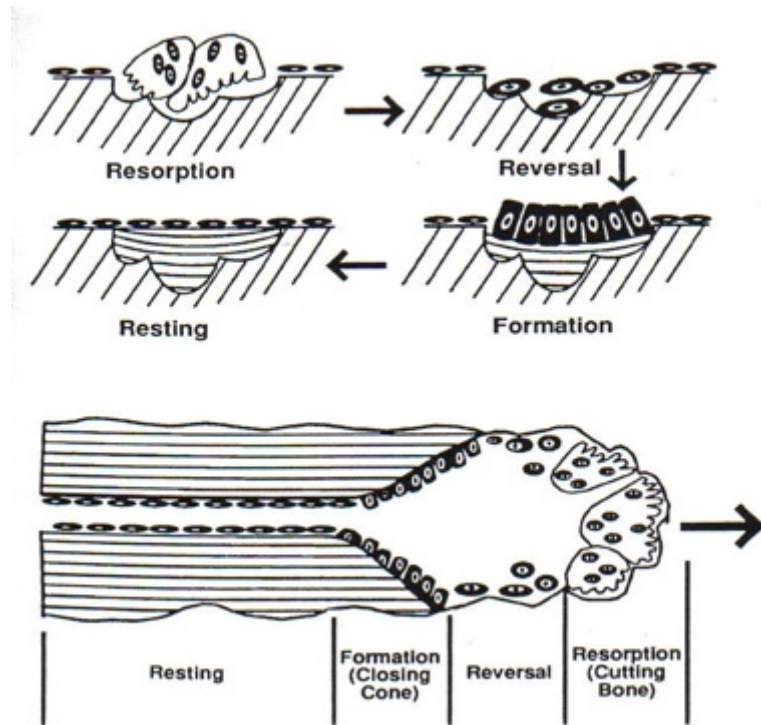
### Fundamentals of Bone Biology

#### *Bone Cells*

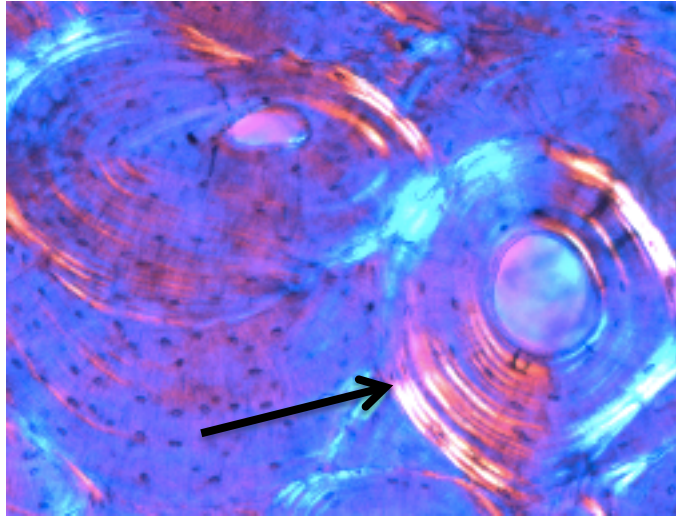
There are three major bone cell types that modulate bone physiology that are relevant to the discussions in this research. *Osteoblasts* are bone-forming cells that release a substance called osteoid, which is rich in ‘bundles’ of collagen (Schultz, 2001). These collagen fibers become mineralized bone due to the formation and deposition of calcium phosphate crystals (Schultz, 2001). *Osteocytes* are osteoblasts that become embedded into the bone matrix in lacunae (or spaces). Numerous canaliculi, or small channels found in bone, spread out from these osteocyte lacunae, facilitating blood/nutrient flow to the interior of the bone matrix. The role of osteocytes has long been debated. The prevailing hypothesis today is that osteocytes play a crucial role in sensing mechanical strain and in subsequently stimulating bone formation or loss (through lowered or increased strain) (Martin, 2003; Jiang et al., 2007). Finally, *osteoclasts* are large multi-nucleated cells that resorb (destroy) bone by acidifying and traveling through the bone matrix. In normal bone, both osteoclasts and osteoblasts travel together through the bone as a single bone multicellular unit, or BMU (Frost, 2003). The BMU has the three-dimensional

shape of a cone (Figure 1) and ensures that all bone that is resorbed by the osteoclasts at the front of the unit is replaced shortly after by new bone formation from the osteoblasts (Frost, 2003). The resulting structures of this destruction and formation process are called *osteons*, which are the predominant structural unit of adult compact bone (Schultz, 200; Stout and Simmons, 1979; Locke, 2004). An osteon, or Haversian system, consists of a Haversian canal surrounded by circular lamellae ('sheets' of bone) (Figure 2). The Haversian canals contain blood vessels and a nerve. The osteocyte lacunae and canaliculi are located in the parallel rings of lamellar bone around the Haversian canal. Osteons are present in trabecular bone as well, but are referred to as hemiosteons due to their half moon appearance as they lie on the surfaces of the struts (Parfitt, 2003).

This review of the micro-anatomy of bone is brief and is limited to topics that will be referred to in subsequent sections. The remaining review of bone biology will deal mainly with the macro scale and larger processes that affect bone growth and maintenance in adulthood.



**Figure 1** - Schematic representation of remodeling. The top image represents remodeling in trabecular bone or on periosteal and endosteal surfaces. The bottom image is of intracortical remodeling. In both, osteoclasts (the large multi-nucleated cells) begin the remodeling process by removing bone. Osteoblasts soon follow and form bone where it was resorbed. (Adapted from Dempster, 2002: 317)

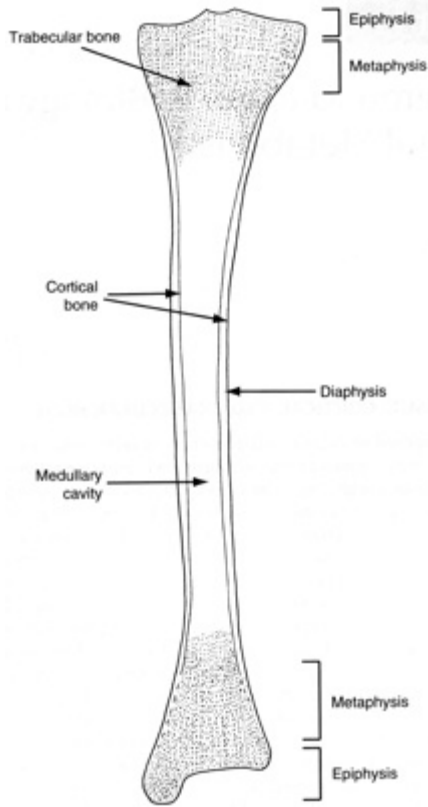


**Figure 2** - Secondary osteon (Black Arrow): notice the central Haversian canal and concentric lamellar rings (Under polarized light, x100 magnification)(Image captured by author).

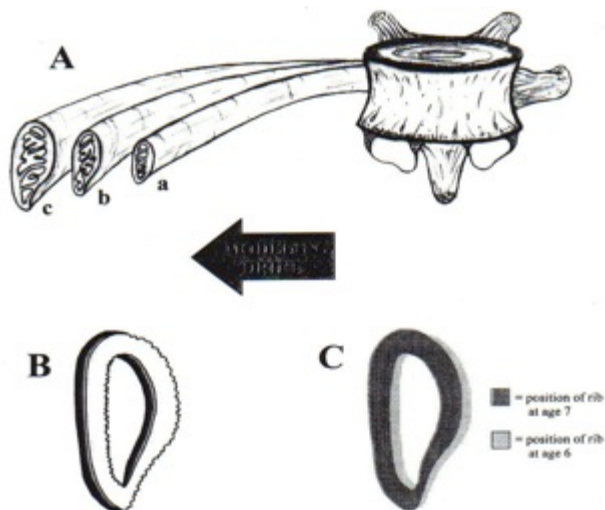
### *Bone Structure and Form*

At the tissue level, most bones of the body are comprised of outer dense cortical bone and an inner network of connected rods, plates and struts called cancellous or trabecular bone (Figure 3). Discussions of cortical bone morphology often make reference to envelopes, or surfaces (Garn, 1970; Seeman, 1997; Dempster, 2002). For most discussions in bioarchaeology, the periosteal, intracortical, and endosteal surfaces are the regions of interest. Specific reference to these surfaces in discussions of bone biology is essential as bone cells react and behave differently at each of these sites (Seeman, 1997), particularly in relation to age and sex. Trabecular bone can be considered distinct from cortical bone based not only on its morphological/structural difference, but also on its higher metabolic activity (Compston, 1999), functional adaptation (Martin and Burr, 1989), and relationship with hematopoietic tissues (Gurevitch and Slavin, 2006).

Bone grows and changes its shape depending on the age of the person. During somatic growth, bone *modeling* is the dominant process that is responsible for enlarging bones and providing their geometric shape (Robling and Stout, 2003). Modeling occurs primarily through drifts, where bone is deposited on the periosteal surface by osteoblasts and resorbed on the endosteal surface by osteoclasts to create expansion and architectural changes in the bone (Frost, 2003) (Figure 4). Sometimes bone is deposited endosteally and resorbed at the periosteal surface (Robling and Stout, 2000). During modeling osteoblasts and osteoclasts work independently of each other. Bone modeling drifts occur until the cessation of growth when *remodeling* becomes the core mechanism of bone maintenance. A key feature of modeling is that growing bone is much more responsive (i.e. plastic) to mechanical loading than adult bone that only remodels (Parfitt, 2003).



**Figure 3** – Cross-section of bone highlighting locations of cortical and trabecular bone (example from tibia) (Adapted from Brickley and Ives, 2008: 22).



**Figure 4** - Schematic representations of bone modeling (adapted from Robling and Stout, 2000: 188)

The functions of bone remodeling are more varied. Remodeling is primarily thought to repair old and damaged bone (micro-fractures) in order to restore mechanical competence (Parfitt, 2003). Mechanical competence may be jeopardized by micro-strains or cracks (Martin, 2003), but also changes in the material properties of bone, such as bone becoming hyper mineralized and thus brittle (Vajda and Bloebaum, 1999). Remodeling is also thought to provide more or less bone mass in a given area in response to mechanical demands; to alter architectural properties in response to mechanical loads; and to aid in plasma calcium homeostasis (Dempster, 2002; Parfitt, 2003).

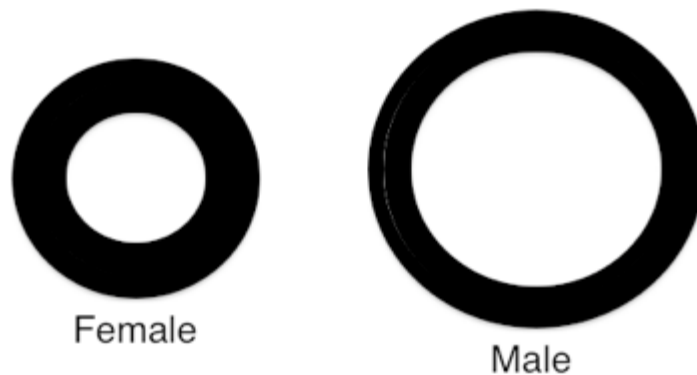
Remodeling occurs by the triggering of the effector cells (precursors to osteoblasts and osteoclasts) in response to mechanical and/or chemical stimuli (the mechanical/chemical mechanisms are not entirely clear) (Turner, 1999; Parfitt, 2003) (Figure 1). In a balanced system, all bone that is resorbed by the BMU is replaced by an equal amount of new bone. However, the remodeling process usually results in a small net loss of bone (Parfitt, 2003; Cho and Stout, 2003). Bone health is affected when the remodeling balance is uncoupled to an extent where there is either too much formation of bone or too little (Cho and Stout, 2003).

The mechanostat theory of bone maintenance is currently considered the best mechanism that explains how bones change their shape throughout life, although others have challenged the prevailing hypothesis (Turner, 1999; Lovejoy et al., 2003, Pearson and Lieberman, 2004). It has long been known that mechanical strains placed on bone result in metabolic responses in loaded bone (e.g. Wolff, 1870; Roux; 1885; Koch, 1917, Jansen, 1920, see Hall, 2005 for a detailed review). This initial observation has become known as “Wolff’s Law” (Ruff et al., 2006; Barak et al., 2011). Wolff’s law is a model that predicts that the orientation of trabecular bone will be in line with the greatest direction of strain on the bone. However it was not until the early 1980s that more concrete explanations emerged (see Frost, 2001 and Frost, 2003 for excellent reviews of the concept). Harold Frost’s early work (Frost, 1966; 1969), as well as more recent additions by Frost (1996; 2001a; 2003a; 2003b) and other researchers (Parfitt, 2003; Schoneau et al., 2003; Rauch, 2005), have shown that bone is very dynamic and complex organ, able to change in direct response to the physical loads placed upon it. The mechanostat is a negative feedback system that controls the relationship between bone architecture and strength and the size or magnitude of the loads and strains placed on that bone (Frost, 1987c; 2003). This model recognizes the body’s tendency towards conservatism, where skeletal weight is sought to be minimized but strength maximized (Martin, 2003). This conservatism is thought to be controlled by ‘set points’ in bone, which are thresholds that must be crossed to elicit morphological change (Frost, 2001). Whether this set point is genetically or biomechanically controlled is still a matter of debate for some (e.g Lovejoy et al., 2003).

The relationship between muscles and bone strength is also worth noting. Countless examples in mammals exist where strong muscles accompany strong load-bearing bones and weak bones are nearly always found in association with weak muscles (Frost, 2003b). The importance of muscle mass and strength has recently gained prominence in bone research, so much so that bone and muscle are now commonly viewed as a single functional unit (Schoenau, 2005). Bone growth, maintenance and loss must then be interpreted in the context of muscle mass if possible or level of physical activity indirectly. In the mechanostat hypothesis, modeling “decides” how much (and how strong) needed bone based on mechanical loads, while

remodeling operates as a threshold (the set point) to regulate if the original amount of bone is still needed for mechanical competence at current biomechanical strain levels (Frost, 2003a). What is key to note in this discussion is that physical activity has a beneficial effect on increasing bone mass and strength and that this relationship is most pronounced during modeling, although it does continue during remodeling as people enter adulthood (Ruff et al., 2006).

While remodeling and the principles of the mechanostat hypothesis apply to all populations, males and females differ in their skeletal development in functionally important ways. Males tend to have a longer period of somatic growth, resulting in larger bones and muscles (Riggs et al., 2002). If we consider the deep relationship between muscles and bone in assessing bone strength (Ruff, 2006), this longer period of growth translates into a potentially marked advantage in bone health. This longer growth period in males also accounts for their generally higher peak bone mass, or the maximum amount of bone someone will attain in their lives (Riggs et al., 2002). As bone is modeled in growing males, long bones are preferentially made wider by increased periosteal bone deposition (Ferretti et al., 2003). Females on the other hand have less periosteal expansion but increased endosteal formation of bone (Ferretti et al., 2003) (Figure 5). So while *percent* cortical areas may be similar, the distribution of bone through geometric space is not, and operates in biomechanically and metabolically different ways. Hypotheses about why males and females differ in growth profiles are addressed in the final section of the chapter.



**Figure 5** – Bone growth between the sexes. Notice the thicker endosteal area in females and greater periosteal deposition in males.



The evolutionary and developmental determinants of bone form and function are also important to consider, as they are related in complex ways (Hall, 2005 for a highly detailed review) and are important in modern considerations of our own human skeletal biology, particularly in understanding inter-population variability. Erikson et al. (2002) have shown that the material properties of the femur (across numerous animal species and taxa) have remained fairly constant over the past 475 million years of evolution. While not demonstrated yet, it suggests that the material properties of bones other than the femur may have also changed little through time as well. The implication is that challenges to functional adaptation (see Martin, 2003; Ruff, 2005) have been met more by changes in size and shape rather than changes in material dynamics (Erikson et al., 2002).

In summary, the formation of the skeleton is dependent upon modeling earlier in life and is maintained by the remodeling process after major growth periods have ended. Both of these processes that guide the formation and maintenance of the skeleton are controlled by the mechanostat, which is best described as a feedback loop governing the size and shape of bones in relation to the external forces acting upon them. Further, while the shape of bones can vary significantly between individuals and populations, all anatomically modern humans share fundamentally the same material properties of bone, which allows bioarchaeologists the opportunity to look at human bone health as a potential product of biocultural change and not changes in the fundamental nature of bone (Erickson et al., 2002). In the following two sections, the discussion of bone biology is expanded to examine bone maintenance and loss through the life cycle in both modern and archaeological contexts.

## **Bone Loss in the Present**

### *The Social Cost of Osteoporosis*

Osteoporosis is currently one of the major diseases affecting people over the age of 50 throughout the world, but disproportionately in Western societies. In the United States alone, it is currently estimated that 10 million people have osteoporosis, while another 34 million suffer from osteopenia (low bone mass) (Becker et al., 2010; Dempster, 2011). By 2020 it is projected that 61 million people in the United States will have either osteopenia or osteoporosis (Dempster, 2011). The principle medical and socio-economic costs of osteoporosis are related to fractures, which currently cost over \$20 billion annually in the United States (Dempster, 2011). Sufferers of fragility fractures are commonly susceptible to post-operative infection and complications, venous thromboembolism, disabling pain, pneumonia, and general physical disability (Becker et al., 2010; Dempster, 2011). Risk of death from fracture or from subsequent medical procedures (such as surgery) is also a significant concern (Dempster, 2011). For example, hip fractures raise mortality risk by up to 20% within the first year after a fracture (Dempster, 2011). Additionally, about one third of hip fracture patients must be placed in a long-term care facility (Dempster, 2011). The social costs are often as high as the physical ones. After a serious fracture, many individuals suffer from psychological deterioration, depression, and face considerable strain on their relationships (Becker et al., 2010; Dempster, 2011). Part of the social strain the disease causes emerges out of the very long period of recovery, financial costs of recovery, and the lack of mobility and independence (Dempster, 2011). Unfortunately, the precursor to osteoporosis,

osteopenia, is largely asymptomatic and the first sign of the disease is fracture, with sudden financial, physical and emotional costs. In light of these deep tolls on society, much more work remains to be done in order to significantly curtail the prevalence of osteoporosis in future generations.

### *Defining Bone Loss*

Osteopenia and osteoporosis are both metabolic bone disorders and are closely related, yet they lie on different points of a spectrum. Osteopenia is generally defined as a loss of bone mineral density (BMD) occurring at a greater rate than what is statistically normal (Mundy, 1995), which is defined as a BMD of 1.0 to 2.5 standard deviations (SD) below the normal average for healthy young (Caucasian) adults (Kanis, 1994; Ross et al., 1999). Osteopenia is also clinically defined by the absence of fragility fractures in bone (Mundy, 1995). Osteopenia itself is not strictly pathological in nature, but it does present a significant increase in the future risk of fragility fractures (Mundy, 1995; Dempster, 2011) and is thus a major health concern.

Osteoporosis is also defined as abnormal bone loss and its most severe form is accompanied by the presence of fragility fractures that occur with only minimal trauma (Birnbaum, 1992; Mundy, 1995; Stini, 1995; Center and Eisman, 1997; Ross et al., 1999; Melton et al., 2003). Osteoporosis is classified by bone mineral density (BMD) measurements determined by dual-energy x-ray absorptiometry (DEXA) and are operationalized in terms of fracture risk (Center and Eisman, 1997). An individual is said to be osteoporotic if they have a BMD of at least 2.5 SD below the norm (Kanis, 1994). There is also a classification of severe osteoporosis, which requires both low BMD and the presence of fragility fractures (Kanis, 1994; Ross et al., 1999). These definitions of osteopenia and osteoporosis are somewhat arbitrary as bone loss is a natural process of aging and thus differentiating normal processes from pathological bone loss can be difficult; it is a difference of degree and not of kind. Additionally, bone loss, in terms of mass, does not necessarily equate with fracture (Ross et al., 1999; Frost, 2001b; Heaney, 2003). Osteoporosis also has multiple causal factors, and accordingly, parsing out their respective roles is problematic.

Osteoporosis is further classified into primary and secondary osteoporosis (Mundy, 1995). Primary osteoporosis is the most frequent form (95%) and occurs without the influence of other pathological processes (Mundy, 1995). Secondary osteoporosis is derivative in nature and is a result of bone loss and fracture stemming from a separate pathological condition, such as Cushing's syndrome (Mundy, 1995), leukemia and immobilization (Brickley and Ives, 2008). This distinction between primary and secondary forms of osteoporosis applies to osteopenia as well (Mundy, 1995). This poses a problem for bioarchaeologists as these secondary causes of osteopenia and osteoporosis may be difficult or impossible to detect (Schultz, 2003). Our understanding of osteoporosis is further complicated by the fact there are two types of the primary form of the disease.

Primary osteoporosis is also subdivided into two types (Riggs and Melton, 1983). Type I osteoporosis refers to skeletal changes in women in midlife, and reflects the sudden drop in estrogen during menopause (Birnbaum, 1992; Stini, 1995). Type II, or 'old age', osteoporosis is

prevalent in both aging men and women. Diagnostically, both types of osteoporosis display unique manifestations, although considerable overlap often complicates precise diagnosis (Stini, 1995). Type I osteoporosis tends to affect trabecular bone over cortical bone (Birnbaum, 1997; Riggs et al., 1998) and is most often associated with the ‘typical’ vertebral crush, Colle’s, and hip fractures (Mundy, 1995). Riggs and Melton (1983), who originally proposed the Type I and Type II distinction, argued that Type I “osteoporosis results from [estrogen] deficiency plus some additional factor, operative only in the presence of [estrogen] deficiency, that produces an exaggeration of the rate and duration of the rapid postmenopausal phase of bone loss” (Riggs et al., 1998: 770). These additional factors are hypothesized to be cytokine activity on estrogen receptors, polymorphisms for increased number or altered function of estrogen receptors, and impaired renal function leading to calcium loss (Riggs et al., 1998). In Type II osteoporosis, cortical bone is believed to be more affected, with fractures of the hip, pelvis, proximal humerus, and proximal tibia becoming more common (Birnbaum, 1992). Type II osteoporosis appears to be highly correlated with the aging process as it ultimately affects nearly everyone (Riggs et al., 1998). However, both types of the disease can manifest themselves in very similar ways as many of the types of fractures overlap, as do the age ranges where osteoporosis normally begins (Center and Eisman, 1997). As a result, the recognition of two distinct types of osteoporosis is considered by some to be a false distinction without adequate clinical and epidemiological support (Center and Eisman, 1997). In bioarchaeology, the distinction between the two types is difficult as diagnostic tools are limited and the methods for age estimation do not allow for accurate and precise age assessments for individuals over the age of 50 (Jackes, 2000). Instead, bone loss that appears advanced for a particular population is treated as osteopenia, while the term osteoporosis (without assignment of type) is withheld for individuals showing clear signs of fragility fractures (Brickley and Ives, 2008).

### *Risk Factors for Osteopenia and Osteoporosis*

The consensus in the clinical literature is that peak bone mass is one of the more crucial factors in determining osteoporotic and osteopenic risk. Peak bone mass is defined as the maximum amount of bone volume obtained during growth (Center and Eisman, 1997). Failure to attain a high peak bone mass presents a greater fracture risk as fractures may occur earlier from the reduced bone mass at the onset of age related remodeling changes (Brickley and Ives, 2008). Peak bone mass reaches its apex during the third decade of life and remains fairly constant until the fourth (Center and Eisman, 1997), although the timing of peak bone mass may vary between individuals by up to a decade (Orwoll et al., 2001; Raisz and Seeman, 2001; Bonjour et al., 2001). Peak bone mass is determined by a suite of factors including genetics, childhood health, nutrition, physical activity during growth and development, and sex steroids (Mundy, 1995; Brickley and Ives, 2008). A certain amount of bone loss after peak bone mass has been reached is a natural and normal phenomenon and is not considered pathological although a number of factors can aggravate this loss of bone (Center and Eisman, 1997; Stini, 1995; Mundy, 1995).

Genetic studies relating to bone growth have received a great deal of attention (Sinclair and Dangerfield, 1998; Seeman, 1999; Stewart and Ralston, 2000; Lovejoy et al., 2003). Parsing out the genetic determinants of bone form, mass and function may provide important answers as to why populations and individuals differ so greatly in bone growth and loss, given that many

skeletal biologists posit that genetics are largely responsible for the attainment of peak bone mass (Bonjour et al., 2007; Makovey et al., 2007; Shaffer et al., 2008) and for longitudinal (length) bone growth (Lovejoy et al., 2003). Despite considerable research on genetic factors of bone growth (Sinclair and Dangerfield, 1998; Seeman, 1999; Stewart and Ralston, 2000; Rosen et al., 2002), the interplay between genetics and environment remain unclear (Seeman, 1999). In fact, to straightforward genetic contribution or relationship to age-related osteoporosis has been conclusively identified to date (Brickley and Ives, 2008). While osteoporosis does seem to run in families (Eisman, 1999), it is still unknown precisely how heritability changes along with the environment (Seeman, 1999). Cooper et al. (2006) have added that the observed heritability of osteoporosis may have less to do with genetics, and more to do with familial, intergenerational factors such as diet, lifestyle and physiology. Seeman (1999) has noted that heritability should not be perceived as fixed or constant. For example, a statement that 80% of bone mass is genetically determined while 20% is the result of environmental factors is flawed (Seeman, 1999). Moreover, if genetic variance does not change, and environmental variance increases, heritability decreases (Seeman, 1999). If this is true, then heritability is a fluid determinant of bone growth, one that changes with gene flow/drifts, changing environmental conditions, and over the life course (Brickley and Ives, 2008).

Reference to ‘racial’ differences in peak bone mass (Key and Bell, 1999; Nelson and Villa, 1999; 2003) have also garnered a great deal of attention. For example, African-Americans generally reach a higher peak bone mass and experience less bone loss than Caucasians of European descent (Key and Bell, 1999; Cho and Stout, 2003; Nelson and Villa, 2003). Higher bone mass in African-Americans may be the result of increased levels of circulating parathyroid hormone, vitamin D and more efficient conservation of dietary calcium (Slemenda et al., 1997; Heaney, 1999; Bilezikian and Silverberg, 2001). While the evidence for racial differences is compelling, Nelson and Villa (2003) are right to caution that differentiating between racial ‘groups’ is problematic, and that ethnicity may provide a more nuanced understanding of populations. By replacing race with ethnicity, researchers would be more able to account for biocultural contributors to osteoporotic risk, from diet and geographic origin, to religious practices and gender roles (Nelson and Villa, 2003). This is an important difference, as the data has shown. For example, if the high peak bone mass in African-Americans is purely genetic, it should be expected that any population of African origin would also have high peak bone mass (Nelson and Villa, 2003). Numerous studies in South Africa and Gambia (Solomon, 1979; Prentice et al., 1990; Patel et al., 1992; Daniels et al., 1995; Aspray et al., 1996) have shown the opposite, that peak bone mass in those groups is not greater than age-matched Caucasians living in Africa, and that in some cases, actually follows below the Caucasian values. In summary, our knowledge of genetic influences on bone mass remains elusive. While a genetic component to bone mass is present, the relationship is not straightforward and seems intimately linked to biocultural and environmental changes over the life course. Another risk factor that is worth mentioning is the ongoing debate about the roles of bone quantity versus quality and fracture risk.

The reliance on bone *quantity*, via DEXA, as the historic (and current) diagnostic gold standard is important as it gives clinicians gradients by which to judge advancing bone loss. However, accumulating evidence from observational studies has shown that DEXA does not predict fracture risk as well as originally thought (Ulrich et al., 1999; Riggs and Melton, 2002; Frost, 2003; Grynbas, 2003; Burr, 2004; Hudelmaier et al., 2004). In fact, in the clinical setting,

Sievänen et al. (2007) have shown that paradoxically, the overall percentage of various fragility fractures directly attributable to low BMD scores remains quite modest (up to 44% at most). When looking at all risk factors, BMD may only comprise about 15% of the risk for fracture (Sievänen et al., 2007). Ultimately, while BMD scores offer good predictive value of fracture risk at the population level, they perform very poorly at the individual level (Järvinen et al., 2007).

More recently, researchers recognized that bone strength, or a bone's ability to resist fracture, is a product of both its quantity, but also its *quality* (organization and structure) (Dempster, 2011). Measures of bone quality include the organization and connectedness of trabecular bone, changes in mineralization, remodeling history of cortical bone, microdamage and cross-sectional geometry (Bouxsein and Karasik, 2006; Carballido-Gamio and Majumdar, 2006; Kehoe, 2006). A number of obstacles have prevented clinicians from better integrating the qualitative factors into the diagnostic process. The first is cost, as DEXA is relatively inexpensive and is widely available (Brunader and Shelton, 2002). Many of the qualitative measures require advanced medical imaging such as Magnetic Resonance Imaging or Computed Tomography, which may not be widely available and are much more expensive to run and maintain. Sievänen et al. (2007) also presented a critique of the theoretical basis for bone quality. Sievänen et al. (2007) remind the reader that bone quality must be conceptually tied to bone quantity, as a BMD score is an aggregate measure of virtually everything that is measured. Secondly, Sievänen et al. (2007) noted that the bone quality concept is too nebulous and has had little benefit to patient outcomes because of a lack of standard diagnostic criteria. It is unlikely that any single measure will ever predict fracture risk with absolute certainty, given that fracture risk involves a great many factors that reside outside the skeleton such as propensity to falling (Järvinen et al., 2007). Sievänen et al. (2007) suggest that researchers should be more concerned with finding a measure of whole bone strength *in vivo*, rather than focusing on smaller constituent parts of whole bone strength. Fortunately, recent research in finite element analysis (FEA) (estimations of bone strength using computer models) is moving rapidly towards achieving this goal. Imaging modalities, usually MRI or peripheral quantitative computed tomography, capture images of three-dimensional bone structure, while values for bone strength are derived from experimental tests of material properties (Kazakia and Majumdar, 2006). In FEA studies, virtual tests of compression (mimicking *in vivo* forces) are then conducted on the MRI images to determine whole bone strength (Kazakia and Majumdar, 2006). Results of FEA studies have been very promising as FEA is a better predictor of fracture risk than BMD alone, or even combined with cross-sectional geometry data (Kazakia and Majumdar, 2006).

While the general concerns about the conceptual and practical problems of investigating bone quality put forward by Sievänen et al. (2007) have some merit, there is still much to be gained from examining individual, qualitative factors of whole bone strength. Measures of bone quality such as trabecular architecture, mineralization, bone turnover, microdamage, and cross-sectional geometry may not all be fully *independent* from bone mass (Sievänen et al., 2007), but they still comprise a suite of characteristics that can be explored to learn about how their interactions contribute to fracture risk. For example, even if bone mass is held constant, risk of fracture increases with age (Hui et al., 1988). Other studies have found near complete overlap in BMD scores between groups of individuals with and without fragility fractures (Melton et al., 1989). Experimental studies have also shown that small losses in bone mass have non-linear effects on bone strength, resulting in far greater loss of strength than the loss of bone mass alone can

explain (Wall et al., 1979; Mosekilde, 1990). Further, If BMD and fracture risk are highly correlated, why do very modest gains in BMD result disproportionately large reductions in fracture risk (Riggs and Melton, 2002)? These studies strongly imply that many of the measures that fall under the term “bone quality” are at least partially independent of bone mass. A perfect example of this is microdamage. Small cracks in cortical begin to accumulate rapidly in individuals over 40 (Schaffler et al., 1995). This accumulation of damage without loss of mass puts bone at risk for fragility fractures because the remodeling process becomes less and less effective at repairing damage with age (Grynpas, 2003). This also contributes to the hyper-mineralization of bone, rendering it brittle and prone to fracture (Currey, 1984; Vajda and Bloebaum, 1999). Cross-sectional geometry is also to some extent independent of bone mass. Long bones from two different individuals with identical BMD scores may have different bending strengths due to the cross-sectional distribution of bone around the neutral axis (Seeman, 2007). In the vertebrae, the bone density that is lost between males and females is similar, but males tend to lose bone through trabecular thinning, while females lose whole struts and thus connectivity (Seeman, 2007). The loss of connectivity rather than a more even thinning out of trabeculae places females at greater risk for vertebral fracture, despite having lost a similar amount of bone (Seeman, 2007). In summary, although the arguments proposed by Sievänen et al. (2007) about the utility of the bone quality concept have some merit in regards to diagnostic concerns, other researchers have clearly established that bone quality has tremendous value in understanding why bones break in some people, and not in others (Kazakia and Majumdar, 2006).

Whole bone strength, measured as a product of bone quantity and quality, is then a vital part in to understanding fracture risk (Dempster, 2011). But what influences whole bone strength? Clinicians identify a suite of biologically based risks, including advancing age, personal and family history of osteoporosis, race, body type (thinness), hypogonadism, and late onset sexual maturity (US Department of Health and Human Services, 2004; Pasco et al., 2005; Becker et al., 2010). Parallel to these risks are lifestyle factors that also contribute to whole bone strength. One of the more prominent lifestyle risk factors is nutrition, specifically excessive salt intake, low calcium intake and vitamin D deficiency (US Department of Health and Human Services, 2004 Pasco et al., 2005; Becker et al., 2010). A history of low or inadequate physical activity is also a serious concern (US Department of Health and Human Services, 2004 Pasco et al., 2005; Becker et al., 2010). Finally, cigarette smoking, high caffeine intake and regular alcohol consumption have also been shown to reduce bone strength (US Department of Health and Human Services, 2004 Pasco et al., 2005; Becker et al., 2010). While low bone mass is thought to present the highest risk for fracture, the more of these biological and lifestyle risks that are present, the greater the fracture risk will likely be (Dempster, 2011). Many of these risk factors are not applicable in the archaeological record, but some of the key contributors that are accessible, like diet, physical activity and reproductive history, are discussed in more detail in the final section of this chapter.

The prevalence of osteoporosis in Western societies is a serious ongoing concern for the medical community, with escalating financial, physical and social costs. By its heterogeneous nature, osteoporosis requires researchers to investigate multiple avenues of bone biology. It is in this complexity that bioarchaeology is able to enter the discussion with new perspectives from populations quite unlike modern Western societies to provide much needed perspective and context on human variation.



## **Bone Loss in the Past**

### *Measuring Bone Loss in the Past*

In the clinical setting, dual energy X-Ray absorptiometry (DEXA) analysis has become the dominant methodology in which research on understanding premature bone loss is conducted (Cummings et al., 2002). DEXA is also used in general studies of bone biology such as muscle/bone interactions (Ferretti et al., 2000) and growth and development (Sagesse et al., 2002; Gafni and Baron, 2007). DEXA is not an imaging technique in the strict sense of the word, rather an area of interest is scanned via two sources of radiation that are then absorbed by bone and soft tissues (Brickley and Agarwal, 2003). The bone mineral content in the area of interest is then scored as a density measure (e.g. g/mm<sup>2</sup>) so that bone mass can be estimated (Brickley and Agarwal, 2003). DEXA was originally developed to find a reliable way to predict fracture risk due to early bone loss (Peel and Eastell, 1995; Brickley and Agarwal, 2003). Brunader and Shelton (2002) have pointed out that DEXA provides high precision bone density data with a low radiation dose that can be used on all biologically meaningful areas of bone loss (across the skeleton as a whole). This is the main reason why DEXA became the gold standard. Compared to CT or MRI imaging, it is also cost very effective (Brunader and Shelton, 2002).

The ability to assess bone density is an important tool in bioarchaeological research as it allows hypotheses about age and sex differences to be tested (González-Reimers, 2002; González-Reimers et al., 2004; McEwan et al., 2005; Mays et al., 2006). Evolutionary changes in bone mass and the resultant implications for bone strength have also been examined (Nelson et al., 2003). Studies of ontogenetic changes in bone mass have also relied heavily on bone mass (Pearson and Lieberman, 2004). Lifestyle and cultural factors can also be compared between archaeological populations (Agarwal and Stuart-Macadam, 2003), as well as modern ones (Nyati et al., 2006). While DEXA is still advocated by some researchers (González et al., 2002; González-Reimers et al., 2004, Mays et al., 2006), there are some notable limitations to its bioarchaeological application.

The major assumptions of the method are that soft tissue is present and that all minerals encountered are hydroxyapatite crystals (the dominant mineral matrix in bone) (Brickley and Agarwal, 2003). These are standard expectations in clinical studies, but DEXA imaging on archaeological skeletal material must overcome the fact that there is no soft tissue present and that diagenesis (chemical exchange between the bones and the soil) may have altered the bone mineral matrix (Bennike et al., 1993; Kneissel et al., 1994; Brickley and Agarwal, 2003; González-Reimers et al., 2004). The absence of soft tissue can be dealt with water, rice, and other materials that can mimic human tissue for the purposes of a DEXA scan (Brickley and Agarwal, 2003). While not ideal, they are generally considered to be adequate when the scans are calibrated accordingly, although they are not directly comparable to scans of living patients as calculation errors remain (Brickley, 1998). Another critical issue is that DEXA scores that are produced as averages over a given area are problematic. Body size introduces error, such that larger bones provide much larger areal BMD scores than smaller bones. This is misleading, because if you correct for body size and take volumetric measurements, the smaller bone (in size) may have a higher bone density (Brickley and Agarwal, 2003; Damilakis et al., 2007). Researchers have improved DEXA scanning to where they can approximate true volumetric

density, but only quantitative computed tomography can provide real volumetric measures (Damilakis et al., 2007). Brickley and Agarwal (2003) note that volumetric measures of BMD have not improved risk assessment. Diagenesis, or the chemical exchange between bone and the surrounding soil, is a more serious confounding factor. There is no accurate way to assess the level of diagenesis in a bone without chemical or histological investigation, both of which are destructive (Brickley and Agarwal, 2003). With the severe limits diagenesis places on the reliability of DEXA scans on archaeological skeletons, researchers have turned to alternative methods to investigate bone maintenance and loss in the past.

One of the simplest ways of investigating age-related bone loss in the past is through direct visual examination of the skeletons. The most typical fragility fractures in people with osteoporosis are of the radius (Coles'), vertebral, and hip and are all readily observable on archaeological bone (Brickley and Agarwal, 2003). Simple visual examination has revealed the presence of fragility fractures in a number of archaeological case studies (Roberts and Wakely, 1992; Foldes et al., 1995; Mays, 1996; Dequeker et al., 1997; Mays, 2006). Although visual examination of archaeological skeletons provides the most direct and efficient assessment of fractures, there are a number of limitations to simple observation. First, there is generally no way through visual examination to distinguish if the fracture was a cause of primary or secondary osteoporosis (Brickley, 2000). Second, the timing of the fracture is often unclear, and may be no way related to advanced bone loss if it occurred earlier in the life of the individual (Brickley, 2000). Finally, fractures that may have resulted from traumatic events run the risk of being diagnosed as fragility-related fractures (Brickley and Agarwal, 2003).

Standard radiography has also played a role in expanding our knowledge of bone loss. In bioarchaeology, a widely cited study by Garn (1970) demonstrated through metacarpal radiogrammetry (radiographs of the metacarpals) that a strong correlation exists between the thickness of the cortical bone in the metacarpals and long bones with overall body bone density. The study also established that males and females do not have identical cross-sectional areas (Garn, 1970). Garn's (1970) hypothesis was that this difference arose in puberty with the release of sex hormones. As discussed earlier, this work has since been validated (Riggs et al., 2002; Ferretti et al., 2003; Saxon and Turner, 2005). This simple method has been used in subsequent research (Mays, 1996; Brickley and Agarwal, 2003; Ives and Brickley, 2004; Mays, 2006; Glencross et al., 2008) (see Chapter 5 for a detailed review). The work done with simple X-rays (and eventually computed tomography) has helped develop some basic foundational knowledge to the study of bone biology.

Simple radiography can also be used to measure the loss of trabecular bone in the area known as Ward's Triangle in the proximal (the neck) femur, or the Singh Index (Singh et al., 1970; Brickley and Agarwal, 2003). The proximal femur is X-rayed and the films are compared to written descriptive stages of bone loss (Brickley and Agarwal, 2003). While this method is a simple and cheap way to assess trabecular bone loss non-invasively, and has been used archaeologically (Mielke et al., 1972), it was found that inter-observer repeatability is a significant problem (Brickley, 1998). The usefulness of the Singh method in accurately reflecting trabecular bone loss and fracture risk, irrespective of repeatability, has also been recently questioned recently (Nazarian et al., 2007; Sah et al., 2007). The central problem is the regional variability in bone strength throughout trabecular bone in femoral neck (Nazarian et al., 2007). However, Nazarian et al. (2007) have developed a method using advanced computed



tomography to account for this variability and produce a more useful index to predict fracture risk. The standard Singh index is still considered of little clinical value and is rarely used in bioarchaeology.

Cortical bone histomorphometry provides another highly useful investigative tool as it examines bone at the tissue level. Histomorphometry measures microscopic changes in bone and its theoretical principles are based on the fact that bone is in a constant remodeling state (Wu et al., 1970; Stout and Simmons, 1976; Stout and Lueck, 1995; Mulhern, 2000; Cho and Stout, 2003; Robling and Stout, 2008). The primary advantage of histomorphometry is that it can assess the metabolic activity and balance of bone directly (Brickley and Agarwal, 2003). While living bone cells are no longer present in archaeological bone, the products of their actions (osteons and their fragments) remain and can be counted and measured to estimate the level of remodeling activity (Mulhern, 2000; Cho and Stout, 2003). This can aid in the differential diagnosis (refining diagnoses down from multiple possibilities) of osteoporosis if fragility fractures are not present (see Chapter 5). Although the advantages of histomorphometry are numerous (age determination, changes at the tissue level, pathological diagnosis), the methodology does present some challenges to bioarchaeologists. The technique requires the production of histological bone thin sections, a destructive process that may limit the availability of the method if descendent groups are adverse to destructive procedures. The production of thin sections also requires expensive laboratory equipment and materials, although simpler and cheaper alternatives are available (Beauchesne and Saunders, 2006). Additionally, diagenetic effects are frequently present (Hackett, 1981; Schultz, 2003) and limit sample size. There are theoretical challenges as well, as the static bone structures that are examined represent a complex interplay between normal aging, biomechanical forces, and metabolic activity (Robling and Stout, 2008). Even with these challenges, histomorphometry has become an important part of the bioarchaeological toolkit in the investigation of bone loss in the past.

The interpretation of biomechanical data from long bones obtained via cross-sectional geometry is also a major research area in bioarchaeology (Ruff, 2000). Biomechanics is the application of engineering principles, primarily beam theory, for the structural analysis of bone (or other biological material) (Robling and Stout, 2003), often to determine their mechanical strengths through geometric space (Ruff, 2000). Bending strengths and resistance to torsional forces are estimated using cross-sectional area, and must control for bone length and body mass to form biocultural interpretations based on physical activity (muscle force) (Robling and Stout, 2003). Cross-sectional geometry is important because it can inform us about growth and development, evolutionary trends, sex differences, and age changes (Ruff, 2000; Ruff et al., 2006). The effect of lifestyle factors, such as activity patterns can also be inferred (Stock and Pfeiffer, 2001) and thus behavioral patterns in the past can potentially be reconstructed. Cross-sectional geometry can also be examined alongside histomorphometry of cortical bone to explore biocultural effects on the skeleton at the structural and tissue levels simultaneously (Robling and Stout, 2003). Another advantage of cross-sectional geometry is that it is almost always conducted non-invasively, although this was not always the case (e.g. Lovejoy and Trinkaus, 1980; Burr et al., 1981; Burr and Piotrowski, 1982; Drusini et al., 2000). The primary limitation of using cross-sectional geometry is preservation (Robling and Stout, 2003). Skeletons are often incomplete, limiting the availability of cross-sectional measures or long bone lengths. Cross-sectional geometry also requires some form of medical imaging analysis, usually computed

tomography. Access to CT imaging may be limited in certain countries. However, photographic images of broken cross-sections can be taken and present a viable option (Ruff, 2008). Additionally, if only radiographs are available, cortical thickness measures from the radiographs can be combined with molds of the outer cortex of the bone to estimate the true cross-sectional shape of the bone to within 5% of direct measures using CT (Ruff, 2008). Finally, it can be quite difficult in cross-sectional area studies to tease apart the biomechanical, ontogenetic and life history influences have on the cross-sectional shape of long bones (Agarwal, 2008).

The quantitative and qualitative analysis of trabecular bone tissue has become a standard method for measuring bone maintenance and loss in the past (Agarwal, 2001; Agarwal et al., 2004; Agarwal 2008). Trabecular bone samples can be examined as thin sections, photographs of thick sections, with radiographs, or through more complex imaging methods such as computed tomography (CT) (Brickley and Agarwal, 2003). Computed tomography has a number of advantages as it provides a non-destructive avenue to three-dimensional images of trabecular bone. The distinction between 2-D and 3-D methods of analysis have important biomechanical implications for trabecular bone analysis. For example, the assessment of trabecular connectivity is only possible using 3-D methods and clinical studies have shown that trabecular architecture, independent of mass, is an important part of trabecular bone strength (Kleerekoper et al., 1985) and bone quality (Brickley and Agarwal, 2003). The primary advantage of trabecular bone analysis is that it can provide measures of both bone quantity and quality (Brickley and Agarwal, 2003) (see Chapter 5). Analyses of trabecular bone are vitality important in bioarchaeology because trabecular bone is more metabolically active and is thus more sensitive to premature bone loss (Compston, 1999). The primary limitation of trabecular image analysis is the availability of medical imaging facilities (Brickley and Agarwal, 2003). Agarwal (2008) also points out that the processing and interpretation of trabecular images often requires expertise and specialized image analysis software.

### *Fragility Fractures in the Past*

While bone loss and osteoporosis in the past has been investigated using all of the methods just described, the prevalence ultimately remains unclear (Brickley and Agarwal, 2003; Agarwal, 2008; Brickley and Ives, 2008). Archaeological skeletal samples have shown conflicting patterns of bone loss, with some demonstrating similar patterns of age and sex-related bone loss, while others do not, and many differing from the typical age- and sex-related patterns of bone loss and fragility observed in modern Western populations (Agarwal 2008). For example, bone loss is often seen in young age and in both males and females, and there is a low prevalence of fragility fracture in comparison to modern populations (Lees et al., 1993; Ekenman et al., 1995; Agarwal and Grynepas, 1996; Weaver, 1998; Holck, 2007; Agarwal and Grynepas 2009). Bone loss with age is often very similar between the sexes (Ekenman et al., 1995; Brickley, 2002; Agarwal et al., 2004; Holck, 2007), a pattern that deviates substantially from Western societies today, where females experience far greater bone loss and higher rate of fracture (Agarwal, 2008). However, Brickley and Agarwal (2003) caution that a number of confounding factors must be accounted for when assessing the prevalence of fragility fractures in the past. First, the lack of fragility fractures may be a reflection of the hidden heterogeneity in frailty of archaeological skeletal populations, where the individuals we observe in old age

represent a ‘healthier’ group that survived, and thus do not represent the population as a whole (Wood et al., 1992; Brickley and Agarwal, 2003). There is also the concern that less people reached old age in the past, although Jackes (2000) has shown that this concern is more a product of high infant mortality and that human longevity has remained largely unchanged. Even with these concerns in mind, the inter and intra-population variation in bone loss in the past strongly support the position that patterns of bone loss across dramatically different cultures should not be expected to follow that of modern Western societies. Furthermore, there have been rapid changes even in modern populations; Melton (1995) has shown that the incidence of fragility fractures doubled between 1975 and 1995 in Western societies. Thus, the current epidemiological patterns of osteoporosis are indeed a “recent phenomenon” (Robling and Stout, 2003: 189), and speak strongly to the malleability of the human skeleton throughout the life cycle.

Differences in bone quality may lie at the heart of the discrepancy between populations. Many studies clearly demonstrate that osteopenia was present in the past (Eriksen, 1976, 1980; Richman et al., 1979; Thompson and Gunness-Hey, 1981; Martin and Armelagos, 1985; Cho and Stout, 2003). However, many researchers have observed that fragility fractures were uncommon (Lees et al., 1993; Ekenman et al., 1995; Agarwal and Grynepas, 1996; Weaver, 1998; Holck, 2007; Agarwal and Grynepas 2009). This may indicate that qualitative differences are important in understanding divergent patterns between populations, as loss of bone mass is a common feature. Ongoing clinical research (Felsenberg and Boonen, 2005; Carballido-Gamio and Majumdar, 2006) has supported this emphasis on bone quality, and there is no doubt that bone quality, in concert with bone quantity, plays a vital role in determining osteoporotic risk.

## **Biocultural Factors of Bone Loss**

### *Hormonal Changes and Bone Growth*

Sex hormones are integral to skeletal growth and development (LeBoff and Glowacki, 1999; Warren, 1999; Wang et al., 2004). The role of estrogen will be emphasized here because of its role in postmenopausal bone loss in women, but testosterone is also vital to skeletal maturation (LeBoff and Glowacki, 1999). Estrogen is a sex hormone composed of both steroidal and nonsteroidal components that are capable of inducing estrus (Weitzmann and Pacifici, 2006). The various components of estrogen are produced in different sites in the body, including in fat, the placenta and in the ovary (LeBoff and Glowacki, 1999). Adipose tissue produces the greatest amounts of estrogens (LeBoff and Glowacki, 1999). Estrogens have important direct effects on skeletal growth but they also interact with other hormones such as PTH, IGF-I and testosterone (Leboff and Glowacki, 1999; Riggs et al., 2002). The biochemical synthesis and metabolism of estrogen will not be discussed in greater detail here (see LeBoff and Glowacki, 1999; Riggs et al., 2002; Saxon and Turner, 2005; Weitzman and Pacifici, 2006 for detailed discussions of estrogen biochemistry). The emphasis of this section will be placed instead on the effects that estrogen has on the growing and aging skeleton. The term estrogen will be used here with the recognition that it implies a series of hormones (estrone, estriol and estradiol).

Estrogen and testosterone are part of a larger hormone family (sex steroids) that has a crucial role to play in skeletal growth (Riggs et al., 2002; Weitzmann and Pacifici, 2006). During the last twenty years of research it has been shown conclusively that estrogen has a direct effect

on bone cells (Weitzmann and Pacifici, 2006). All the major bone cells described in the biology review section (osteoblasts and osteoclasts) have receptors ER $\alpha$  and ER $\beta$  that sense and respond to estrogen fluctuation (Weitzmann and Pacifici, 2006). Interestingly, these receptors are not distributed throughout bone homogeneously. Weitzmann and Pacifici (2006) note that ER $\alpha$  is predominant in cortical bone, while ER $\beta$  is far more common in trabecular bone. ER $\alpha$  is thought to be the more active or sensitive receptor in bone cells (Weitzmann and Pacifici, 2006). Perhaps most importantly, estrogen appears to preferentially induce bone formation on the endosteal surface via ER $\alpha$ , while signaling through ER $\beta$  to inhibit periosteal apposition (Saxon and Turner, 2005).

At the organ level estrogen acts to help conserve bone mass (Riggs et al., 2002). This is accomplished at the tissue and cellular levels by reducing the activation frequencies of basic multicellular units (osteoclasts and osteoblasts), or the rate at which new BMUs are created to remodel bone (Riggs et al., 2002; Weitzmann and Pacifici, 2006). The increased activation frequency in menopause due to a drop in estrogen that is accompanied by a longer period of resorption and a shortened formation period (Riggs et al., 2002). This in turn prevents the osteoblasts from completely filling in resorption cavities, resulting in a net loss of bone (Riggs et al., 2002). This is particularly damaging in trabecular bone as this unbalanced remodeling perforates the trabeculae and greatly advances loss in connectivity and mechanical competence (Riggs et al., 2002). The protective effect of estrogen is accomplished in part by reducing the formation of osteoclasts as well as their lifespan (Riggs et al., 2002). Controversy exists over the effect of estrogen on osteoblasts, but Riggs et al. (2002) suggest that estrogen may have the opposite effect on osteoblasts that it does on osteoclasts. Thus, estrogen conserves bone mass by reducing the active remodeling space, which decreases porosity in cortical bone and preserves trabecular integrity in trabecular bone (Weitzmann and Pacifici, 2006).

Martin (2003) hypothesizes that the protective effect of estrogen is a result of a compromise between functional and metabolic demands of female mammals (the demands of pregnancy and lactation). Pregnancy and lactation require that some bone be metabolized (Martin, 2003; Agarwal and Stuart-Macadam, 2003). Bone that is lost on the endosteal surface will contribute far less to a reduction in mechanical competence than bone that is lost on the periosteal surface (Martin, 2003). Since trabecular and endosteal bone is more metabolically available (Compston, 1999), the overall picture we see in how females deposit bone makes evolutionary and biomechanical sense.

Prior to puberty, girls and boys are nearly identical in terms of skeletal size and volumetric bone mineral density (Riggs et al., 2002). The rapid increase in sex steroids at puberty explains a large portion of the difference we see post-pubescence (Riggs et al., 2002; Wang et al., 2004). Serum levels of estrogen are responsible for the pubertal growth spurt, as well as the end of growth when epiphyseal ends fuse (Riggs et al., 2002). It is believed that estrogen may also contribute to longitudinal growth in addition to the well-established appositional growth (Martin, 2003).

Girls begin puberty typically before boys do and they have higher serum levels of estrogen (Riggs et al., 2002). This means that they grow sooner and faster than boys. The elevated level of estrogen in girls compared to boys also helps explain why bone is preferentially

deposited at the endosteal surface rather than on the periosteal surface, as boys do. Recall that ER $\beta$  receptors in bone cells along the periosteal surface inhibit periosteal expansion, while endosteal ER $\alpha$  receptors signal for deposition of new bone (Saxon and Turner, 2005). It has been noted that postmenopausal women once again resume a greater rate of periosteal apposition similar to men, but endosteal resorption is also increased because the protective effect of estrogen (on activation frequency) has been lost (Saxon and Turner, 2005). Furthermore, it has been found in animal studies that androgens in males help to increase periosteal expansion while they appear to have little effect endosteally (Saxon and Turner, 2005). This has also been supported in human studies (Orwoll, 1999). The rate of osteoporotic fracture for women is much higher than men because men preferentially deposit bone along the periosteum as they age, leading to larger cross-sectional areas and increased bone strength (Saxon and Turner, 2005).

### *Biocultural Interactions and Bone Loss*

As new data continues to be produced on how and why males and females develop differently from a strictly biological perspective, a number of researchers are realizing and exploring the role of culture in shaping these intrinsic patterns (Worthman, 1995; Bogin, 1999; Agarwal and Stuart-Macadam, 2003; Fausto-Sterling, 2005). The Western experience of osteoporosis is not universal because the risks women face for developing osteoporosis are greatly mediated by extrinsic and independent factors from the menopause-induced drop in estrogen levels (Agarwal and Stuart-Macadam, 2003; Agarwal, 2008). For example, nutrition, physical activity (operating through gender roles), and child rearing practices are heavily cultured variables that can alter growth and development through hormonal shifts or other pathways. Clearly these variables should be explored in any bioarchaeological skeletal analysis if biocultural interpretations are being used. This section will first show how cultural practices relating to pregnancy and lactation can affect hormone levels. The second aim of this section to provide more examples of biocultural bone growth through nutrition and physical activity. Changes in females are emphasized in this section over males because the focus is on how bone growth and development can contribute to post-menopausal osteoporosis, which is typically perceived as universal and female specific. It should be noted that most of the arguments presented below apply to males as well, with the exception of pregnancy and lactation.

Worthman (1995) has noted that hormones are not shielded from the external world. For example, stress can cause delayed or altered growth and interfere with proper digestion (Worthman, 1995). As a result, hormones help generate phenotypes through a development process unique to each individual and via a series of changing contexts, and cannot be attributed to direct genetic causes (Worthman, 1995). Worthman (1995) argues that there are three reasons for this: genetic diversity, developmental adaptability (plasticity), and the responsiveness of hormones to environmental stimuli, which guide the entire developmental sequence. So while sex hormones may strongly influence the nature of male and female bone morphology as a general rule, those hormonal influences are not by any means fixed across individuals or populations.

The ages at which menarche and menopause occur are also not universally standard (Sievert, 2006). The age at menarche has been decreasing with time (in industrialized societies) (Eveleth and Tanner, 1990, cited in Sievert, 2006). This is likely directly related to the increase in

body fat in industrialized societies as body fat has a significant effect on estrogen levels and age at menarche (Murphy and Carroll, 2003). The age of onset of menopause has been more stable, but its manifestation is highly variable (Sievert, 2006). The earlier onset of menarche has important repercussions for growth and its delayed effects on bone loss at menopause. Parallel to these issues are the number of menstrual cycles a woman has during her lifetime, as this will affect lifetime exposure to estrogen. This can be tremendously varied. For example, women in non-industrialized societies typically have around 48 menstrual cycles while women in Western industrialized societies average approximately 420 cycles (Trevathan, 2007). This diversity in experienced menstrual cycles is primarily a product of pregnancy and lactation history. Women in non-industrialized societies typically have on average more children than women in industrialized societies (Agarwal and Stuart-Macadam, 2003). Estrogen levels are increased during pregnancy, up to 100 fold (Wizemann and Pardue, 2001), which may provide a protective effect on the maternal skeleton (Agarwal et al., 2004). Periods of lactation are also typically much longer in non-industrialized societies, which are accompanied by a probable long period of lactational amenorrhea (Agarwal and Stuart-Macadam, 2003). Agarwal and Stuart-Macadam (2003) suggest that these patterns were similar in the past, that in fact they have defined our evolutionary past. Deviation from this pattern through new cultural norms in Western societies may help explain why the evidence of a high prevalence of postmenopausal osteoporosis does not exist in archaeological populations (Agarwal and Grynbas, 1996; Agarwal, 2008).

Shahtaheri et al. (1999) have shown that while pregnancy does create temporary loss of bone, this loss is completely compensated for by the creation of a more complex (but thinner) latticework of trabeculae by late pregnancy. The mechanisms by which new trabeculae are created (post-modeling) remain unclear according to Shahtaheri et al. (1999). Perhaps the increased levels of estrogen in pregnant women are responsible. It may be that the protective role  $ER\alpha$  has on preserving endosteal bone has another similar role to play during pregnancy. The synergistic relationship between estrogen, mechanical loading and bone growth Martin (2003) argued for may also be at play here. It has been shown as well that multi-parous women and women who breast feed for extended periods (greater than 6 months) suffer no long-term negative effects in terms of bone health (Lenora et al., 2009). Multi-parity and extended lactation appear to contribute to skeletal health rather than jeopardize it.

The reproductive history of modern Western women may actually have a wider impact on bone loss. As was mentioned previously, modern Western women typically have many more menstrual cycles in their lives compared to women in non-industrialized societies (Trevathan, 2007). Gurevitch and Slavin (2006) have proposed that this chronic (what they term ‘excessive’ need for blood) bleeding is in fact largely responsible for the prevalence of osteoporosis today. The model Gurevitch and Slavin (2006) present can be summarized as follows: chronic bleeding puts strain on the hematopoietic tissues (blood producing) that respond by stimulating bone development (as in the case of anemia). This increase in osteogenic progenitor cells then triggers the development of osteoclast recruitment (Gurevitch and Slavin, 2006). Endosteal resorption follows and the marrow cavity is expanded (Gurevitch and Slavin, 2006). It is thought that the expansion of hematopoietic areas is meant to help the body “keep up” with the demands of chronic bleeding (Gurevitch and Slavin, 2006). This cycle is exacerbated as we age given the “gradual depletion of both stromal and hematopoietic progenitor cells” (Gurevitch and Slavin, 2006). When the protective aspects of estrogen are removed during menopause, the result is a

rapid acceleration in endosteal expansion, contributing significantly to the risk of developing osteoporosis (Gurevich and Slavin, 2006). While this hypothesis remains to be tested much more rigorously, it demonstrates the profound effect cultural practices can have in shaping the body. Reproductive factors unquestionably play an important role in explaining changing patterns of bone maintenance and loss through time. However, physical activity and diet/nutrition are nearly as crucial.

The dramatic subsistence changes that mark the start of the Neolithic involved increasing sedentary lifestyles. Studies of cross-sectional geometry have explored how this transition affected bone strength. Individuals in agricultural communities typically show reduced bone strength as the cross-sectional areas are smaller compared to hunter-gatherers (Larsen, 2002; Ruff et al., 2006; Ruff, 2008). However, activity patterns and work loads were still demonstrably more demanding than those experienced by modern Westernized societies (Agarwal, 2008). Some archaeological agricultural communities even show greater cross-sectional bending strengths than hunter-gatherer populations (Bridges, 1991). It is hypothesized that demanding day-to-day workloads in the past could have helped protect individuals from fragility fractures (Agarwal, 2008). Life long physical activity could not only have improved bone cross-sectional geometry, but musculature as well. Given that a large proportion of fracture risk involves propensity to falls (Sievänen et al., 2007), a more robust musculature would have aided balance and also helped protect the body in the event of a fall (Englund et al., 2011). Physical activity may also affect cortical and trabecular bone in different ways. For example, in the medieval Wharram Percy population, cortical patterns of bone loss appear advanced and authors suggest that physical activity was not sufficient to protect individuals from bone loss (Mays, 1996; Mays et al., 1998; McEwan et al., 2005). However, patterns of trabecular bone loss in vertebrae showed no change in trabecular structure from middle to old age, for either sex (Agarwal et al., 2004). Agarwal et al. (2004) point out that there are very few fragility fractures at Wharram Percy, suggesting instead that physical activity could have indeed protected against fracture, despite more advanced cortical bone loss. Interpreting the influence of physical activity is also complicated by the fact that the body responds with varying sensitivity with age (Pearson and Lieberman, 2004; Ruff et al., 2006). There is also a lack of consensus about the necessary loads required to improve bone strength (Agarwal, 2008). Research has shown that the skeleton is most responsive during growth and development, primarily in adolescence, and that the body's response to physical activity diminishes afterwards (Pearson and Lieberman, 2004; Rittweger, 2006). Some authors have shown that high strains can alter bone mass and geometry in older age groups (Rittweger, 2006; Ruff et al., 2006). Ultimately, physical activity has a complex relationship with bone strength, as it affects not only cross-sectional geometry and trabecular architecture, but tissue-level properties as well (Agarwal, 2008). For example, a study of the Pecos archaeological sample by Burr et al. (1990) demonstrated that while endocortical bone loss was observed in both sexes, osteon sizes were smaller, allowing for a greater density of osteons throughout the cortex. The authors suggest that increased osteon densities and increased periosteal deposition could have helped retain bone strength, even with substantial endocortical bone loss (Burr et al., 1990). Ideally, future studies of the effects of physical activity on bone maintenance and loss should include multiple measures to provide a more robust representation of bone strength.

One of the primary advantages of considering physical activity in studies of bone

maintenance and loss is that day-to-day work is a gendered practice and is certainly mediated by culture. Past populations present exciting opportunities to compare cultures with egalitarian distribution of work to those with highly gendered work roles (see Chapter 7). For modern Western women, greatly reduced levels of physical activity (on average) have resulted in decreased muscle mass throughout their lives. It is thought that this substantial reduction in muscle mass is a main contributor to Type I osteoporosis (Frost, 1997; 2000; Riggs et al., 2002). Lower levels of physical activity have also contributed to advanced bone loss in males (Khosla et al., 2008), but males are more protected by longer growth periods and the preferential deposition of bone on the periosteal surface, which improves bending strength (Martin and Burr, 1989). The problem of lowered physical activity is compounded by the fact that many women today start and end growth earlier, which lowers peak bone mass and cross-sectional geometry, which extends the period of bone loss before menopause. When menopause begins, the drop in estrogen levels are more detrimental than they would be otherwise because the quantity and quality of the bone was worse at that same point in time than in past populations or as we see in many non-industrialized societies today due to muscle/bone inactivity.

Dietary practices are also highly related to skeletal growth and development. Much like the Neolithic transition had a profound effect on physical activity, dietary changes during this period were equally substantial. The change from a hunter-gatherer diet to one based on agriculture altered human nutrition in a number of important ways. First, agricultural diets tended to lack nutritional variety, although this would have varied between populations and regions (Larsen, 2002; Nelson et al., 2003). Secondly, many agricultural diets were based on grain, which increased phytate in the diet (Larsen, 2002; Nelson et al., 2003). Phytate binds with calcium and would have further reduced the bioavailability of calcium in the body (Larsen, 2002; Nelson et al., 2003). While calcium is clearly an important nutrient for bone health (Rizzoli et al., 2008), calcium is only a single component in a suite of nutrients that have a positive effect on bone health. For example, recent clinical work has cast some doubt on the importance given to calcium in the public consciousness. Calcium supplementation has been shown to be insufficient in preventing bone loss (Dawson-Hughes, 1991; Elders et al., 1994), fractures (Cummings and Klineberg, 1994; Feskanich et al., 1996) and is in fact correlated with hip fractures (Abelow et al., 1992; Feskanich et al., 1996). This paradox is thought to be explained by chronic deficiencies in vitamin D in North American and European countries (Vieth, 2005; Agarwal, 2008), along with parallel increases in obesity and sedentary lifestyles (Nelson et al., 2003). Vitamin D is essential for the proper mineralization of newly create bone tissue (Vieth, 2005) and is an ongoing concern in the treatment of osteoporosis (Rizzoli et al., 2008). Although examples of vitamin D deficiency exist in the past, they are very uncommon (Ortner, 2003). Regular exposure to the sun from working primarily outdoors probably contributed the most to limiting vitamin D deficiency in the past (Mays, 2006).

Malnutrition in agricultural communities in the past has also been extensively considered as contributing to bone loss. Numerous studies, using multiple methods, have hypothesized that chronic undernutrition in the past was a primary cause of bone loss in the past (Ericksen, 1976; Martin and Armelagos, 1979; Martin, 1981; Mays, 1996; Mays, 2006). Peak bone mass seems to be lower in past (Mays, 1996; 2006) and bone loss is often observed in younger ages (Agarwal et al., 2004). However, it is also difficult to separate the effects of undernutrition with the unsanitary living conditions, and higher pathogen loads common in the past (Agarwal, 2008).



Another aspect of nutrition that has been considered to explain the rapid increase in the prevalence of osteoporosis in modern Western societies is the over-consumption of protein. Anthropologists have observed that Inuit populations have shown strong correlations between their high protein diet and bone loss (Mazess and Mather, 1975; Thompson and Gunness-Hey, 1981; Pfeiffer and Lazenby, 1994; Nelson et al., 2003). The relationship between protein and bone health is complex however, and depends on the total diet, and not protein consumption alone. Clinical studies of protein consumption and bone health have suffered from a lack of standardized methods. This greatly hinders any useful comparison between the disparate studies. For instance, sample sizes range in size from 4 (Spencer et al., 1983) to 86, 000 (Feskanich et al., 1996) individuals, and some studies (Spencer et al., 1983; Roughead et al., 2003) only measured physiological changes for a few weeks. Similarly, longitudinal studies are rare, and when accomplished, suffer from inconsistent reporting of dietary intakes (Cooper et al. 1996). Part of the lack of standardization is that there exists no operationalized definition of a high protein diet. The range of 'high' protein diets ranges from below 100g a day to well over 200g. Considering that every study uses different levels of protein (in addition to variable levels of other macro and micronutrients), it makes meaningful comparisons and repeatability very difficult.

Compounding the problems of methodological variation and weakness, there exists an incomplete understanding of the complex interactions between the various micronutrients found in our generally omnivorous diet. For instance, although it is generally accepted that excess protein leads to an increase in endogenous body acid, it has been argued the high phosphorus content in meat protein counteracts this negative effect, thus neutralizing the detrimental effects of the high acid load (Spencer et al., 1983; Cooper et al., 1996). However, Feskanich et al. (1996) stated that although phosphorus may have a beneficial effect in combating acidosis, it decreases the production of vitamin D, an essential vitamin for bone mineralization. Furthermore, Wohl et al. (1998) have shown that fat, a significant component of the Western diet, can have detrimental effects on the production and maintenance of cancellous bone. Ultimately, the nature of this debate highlights the fact that the methodological trend of isolating one or a few micronutrients at a time for study is flawed, and that future research will have to incorporate methods that can evaluate the interactions between wide varieties of micronutrients, as are found in 'free-living' diets. This is a significant challenge that will not be resolved for some time given the complexity involved in such a situation.

If gender roles and behavior are malleable, then so are hormonal milieus. Consequently, this requires that we move beyond a consideration of static and universal biological sex differences to one where culture is considered to have a real effect on the body. In turn, this may drastically change our perception of what normal growth and aging processes are like. The following chapter builds on the examples outlined in this chapter to define the life course approach. The life course approach is the main theoretical pathway used in this dissertation and it attempts to link biocultural influences, such as nutrition, activity and reproductive history with bone growth and development, as well as bone maintenance and loss throughout the entire life cycle.

This section has demonstrated how biological and cultural variables intersect by focusing on differences in sex-related growth and development patterns, and what that might mean for

explaining variation in bone loss across cultures. The purpose here has been to challenge assumptions about the fixity of biologically regulated growth and development patterns and that women are bound to face post-menopausal osteoporosis, for there is compelling evidence to the contrary (Farwell and Molleson, 1993; Agarwal, 2001; Agarwal and Stuart-Macadam, 2003; Nelson et al., 2003; Fausto-Sterling, 2005; Agarwal, 2008). In doing so, this will help highlight the theoretical and practical utility of the life course model used to investigate bone maintenance and loss in this research (see Chapter 3).

## Chapter 3 - Lifecourse Theory in Bioarchaeology

### Biocultural Perspectives in Bioarchaeology

Popular descriptions of the human skeleton often construe it as a dry and inert material, essentially unchanged over the lifetime. In actuality skeletons are a dynamic, living tissue that has the ability to shape itself over the life course. The dynamic nature of the skeleton resides in its basic biology – at its cellular level, bone tissue is able to respond to the physiological and biomechanical needs of the body. The fact that the skeleton can respond and adapt to the biological and cultural environments forms the basis for the central tenet of bioarchaeology. The well-established biocultural approach in bioarchaeology emphasizes the importance of the interaction between humans and their larger social, cultural, and physical environments, recognizing that the skeleton is influenced by environmental variables (Larsen, 1997; Steckel and Rose, 2002; Zuckerman and Armelagos, 2011). This approach has been the cornerstone of bioarchaeology in investigating patterns of skeletal health and disease (Armelagod et al., 1972; Mays, 1996; 1999; Agarwal et al., 2004; Cho and Stout, 2003; Paine et al., 2009; Klaus and Tam, 2009) and is particularly useful in studies that seek to sort out the influences that may have affected bone aging and bone loss in past populations because of the multifaceted nature of bone loss (see Chapter 2).

However, even within biocultural models, environmental and cultural effects on skeletal maintenance and bone loss are often viewed as secondary modifiers that are subservient to biology. For example, while lifestyle factors such as reproductive behavior (parity and/or breastfeeding) (Poulsen et al. 2001; Turner-Walker et al. 2001; Mays et al. 2006) or diet (Martin 1981; Martin and Armelagos 1979, 1985) are considered to influence bone maintenance in the past, they are still only considered as isolated agents that exacerbate inevitable biological (hormonal or genetic) changes to bone loss. As such, indications of bone loss or osteoporosis in the past are often regarded to reflect the irreversible course of menopause and aging (Mays 1996; Mays et al. 1998; Macho et al. 2005). Further, bioarchaeologists often hypothesize about the influence of environmental factors on bone morphology over a short period of time during the life of an individual(s) or during a distinct phase of the life cycle (typically the adult and post-menopause phase). This is in part due to the nature of archaeological samples that obviously do not permit looking at changes in morphology longitudinally over a given individual's life cycle. Skeletal samples permit only cross-sectional studies of bone loss and fragility and generally attract focus on individuals with unusual pathology, rather than lend themselves to life course approaches in the study of bone health. The result, however, is that while bone loss and fragility fracture have been widely reported and studied in bioarchaeology, they are regarded primarily as the result of skeletal degeneration that reflects senescence of the body (Agarwal 2008). In bioarchaeological studies the focus on bone maintenance and loss is at the end of the life cycle, particularly in females. The *a priori* assumption is that it is inevitable that women will lose bone and have more fragile skeletons (Agarwal 2008).

The assumption that bone maintenance and bone loss is tied entirely to menopause and old age is well perpetuated in popular biomedicine. While the level of sex steroids plays a vital role in bone maintenance across the life cycle in both sexes, particularly in old age, it is

increasingly well known in clinical and epidemiological studies that there are many other biological and environmental influences on bone health that can change the outcome of bone loss and fragility. For example, biomechanical influences (physical activity), reproductive behaviors, diet and nutrition are just some of the factors now known to interact and potentially change the course of adult bone maintenance and loss (Sowers and Galuska 1993; Stevenson et al. 1989; Ward et al. 1995). While bioarchaeologists have strived to investigate environmental influences on bone health in past populations, it seems they are tied to the notion that the biological influences of menopause and senescence are primary. This may be related in part to fact that bioarchaeological approaches to bone maintenance and aging are also shaped within, and struggle against, the larger framework of biological anthropology that gives primacy to biology and the gene in explaining bone morphology. In these developmental biological frameworks the morphology of the skeleton is seen as limited by regulatory mechanisms and a set range of possible responses in human tissue (Lovejoy et al. 2003). While insights from development biology have been revolutionary in our analyses of the evolution of the human primate skeleton, they should not overshadow the importance of postnatal influences on bone morphology during growth and aging. These non-predetermined influences do not act in isolation, and often act synergistically with one another and with biological (genetic, hormonal) influences on bone morphology. More importantly, these influences act throughout the life course, beginning even *in utero*, to shape the skeleton (Cooper et al. 2006; Winsloe et al., 2009). The adult-aged skeleton, in both its strength and frailty, is the creation of life history and trajectories taken during growth.

This chapter outlines the lifecourse approach utilized in this dissertation by tracing its theoretical foundations. The lifecourse approach functions as a general set of guiding principles, as its purpose is to examine alternative perspectives on human morphology that are the result of development and plasticity, and how these perspectives can be applied to understanding growth and aging of the human skeleton. As the lifecourse approach is firmly set within a biocultural framework, theoretical issues of biocultural theory are reviewed first. This is followed by a discussion of life history theory in biological anthropology and its connection to the plasticity concept. Next, the processes of growth, development and plasticity are explained in the contexts of both clinical and bioarchaeological research. This review of developmental plasticity is crucial as it forms the central the application of the life course approach in this project and has not been articulated to date in bioarchaeology. The few previous applications of developmental approaches in bioarchaeological studies of bone maintenance and loss in past populations are also reviewed. The chapter concludes with a review of new directions in the study of maintenance and aging of the skeleton that are possible with the integration of ideas in both biological and social theory on the role of ontogenetic process and embodied lived experience in the construction of skeletal form.

### **Problems with Biocultural Models in Bioarchaeology**

Biocultural approaches have emerged out of the past forty to fifty years of internal debate as effective unifying models in biological anthropology (Goodman and Leatherman, 1998; Zuckerman and Armelagos, 2011). Prior to the development of biocultural approaches, research emphasized description and (often racial) categorization (Zuckerman and Armelagos, 2011). Biocultural approaches have faced numerous challenges within biological anthropology. First, defining biocultural theory is surprisingly difficult because there simply is no standardized

usage; biocultural theory exists more as a generalized approach to anthropological work, rather than as a well-defined theoretical perspective (Dressler, 1995). Biocultural models range from the very ecologically (external to culture) based (Hanna et al., 1989; Beall and Steegman, Jr., 2000) to relying almost exclusively on socio-cultural data (Scotch, 1963). When infectious diseases are the subjects of research, epidemiological models are often blended with biocultural data (Kuh and Ben-Schlomo, 1997; Sattenspiel, 2000) since social behavior may be contributing to infection rates. Dressler (1995) has argued that while concern for the integration of culture and biology has a long tradition in the various sub-fields of anthropology, anthropologists have failed to create concrete biocultural models. The root of this problem seems to be methodological: how can we evaluate knowledge claims from seemingly opposite ends of the spectra, between the biological and the cultural (Dressler, 1995)? How can political or economical forces be weighed against ecological or biological ones (Dressler, 1995)? How do socio-political variables operate with or through biology (Dressler, 1995)?

Biocultural approaches to anthropological work are also confronted with problems of scale, of how to move from local contexts to regional ones. If cultural forces on biology are to be taken seriously, it requires a detailed analysis of local culture in order to frame biological interpretation (Singer, 1989; Dressler, 1995). Typically this takes the form of ethnography, but can be conducted by archaeological or historical analysis as well. The problem arises when attempting to move from local understandings to large-scale comparisons between populations. Methodologically, local studies may have been conducted differently using different variables or scales of analysis. For instance, certain social stressors may apply to one group but may be irrelevant or minimal in another (Dressler, 1995). If we take cardiovascular disease as an example, Dressler (1995) has argued that lifestyle incongruity brought upon by social change (such as immigration) is a common variable cross-culturally but the specifics change significantly between cultures so that no single model can be applied to all. Adopting a more generalized research protocol may allow for the fitting of more cultures together, but the risk is that important knowledge of local cultural practices may be lost. In bioarchaeological studies, comparisons between populations are further challenged by the fact that the archaeological and skeletal evidence available may vary substantially between groups. If the Imperial Roman period is taken as an example, local biocultural practices may be lost to the archaeological record, forcing bioarchaeologists to use more general knowledge of Roman biocultural forces at the local level. The danger here of course is that those local forces deviated in important ways from the larger scale ones.

Biocultural models also face the problem of the definition and use of the term culture itself. Literature reviews from both the subfields of biological anthropology and medical anthropology clearly illustrate the tension between environmental and socio-political biocultural models. A frequently cited debate (Singer, 1989, 1992; Wiley, 1992, 1993) in *Medical Anthropology Quarterly* highlights this divide and provides a meaningful starting point for this discussion. Singer (1989) provided a stinging critique of what he called medical ecology as biocultural analysis. Singer's (1989: 223) central argument and point of frustration was that medical ecology had failed to "consider fully or accurately the role of social relations in the origins of health and illness". Singer (1989) claimed medical ecology allowed for only token recognition of social forces, placing poorly defined ecological or environmental models at the foreground of interpretive models. A central critique then is that culture is often used to mean

‘context’ in broad, meaningless terms (Dressler, 2005). This is a position also taken by Goodman (1998), who argued many studies fail to ask genuine biocultural question because they treat societies as simplistic, functionally integrated wholes, or focus too heavily on evolutionary questions framed in ecological models. Singer (1989) viewed this tendency towards ecological models as merely gesturing and not a genuine recognition of real cultural forces that affect biology.

Wiley’s (1992) reanalysis of Singer’s (1989) critiques are worth discussing. The bulk of Wiley’s (1992) argument is meant to clarify what she perceives as Singer’s failure to accurately represent adaptation and its utility in biocultural research. Wiley (1992) emphasized the historical nature of evolutionary models and the diachronic interpretive lens they provide. For Wiley (1992), adaptation should also be viewed as a dynamic process, not a static a priori (and even circular) one as viewed by Stringer (1989). The use of adaptation as an analytical tool is still met with difficulties today. For example, it is still unclear if delayed growth in response to physiological stress is adaptive or not (Reave and Sherman, 1993; Arendt, 1997; Formicola and Giannecchini, 1998; Witt et al., 2004; Kemkes-Grottenthaler, 2005; McDade et al., 2008). Adaptation is a difficult concept to apply methodologically because its meaning can change according to the scale of the research (Reave and Sherman, 1993). Nevertheless, Wiley (1992) stressed that ecological models (including natural selection and adaptation) added to the diversity of theoretical perspectives and did not threaten more ethnographically oriented models. Wiley’s (1992) arguments were supported Goodman and Leatherman (1998), who felt the ecological approach had in fact challenged biological anthropologists to integrate environmental and adaptive forces with social and cultural ones. As Zuckerman and Armelegos (2011) state, ecological models supported a re-envisioning of the human environment to include cultural, environmental and biological components as an integrated whole.

The argument between Singer (1989, 1992) and Wiley (1992, 1993) do not reflect an isolated debate (Goodman, 1996; Goodman and Leatherman, 1998; Armelagos and Van Gerven, 2003; Zuckerman and Armelagos, 2011), or one that is at an end. Segal and Yanagisako (2005) have recently taken a cynical view regarding the challenges of biocultural approaches and have called for the complete separation of subdisciplines, eradicating any attempts at a holistic approach as it is one that characterizes attempts at biocultural integration as “reductive, deterministic, and preferential to biological and adaptationist interpretations rather than more sociocultural approaches to the detriment of both” (Zuckerman and Armelagos, 2011: 16). Fundamentally, this debate over the socio-political and the biological, ecological or environmental, is unnecessary and somewhat of a red herring. There is no fundamental reason why evolutionary explanations for bone form and function cannot be contextualized along with knowledge of smaller scale cultural forces such as gender roles or foodways in bioarchaeology. As Wiley (1993) noted, emphasizing environmental/evolutionary or socio-political models of biocultural inquiry have more to do with the scale of analysis and the questions being asked than with any predetermined worth of either approach. Contrary to the position of Segal and Yanagisako (2005), the real strength of the biocultural model is that “it explicitly considers social and cultural components of the environment, as well as physical, in regards to human adaptation” (Zuckerman and Armelagos, 2011: 20).

## **Building a Useful Biocultural Framework**

From the review in the previous section, it becomes clear that any biocultural approach must somehow seemingly balance several competing factors. The temptation is to balance these factors in accordance with what is considered useful within each sub-discipline. Yet on this path we continue to foster the split within the wider discipline that needlessly pits the physical realm vs. the cultural realm (Ingold, 1998). Räsänen et al. (2006) have also formulated a solid argument for an ethical imperative against hyper-specialization in parsing out causative agents in complex diseases. Specialization is not necessarily detrimental, but social responsibility dictates that various lines of evidence be explored and brought back together if causation of complex disorders are to be discovered (Räsänen et al., 2006). Biocultural approaches also facilitate reintegration of bioarchaeological research back into broader anthropological whole (Blakely, 1977). In citing Martin (1998), Zuckerman and Armelagos (2011) argue that biocultural models are essential if anthropological practice is to remain relevant to contemporary society. Biocultural models are well suited to denaturalizing causes of human disease and suffering, and in doing so can uncover underlying social contexts and factors that contribute to differential mortality and morbidity in the past so that we can challenge them in the present (Zuckerman and Armelagos, 2011).

A number of researchers have recognized these problems and have suggested better ways of conducting biocultural research. Dressler (1995) suggests that we more frequently employ multivariable models, analytic diagnosis and multiple scales of analysis. Multivariable models are used to cover the diversity of data types we deal with, particularly when attempting to combine the biological and cultural (Dressler, 1995). Concurrent with a set of meaningful and relevant data points, analytic diagnosis provides the realization that “particular characteristics of a data set will influence the results obtained in the analysis of that data set, and it is important for the reasonable interpretation of those results that those characteristics be understood” (Dressler, 1995: 50). While not explicitly worded in this way, Dressler (1995) is making an argument for a self-reflexive thought throughout the research process. Multilevel hypotheses are perhaps the most important of the suggestions presented (Dressler, 1995; 2005). Here, Dressler (1995) is arguing for what are essentially hypotheses that link grounded small scale or local analyses to larger global concerns. Dufour (2006) has argued similarly, drawing attention to the fact that if we are to understand the complex interaction between biology and culture, it is crucial to define and measure multiple potential causal pathways. To summarize this point, delving deep into the local does not necessarily prevent future larger scale comparisons if multiple and meaningful variables are chosen within each locus that can be linked through deliberate and carefully formed hypotheses. Dressler’s (1995) general outline of research elicits similarities in Wiley’s work on tacking between multiple scales of analysis and on her criteria for the proper use of analogical reasoning in archaeology (2001).

In summary, the idea that culture and biology are enmeshed is not new to biological anthropology (Boas, 1912; Durkheim, 1951; Henry and Cassel, 1969). Any failures to implement biocultural models in large part reflected the internal struggles and growing pains of biological anthropology, and later, bioarchaeology (Zuckerman and Armelagos, 2011). Since the emergence of biological anthropology, the de-emphasis of analytical approaches, which include integrative biocultural interpretation, appears to stem from a historically and deeply rooted methodological

inclination towards description over hypothesis testing (Lovejoy et al. 1982; Hoppa and Fitzgerald, 1999; Armelagos and Van Gerven, 2003). Moreover, actually applying biocultural approaches is quite difficult due to the often-limited contextual evidence available to bioarchaeologists when compared with socio-cultural work (Dressler, 1995; Djurić-Srejić and Roberts, 2001; Dufour, 2006). The nature of skeletal material itself can also present challenges. Biased preservation and excavation, along with imperfect techniques to assign age and sex to skeletons continue to hinder research (Jackes, 2000). Some authors (Goodman, 1998; Armelagos and Van Gerven, 2003) have also criticized the discipline for failing to simply start asking bioculturally-oriented questions. Despite these persistent challenges, there is significant evidence to make the argument that the overly descriptive tendencies of bioarchaeology are fading. Many papers are now much more actively engaged with the social, historical and archaeological contextualization of skeletal remains (Knudson and Stojanowski, 2008). Zuckerman and Armelagos (2011) have reported that biocultural research has expanded substantially into new areas of inquiry, including social and economic contributors to disease, social identity, disability, gender, queer theory, embodiment, and sexuality (Shakespeare, 1999; Armelagos and Harper, 2005; Sofaer, 2006; Bentley et al., 2007; Geller, 2008; Ortner and Schutkowski, 2008; Barrett and Blakey, 2011; Hollimon, 2011). Even longstanding research foci like biodistance studies are being re-evaluated in light of refining our understanding of biological and social identities (Nystrom, 2006; Stojanowski and Schillaci, 2006). The bioarchaeology of children and childhood has also seen a great upswing after decades of neglect (Lewis, 2007). The current breadth of research questions in bioarchaeology support the assertions of Stojanowski and Buikstra (2005) who claim that this historical schism between descriptive, methodological research and analytical, biocultural approaches have reached a form of balance in the literature. Bioarchaeology is now well poised to advance as it is well supported by both theory building and important foundational descriptive research.

### **Life History Theory and Skeletal Plasticity**

Life history theory was quite influential within the ecological approach, particularly the work of Baker et al. (1986). The model by Baker et al. (1986), and others like it, can be simply stated as seeking to understand how development occurs from embryonic stages into adulthood in the context of genetics, the metabolic demands of all the internal systems (immunological, hormonal, etc) and exterior forces such as diet and physical activity (Baker, 1984; Baker et al., 1986; Bogin, 1999; Bogin and Rios, 2003). Four major themes stand out when reading studies that employ life history models: the cumulative process of growth, developmental and phenotypic plasticity, genetics and evolutionary history, and finally the concept of tradeoffs.

The study of growth as a cumulative process in the production of the adult body seems obvious at first, yet much of how this happens remains unclear. For example, peak bone mass, or the maximum amount of bone attained during life, has long been considered to be a major determinant in assessing osteoporotic risk in old age (Bonnick 2002; Brunader and Shelton, 2002; Cummings et al., 2002). While genetics seem to contribute to peak bone mass (Seeman, 1999), non-genetic factors of skeletal growth during childhood and adolescence are also thought to be highly influential. In contrast with most studies, recent work by Gafni and Baron (2007) casts doubt on this 'deposit-banking' model of bone growth. Gafni and Baron (2007) used animal models to show that variations in bone density accrual during the greatest periods of growth did



not persist late into adulthood. These data, and other related studies (Slemenda et al., 1997; Bonjour et al., 2001; Gafni et al., 2001; Schoenau, 2004), suggest that the effects of childhood growth on adult bone mass are perhaps not best understood as simple linear or cumulative relationships, at least without further careful consideration and testing. However, the predominant view is that adult skeletal morphology and cross-sectional geometry are intimately linked with the earlier stages of growth and development (Javaid and Cooper, 2002; Ruff, 2005; Javaid et al., 2006; Ruff et al., 2006). Differential pathways of growth and development have also been compellingly linked to variation in hormone levels (Worthman, 1995), immunological variation (McDade, 2005), and in defining adult body size and weight (Baker et al., 1986; Gluckman and Hanson, 2004). These competing perspectives on the persistence of growth and development effects into adulthood highlight that the growth and development process is still incompletely understood and deserving of continued study.

One commonality that holds most of these studies together is the idea that all factors of growth, from musculo-skeletal (Cooper et al., 1997; Cooper et al., 2001; Ruff, 2005), and endocrine (Worthman, 1995), to immunological (McDade, 2005) are subject to shaping in response to environmental (including cultural) conditions. This is not to say that genetics are not important. By some estimates, the human genome is thought to be responsible for up to 85% of adult bone mineral density (BMD), with some variation between different skeletal elements (Gafni and Baron, 2007). Dental development is also highly “controlled” by genetic heritage (Sinclair and Dangerfield, 1998; Cardoso, 2007). Some would go so far as to say that genetics overrides any other contribution to growth in every meaningful way (Lovejoy et al., 2003). However, many researchers who use life history models to understand growth and development take a more nuanced view of the process, recognizing that skeletal growth, and growth of other complex systems act as an interplay between epigenetic and genetic processes. The interplay between genetic (and evolutionary history) and epigenetic forces can be partially understood through the concept of tradeoffs.

Tradeoffs form an important analytic lens into the life history of an individual as our bodies have evolved as finite systems, with competing physiological demands and a limited energy supply. The result is that the human body will preferentially maintain or favor one system over another in order to preserve what it perceives as greater health for the individual (Bogin, 1999; Bogin et al., 2006; Cameron, 2006; Ellison, 2006; Kuzawa, 2006; Schell, 2006). Looking at life history and tradeoffs understandably requires knowledge of evolutionary paths to the current state of being (Mace, 1999), as tradeoffs may change through time as selective pressures change. Theoretically, exploring the nature of tradeoffs is exciting because it forces us to reconsider the role of adaptation. Is sacrificing skeletal growth during periods of malnutrition adaptive, or should it be considered pathology (Reave and Sherman, 1993; Arendt, 1997; Bogin, 1999; Formicola and Giannecchini, 1999; Witt et al., 2004; Bogin et al., 2006; Cameron, 2006; Ellison, 2006; Kuzawa, 2006; Schell, 2006; McDade et al., 2008) since there is reason to believe that shorter people do not live as long (Kemkes-Grottenhaler, 2005)? Yet from evolution’s perspective, it may be adaptive to sacrifice some growth to safely reach reproductive age if the body is stressed early in life. Modern medical advancements that extend average human longevity complicate matters. The adaptive gain of reaching reproductive age through tradeoffs may then become very maladaptive at middle and old age when the reduced growth greatly increases the risk of developing osteoporosis if indeed high peak bone mass is a significant

determinant of reduced fracture risk (Bonnick, 2002; Brunader and Shelton, 2002). As longevity increases through medical intervention in developing countries it is likely this issue will increasingly come into play in these regions (Cooper et al., 1992). Other similar problems in different bodily systems have also been documented (Barker 1998; Barker 2001; Gluckman and Hanson, 2004).

Understanding tradeoffs is not a simple matter; evolutionary adaptation must be considered along with somatic adaptation (developmental plasticity) during a single lifetime. These long and short-term modifications often conflict and are potential sources of causation for diseases in the elderly today since life expectancy continues to increase. Exploring tradeoffs allows us in some sense to look at tensions between evolutionary forces and cultural ones, as tradeoffs will vary across time and space according to local cultural patterns. For example, modern lifestyles in the West are radically different than the general pattern humans have been following through our evolutionary history, at least in terms of diet, activity and child rearing practices (Agarwal and Stuart-Macadam, 2003), presenting a new set of conflicts and tradeoffs created between evolutionary heritage and developmental plasticity that must be understood. This is compounded by the fact that a mother's health has dramatic repercussions on the skeletal health and development of her child through fetal programming (Barker et al., 2001; Cooper et al., 2001; Gluckman and Hanson, 2004).

While the theoretical innovations of life history theory are exciting, biological anthropology, and bioarchaeology in particular, are faced with methodological problems in adapting life history models to skeletal data. Most life history case studies do not investigate the growth and health of the skeletal system. Variables that are most important in life history models include anthropomorphic measurements of the mother, the subject's birth weight and the subject's anthropomorphic measurements through various growth stages, detailed dietary history, and history of the environment during growth, including socio-economic status (Little and Haas, 1989; Sinclair and Dangerfield, 1998; Bogin, 1999; Stinson et al., 2000). Most of this information is lost to the bioarchaeologist. Nevertheless, some important work has been done using life history theory in biological anthropology using archaeological populations. For example, Klaus and Tam (2009) have developed a biocultural model for the effects of systemic stress on key biological systems, such as female reproduction and overall growth suppression. This model involved linking measures of juvenile stress, such as enamel hypoplasias, with markers of adult stress, which included non-specific periosteal infections and female fertility (via age-at-death distributions) (Klaus and Tam, 2009). The application of the core principles of life history theory are clearly possible (Klaus and Tam, 2009) and will undoubtedly provide exciting new pathways of research. One of the most direct ways life history can be approached in bioarchaeology is through the related concept of developmental plasticity.

### **Skeletal Growth, Development, and Plasticity Across Disciplines**

Plasticity, growth, and development are essential to understanding patterns of phenotypic variability. *Growth* is generally understood to reflect stepwise or progressive changes in size and morphology during the development of an individual (Scheuer and Black 2000). While growth is generally correlated with chronological age, differences in rates of growth are common between individuals of equal chronological age due to divergent life-history trajectories (Scheuer and

Black 2000). While growth in size is correlated with biological maturity, they diverge enough so that “individuals reach developmental milestones, or biological ages, along the maturity continuum at different chronological ages” (Scheuer and Black 2004:4). Growth is then best seen as the enlargement and differentiation of tissues advancing with chronological age, while *development* comprises the pathways of biological milestones along the life course. Given that rates of growth and the timing of developmental changes differ between individuals, numerous debates have formed over what constitute normal growth and development trajectories (Bogin 1999; Worthman and Kuzara 2005). Differential growth rates have also been studied to understand health (Clark et al. 1986; Mays et al. 2008; Klaus and Tam 2009), adaptive fitness (Lasker 1969; Roberts 1995; Schell 1995; Worthman and Kuzara 2005; Lewis 2007) and the evolutionary significance of growth rates (Ruff et al. 1994; Nelson and Thompson 1999; Ellison 2005). The complexity and debate on the role of growth and development is exciting as it allows us to explore how gene–environment relationships operate to produce a wide range of phenotypes at different stages of the life course.

In contrast to growth and development, *plasticity* is a much broader concept that is much more difficult to grasp, as there are inconsistencies across disciplines in how the term is used to describe its role in the formation of the adult phenotype through developmental processes. Most of the confusion with the concept of plasticity resides in its conceptual link to adaptation (Lasker 1969; Roberts 1995; Schell 1995). Prior to the 1950s and 1960s the working definition of plasticity was simply an understanding that human morphology appeared to be malleable during growth and development (Bogin 1995). Yet this vague conceptualization of plasticity was purely descriptive and was not amenable to hypothesis testing. Dobzhansky (1957) was one of the first to view plasticity as a form of adaptation. In this view natural selection produces genotypes “that permit their possessors to adjust themselves to a spectrum of environments by homeostatic modification of the phenotype” (Roberts 1995:2). Lasker (1976) is considered to have truly merged plasticity with adaptation and in the process redefine the plasticity concept altogether. Lasker’s (1969) view of plasticity operated within three modes of adaptation. The first of these was natural selection itself, where the selection of genotypes directly influences the genetic spectrum of the population (Roberts 1995). The second form of adaptive plasticity, acclimatization, is a non-permanent physiological and behavioral response that adapts an individual to the immediate environment (Roberts 1995). The third and most important mode (in this discussion) is developmental or ontogenetic adaptation (Roberts 1995). The key features of ontogenetic modifications are that plastic responses operate through growth and development, and that the changes are not reversible and also not heritable (Schell 1995). Numerous others have studied variation in human phenotypes through the lens of plasticity and while they all have their own definition of plasticity, adaptation and a concern with trade-offs are central to most (see Worthman and Kuzara 2005 for an excellent review). While a concern for adaptation has been helpful in trying to fit plasticity into the framework of modern Darwinian thought (e.g. McDade et al. 2008), a broadening of focus would offer a better understanding of the process of plasticity and its role in development.

Understanding plasticity in the developmental context beyond a strictly adaptationist model has been put forward by a number of researchers (Cooper et al. 2006; Lewontin 2001; Oyama 2000a; Sofaer 2006; Worthman and Kuzara 2005) often using terms such as developmental plasticity, developmental systems theory or approach (DST/DSA), and

developmental dynamics. All of these approaches share a general concern with the developmental processes in embryogenesis, fetal growth, early postnatal growth, and adolescence that give rise to variation through plastic responses. While these areas of research have much common ground, there are differences in nomenclature and conceptual divides about the limits of plasticity. Plasticity studies working primarily in fetal development (e.g. Hallgrímsson et al. 2002) are conceptualized differently than research that extends plasticity to include infancy, childhood, and adolescence (Fausto-Sterling 2005). In essence, this mirrors the larger tension between the two most prominent approaches, evolutionary developmental biology (EDB) and developmental systems theory (DST). Both are concerned with understanding how plasticity operates rather than solely looking at the products and evaluating their adaptive fitness and both give an alternative to reductionist approaches. However, the EDB perspective is limited primarily to embryology/fetal development and is less concerned with post-birth plastic and developmental changes (Robert et al. 2001; Hallgrímsson et al. 2002). Further, in EDB genes are given primacy during development as they are seen to supply the material needs of development (Robert et al. 2001); genes can exist without development, but there is no development without genes. EDB does emphasize the importance of variation, with the goal to observe patterns of variability to better understand underlying developmental systems that can ultimately be linked to how development intersects with natural selection and evolutionary change (Hallgrímsson et al. 2002). Perhaps most importantly, variation in developmental processes is studied in the context of conservation of form, where “individual variation is minimal and seemingly constrained” (Robert et al. 2001:959). The developmental systems theory (DST) or approach diverges from EDB in many ways. DST contrasts with EDB in that variation is primarily focused on in terms of plasticity rather than conservation of form. Developmental information is believed to reside neither in the genes nor the environment, but rather in the interaction of the two (Robert et al., 2001). As such, genes have no primacy in the DST model and plasticity is the defining feature of the *development system* that is defined as the interplay of all influences on development including the “molecular, cellular, organismal, ecological, social, and biogeographical” (Robert et al. 2001:954). As such development is seen to extend well into postnatal growth (Robert et al. 2001; Worthman and Kuzara 2005). There are a number of examples of this, including neurological growth (Kamm et al. 1990) and immune functions (Worthman 1995).

Common ground between EDB and DST approaches may be found in the study of epigenetics (Robert et al. 2001). While there are many definitions of epigenetics, it can be broadly defined as the study of genetic and non-genetic interactions on development (Robert et al. 2001; Hallgrímsson et al. 2002). Robert et al. (2001) suggest that epigenetics may be the “practice of what DST proposes,” a place for scientific testing of DST. While both DST and EDB approaches advocate for both acceptance of genetic and non-genetic influence during developmental processes, DST goes one step further in suggesting that inheritance is also epigenetic (Robert et al. 2001). For DST theorists again the gene is not the only player in inheritance, and instead inheritance is extended to include ecological, social resources, or other interactants that influence development (Oyama 2000b). As such, epigenetic processes are seen as heritable and are constructed and reconstructed during each life cycle.

Whether non-genetic influences are heritable, particularly in skeletal morphology, continues to remain uncertain. This uncertainty, of whether or not non-genetic forces

significantly shape postnatal and intergenerational skeletal morphology, has limited the theoretical explorations of plasticity and development in bioarchaeology. Moreover, EDB paradigms in biological anthropology essentially greatly minimize the role of environmental and postnatal influence on the plasticity of morphology (Lovejoy et al. 2003). The focus for studies of bone plasticity in biological anthropology has thus been primarily on evolutionary and adaptive change, rather than postnatal development over the life course. One theoretical with adaptationist models is that they run the risk of naturalizing social processes (Gould and Lewontin, 1979; Orlove, 1980; Singer, 1996). Adaptationist models also minimize the role of developmental processes themselves. Pritchard (1995) has noted that plastic responses in a given tissue or tissues not only react to external stimuli but also generate their own effects in other tissues. In this context, plasticity during development is a generative force in shaping the body as much as a reactive one, and should then be viewed as more than a side-note or byproduct of discussions on gene–environment dynamics. Furthermore it is unclear how plasticity can successfully modulate and affect existing genetic networks in widely different developmental and environmental landscapes rather than relying on the evolution of novel genes or genetic pathways to produce phenotypic variation (Young and Badyaev 2007). The interdependency between genes, development, and environment are at the heart of the matter in understanding plasticity. The aim of the following section is to review how theoretical approaches to plasticity and development been developed over time in anthropology and bioarchaeology, and specifically how developmental approaches can help us better understand bone maintenance and aging across the life course.

### **Concepts of Plasticity in Anthropology and Bioarchaeology**

The formal history of the study of plasticity in anthropology can arguably be said to have begun with Boas (1912), although earlier studies do exist that similarly observed generational changes in growth in migrants (Baxter 1875; Bowditch 1879). Through detailed anthropometric measurements of body size and shape Boas (1912) observed that the children of new immigrants (of European descent) to the United States displayed different growth patterns than their parents. Moreover, he noted the change was accentuated with each generation (Boas 1912). In an earlier work commissioned by the U.S. Congress, Boas (1910:53) remarked “we must speak of plasticity (as opposed to permanence) of types.” Boas’s 1912 article was pivotal as it presented solid evidence that environmental changes, which included changing cultural milieus, could produce changes in body size and shape in future generations. Growth and adult stature was seen as more than the sole product of genetic heritability. Boas’s work was supported by Shapiro’s (1939) often-cited growth study of Japanese children in Japan and Hawaii that also showed significant differences in growth, stature, and development, which he also attributed to environmental triggers. Numerous migrant studies have repeatedly confirmed the correlation of changing environments to changes in growth and development (Goldstein 1943; Lasker and Evans 1961; Kasl and Berkman 1983; Baker et al. 1986; Bogin 1995; Bogin and Rios 2003). Plasticity studies were not limited to migrants only; plasticity was studied within cultures as well to account for the fact that those who stayed behind might have differed in some important ways (e.g. Mascie-Taylor 1984). Plasticity was also studied through observation of so-called natural experiments (Roberts 1995). For example, Roberts and Bainbridge (1963) observed a population of three Nilotic tribes living in the same environment but with slight cultural differences.

Somatotype and anthropometric measures demonstrated small but significant differences between the three tribes (Roberts 1977). Roberts (1977) concluded that these differences were environmentally based through ways of life and dietary differences in particular. Similar studies between Polynesian and other traditional cultures have observed similar results in cases of changing or differing socioeconomic conditions between two closely related migrant/sedante groups (Baker et al. 1986; Kasl and Berkman 1983).

Schell (1995) has argued that by 1954 with Kaplan's review of migrant-sedante studies in *American Anthropologist* that plasticity was firmly established as a recognized phenomenon of growth and development. However, even though developmental plasticity was recognized in the field, definitions of what plasticity was, or could do, were variable. A dominant challenge has been defining the concept of plasticity itself. This appears to have become an interpretive problem only after Lasker (1969) permanently tied plasticity to adaptation. There is much we do not know about the adaptiveness or relative benefit of plastic modifications made during growth and development, in part because of the difficulty of interpreting growth patterns (Schell 1995; Humphrey 2000; Saunders 2000; Worthman and Kuzara 2005; Lewis 2007). However, two general interpretations have been put forward in attempts to understand variation in growth and development. Both models address the issue of morphological variation and compare stress and health among and between cultures from an adaptationist perspective, but from very different theoretical positions.

The first of these interpretations is the "medical model" common in public health policies, pediatrics, and nutritional science (Schell 1995). The medical model views growth as a reflection of health, and with this it literally becomes a measure of health and consequently, of adaptation (Schell 1995). Growth to the full extent of an individual's genetic potential is interpreted as good health while slow or stunted growth signifies ill health (Schell 1995). The implicit assumption is that the body will always reach its full genetic potential if no boundaries are presented. Assessing this with archaeological skeletons is difficult, given that retarded growth and development may not show clear outward signs (Humphrey 2000).

The opposing model is termed the human adaptability paradigm (HAP) (Schell 1995). The HAP views growth and development as the *mechanism* of plasticity (Schell 1995). In other words, "growth patterns can be a mode of adaptation" and in "this context growth is a *means* of achieving an adapted state rather than a result of that adaptation" (Schell 1995: 223, emphasis in original). The problem here is that the modifications that reduce stress/strain can be seen as adaptive but they cannot be proven so in a strict sense (Bogin 1995; Schell 1995). Further, the medical and HAP models conflict because growth cannot be both a measure and a means of adaptation (Schell 1995). As such, the models are mutually exclusive. Clearly this poses a problem for which model to use in bioarchaeology. To some degree this may depend on what influences or stressors are being considered as causes for the observed plastic changes. Schell (1995) has offered that the medical model may be better suited to interpreting plasticity as a feature of human-made environmental changes, such as slums, where nutrition is poor and disease load high, while the HAP may be beneficial for interpreting plastic responses induced by the physical environment. However, it is unclear how to structure bioarchaeological research questions and analyses when typically both human-made and naturally occurring environmental factors are at play. While both these models have contributed significantly to studies of growth

and development, neither works fully when applied to bioarchaeological analyses, particularly to the interpretation of patterns of bone maintenance and loss.

Considering development as a generative force (Pritchard, 1995; Oyama 2000a), rather than a “reading out” of genetic material during key periods of growth, may help us better understand how the human body and skeleton is shaped and reformed throughout life. Recent biomedical and epidemiological studies have specifically explored how plasticity during growth and development can influence aspects of lifelong bone health, such as bone mineral density and loss. For example, infant and adolescent growth spurts seem to be highly influential in defining bone quality and quantity at later life stages (Cooper et al. 2001, 2006; Javaid and Cooper 2002; Miller 2005; Javaid et al. 2006). Peak bone mass (the maximal amount of bony tissue accrued during growth) is generally thought to be mostly inherited (Duncan et al. 2003), but Cooper et al. (2002: 391) remark that “only a small proportion of the variation in individual bone mass” is accounted by genetic markers. Seeman (1999:91) has also noted that the contribution of heritability in bone health is not a constant proportion, and that statements claiming “80 percent of areal BMD (bone mineral density) is genetically determined leaving only 20 percent to modify” is flawed. Heritability is a complex, fluid measure based on a relationship between population and environment variance (Seeman 1999). As age, height, gender, and body composition vary, so do heritability measures of bone mass or density (Seeman 1999). Cooper et al. (2006) posit that environmental cues early in life interact with the genome to create the boundaries of growth and development for a given individual. It has been hypothesized that these types of developmental boundaries or trajectories may originate in expectation of future environmental conditions and serve as predictive adaptive responses (Gluckman and Hanson 2004). For example, fetal programming by maternal under-nutrition is a risk factor for low birth weight (Cooper et al., 2002). Low birth weight is strongly correlated with lower levels of basal level growth hormones, even during adult life, placing the individual at risk for lower peak bone mass, reduced mineralization, and an elevated rate of bone loss later in life (Cooper et al. 2002; Dennison et al. 2005). Numerous epidemiological studies have shown that impaired fetal and childhood growth place individuals at risk for fragility fractures later in life (Cooper et al. 1995, 1997, 2001; Gale et al. 2001; Cameron and Demerath, 2002; Dennison et al. 2004; Dennison et al. 2005). These studies emphasize the dramatic role of environmental influences on phenotypic plasticity in early life, and more importantly underscore how this early exposure can change the trajectory of development and aging of skeletal morphology throughout life.

### **Studies of Skeletal Plasticity in Bioarchaeology**

A number of studies have made important strides in using plasticity as an analytical tool, particularly in the study of temporal and spatial differences in skeletal morphology as related to influences such as nutrition, activity or disease (Bogin 1999; Larsen 1997; Lloyd and Cusatis 1999; Knüssel 2000; Saggese et al. 2002; Schwartz et al. 2003; Rauch 2005; Prentice et al. 2006; Ruff et al. 2006; Skerry 2006; Lewis and Gowland 2007; Mcdade et al. 2008). Most notably, patterns of long bone growth in archaeological skeletal samples have been widely used as a proxy for comparing health and stress statuses between and among populations (Humphrey 2000; Saunders 2000; Kemkes-Grottenthaler 2005; Lewis 2007; Mays et al. 2008). In studies of

bone maintenance and loss in bioarchaeology the focus has been primarily on the influence of nutrition and levels of physical activity in either encouraging, or protecting against, the onset of age- and sex-related bone loss and fragility (Agarwal 2008; Agarwal and Glencross in 2011). There has been some study of the affect of early growth and development on the maintenance of the mature skeleton in archaeological samples. For example, the classic studies of bone loss in prehistoric Sudanese Nubia were some of the first studies to consider and compare bone growth and maintenance in both juvenile and adult skeletons. Armelagos et al. (1972) suggested that the significant cortical bone loss in the femur found in young-aged female Nubians, as compared to males, was likely due to early growth disturbance and stress as young adults during pregnancy and lactation. Similarly, a study of cortical bone growth maintenance in prehistoric juvenile Nubians from the Kulubnarti site found that while bone mineral content increases after birth, processes of modeling combined with likely periods of nutritional stress, cause a reduction in percent cortical area during early and late childhood (Van Gerven et al. 1985), although this study does not comment on the role of early bone maintenance on later femoral bone loss. Two recent studies have focused on the structural variation of trabecular bone during ontogeny. Kneissel et al. (1997) examined the ontogeny and aging patterns of vertebral trabecular bone in a juvenile and adult skeletal sample from Medieval Lower Egyptian Nubia. The authors found the largest bone trabecular volume during adolescence when the rod-like trabeculae of childhood begin to change to plate-like structures. In addition, age-related loss of trabecular structure was observed in adults, with changes occurring earlier than those seen in modern populations (Kneissel et al. 1997). Gosman and Ketcham (2009) also examined patterns of ontogeny in trabecular bone in their study of tibial bones from the prehistoric Ohio Valley, particularly noting changes in trabecular structure and connectivity from growth to skeletal maturity and with increasing ambulatory activities.

More recent studies have attempted to more directly correlate growth patterns and developmental stress, with variation in skeletal morphology and bone loss. For example, a study by Rewekant (2001) examined the correlation of adult cortical bone loss with indicators of growth disturbance (specifically compression of the skull base and vertebral stenosis) in two Polish medieval populations with differing socioeconomic status. Rewekant (2001) found greater adult age-related cortical bone loss in the metacarpal in the population that also showed greater disturbance of bone growth during childhood. Interestingly, lower sexual dimorphism in measurements of metacarpal cortical bone and skull base height were also found in the population that appeared to have suffered greater environmental stress during growth. This study suggests a relationship between the disturbance of growth and the achievement of peak bone mass, as well as the age- and sex-related patterns of bone loss later in life. Similarly, McEwan et al. (2005) examined the correlation of bone quantity in the radius to overall growth patterns and indicators of growth disturbances typically attributed to poor nutrition (specifically Harris lines, and cribra orbitalia) in juvenile skeletons in a medieval British sample. The authors found that while bone mineral density (BMD) was well correlated to overall growth, cortical index (a measure of total cortical bone) was not (McEwan et al. 2005). This again suggests that some aspects of bone maintenance such as the overall amount of cortical bone may be compromised during development under the influence of environmental (nutritional) stress with a lasting effect on cortical bone content and morphology well into adulthood (Mays 1999; McEwan et al. 2005). There has also been focus on influences after childhood, into young adulthood that may play a significant role in later bone fragility. For example, several bioarchaeological studies within and



between skeletal populations suggest that physical activity during adulthood can result in a conservation of bone quantity during life and offer protection against the affects of bone loss in old age (Lees et al. 1993; Ekenman et al. 1995). The opposite has also been noted, with observations of decline in bone quantity and strength in more sedentary agricultural populations as compared to physically active hunter-gatherer groups (Ruff et al. 1984; Larsen, 2002; Ruff et al. 2006), although this observation is not universal as workloads were likely variable in agriculturalists depending on region and local terrain (Larsen, 2002; Nelson et al. 2002). While it is known that bone tissue responds to mechanical loading, the biomedical literature is unclear on what type and level of physical activity or exercise is needed to affect bone mass and more importantly bone strength into adulthood. There may be an ideal “window of opportunity” for physical activity to contribute to the growth and robusticity of the skeleton during the acquisition of peak bone mass (Pearson and Lieberman 2004), but it seems likely that some high strain stress activity may still be effective at older ages (Rittweger 2006). Reproductive behavior is another factor that may influence the trajectory of bone maintenance and loss in older age. Several studies have suggested that young age females in the archaeological record show evidence of bone loss that is result of physiological stress on the skeleton due to pregnancy and/or breastfeeding (Martin and Armelagos 1979, 1985; Martin et al. 1984, 1985; Poulsen et al. 2001; Turner-Walker et al. 2001; Mays et al. 2006). However, it can be argued that the loss of bone in reproductive-age women in the past was transitory, and that bone loss during reproduction would have little or no affect on long-term bone fragility in women who would have survived to old age (Agarwal and Stuart-Macadam 2003; Agarwal et al. 2004; Agarwal 2008; Agarwal and Grynepas 2009). In fact, high parity and prolonged breastfeeding in some past populations would have provided women in the past with a very different hormonal milieu and steroid exposure that could have offered protection against the sudden postmenopausal drop of hormones experienced by modern women (Weaver, 1998; Agarwal et al., 2004; Agarwal, 2008).

All of these studies take vital steps in exploring the role of development in bone morphology and maintenance, and emphasize the importance of earlier life experiences on the strength and fragility of the aged skeleton. While influences such as nutrition, physical activity, and reproduction are critical to understanding bone growth and maintenance, it is increasingly evident that what is really important is how these influences are played out over the life course, and the cumulative effect that they may have on the skeleton at the end of life.

### **The Lifecourse Approach in the Study of Bone Maintenance and Loss**

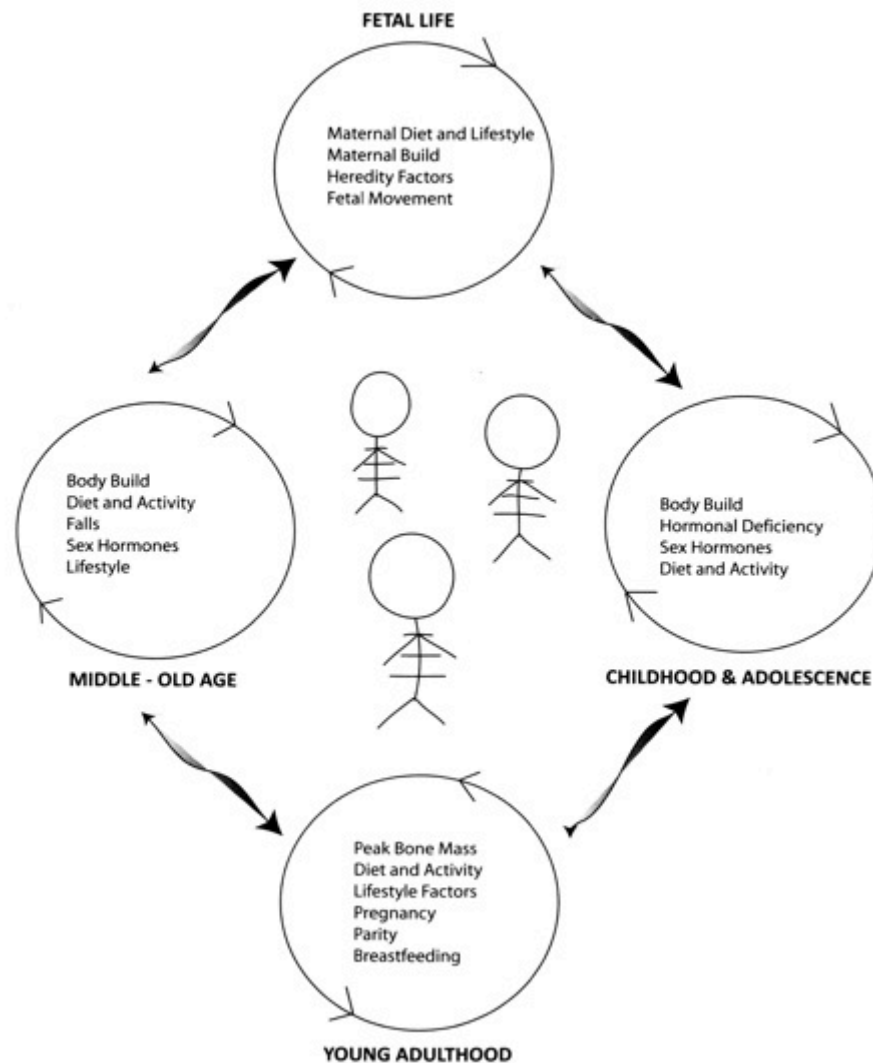
As outlined in Chapter 2, despite the numerous studies of bone aging and osteoporosis in bioarchaeology, the biocultural factors that result in substantial variation in bone loss between populations in the past remain unclear (Agarwal and Grynepas 1996; 2009; Agarwal 2008). Some archaeological populations of similar temporal or spatial origin show similar patterns of bone loss, while others do not. However, most differ from the typical age- and sex-related patterns of bone loss and fragility observed in modern Western populations (Agarwal 2008). Unlike living groups today, bone loss is often seen in young age and equal in both males and females, yet there is a low prevalence of fragility fracture in comparison to modern populations (Agarwal and Grynepas 1996; 2009; Agarwal 2008; Brickley and Agarwal 2008). The explanation for these observed patterns in the bioarchaeological record is complex, and the use of often incomplete

and biased skeletal samples is a persistent issue in the analysis of any indicator of health and disease in the past (Wood et al., 1992, Wright and Yoder, 2003; Jackes, 2011). However, the variable patterns of bone maintenance fragility in the past should not come as a surprise, given that groups in the past would have had very different biosocial histories from our own. The morphological variability in bone maintenance and loss we see in archaeological populations is an important research avenue, because it perfectly illustrates the biocultural nature of plasticity and development of the body. Yet, recent bioarchaeological studies have used familiar patterns of bone loss in the past to ratify traditional paradigms of aging in the female skeleton, while discounting patterns that simply do not fit *a priori* expectations. The purpose here is offer new ways of interpreting patterns of bone loss in the past to begin to make more meaningful interpretations of bone growth, maintenance, loss, and aging in both past and present day societies.

What is these data suggest is that bone maintenance and loss is the result of ontogenetic processes over the life course, with trajectories of bone maintenance laid out in early growth, refined during adulthood, and played out and modified within the everyday individual and generational choices of behavior and life experience (Figure 6). Observing one snapshot of bone maintenance at one scale (such as bone loss only in adulthood; one area of the skeleton, or using one methodology) will give a skewed perspective on the complex and unique path that has created the observed bone morphology (see chapter 5 as well). Figure 6 shows a diagrammatic model of the plasticity in development and maintenance of the skeleton over the life course. Circles represent major periods of the biological life cycle (fetal life, childhood and adolescence, young adulthood, and middle/old age) each containing examples of some of the major influences within each life stage in human skeletal development. Influences within each stage are interdependent (represented with arrows around each circle), and influences in each stage are cumulative and dependent on influences in earlier life stages (represented as arrows between circles). Cumulative influences shape skeletal morphology, and affect bone maintenance and bone loss. These influences account for the variation in skeletal morphology and maintenance observed over an individual's life course, as well as within communities/populations (represented by varied skeletal figures in middle of model). Note, interdependent arrows are shown even between middle/old age and the fetal life stage, as representation of the potential intergenerational effect of individual and population life history to skeletal morphology in subsequent generations.

This life course perspective of plasticity and development of the skeleton is partially grounded in the DST approach to the growth of organisms discussed earlier. DST approaches emphasize the interaction of both environmental and biological influences on the development of the organism that occur over the entire life cycle (Oyama 2000a). Fausto-Sterling (2005) has applied this general model specifically to understanding skeletal morphology and osteoporosis in modern humans. Borrowing from life course approaches that have been used in the study of chronic diseases, Fausto-Sterling (2005) highlights the cumulative nature that influences have on bone health, and suggests that prior events during life can alter the trajectory of bone development in later points of the life cycle. Life course approaches extend this model of "critical periods" in fetal development (Ben-Shlomo and Kuh 2002), and suggest there could also be modifiers on bone form and health later in life. The concept of the body as a product of

developmental context (both biological and social) is not limited to DST and life course approaches, and is also found in archaeological perspectives of embodiment.



**Figure 6** – Bioarchaeological life course model of bone growth, maintenance and loss (Adapted from Agarwal and Beauchesne, 2011: 324)

Ingold (1998) has argued that the body is a developmental system that is contextually dependent, and that more importantly, humans grow and are active in their development through engagement with the social world. This engagement with the world in which bodies are situated can be both conscious (with agency) and unconscious (Krieger 2001, 2009), and dilutes the belief that organisms are primarily passively built by their genetic code. In her discussion of skeletal markers of gendered behavior in archaeological skeletons, Sofaer (2006:161) notes that while it may be difficult to directly correlate skeletal markers with distinct activities or lifeways

in the past, “plasticity of the body means that the body is never pre-social and is contextually dependent”. There is no pristine bodily state that is outside of the environmental and cultural context in which it operates (Oyama 2000a). This is not to say that the plasticity and development of organisms are limitless (Oyama 2000a; Sofaer 2006). Bone's ability to shape itself is bound by, among other things, genetics, environment, age, and sex (Oyama 2000a; Hallgrímsson et al. 2002, Lovejoy et al. 2003; Pearson and Lieberman 2004; Ruff et al. 2006; Sofaer 2006). For example, processes such as canalization and developmental stability tightly control fetal skeletal development (Hallgrímsson et al. 2002). However, novel or stressful environments can reduce the ability of these processes from limiting variation (Young and Badyev 2007).

While the traditional view gives the gene formative power as keeper of the plan or code, the developmental perspective sees the gene not as an information-containing device, but as an information-generating device that depends on immediate environment (Oyama 2000a). While bioarchaeologists do not dismiss the notion that genes and the environment interact, it is that the flow of information in development is thought to move outwards in one direction from the genome, which then interacts with the environment (see Oyama 2000b). This leads to the idea that there are “two kinds of developmental processes, one controlled primarily from the inside and another more open to external forces” (Oyama 2000b:21). What this means for bioarchaeology, is that while the coded forces of bone physiology and senescence play vital roles in bone growth and maintenance, they need to be viewed as interwoven in a larger developmental process driven by cumulative life experience. While it may be suggested that the focus on life experience limits the exploration of bone morphology and health to the individual context, these theoretical approaches to the body, and to development over the life course, are inherently intergenerational.

Epidemiological life course approaches contextualize early life exposure in structures that include the role of parents, grandparents, households, and communities (Ben-Shlomo and Kuh 2002). Here, biological and social risk is seen as playing across entire generations. DST approaches go one step further, extending what we traditionally think of as heredity. Inheritance is seen as more than the passing of a trait or blueprint, but instead the transmission of entire developmental contexts, which can include genes, cellular machinery as well as social and ecological systems (Oyama et al., 2001; Robert et al. 2001). Social and environment context are seen as potential intergenerational influences on the phenotypic variation of the skeleton. As such, skeletal variation in bone maintenance and loss potentially could be the result of developmental processes that have acted at the level of the individual, generations, or entire communities. This has great relevance for how bioarchaeologists observe variation in not only bone maintenance, but also all aspects of bone morphology.

## **Chapter 4 - Velia and the Imperial Roman Context**

The skeletal material used in this dissertation originates from the archaeological site of Velia, on the southwestern coast of Italy, and date to the Roman Imperial period. The people of Velia were mostly from the non-elite classes (Craig et al., 2009), and consequently, the historical and archaeological context of the site is framed by a concern for what the lives of non-elites in ancient Rome were like. Scholars interested in ancient Roman culture have historically been fixated on studies of elites and their products (Dixon, 2001; Toner, 2002; 2009; Alcock and Osborne, 2007). The culture and rule of elites did define many aspects of Roman society (Aldrete, 2004), but historical or anthropological study of Roman elites does not address the daily lives and experiences of the vast majority of the Roman people (Toner, 2009). It is estimated that nearly 99 per cent of the population controlled by Rome was comprised of non-elites (Toner, 2009). The ‘democratization’ of the past has become a research goal for many historians and they have attempted to reinterpret traditional sources of evidence for clues into how most Roman people actually lived rather than rely on biased elite accounts (Toner, 2002). Bioarchaeological contributions to Roman studies, such as pathological and dietary information obtained from skeletons (Garnsey, 1988; Jackson, 1988; Garnsey, 1999; Hope and Marshall, 2000; Toner, 2002) have been exciting developments as they come from directly ancient Romans themselves and can be used as foils against elite descriptions of life in the historical record.

This chapter synthesizes the work on daily life in Ancient Rome from a variety of disciplines. This synthesis is ultimately geared to inform the bioarchaeological analyses of the skeletal sample from Velia (see Chapter 7), and for future researchers interested in the daily lives of Romans. This chapter also focuses temporally on the first and second centuries AD, as the Necropolis of Velia dates to this period. Defining the popular culture and daily life of non-elites is a challenging task, as the millions of non-elite Romans did not form a unified class or subgroup (Toner, 2009). With the social complexity of Roman popular culture in mind, this discussion of daily life will review societal structure, work, diet and nutrition, gender roles, family and reproductive history because these form important biocultural influences on growth and development and overall health of the skeleton later in life. In addition, Velia functioned as a thriving port city, and so an analysis of urbanization and what it meant to live in a Roman city will be discussed. The special character of port cities like Velia, notably Ostia, will also be explored. One of the goals is to attempt to contrast life in the city against what most Romans experienced, a life of agriculture on the farm (Toner, 2009), and what that might mean for the health and well-being of people living in and out of Roman cities. The chapter concludes with an archaeological summary of Velia, as well as the complete profile of the skeletal sample.

### **Rome in Historical Context**

The city of Rome is located approximately half way down the Italian peninsula and is situated along the banks of the Tiber at a rather large C-shaped bend in the river (Connolly and Dodge, 1998; Aldrete, 2004) (Figure 7). The Tiber runs from the Apennine mountain range in the East to the Mediterranean (Aldrete, 2004). The bend in the river acted as a natural fording point and was a logical choice in the foundation of the original settlement (Tingay and Badcock, 1989; Connolly and Dodge, 1998). The Tiber also provided means for transportation, irrigation,

drinking water, and fishing (Tingay and Badcock, 1989; Connolly and Dodge, 1998). Other aspects of the physical geography of Rome were also important to its site location. Numerous hills surround the river crossing. The hills provided defensive capabilities as well as protection against the frequent flooding of the Tiber (Stambaugh, 1988; Tingay and Badcock, 1989; Connolly and Dodge, 1998; Aldrete, 2004). All of the geographical features made the site of Rome an attractive place to establish a new community.



**Figure 7** - Map showing the location of Rome and Velia (courtesy of Dr. Luca Bondioli)

Numerous origin myths of Rome have been created. The most common of these is the myth of Romulus and Remus, twin sons of the Vestal Virgin, who had been raped by the god Mars (Tingay and Badcock, 1989; Creighton, 2000; Aldrete, 2004). The twins were abandoned by their mother along the Tiber, only to be rescued and nursed by a lone she-wolf (Tingay and Badcock, 1989; Creighton, 2000; Aldrete, 2004). The young twins were later discovered by a shepherd and raised as his own (Tingay and Badcock, 1989; Creighton, 2000; Aldrete, 2004). As adults, the twins returned to the site of their abandonment along the Tiber to establish a new city (Tingay and Badcock, 1989; Creighton, 2000; Aldrete, 2004). The brothers are reported to have begun a fierce argument over who would be king, resulting Romulus murdering his brother in blind rage (Tingay and Badcock, 1989; Creighton, 2000; Aldrete, 2004). Rome thus received its name from the victor of the argument. Aldrete (2004) reminds us that this myth is somewhat unique in that the pivotal events involve rape and murder. It also introduces important themes that will recur many times throughout Roman history, such as conflict between men and a desire for power (Aldrete, 2004). Even among commoners sharing the same difficult lot in life, neighbors were seen as competitors (Toner, 2009). Conflict was a theme that coursed through all levels of Roman society.

Archaeological evidence places the foundation of Rome at around 1000 BC (Mackay, 2004), although the traditional historical founding date is accepted to be 753 BC (Stambaugh, 1988; Creighton, 2000; Aldrete, 2004). Rapid population increases and urbanization began early at the site shortly after its establishment (Aldrete, 2004; Mackay, 2004). This initial period is classified as dynastic or monarchical as Rome was first ruled by numerous kings (Mackay, 2004). The last king of Rome, Tarquinius Superbus (“the Arrogant”), was removed in 509 BC and the Republic was formed (Aldrete, 2004; Mackay, 2004).

The early Republican period was marked by rapid development and a conscious effort to supersede the accomplishments of the monarchical period (Aldrete, 2004). A pivotal event for the early Republic was the near-sacking of Rome by the Gauls in 390 BC (Mackay, 2004). In some sense this defeat re-militarized Rome as fortifications, which were absent previously, were immediately built around the city (Aldrete, 2004). Defense became a significant concern. By 264 BC all of Italy had been colonized and expansion continued outwards (Mackay, 2004). In the second half of Republic, Rome faced many wars, including numerous conflicts with Carthage, The Macedonian war in Greece, the Punic wars in Spain, and a massive slave uprising led by Spartacus to name some of the better known events (Scullard, 1980; Aldrete, 2004; Mackay, 2004). Rome had become a world power and was struggling to find balance both internally and with its colonies (Scullard, 1980).

Towards the end of the Republic a delicate truce between Caesar, Crassus and Pompey had formed (Shotter, 2005). Caesar desired a consulship, but required an alliance with the other two influential men in order to secure it (Shotter, 2005). The first triumvirate, as it is known, began to crumble rapidly after its formation as each man vied for more control over Roman lands, resources and people (Aldrete, 2004; Shotter, 2005). Civil war erupted and initiated what would become the downfall of the Republic (Shotter, 2005; Mellor, 2006). Caesar emerged victorious and named himself dictator for life and was assassinated shortly afterwards (Aldrete, 2004; Mellor, 2006).

Caesar’s heir, Octavian (Augustus), would form an alliance with Mark Antony and Lepidus, two other politically and militarily influential leaders (Mackay, 2004). Octavian was to

rule the west (including Italy), Antony the east and Lepidus Africa, notably Egypt (Creighton, 2000; Mackay, 2004). Tensions between Octavian and Lepidus arose quickly as Lepidus used the Egyptian grain resources essential to the survival of the Empire as political leverage against Octavian (Creighton, 2000). Civil war broke out between Octavian and Lepidus, with Octavian emerging the winner (Creighton, 2000). Octavian's increasing influence and power worried Antony and so he quickly attempted to eliminate Octavian as a competitor (Creighton, 2000). Antony's military engagements ultimately failed and Octavian took full control of the Roman world (Mellor, 2006). The Imperial period had begun.

At the beginning of Augustus' reign (27 BC-14 AD), the Empire had nearly reached the extent of its maximum territorial expansion (Creighton, 2000). Only Britain and Dacia were made new provinces during the first two centuries of the Empire (Creighton, 2000), as Octavian's goal was to restore Rome to its former glory rather than expand substantially. Octavian's motivation for renewal was based the fact that Rome had fallen into disrepair during the political and military struggles at the end of the Republic. Octavian began a series of public works meant to renovate and expand the city, and is famously claimed to have stated, "I found the city made of brick and left it made of marble" (Aldrete, 2004: 18).

The series of Emperors that followed were not uniformly Octavian's equal. Nero's insanity and cruelty is legendary, although Mackay (2004) has argued that this characterization is overstated. The first hundred years of the Imperial period have been described as relatively stable and prosperous (Schwartz, 1998; Aldrete, 2004), but other historical reconstructions produce a more tumultuous picture (Heckster, 2006). In 64 AD a catastrophic fire stuck Rome and burned large portions of it to the ground (Creighton, 2000; Aldrete, 2004). Nero would commit suicide shortly after, an act that caused another civil war as powerful senators grasped at a chance at playing Emperor (Heckster, 2006). This period of instability followed until Vespasian established the Flavian Dynasty in 70 AD (Mackay, 2004). This period is regarded as the Pax Romana, or period of Roman peace (Schwartz, 1998; Mackay, 2004). Civic expansion and florescence continued until the death of Marcus Aurelius, who is considered the last of the great rulers of the Flavian dynasty during the second century AD (Aldrete, 2004). Aurelius' son Commodus took control of the Empire and this point marks a long period of instability that would last nearly a hundred years (Mackay, 2004).

## **Daily Life in Imperial Rome**

The aspects of daily life that are discussed below were selected because they provide useful information on building a holistic biocultural analysis that integrates many aspects of daily life that have direct consequences on health. The sections on societal structure and gender roles are meant to highlight how Roman society was divided along class and gender lines. The exploration of diet and nutrition is crucial as it provides insight to both culture and health of everyday Romans. Roman attitudes toward family and children will be discussed given that children form an important anthropological source of information about a society, both biologically and culturally. In the maternity and childbirth section the aim is to try to get a hold on the reproductive histories of Roman women as well as typical weaning times and breastfeeding beliefs. This is an important line of cultural evidence as reproductive history of the



mother and the development of an infant are thought to greatly affect the skeleton and adult health (Bogin, 1999; Gluckman and Hanson, 2004).

### *Societal Structure – The Haves and Have-nots*

The class distinction between slaves and freedmen defined one of the starkest social differences in Roman society. In Italy, 15 to 25 per cent of the population would have been slaves, but this number would drop to 10-15 per cent elsewhere in the Empire (Toner, 2009). Slaves were considered chattel, little more than living farm tools (Scullard, 1980; Tingay and Badcock, 1989; Toner, 2002). Slaves were also judged to have little ethical worth and were portrayed regularly as thieves, unreliable, unruly and morally wicked (Mathisen, 2003). All genders, ages and nationalities were used as slaves (Tingay and Badcock, 1989; Toner, 2002). However, the experience of slaves was not uniform, with some able to live (relatively) decent lives with a fair amount of autonomy, perhaps even their own family life (Garnsey and Saller, 1987). One example of this ‘gentler’ form of slavery is in the port city of Ostia, where slaves born into the household and may have been shown considerable warmth and care (Meiggs, 1960). This form of slavery was usually confined to the city (Garnsey and Saller, 1987); slavery in rural areas normally resulted in a very physically and emotionally difficult life (Toner, 2009). It is unclear exactly how the treatment of slaves may have operated at Velia. Velia was a port city, but also a city that relied on agriculture within city walls (Craig et al., 2009). The population of Velia was in all likelihood of a lower social class (Fiammenghi, 2003), so slaves probably experienced a life closer to the “rural type” than what many may have enjoyed at Ostia.

Psychological oppression, physical/sexual abuse, and the dissolution of families was a lifetime experience for many Roman slaves and was part of Roman culture throughout its history (Garnsey and Saller, 1987; Toner, 2009). Nevertheless, manumission, or physical freedom, was available (Casson, 1998). Some could even earn a salary to purchase their eventual freedom (Casson, 1998). In either case, a freed person was usually considered a client of the former owner (see below) and still provided public service to them. First generation freed persons had nearly no option of social mobility, but their children could advance in social rank if they were wealthy enough and owned land (Meiggs, 1960; Garnsey and Saller, 1987; Casson, 1998). However, the lives of most freed, non-elite people were extraordinarily difficult, to the extent that only their official status as “free” separated them from the lives of slaves (Tingay and Badcock, 1989; Toner, 2002). As Toner (2002: 62) has stated, “freedom didn’t fill your belly”. Even for the newly freed, approximately 10 per cent would be essentially destitute (Toner, 2009). This figure could increase to nearly two thirds of commoners in times of economic and political crisis (Toner, 2009). In the end, freedom was a political reality, not an economic one.

Another social distinction to consider is between skilled and unskilled labour in Roman society. Those with skilled labor could fill out an economic niche and could support themselves fairly well in most cases (Stambaugh, 1988; Toner 2002; Aldrete, 2004). On the other hand, unskilled individuals were treated much like oxen or cattle, destined to menial jobs usually involving substantial physical labor (Toner, 2002; Aldrete, 2004). As such, unskilled people were paid less and experienced much harder lives as a general rule. In summary, the quality of life of slaves or common free people had a lot to do with more than social hierarchy.

Slavery aside, throughout its history Roman society has been deeply divided along lines of rank (Garnsey and Saller, 1987; Dupont 1993, Adkins and Adkins, 1994; Casson, 1998; Dixon, 2001; Toner, 2002; Aldrete, 2004). Personal wealth certainly reflected the divisions within society, but it was not the central basis for those differences. The first and most important distinction was whether or not an individual was classified as a citizen (Dupont, 1993). This distinction was strongly present in the Republic and continued into the Empire. Until the beginning of the third century AD, when citizenship was granted to all adult males, only a very small percentage of individuals could be considered full Roman citizens (Aldrete, 2004). If the figures provided by Aldrete (2004) are more or less accurate, only about 10% of Roman people throughout the Empire were citizens prior to the third century AD. Citizenship could only be granted to free adult males (Dupont, 1993; Casson, 1998; Aldrete, 2004) and so slaves, women, and children would never qualify. Citizenship required service during periods of war, but it also granted some form of protection under the Roman judicial system, something non-citizens usually did not experience (Aldrete, 2004).

Personal monetary wealth was certainly used to gain advantage in Roman society and did mark some form of class boundary within the elite orders. To begin, wealth was certainly required to belong to the highest political offices. To hold any of the various forms of Magistrates (Senatorial order), a Roman citizen had to be worth at least 1,000,000 sesterces (Garnsey and Saller, 1987). The Equites had to have a personal wealth of at least 400,000 sesterces (Garnsey and Saller, 1987). The requirement for Decurions was more fluid, but seemed to be around 100,000 (Garnsey and Saller, 1987). For a sense of scale, the richest in the Roman world, who were only around 200,000 individuals out of millions, had an estimated total wealth of around 100 million sesterces (Toner, 2009). These sums of money were vastly above what the average Roman was worth, approximately 25,000 times the average subsistence income, and had more to do with establishing rank among the elite rather than between the elite and the humble. This last point is important. The consensus among historians seems to be that there was no identifiable middle class in Imperial Rome (Runciman, 1983; Garnsey and Saller, 1987; Toner, 2002; Aldrete, 2004), although Casson (1998) argues that the size and diversity of the Roman economy would have lent itself to the development of a middle-class. Certain populations, such as the people of Isola Sacra, have been classified as middle-class as well (Meiggs, 1973; Garnsey, 1999). Another exception may be soldiers, but even with salaries that were above the average worker, they do not comprise a middle class in the modern economic sense (Garnsey and Saller, 1987). Runciman (1983) has argued that the Roman economy was capitalist in the market sense, but that that a middle class was never produced because Roman society was never truly democratic. All in all, historians know very little about the Imperial Roman economy because there “are no government accounts, no official records of production, trade, occupational distribution, taxation” (Garnsey and Saller, 1987: 43). Large-scale systematic reconstructions of the Roman economy are thus not feasible or are tentative at best (Garnsey and Saller, 1987), but some aspects of the economy and distribution of wealth are known. Moreover, elite culture also distanced itself from common people by an “upper-class culture of learning”, or *paideia* (Toner, 2009: 3). The *paideia* was structured to deliberately exclude the uninformed through the practice of impenetrable jargon relating to the arts, academic discourse, and law (Toner, 2009). As a final note along these lines, the primary source of all elite wealth was land (Garnsey and Saller, 1987). This is probably because around three quarters of all labor went into the production of food (Garnsey and Saller, 1987). Land ownership was also expensive (Garnsey and Saller, 1987) and it left most independent farmers at the subsistence level. The Roman elite held numerous plots of

land from which they could live comfortably off the work of freedmen and slaves who operated their estates (Aldrete, 2004).

The Patronage system was one of the ways that Roman society was able to link together these disparate social divisions. This social mechanism worked as follows: men of high social status (Patrons) would serve a number of his social inferiors (clients) through financial or legal assistance (Garnsey and Saller, 1987; Dupont, 1993). The assistance or protection clients received would be returned to the Patron through public acts that would enhance his reputation and prestige (Aldrete, 2004). This could take the form of public speeches or even enthusiastic clapping at a public gathering hosted by the patron (Aldrete, 2004). To some extent, this system may have helped alleviate tension between the very distinct social groupings of Roman society. Finally, in a culture so obsessed with rank, it served as continuous public display of social positioning (Garnsey and Saller, 1987). In Roman society status was highly dependent on the social perception of honor and moral worth and it appears that the patronage system served to reinforce positive social perceptions of the Patron (Garnsey and Saller, 1987). As Dupont (1993: 31) has stated, “[w]ealth was of no use except in public life”.

In summary, social structure in Rome was divided along many lines and resulted in a fairly complex connection of relationships. Status and social relations were derived from land ownership, citizenship, wealth, freedom (or lack thereof), social prestige/morality as well as gender and age (more on these last two points later). As a result, this system created some paradoxes. For instance, slaves and freed people in the city occasionally had more room for social mobility and may have had it easier than free, but humble peasantry in the countryside. In large part, this had to do with skilled slaves having an economic advantage over those without special skills to sell (Garnsey and Saller, 1987). In many cases, favored slaves or ex-slaves in the city also had the benefit of the patronage system, and were thus able to obtain capital for businesses or other investments (Meiggs, 1960; Garnsey and Saller, 1987). City slaves were often also well connected through prestigious families, something humble freedmen could often not also claim (Garnsey and Saller, 1987). The point here is that social positioning was not linear and depended on a number of social and economic factors. As a final point, the lives of elites, as well as the popular culture of the common people would have changed gradually over time, as it does now (Toner, 2009), and so some of the dynamism of social change is lost in a more static summary of this nature. However, the descriptions of common and elite life are structured to represent the average experience, and represent a majority of the people living in the Imperial period.

## **Work in Ancient Rome**

The majority of the Roman population worked in the fields supporting themselves and nearby cities (Garnsey and Saller, 1987; Tingay and Badcock, 1989; Dupont, 1993; Casson, 1998; Erdkamp, 1999; Toner, 2002; Aldrete, 2004). As mentioned previously, farming was the basis of economic wealth and it defined the lives of most Romans, and was important for Velians as well (Craig et al., 2009). Non-elite farms tended to be rather small. Archaeological reconstructions show that the average farm was around 1.6 hectares, which is roughly what one family could manage successfully (Aldrete, 2004).

Erdkamp (1999: 572) has stated that the “rural world in its entirety remains largely hidden in the dark”. There is much we do not know about the life and work of rural commoners. Historians have noted the difficulty of farming in the Roman period (Garnsey, 1988; Erdkamp, 1999). Oxen were expensive beasts, so much so that often one team was shared among an entire village (Aldrete, 2004). This meant that most of the tending of the fields was done by hand using rudimentary tools (Aldrete, 2004). Everything from weeding, to harvesting, to threshing was a product of manual labor (Aldrete, 2004). In addition to these challenges, Roman farmers faced low seed to reap ratios (1:4) because they simply scattered seeds by hand (Aldrete, 2004). For comparison, modern farmers have yields of up to 1:50 (Aldrete, 2004). This problem was compounded by the fact that crops were lost to flood, cold, vermin and so forth (Garnsey, 1988). Inter-annual variability in crop yields was high in many areas (Garnsey, 1988). Thieves were also a common issue (Aldrete, 2004). The result was that a year of difficult toil would generally only provide just enough for the family, perhaps 5-10% surplus if they were lucky (Garnsey and Saller, 1987; Aldrete, 2004). Olive and grape production demanded far less physical labor and harvesting was generally done by the elderly and children (Aldrete, 2004).

The relatively small number of Romans who were not agriculturalists (or serving in the military) lived in cities (Aldrete, 2004). Numerous options were available for work, many of these divided along class lines (Aldrete, 2004). Work for upper class Romans was generally politically related (Aldrete, 2004). These professions were not salaried positions; reimbursement came in the form of social prestige (Dupont, 1993; Aldrete, 2004). Wealth was maintained from profits off of their estates (Aldrete, 2004). The lower classes were essentially divided into skilled and unskilled labour, as described previously (Brunt, 1980; Erdkamp, 1999; Aldrete, 2004). Aldrete (2004) notes that over 200 professions are known from inscribed tombstones in Rome. Many of these are highly specialized, filling very specific niche markets. Some of the more esoteric examples include a tailor who specialized in embroidery using only feathers, as well as a hair removal technician who plucked out underarm hair for a small fee (Aldrete, 2004). Despite this apparent diversity of work, most people in Rome and other cities would have been unskilled labour, many of whom were also free-born (Brunt, 1980). Even subsistence farmers had to occasionally sell themselves as unskilled laborers to support the farm in meager times (Erdkamp, 1999). The lives of unskilled labor were harsh and uncertain. While skilled Romans could count on a fairly regular work load and thus pay, unskilled labour had to actively find someone to hire them each day and as a result work was patchy and financial rewards were meager, particularly in the city (Brunt, 1980). Work may have been seasonal as well (Craig et al., 2009). A large pool of unskilled labour was very advantageous for the wealthy; slaves had to be maintained and the loss of slaves could become expensive (Brunt, 1980). There were no such worries with unskilled freedmen (Brunt, 1980).

At this point it is worth discussing Roman notions of work and morality. Roman attitudes to work reflect in some way the argument by Toner (2002) that elite and humble Romans shared very different cultures. Upper class Romans simply did not like or want to work (Aldrete, 2004). Physical labour or even commerce was considered crude and vulgar (Aldrete, 2004). This attitude was extreme. The elite considered “only those people who were so rich that they did not have to do anything to earn a living...fully human and civilized” (Aldrete, 2004: 190). The only exception for non-degrading forms of work for the elite was farming (Aldrete, 2004). This was why elite status essentially required land-ownership, as wealth gained via commercial success was morally questionable. Of course, few elites ever actually worked on their estates (Aldrete,

2004), owning and living off the profits of the land was enough to show moral virtue. The division of work between the classes was probably quite distinct, but Robinson (2005) presents evidence from Pompeii that suggests this boundary may have been more fluid in actual practice. Robinson (2005) used spatial analysis from areas of textile production, the hospitality industry (taverns, inns), property rentals and agriculture to show that elite and non-elite spheres of influence often crossed. The moral underpinnings that greatly separated these two economic worlds still functioned in most cases, but the day to day activities of the upper and lower classes often bled into each other. For example, elites would finance and build businesses in higher density, poorer regions of Pompeii to maximize profitability and to gain clients to raise the public moral standing and political influence of the owner (Robinson, 2005). In return, non-elites could receive shops and businesses as dowries in order to secure ties and alliances between families (Robinson, 2005).

Nevertheless, Roman non-elites had attitudes towards work that were typically very different than the moral repugnancy elites felt at the thought of earning a living. In Pompeii, one tombstone proudly declared, “Profit is happiness” (Aldrete, 2004). Commoners also formed professional associations, called *collegia* (Aldrete, 2004). These worked like business associations where professionals would help each other financially or socially (Aldrete, 2004). Monuments were often erected to display accomplishments (Aldrete, 2004). There is some indication through graffiti that *collegia* were politically influential or at least active in voicing their support for one politician or another (Aldrete, 2004). Guilds were also important and much of what historians know from these guild associations come from Ostia (Meiggs, 1960; Hermansen 1981), and functioned in essentially the same way as *collegia* (Meiggs, 1960). During the Imperial period industry as a whole changed very little (Adkins and Adkins, 1994). Even with these professional associations, technical innovation was slow, and most professions were highly labor intensive (Adkins and Adkins, 1994). However, Delaine (2005) has noted that in Ostia, most commercial spaces were multi-functional and complex. Commercial spaces were also often “marked by differential degrees of visibility and variable control of access requiring different degrees of local or specialised knowledge to navigate” (Delaine, 2005: 45). Business owners were then essential to the day to day functioning and well being of the city, wresting some control away from elite interference in daily life (Delaine, 2005).

### *Diet and Nutrition*

This section will focus on diet in Italy primarily, as dietary practices across the Empire was quite diverse and a complete accounting of these practices would not be informative about life at Velia. The Mediterranean triad of cereals, olives and wine formed the core of the agricultural products in Imperial Rome (Garnsey and Saller, 1987; Garnsey, 1988; Garnsey, 1999). Cereals were generally ubiquitous across the Empire, but olives and wine had a more variable distribution due to the geographic diversity of the conquered Roman world (Garnsey, 1999). In the cereal group, wheat was dominant, but barley was common as well (Rickman, 1980; White, 1988; Garnsey, 1999; Matz, 2002). As a whole, it is estimated that cereals made up approximately 70-75% of the average Roman diet (Rickman, 1980; Foxhall and Forbes, 1982; White, 1988; Garnsey, 1999). Garnsey (1999) has argued that pulses or legumes should also be

considered an important food source in reconstructions of Roman diets. These were essential because they served as a surrogate for meat proteins and filled in nutritional gaps that cereals could not provide (Garnsey, 1999). Meat and fish were consumed, but should not be considered staple foods (Garnsey, 1999; Matz, 2002), although Jackson (1988) has claimed that the large amount of faunal remains in Roman archaeological sites is highly suggestive of regular meat consumption. Brothwell (1988) has noted that literary and archaeological evidence indicates that pork was commonly consumed in the Mediterranean diet from the Neolithic onwards. Sheep and cattle were also raised, but used mainly for utilitarian purposes other than regular meat consumption (Brothwell, 1988). White (1988) has noted that the military provides one exception; records indicate that beef was a regular component of a soldier's diet. Overall, meat as a food source was probably in short supply because the physical geography of the Mediterranean could not support large scale animal husbandry (Garnsey, 1999). Growing seasons are short and many regions are arid or semi-arid (Garnsey, 1999). It then becomes much more economical to farm plants than raise animals, as plants provide a much greater return per energy investment than animals do (Garnsey, 1999). Fruits and vegetables grown locally on the farm also served as important sources of nutrients and fiber and were consumed along the Mediterranean triad (Adkins and Adkins, 1994; Garnsey, 1999; Matz, 2002).

As a whole, the Mediterranean diet is extremely healthy, particularly when compared to modern affluent Western diets (Garnsey, 1999). In contrast to maize or rice based diets, there is no deficiency disease related to heavy wheat consumption (Garnsey, 1999). Protein levels are relatively high in wheat, unlike many other staple foods across the world (Garnsey, 1999). However, cereals do lack some important amino acids, notably lysine (White, 1976), but these can be obtained from meat, fish or pulses (Adkins and Adkins, 1994; Garnsey, 1999). Another important note is that the processing of wheat can change its nutritional value. For instance, poorly sieved flour has higher phytate content due to a higher quantity of bran and germ remaining (Garnsey, 1999; Larsen, 2002). Phytate impedes the absorption of minerals such as iron and calcium and is linked with iron-deficiency anemia, dwarfism and rickets (Larsen, 2002; Nelson et al., 2003). So while wheat is a good staple food, it must be supplemented with additional protein sources and its processing can either enhance or detract from its nutritional value.

Olives and wine also played an important role in the Roman diet (Adkins and Adkins, 1994). The function of olives in the Mediterranean diet was quite varied. Olives served as the key source of dietary fat (Garnsey, 1999). Olives also had a number of utilitarian uses, such as lamp oil or once fully pressed the leftovers could be used as fertilizer (Garnsey, 1999). All social classes consumed wine, but qualitative differences served to mark social boundaries and reify divisions in rank (Lewis and Reinhold, 1966; Adkins and Adkins, 1994). Wine was consumed heavily, by both young and old alike, but was almost always watered down and sweetened with honey when available (Adkins and Adkins, 1994). The total amount of wine consumed daily was still substantial, with some estimates averaging at around one bottle of wine per day (Purcell, 1985). The percentage of alcohol in Roman wine is unknown however (Purcell, 1985). The Roman love of wine may have in fact been a strain of the agrarian economy (Purcell, 1985). Viticulture is a sensitive process and vulnerable to small environmental changes (Purcell, 1985). Large tracts of arable land were used for wine production, reducing the availability of food staples such as wheat (Purcell, 1985). The labor costs involved in wine production were also

quite high, requiring three times the manual labor than traditional farming come harvest (Purcell, 1985).

As mentioned previously, actual work on the land was difficult and yields were often low. Even with little to no surpluses, most farmers had to trade some of their crops for resources they needed and could not produce themselves, such as salt or olives (Garnsey, 1999). So while people aimed at self-sufficiency, it was hardly ever reached (Erdkamp, 1999; Garnsey, 1999). Many farmers also had to deal with creditors, landlords and taxation, placing further economic burdens upon them (Garnsey, 1999). As for the actual productivity of the land, concrete statements are difficult to make. What is known is that that inter-annual variability could be high, creating added risk and uncertainty (Garnsey, 1988). Food crises were also exacerbated through military campaigns as extra resources were required (Garnsey, 1988). Despite all these risks, actual large scale famine events were low as farmers and upper-class Romans had long standing risk-reducing mechanisms in place (Garnsey, 1988), but daily caloric intake was often relatively low (Garnsey, 1999).

Food itself may have been a potential source of illness and paradoxically, medicine (Capasso, 2007). Capasso (2007) analyzed the macro-botanical remains from Herculaneum, a site affected and preserved by the eruption of Mt. Vesuvius. The results indicated that many of the food remains were infected with numerous forms of bacteria, including what is probably salmonella (Capasso, 2007). Contrary to expectations, the skeletons at Herculaneum show very little evidence of non-specific infection (Capasso, 2007). This is unexpected because skeletal lesions indicative of infection are common in ancient societies (Rothschild and Martin, 1993) due to poor sanitation, or in this case, one would think food (Capasso, 2007). Capasso (2007) found that the process of Roman fruit preservation, typically with pomegranates and figs, can result in the development of tetracycline in the dried product. Tetracycline is a natural antibiotic that may have controlled the prevalence of non-specific infection in Roman people, at least at Herculaneum (Capasso, 2007). Direct evidence of tetracycline was present in the skeletal remains (Capasso, 2007). Rates of brucellosis, a bacterium of animal origin, at Herculaneum was still very high (Capasso, 2007). Tetracycline is still used today to treat brucellosis, but it must be administered very early on in the development of the disease to have any effect (Capasso, 2007). So it appears that at Herculaneum, and perhaps elsewhere, food was a potential source of illness, but of medical assistance as well. The dosage of tetracycline was enough to cure most mild infections but not more persistent infections, such as brucellosis.

Garnsey (1999) has raised a number of excellent points about common misperceptions of the Roman diet. Garnsey (1999) noted that scholars have tended to equate the health benefits of the Mediterranean diet today with the good health of Roman citizens. This assumption is false because it does not account for the availability of food on a day-to-day basis and that endemic malnutrition most likely characterized Roman life (Garnsey, 1999). Chronic malnutrition, coupled with the better known, but rare, dramatic and devastating famines, provided a powerful critique of the assumption that the nutritional value of the Mediterranean triad automatically translated into healthy Romans.

The most promising and direct evidence of endemic malnutrition has come from Romans themselves through bioarchaeological analyses of their remains (discussed in more detail further on). Biological anthropologists have long known the value of skeletons in reconstructing diet and nutritional health in ancient populations. Inadequate nutrition can lead to a multitude of diseases

(Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003), including what pathologists call diseases of deficiency (Garnsey, 1999). Some relevant diseases or effects of poor nutrition include dental enamel hypoplasia, porotic hyperostosis and short stature (Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003). All of these are common in Roman skeletal remains, with women and children generally experiencing a higher prevalence (Garnsey, 1999), although this is not always the case (Paine et al., 2009). Further discussions of dietary deficiencies will be expanded in the section devoted to diseases further in the chapter.

One important issue that some Roman scholars have raised is that most of what we know about the Roman diet comes from texts (Cummings, 2008). This poses a problem given that most evidentiary sources were produced by the elite (Toner, 2002). So far, only a handful of studies have conducted isotopic analyses on Imperial Roman skeletons. The most significant study was conducted from individuals from Isola Sacra, the necropolis of Portus Romae, which served as one of Rome's major ports (Prowse et al., 2004). Prowse et al. (2004) found that while diet was mostly terrestrially based (from cereals), higher than expected nitrogen values suggested that marine foods were also quite important. This is unsurprising, as the authors state, fishing was probably an important industry given that the city functioned as a port (Prowse et al., 2004). In a follow-up study, Prowse et al. (2005) showed that there was also a small age and gender bias in the sample diet. Children were denied a varied diet, subsisting almost entirely on poor quality cereals once weaned (Prowse et al., 2005). The gender bias presented itself from consistently lower  $\delta^{13}\text{C}$  and  $\delta^{15}\text{N}$  levels in females, which is suggestive of reduced access to these isotopically enriched marine foods (Prowse et al., 2005). However, a number of females at Isola Sacra consumed more protein than many other males (Prowse et al., 2005), suggesting that preferential access to food may have been more related to elite status than sex or gender. This is consistent with observations that what was socially proscribed by the elite may not have been followed strictly by the poor.

Cucina et al. (2006) found that wealthy suburban diets may have differed significantly from the expected grain based diet of most Romans. Cucina et al. (2006) examined the skeletal remains of individuals, probably laborers, from the Necropolis of Vallerano, a suburb of Rome dated to the 2<sup>nd</sup> and 3<sup>rd</sup> centuries AD. Evidence of physiological stress was high in the population (Cucina et al., 2006), which is in accordance with Garnsey's (1999) depiction of endemic malnutrition. What was unique about this population was that oral pathologies normally associated with carbohydrate rich diets were very low (Cucina et al., 2006). This finding led the authors to suggest that perhaps more meat was being consumed in this suburb (Cucina et al., 2006). No isotopic study was conducted, so this remains in question. A recent study by Cummings (2008) showed elevated patterns of animal protein consumption in a large Romano-British sample. While the Cummings (2008) sample was not near the heart of Rome or part of the traditional Mediterranean region, the findings are worth consideration in light of the Cucina et al. (2006) and Prowse et al. (2004) findings. It is possible then that in some circumstances or regions meat consumption, including fish was higher than previously thought. This line of argument is also consistent with the archaeological evidence Jackson (1988) has presented on faunal remains.

What can be concluded from osteological and isotopic analyses is that consumption of the typical Mediterranean 'triad' diet cannot always be assumed. Regional differences in social hierarchies, demographics, and gender roles most likely resulted in a complex and uneven



distribution of diets across the Roman world. Fortunately, the isotopic reconstruction for Velia is available (Craig et al., 2009) and is summarized in detail in the section on Velia at the end of the chapter.

### *Health and Disease in the Roman Era*

Roman society was highly stratified and spread across a variety of social and geographical contexts. This provides a challenge for any analysis of how disease shaped the lives of Roman people as access to food, care and exposure to various disease vectors would have varied across the population. To address this issue, differential risks between cities and rural areas will be explored first. Prevalence of disease along gender and age lines will then be explored.

Cities were crowded places to live by most accounts. While commoners suffered the most, the elite did not escape hardship. The rich did have better access to food, but poor sanitation/hygiene exposed all classes to infectious disease (Toner, 2002). Waste management was one of the major problems in Rome. Most people did not have latrines to use in their homes and so they simply dumped what they wished from their windows (Jackson, 1988; Stambaugh, 1988; Aldrete, 2004). There were laws against this practice, but it appears the practice went on unabated (Aldrete, 2004). Disposal of the dead was also a logistical problem (Stambaugh, 1988). While there were specialists and sanitation/religious laws that required the proper removal and treatment of the dead (Hope and Marshall, 2000), many reports suggest that the number of dead was so chronically high as to pose consistent practical problems of removal (Toner, 2002; Aldrete, 2004). As a whole, human waste, dead animal and human bodies, and household garbage were normal sights among Rome's crowded streets (Toner, 2002). Many roads were unpaved, creating a muddy and probably dangerous sludge during rainy periods (Aldrete, 2004).

Overcrowding and the filth produced throughout Rome's streets would have created an environment greatly facilitating the transmission of infectious diseases. The first of these are enteric viruses found in feces (Aldrete, 2004). These commonly cause gastroenteritis, but some forms can lead to meningitis and hepatitis A (Aldrete, 2004). Human and animal wastes also carry bacteria such as *E.coli* and salmonella (Aldrete, 2004). *E.coli* infection causes severe gastroenteritis and diarrhea (Aldrete, 2004). Salmonella poisoning commonly leads to gastrointestinal problems, but can also affect the respiratory, cardiovascular and nervous systems (Aldrete, 2004). Protozoans such as *Giardia lamblia* must have also been common, causing infection and diarrhea (Aldrete, 2004). Parasitic worms of all forms were also common (Aldrete, 2004). These cause a variety of health problems, but they are generally considered as a whole to weaken the individual and raise susceptibility to infection, but death can also occur (Aldrete, 2004). It is not clear whether or not cholera was endemic in Rome or other Roman cities because of the vagueness of ancient description of disease symptoms (Aldrete, 2004). If present however, the effects of cholera would have been profound, resulting in high amounts of mortality and morbidity. Lastly, malaria was probably an endemic problem as well, at least in Rome, since the city was built in marshland (Aldrete, 2004). The low-lying areas of Rome were drained by the Imperial period, but malaria was probably still a common aspect of daily life. This is an important consideration as endemic malaria can cause thalassemia, a condition that can produce

skeletal lesions that mimic other etiologies, such as nutritional deficiency or infectious disease (Keenleyside and Panatoyova, 2006).

Direct skeletal evidence of health in the Roman era is not widely available (Cucina et al., 2006), and compared to the wealth of knowledge on the politics, architecture and general history of Roman culture. The bioarchaeological studies that do exist are valuable as they provide excellent evidentiary sources scholars of Roman history can use to test documentary accounts of how disease shaped Roman society. There are some factors of inherent skeletal biology however that weakens them as sources of information about disease. As a general rule, bones grow and react to infection relatively slowly (Isacan and Kennedy, 1989; Ortner, 2003). This means that acute diseases that kill quickly will not leave markers on skeletons that can be read by bioarchaeologists or paleopathologists (Isacan and Kennedy, 1989; Margerison and Knüsel, 2002; Ortner, 2003). Moreover, there are some diseases, such as bubonic plague, that normally do not leave traces of their presence whether they were acute or chronic (Margerison and Knüsel, 2002). What skeletal evidence is particularly good for is a log of long term, chronic stress on the body. These can be nutritional deficiencies, metabolic disorders, and infection (Isacan and Kenedy, 1989; Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003). Many diseases are also unique to connective tissues and can be seen archaeologically (Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003). The major caveat here is that the individual had to have lived past the initial insult in order for the skeleton to react.

Diseases caused by poor hygiene and crowded conditions can be analyzed indirectly with skeletal remains. For example, direct evidence of enteric viruses or parasitic protozoans or worms will not be found on skeletons, although evidence for these can be found in soil (Reinhard, 1992; Bathurst, 2005). What we find on skeletons directly is evidence of the metabolic stress, usually anemia, caused by pathogens such as cribra orbitalia and porotic hyperostosis, as well as malnutrition (Garnsey, 1999). Iron deficiency has also been linked to these skeletal lesions (Ortner, 2003). These conditions cause abnormal enlargement of the spongy bone and marrow spaces in the eye orbit and cranium respectively (Ortner, 2003). The longstanding hypothesis has been that the skeleton does this to increase production of red blood cells during an anemic state (Ortner, 2003). However, Walker et al. (2009) have demonstrated through biomedical and bioarchaeological studies that humans respond to iron deficiency not by increasing red blood cell production, but by limiting it (Walker et al., 2009). Rather, they suggest that porotic hyperostosis and cribra orbitalia are caused by megaloblastic anemias resulting from depleted maternal vitamin B<sub>12</sub> during breastfeeding and the onset of weaning (Walker et al., 2009). Additionally, the generally unsanitary conditions in the past often contributed to an infants nutrient depletion in infants from regular periods of gastrointestinal infection (Walker et al., 2009). Evidence of porotic hyperostosis and cribra orbitalia are common in Roman skeletal samples (Garnsey, 1999). Enamel hypoplasia, which are growth defects on dental enamel obtained during infancy and childhood, are also common in Roman skeletons (Garnsey, 1999). Short stature is also a sign of metabolic stress (Larsen, 1997). All of these markers are termed unspecific, in that they are directly related to nutritional or physiological stress, but of unknown origin or specificity (Larsen, 1997; Ortner, 2003). While this limits the level of detail that bioarchaeologists can achieve, it remains a rich source of data on the health of Roman people.

Urban poverty was also a factor that should be considered when assessing the health of the average city dweller. Some estimates place the percentage of homeless at around 10 per cent,

but this could rapidly expand to nearly two-thirds of the population, depending on economic conditions (Toner, 2002; 2009). Debt in the countryside often led people to migrate to urban centers or large villas for work (Erdkamp, 1999; Toner, 2002). Unfortunately, this eroded family ties through geographical isolation, which contributed to the cycle of poverty (Toner, 2002). Some of the effects of poverty included poor diet, nutrition and hygiene (Toner, 2002). Even access to proper clothing was also a problem. For most Romans, not only the abject poor, clothing was expensive and limited (Toner, 2002). Clothing often served as bedding as well, and fleas were probably common (Toner, 2002). Archaeological and ethnographic evidence from other pre-industrial cities today suggest that many poor Romans probably had to sleep outside, among the refuse in the streets or next to the Tiber (Toner, 2002). Mental health likely suffered as well (Toner, 2009). Frequent exposure to disease, either in oneself or seen on others, was a social reality.

Historical information on disease loads and the social realities of disease in rural areas is much scarcer. As a whole, rural regions were likely better than urban centers. Much of what has been described so far is a function of aggregation and crowding, coupled with poor knowledge of hygiene and pathogens (Stambaugh, 1988). Rural areas dealt with these diseases but to what extent is unclear as most studies have explored city contexts. Urbanization typically involves increased physiological stress from environmental factors such as poor sanitation and thus disease (Storey, 1992; Lewis, 2002; Lewis and Gowland, 2007). The diseases most encountered by rural commoners were normally attributed to nutritional deficiencies brought on by endemic malnutrition (Garnsey, 1999).

The health of the population of Urbino presents an excellent example of the hardship endured by many Romans. Urbino is located approximately 45km inland from the present day city of Pesaro, on the northeastern coastline of Italy (Paine et al., 2009). The skeletal sample from Urbino is from the Imperial period and is essentially contemporaneous with Velia. The Urbino population is also thought to have been largely comprised on non-elites (Paine et al., 2009). Overall, the health of the Urbino people can be classified as quite poor. Average adult stature was very short, 5'4" for males and 4'11" for females (Paine et al., 2009). Paine et al. (2009) note that this is shorter than most other sample of Italian origin and that this degree of growth stunting represents chronic malnutrition. Life expectancy was usually low, at 24-26 years of age (Paine et al., 2009). All of the adults at Urbino had dental enamel hypoplasia defects (Paine et al., 2009). This fits quite nicely with arguments by Garnsey (1998) that chronic nutritional deficiencies were a fact of life in the Roman world. Also, males generally had higher rates of skeletal lesions (cribra orbitalia, porotic hyperostosis, periostitis) than females (Paine et al., 2009). This observation is consistent with modern studies that show morbidity and mortality favor females over males (DeWitte, 2010). As a whole, the high prevalence of these lesions, and at young ages (below 40), strongly suggest that day-to-day life was physically challenging (Paine et al., 2009). For example, Schmorl's nodes do not typically afflict the young as they are normally produced by herniated disks in older people (Paine et al., 2009). In most cases, the presence of Schmorl's nodes in young individuals is a product of heavy manual labor (Paine et al., 2009). The context of health at Urbino is not unique, similar findings have been found at other Imperial Roman sites (Salvadei, et al., 2001; Facchini et al., 2004; Cucina et al., 2006). Paine et al. (2009) conclude that lived a life full of physiological stressors and that they experienced a generally poor quality of life that was defined by chronic health problems.

The health of the populations of Herculaneum and Pompeii have also been studied (Bisel and Bisel, 2002) and present an exciting case study as they represent a snapshot of a living population, and not a mortuary sample. Average adult height was slightly greater than at Urbino (Laurence, 2005). Periods of acute stress were common, with 80 per cent of the sample from Pompeii, and 50 per cent of those from Herculaneum with evidence of enamel hypoplasias (Laurence, 2005). This is substantial, but reduced from Urbino's population, who all had evidence of enamel hypoplasias (Paine et al., 2009). Chronic infection was also common at both Pompeii and Herculaneum, with around 30 per cent of individuals showing skeletal lesions of varying origin (Laurence, 2005). Henneberg and Henneberg (2002) have noted that syphilis was present, and that tuberculosis may have been endemic in these populations. Arthritic conditions were also prominent, affecting approximately 30 per cent of the population (Laurence, 2005). The demographic makeup of these two populations is not entirely certain, but the disparity in health and stature between some of them suggests a mixture of elite and non-elite groups (Laurence, 2005). However, even with the presence of elite individuals, stature and disease prevalence was similar to Urbino.

The picture that emerges from an analysis of disease prevalence in Ancient Rome is one of high morbidity. And yet, there is evidence to suggest that some sectors of society were less affected. A recent bioarchaeological study by Killgrove (2008) has revealed that in Casal Bertone, a district of Imperial Rome, individuals appear quite healthy. Developmental defects in the dentition and skeletal markers of dietary or parasitic stress were extremely low, particularly when compared to other bioarchaeological studies of Imperial Romans. It is unclear why exactly these individuals were apparently so much healthier. Killgrove (2008) is continuing work to see if status differences can be attributed to the improved health of the people, or what other causes may have led to this unusual scenario.

There is historical and bioarchaeological evidence to suggest that disease loads were not uniformly distributed through society, but rather along age and gender lines. As mentioned previously, there was differential access to food that favored men first and women second (Garnsey, 1999). Pregnancy also added extra strain because of potentially inadequate food intake and physical labor (Toner, 2002). A high degree of physical labor on an already weakened body only burdens it further and increases susceptibility to disease (Garnsey, 1999). One would expect that pregnancy combined with these factors probably resulted in women being at higher risk of infection than men, although Paine et al. (2009) and DeWitte (2010) have shown that this assumption may be flawed. However, there is some bioarchaeological evidence to support a bias against females. Cucina et al. (2006) found that enamel hypoplasias were slightly higher in women than men. The incidence of cribra orbitalia is more telling. At the site of Vallerano, the prevalence of cribra orbitalia for women was 85%, compared to 50% for males (Cucina et al., 2006). At Poundbury, a contemporary Roman-British site, females also showed higher rates of non-specific infection for dietary stress, but only moderately so (Farwell and Molleson, 1993). However, the individuals from the eastern cemetery of Roman London showed no sex differences (Conheaney, 2000). Shotton (2004) has argued that Roman-Britain was somewhat unique and enjoyed better agricultural productivity than other Roman provinces. This would help explain the discrepancy between British and Italian sites in rates of pathology between the sexes. As a whole, skeletal evidence from Roman sites indicates that women were afflicted with higher rates of disease than men (Garnsey, 1999), but this should not be assumed (DeWitte, 2010; Paine et al., 2009).

Roman children were also at extraordinarily high risk of infection and premature death. Exposure to the unsanitary environment that was typical of the Roman city posed great risks to the newborn. Infant mortality was high, with estimates that one quarter of children did not see their first birthday and that potentially one half did not reach the age of ten (Garnsey and Saller, 1987). Life expectancy in general was low, averaging around thirty years (Garnsey and Saller, 1987). If you lived to twenty, life expectancy increased to around forty years of age (Toner, 2002). Tombstones from Ostia inscribed with the age at death of the deceased indicate that 82% of males and 86% of females died before the age of 30 (Meiggs, 1960). This is not to say Romans were a people without elderly. These data simply indicate that the average Roman lived shorter lives on average than we do today, in large part because the extraordinarily high infant mortality rate greatly reduced life expectancy.

The health of children was compromised in part because of socially proscribed child rearing practices. Recall that mothers were told to withhold colostrum, and without this immune-boosting first milk, the risk of infection is much higher as the mother's antibodies are not transmitted to her child (Garnsey, 1999). Toner (2002) and Garnsey (1999) have argued that Roman weaning practices further endangered infants. These authors state that weaning was either done too early or too late and that children were weaned onto nutritionally poor foods (Garnsey, 1999; Toner, 2002). The last point is true, as isotopic and historical studies have shown (Garnsey, 1999; Prowse et al., 2008). Average weaning times were completed by around three years of age (Fuller et al., 2006; Dupras and Tocheri, 2007; Prowse et al., 2008). Fitzgerald et al. (2006) found that infants were indeed exposed to higher risk around 3 months of age. A second period of high risk was also found between months six and nine, the period typically when the weaning process began (Fitzgerald et al., 2006). This is also a period of rapid somatic growth (Fitzgerald et al., 2006) requiring more than cereals to meet nutritional needs (Garnsey, 1999).

What can be concluded from this review of health and disease in Imperial Rome is that most people were subject to high pathogen loads, particularly poor individuals in urban centers. However, assumptions of poor health need to be tested in bioarchaeological analyses of Imperial Romans however, given the results by Conheeny (2000) and Killgrove (2008). Women and children generally suffered more than men, regardless of context. The differential pathogen load among men, women and children were likely related to gender and age biases that often favored the better health of men.

## **Gender Roles and Family Life**

Family life is an understudied subject in Roman history (Saller and Shaw, 1984). Family represents the most important social (and financial) unit in Imperial Rome (Rawson, 1986) and is reflected the gender and age differences within Roman society at large (Rawson, 1986; Garnsey and Saller, 1987). Imperial Rome was strongly patrilineal, in either the city or in rural areas (Dixon, 2001). Families tended to be nuclear, as young adult males would leave the home to start their own families (Saller and Shaw, 1984; Garnsey and Saller, 1987). Within the family unit, the oldest adult male, or *paterfamilias*, had absolute say over all other family members, clients, workers and slaves (Garnsey and Saller, 1987). Women were expected to be completely subservient, chaste, and above all suppress their own feelings (Toner, 2009). The Roman male

was judged on many things by his peers, but high among these was his *virtus* (Toner, 2002). Toner (2002) states that a proper translation of the *virtus* concept is not entirely possible but can be described as having courage, piety and manliness (Toner, 2002). These aspects were best displayed in public scenarios and places so that one's prestige could be augmented (Toner, 2002). As a whole, the domestic sphere was culturally valued to belong to women (Dixon, 2001). This contrasts with the public realm, which was considered strongly representative of maleness (Toner, 2002).

The stark separation of male and female spheres of life represents elite accounts of family and gender divisions and does not necessarily correspond with the daily life of many Romans (Garnsey and Saller, 1987). Garnsey and Saller (1987), including other scholars (Rawson, 1986; Dixon, 2001), have noted that reconstructing the Imperial Roman family is quite difficult. This is in part a difficulty of language. Modern Western notions of family have been traditionally superimposed on Roman concepts (Rawson, 1986; Garnsey and Saller, 1987). Furthermore, Roman ideas of family are fluid: references to *familia* may take on different meanings in varied contexts (Rawson, 1986; Garnsey and Saller, 1987). Feminist scholars (Rawson, 1986; Dixon, 2001) have argued forcefully that the available sources on family life are incomplete or biased and that a precise description of women's lives in particular is difficult to describe. Many historical sources are also equivocal under critical analysis (Dixon, 2001), in that they can be used to either support or refute a strong male bias in Roman society, or alternatively, the inferior position of women. This weakness of the source material is largely due to the fact that they were produced by men and for men in most cases (Dixon, 2001). One exception has been the thousands of funerary inscriptions across the Empire as these reflect the thoughts of non-elite Romans (Garnsey and Saller, 1987). With these caveats in mind, there are some things that can be said about gender roles, family and work.

Garnsey and Saller (1987) raise the point that most reconstructions of family life, having been conducted by historians of Roman law, reflect moral attitudes of the early Republic rather than the Imperial period. This means that the sharply defined patriarchy described by most accounts may not have reflected daily life in Imperial Rome, but rather legal absolutes that may or may not have been followed closely (Garnsey and Saller, 1988). A good example that contradicts the complete male dominance of society and family can be found with the *sine manu* form of marriage common during the principate (Rawson, 1986; Garnsey and Saller, 1987).

In the *sine manu* form of marriage, the wife did not enter the relationship under the husband's authority like the more traditional form of *cum manu* marriage, common during the Republic (Garnsey and Saller, 1987). Women involved in *sine manu* marriage stayed under the authority and legal power of their own family and *paterfamilias* (Rawson, 1986; Garnsey and Saller, 1987). In this way, Roman women would only be preoccupied with their own family's property and affairs, not their husbands' (Rawson, 1986; Garnsey and Saller, 1987). *Sine manu* marriage even allowed for women to own and control property on the death of their *paterfamilias* (Garnsey and Saller, 1987). This resulted in a surprising amount of legal independence for Imperial Roman women, who were able to divorce as well (Rawson, 1986; Garnsey and Saller, 1987). Ultimately, "the right of the wife to divorce and take much of the dowry with her, together with her independent right of ownership, gave some wealthy women considerable financial leverage and freedom in marriages" (Garnsey and Saller, 1987: 135). The scenarios that Rawson (1986), Garnsey and Saller (1987) and to a lesser extent, Dixon (2001), describe

leaves room for the possibility that Roman women were very active agents in their lives who could act in their own interest and resist male dominance. This can also be found in reexaminations of gendered work and social status.

As mentioned previously, the domestic sphere was equated with women. This equation has also permeated descriptions of typical female work. For instance, weaving and cloth production was thought to be the predominant form of women's work (Dixon, 2001). Although spinning and weaving were symbols of womanhood, mainly due to social constructs of morality and class, it was a largely symbolic practice for elites, slaves did most of the actual weaving in the upper class household (Kleiner and Matheson, 1996). Aldrete (2004) notes that upper-class women were expected to be strong leaders within the household, guiding the care and education of children and the work of slaves. Much of the daily work and efficient running of the household was done by women (Dixon, 2001), and in this respect they had some form of social control in the household.

While elite women were controlled in their selection of work or career, most non-elite women would have helped with skilled labor in more urban areas in order to find regular work, survive and support a family (Scheidel, 1995; Kleiner and Matheson, 1996; Toner, 2002). Kleiner and Matheson (1996) have also argued that the portrayal of the household defining women's lives has been grossly overstated. In all probability, common women must have been multi-skilled to survive (Dixon, 2001; Toner, 2002), and would have been economically and socially active beyond the immediate household. Among commoners, most women would have had to help with farming in rural areas (Toner, 2002). Much of this day to day work for common women would have been physically demanding (Toner, 2002). Life as a commoner was then difficult regardless of gender.

Perhaps the most challenging existence of all in Imperial Rome was that of a child (Harlow and Laurence, 2002). At birth, all children were presented before the paterfamilias for recognition (Garnsey and Saller, 1987; Dupont, 1993). If the paterfamilias claimed the child, he would either pick it up if it was male or in the case of female infants, simply inform the mother that the child was acceptable (Garnsey and Saller, 1987). Children were normally rejected under suspicion of adultery by the wife or if the child was deformed (Garnsey and Saller, 1987). Rejected children were often simply exposed to the elements or sold into slavery (Garnsey and Saller, 1987). While these actions seem harsh, Harlow and Laurence (2002) caution that the characterization of Roman as indifferent to their children is incorrect, as it is in essence a reflection of modern Western tradition onto the past. Romans perceived childhood as a fully separate stage over the life course, one that demanded much from children in light of our current notions of childhood well-being (Harlow and Laurence, 2002). It is important to recognize that the strain placed on children was not out of indifference, but for the perceived benefit of the child so that they may become proper adults in Roman society. In the Roman world view, newborns entered the world soft and shapeless and it was a parent's duty to literally form and mould the developing body into fully human form (Dupont, 2003). Weakness or softness was considered moral weakness and as a result Romans treated their children with severity. At birth, infants were often immobilized for a short period to a plank of wood so that their limbs could not move (Garnsey and Saller, 1987). This was done to ensure proper growth. Infants and children were also bathed in cold water as to not soften them with the feel of warm water (Garnsey and Saller, 1987). Educators, from within or outside the family, would routinely beat children for mistakes

made during lessons (Aldrete, 2004). Children would also be put to work as young as five years old (Redfern, 2007). As Harlow and Laurence (2002) have noted, Dupont (1993) has argued that Romans loved their children immensely and that these practices were not done out of malice. It is also important to remember that infant mortality was shockingly high, estimated to be around 300 per 1,000 births, compared to 10 per 1,000 today in the West (Toner, 2009). Toner (2009) has suggested, following Scheper-Hughes' (1992) work in the slums of Brazil, that the emotional distance between parents and children in Rome may have served as an emotional defense mechanism to guard against attachment. Regardless, children lived risky and harsh lives. This point will be expanded upon in the section on disease in Imperial Rome.

Food was another social product that was divided along gender and class lines. Children were generally weaned on cereals only, which are insufficient to meet their dietary needs (Garnsey, 1999). A greater diversity of foods was only provided as children aged past infancy into more autonomous stages (Garnsey, 1999; Prowse et al., 2008). In adults, a greater diversity and amount of food was given to men over women (Garnsey, 1999), although this did not always hold true for non-elites (Prowse et al., 2005; Craig et al., 2009). Women of marriageable age had their diets reduced to "low or moderate" levels in order to curb their developing sexual appetites (Garnsey, 1999). The food of pregnant women was also regulated. Numerous medical texts advise exercise and to avoid excess food intake as it would complicate the pregnancy (Garnsey, 1999). Garnsey (1999) points out that Roman people were not ignorant of the fetus or that pregnant women required more food. However, warnings against laziness or overeating and of the negative effects of wine were still common however (Garnsey, 1999: 107). Romans were ignorant however of the complex dietary needs of pregnant women, such as the fact that pregnant women require three times the iron intake than men (Garnsey, 1999). As a result, prejudice and ignorance contributed to the poor health of women in many cases.

### *Reproductive Histories of Roman Women*

The reproductive histories of Roman women differed substantially from modern Western experiences. Roman historians have noted that menarche is thought to have occurred in the mid-teens, around 14 years of age (Laurence, 2000), but potentially as late as 16 or 17 (Harlow and Laurence, 2002). The age of marriage for a Roman woman was generally thought to have occurred in the early teens, but new reconsiderations have shown that for non-elite women, marriage most likely occurred in the late teens to early twenties (Kleiner and Matheson, 1996; Harlow and Laurence, 2002). The general consensus among historical reports is that a Roman woman would begin having children shortly after marriage up to her late 30s or even early 40s, by some accounts (Leftkowitz and Fant, 1982). Roman women would typically have had around 5-8 children, with approximately half of those infants surviving to adulthood (Leftkowitz and Fant, 1982; Garnsey and Saller, 1987; Garnsey, 1998).

Breastfeeding was also a socially regulated activity between mother and child. Colostrum, or the first breast milk a mother produces, was thought to be unhealthy because it had not fully developed into mature milk (Garnsey, 1999). Infants were given to a wet-nurse instead as a general rule, although the mother may have also breastfed the child once the production of colostrum had ceased (Garnsey, 1999). This was probably not the case for lower classes that did



not have slaves to use as wet-nurses. The withholding of colostrum was done with the best intentions but the unfortunate result is that Roman infants were denied an important source of food. The lack of immune support (Garnsey, 1999) from colostrum was probably particularly damaging, particularly with the often unsanitary and poor quality of solid food introduced early in life.

Classical sources of evidence indicate that the age of weaning was generally around three years of age (Garnsey, 1999). This appears to be supported by isotopic evidence of Roman children (Fuller et al., 2006; Dupras and Tocheri, 2007; Prowse et al., 2008). Dupras et al. (2001), as well as Dupras and Tocheri (2007), found that at Kellis 2, an Imperial era Roman cemetery in the Dakhleh Oasis, Egypt, children were fully weaned by 3 years of age. In the Isola Sacra population, which represents the people of Portus Romae (Rome's largest port), weaning began by one year of age and finished between the second and third years (Prowse et al., 2008). Fuller et al. (2006) found similar results in Roman-Britain, although later in time during the 4<sup>th</sup> to 6<sup>th</sup> centuries AD. The authors state that weaning was complete in the population by 3-4 years of age (Fuller et al., 2006). The work by Dupras and Tocheri (2007) revealed that exclusive breastfeeding was practiced for 6 months on average before other foods were introduced. This is important, as it may have had positive health benefits on the skeleton for both the mother and child (Kreiger, et al. 1982; Feldblum et al., 1992; Cumming and Klineberg, 1993; Agarwal and Stuart-Macadam, 2003). These studies comprise the few isotopic analyses done on infant remains for the purpose of assessing breastfeeding practices.

## **Daily Life in the Roman City**

Roman culture was an urban culture at heart (Meiggs, 1960; Stambaugh, 1988; Aldrete, 2004), even if the vast majority of its people lived agrarian lives (Toner, 2009). As territorial boundaries expanded during the Republic, Romans built towns and cities to serve as administrative centers and launching points for the dissemination of Roman culture (Aldrete, 2004). Roman cities were remarkably uniform and modeled after Rome itself (Aldrete, 2004). Life in a Roman city formed a unique social and physical context different from life in the country, one that forms an important part of biocultural condition of Velia. It is important however to not overstate the importance of the city; a great majority of the Roman people lived agricultural lives away from urban centers. This is partially true of Velia as well, as local agriculture formed a substantial part of the day-to-day work and contributed significantly to the average person's diet. It is necessary then that both of these contexts be compared as they differ vastly in how they structured daily life.

### *Roman Cities – Splendor and Squalor*

The purpose of this section is explore what Roman cities were like from a more social perspective, rather than an architectural one. Most of this evidence comes from Rome itself, as it remains the most intensely studied Roman city for obvious reasons. Nevertheless, the archaeological and documentary evidence from other cities, such as Ostia, are also data rich

inform much of what Roman scholar know about city life. The regularity and ‘template’ nature of Roman city and town planning (Purcell, 2007) helps in this regard.

At its height during the 1<sup>st</sup> and 2<sup>nd</sup> centuries AD, Rome was truly an enormous city. Population estimates vary, but the consensus narrows on around a million inhabitants during this period (Stambaugh, 1988; Coulston and Dodge, 2000; Patterson, 2000; Aldrete, 2004). Stambaugh (1998) considers the population density of ancient Rome equivalent to modern day Calcutta or Bombay. After Rome’s collapse, no other city in the world would reach its population mark until the middle 18<sup>th</sup> century in London and Paris (Aldrete, 2004). As a result of this massive influx of people, crowding was serious problem in Rome (Stambaugh, 1988; Patterson, 2000). Numerous ancient reports describe the dangers of walking the streets for fear of being crushed by the mob or by animal-drawn carts (Aldrete, 2004). Overcrowding in Rome also made life incredibly noisy (Stambaugh, 1988). There are many ancient reports of constant aggravation throughout the day caused by incessant sound and noise (Stambaugh, 1988). At night, when the streets emptied, traders would finally be allowed to ride into the city (Aldrete, 2004). So even in one’s own home at night there was little escape from chronic noise pollution. Personal safety was often threatened as well. Much more could be said about life in Rome (Stambaugh, 1988; Toner, 2002; Aldrete, 2004; Toner, 2009), but the focus here is on Velia. What should be retained here is that life in a Roman city was difficult. Poor sanitation, fires, and other factors threatened personal safety and health on a regular basis for the non-elite (Toner, 2002; 2009; Aldrete, 2004).

### *Ostia and Roman Ports*

Although a number of studies have examined ports during the Republic and the Empire (Paget, 1968; Houston, 1988; Rickman, 1998; Blackman, 1992; Rife et al., 2007), detailed information on the unique social aspects of daily life in port cities remains elusive. The majority of historical and archaeological work on ports has been in understanding their construction, economic activity (coins, trade goods, etc), and ship building (Paget, 1968; Houston, 1988; Rickman, 1998; Blackman, 1992). Nevertheless, a number of useful distinctions about port life can be drawn. As a whole, ports provided crucial economic and social lifelines across the Empire (Hall and Merrifield, 1986). Although some mechanized tools, such as cranes, were available, the vast majority of the work needed to keep ports operational was done with manual labor (Rickman, 1988). Much of this work was specialized (Mocchegiani Carpano, 1984), including “*saccarii*, carriers of sacks, *phalangarii*, carriers of great amphorae, *mensores*, who measured, *urinatores*, divers for salvage of goods dropped overboard” (Rickman, 1998: 263). Work life in port cities may have been seasonal, with demands for labor peaking in the summer (Rickman, 1988). Freedmen often found themselves out of work in winter months (Rickman, 1988). Rickman (1998) suggests that this may have created some social volatility in ports. Historical documents also indicate that ports required additional safety measures relating to fire hazards, security and general policing (Rickman, 1988). Ports were also diverse places, people from across the Empire would be working and living in close proximity (Rickman, 1998). There have been some suggestions that this may have led to some racial or ethnic tensions (Cracco Ruggini, 1959; 1978; 1980). The degree to which this would have affected day to day life probably depended on the size, location and importance of the port (Hands, 1968; Rickman, 1980). Work

out on the open sea would not have offered any release either. Most trades aboard ships were physically taxing and dangerous (Rickman, 1998). Mental health was likely poor for most Romans (Toner, 2009), and the added tensions of port life may have added to already elevated levels of stress.

The archaeological and documentary evidence from Ostia is one rather large exception from the limited data on the social life of ports and reconstructions based on this city can be used as a model for other Roman ports. Interestingly, there is almost no documentary evidence about Ostia from across the Empire, nearly all the information at hand comes from archaeological work (Meiggs, 1960). Ostia was not another Pompeii, who's outlook was inward and local (Meiggs, 1960). As Rome's major port city at the height of the Empire, Ostia was politically and socially important in connecting the Western and Eastern halves of the Empire (Meiggs, 1960).

The people of Ostia were quite heterogeneous (Meiggs, 1960). Tombstones indicate that Thracians, Egyptians, Pannonian, Sardinian, and Corsican travelers were buried at Ostia (Meiggs, 1960). Numerous records from Ostia indicate Spanish and Greek traders were present in high numbers as well (Meiggs, 1960). Italian Romans were also common, although this is expected (Meiggs, 1960). Some records indicate that many of these foreigners stayed on arrival and married into the local population (Meiggs, 1960). Ostia was also home to many veterans who retired in the city to establish businesses with their military earnings (Meiggs, 1960).

Housing in Ostia was very similar to Rome (Meiggs, 1960; Hermansen, 1981). Early in the development of the city, the domus was quite common as the population was still relatively low (Meiggs, 1960). Things changed during the 1<sup>st</sup> and 2<sup>nd</sup> centuries when Rome reached its apex. Ostia became highly populated, but never reaching the density or magnitude of Rome (Meiggs, 1960). Insulae became the form of housing that most people occupied (Meiggs, 1960; Hermansen, 1981). Insulae at Ostia may have been somewhat unique. Meiggs (1960) states that room sizes were larger than those in Rome, and that their floor plans generally allowed for more light and air to enter the complex. It is possible then that Ostian living conditions were improved over those in Rome. Meiggs (1960) does caution that what we have left to explore archaeologically are likely the best preserved insulae that wealthy families would have occupied. Cheap, poorly constructed wooden insulae would not be well preserved and this may be a source of bias in archaeological interpretations (Meiggs, 1960).

Ostia is somewhat unique in that the strict social boundaries typical of Roman culture were less stringent (Meiggs, 1960). From archaeological and historical data, it seems that freedmen and the "liberality with which slaves were given freedom" formed the foundation of its own subculture (Meiggs, 1960: 217). Slaves were still owned and widely used, but the level of trade and variety of social interaction at Ostia elevated the importance of free people (Meiggs, 1960). The role of freedmen in organizing trade relationships, particularly through large trade guilds, was crucial to their prosperity (Meiggs, 1960). Political involvement by freedmen themselves or their descendants became important to establishing and maintaining their elevated status as well. Finally, the priesthood dedicated to the imperial cult was a profession many freed slaves occupied, thereby increasing their social influence among Ostians (Meiggs, 1960).

The role and social position of women at Ostia was similar to the rest of the Roman world (Meiggs, 1960). All of the proscribed gender roles for Roman women persisted. However, amidst the gender inequality, women were still important to social life in Ostia. Women did have

some upward social movement, primarily through religious life (Meiggs, 1960). Meiggs (1960) notes that funerary inscriptions commonly make references to women in affectionate terms and signify the value of women in maintaining family life.

There is one paradox at Ostia that defies conventional explanation. This exception is the lack of evidence for widespread prostitution (Meiggs, 1960). No building in Ostia has unequivocally been ascribed as being a brothel (Meiggs, 1960). This is odd, given that Herculaneum and Pompeii had many such buildings (Meiggs, 1960). Additionally, out of all the graffiti at Ostia, references to sexual activity or professions are uncommon (Meiggs, 1960). It is improbable that all the people who lived and passed through Ostia lived chaste and celibate lives (Meiggs, 1960), but this archaeological evidence does suggest that prostitution may not have been as institutionalized or publicly acceptable as in other cities such as Pompeii. This is unusual as harbors and prostitution are generally thought to go hand in hand (Meiggs, 1960).

## **Velia - Historical and Archaeological Contexts**

### *History and Function*

The ancient city of Velia was founded as a Greek colony (originally named Elea) around 540 BC (Morel, 2006). It is situated on the west coast of Italy, approximately 80km south of Salerno, in the Campania region (Hutton, 1971) (see Figure 1). The city was under Roman control by the late third century BC and functioned as an important trading centre and port (Crowe et al., 2010). Occupation of the site continued until its collapse in the middle ages (Craig et al., 2009). The diversity of its historical trajectory has led to Velia being described as a “palimpsest of south Italian history” (Hutton, 1971). The local geography of the site is varied, with river valleys to the north and south, and plains to the east (Craig et al., 2009). Although the larger geographical region (the Cilento) has been described as agriculturally poor, “the plains of Velia could readily be exploited for agriculture and arboriculture (the encroachment of marshland is to be dated after classical period), and the uplands would have provided timber and pasture; this adds up to resources of sufficient value to be considered worthy of protection by means of a ring of fortified posts on the hill tops” (Craig et al., 2009: 574).

Craig et al. (2009: 574) note that literary sources, notably the famous geographer Strabo, refer to Velia as a well governed city, and one that made good use of the maritime resources available to it. This is not to say that subsistence off of the land was unimportant. Local agriculture still formed an important part of daily life, like most urban centers at the time (Greco and Schnapp, 1986). In addition, nearly 80 hectares of arable land within the city boundaries was devoted to agriculture on a permanent basis (Greco, 1999). Subsistence practices at Velia depended heavily on the river valleys surrounding the site (Schmeidt, 1970; Bencivenga Trillmich, 1990). The people of Velia grew cereals, olives, and vines in addition to practicing animal husbandry (Crowe et al., 2010). However, Velia was a port city and it is this aspect of Velian life that is most noted (Craig et al., 2009; Crowe et al., 2010). Health and leisure may have been an important of Velia’s history as well (Hutton, 1971). Up until the early Empire, Velia was the site of a well-known healing spa for the Roman aristocracy (Hutton, 1971).

Historians reckon that Velia likely had the foremost port facilities of any city south of Naples at its peak in the Imperial period (Greco, 1975; Morel, 1999). All major port facilities were present at Velia, including the manufacture, repair and service of boats (Greco, 1975; Morel, 1999). Fishing and fishing related commerce and activities were known to have been part of daily life (Marzano, 2007; Craig et al., 2009; Crowe et al., 2010). Economic activity was lively, and Velia's prosperity was apparently well known around the Empire (Pappalardo, 2006). Unfortunately, much of the archaeological evidence is not available due to coastline displacement, differential preservation and anthropogenic factors over the (Craig et al., 2009).

Numerous excavations have been undertaken at Velia, primarily focused on the Acropolis, although much of the foundations of the original structures of the Acropolis were destroyed in the construction of a medieval castle keep that still stands today (Pappalardo, 2006). Houses were constructed somewhat differently at Velia, in all probability to deal with the steep gradient of the slope (Pappalardo, 2006). For many houses, "perimeter walls at the base were polygonal with perfectly matched curves, supporting simple brick structures finished with plasterwork" (Pappalardo, 2006: 42). Houses were simple, having two or three rooms at most, with most of the families activities taking place near the entrance (Pappalardo, 2006).

Excavation of the Necropolis are much more recent, beginning in 2003 and ending in 2006. Approximately 230 burials have been excavated, and the context of the burials and finds from the Necropolis indicate that the cemetery was used between the 1<sup>st</sup> and 2nd centuries A.D. (Crowe et al., 2010). The spatial distribution of burials was seemingly random and spaced over a large area (nearly half a hectare) (Craig et al., 2009). Twenty seven different tomb styles were found, without any clear association with age, sex or location (Craig et al., 2009). The type and amount of artifacts varied by grave, however non of the artifact distributions were considered to be from high prestige families or households and did not suggest any form of noticeable social asymmetry in this population (Fiammenghi, 2003). Ultimately, it is believed that the majority of people living in Roman Velia were from the non-elite classes (Fiammenghi, 2003).

### *Isotopic Reconstruction of the Velian Diet*

Craig et al. (2009) conducted a paleodietary reconstruction of all adults at Velia (N = 119) using stable carbon and nitrogen isotope analysis. Results of the Craig et al. (2009) study show two main dietary profiles for the site, neither of which correlated strongly with any archaeological features or information available for this population. However, Craig et al. (2009) noted that in general, Roman burial contexts do not correlate with the social status of the individual, so it is not unexpected to find the same at Velia. In the larger main dietary group, grain consumption defined the majority of the diet, with little evidence of regular land or marine protein (Craig et al., 2009). In essence this group represents the typical Roman diet that Garnsey (1999) has described. The second subgroup consumed substantially more meat and fish, and in particular, higher trophic level fish (Craig et al., 2009). The dietary division in the Velia population was irrespective of age, and so those who lived longer did not necessarily have better diets (Craig et al., 2009). There was a separation by sex however, with males, on average, consuming more dietary protein than females (Craig et al., 2009). However, the differences between the sexes are quite small (<1‰), and a number of females also consumed a considerable amount of protein, and so clearly the social moors about gendered foodways were not strictly

followed (Craig et al., 2009). Occupational differences between the sexes, with many more males working in and around the fishing industry, in large part explains the observed sex differences in diet (Craig et al., 2009). This was confirmed by Crowe et al. (2010) who demonstrated a link between occupation and diet. At Velia, approximately 35 per cent of the male skeletons displayed external auditory exostoses (EAE), which are bony growths that develop in the ear canal as a response to chronic exposure to cold wind and water (Crowe et al., 2010). No females showed signs of EAE, so this is a case where gender roles were strictly adhered to (Crowe et al., 2010). In this subgroup of males, there is a high correlation between EAE and high protein, marine based diets (Crowe et al., 2010). Subsequently, the individuals with EAE and high protein diets have been interpreted to be fishermen (Crowe et al., 2010).

### *Velia in the Larger Roman Context*

Daily life at Velia represents in many ways the typical Roman experience (Garnsey, 1998; 1999; Toner, 2002; 2009). Diet was by and large standard (Garnsey, 1999), with only a small (N = 17) subgroup consuming significantly larger amounts of meat or marine protein (Craig et al., 2009). Dietary studies are ongoing and should soon reveal more about the process of weaning at the site (Bondioli, 2011, personal communication). Although there was an aristocracy at Velia (Pappalardo, 2006), the skeletal sample represents the non-elite individuals who made up most of the city (Craig et al., 2009). Day to day life was based on manual labor, most likely in the various trades related to the fishing industry or with agriculture in and around the city. The sample also represents in all probability a mix of free and enslaved individuals. Based on descriptions of Ostia (Meiggs, 1960), the population of Velia may have been racially and ethnically diverse. However, Crowe et al. (2010) noted that while Velia was an important port in the south of Italy, it was much smaller in scale compared to the larger ports like Portus Romae (and Ostia), and so Velia may have been more homogeneous than those busier harbors. There are ongoing oxygen isotope studies that should soon reveal immigration patterns at Velia so that this question can be answered (Bondioli, 2010, personal communication). Velia's smaller size may have reduced some of the risks of city life found in Rome, but would not have eliminated them. Sanitation was in all probability still poor and people still worked and lived close together. Finally, while the image Rickman (1988) conjures about ports as unsafe, rowdy places may hold for the larger harbors, historical descriptions of Velia paint a more idyllic picture of a well-governed town famous for its beauty and spas (Hutton, 1971; Pappalardo, 2006). The mounting bioarchaeological evidence from across the Empire (Garnsey, 1998; Salvadei, et al., 2001; Facchini et al., 2004; Cucina et al., 2006; Paine et al., 2009) all indicate that daily life was difficult, no matter the context. The aristocracy of Velia may then have enjoyed the spas and natural beauty of the site, but the non-elite where in all likelihood eking out a living from day to day.

## Velia - The Necropolis Skeletal Sample

**Table 1** - Summary of age and sex distribution of the total Velia population

<b>Age Group</b>	<b>Female</b>	<b>Male</b>	<b>Total</b>
Sub-adult	N/A	N/A	35
Young Adult	10	7	17
Middle Adult	17	22	39
Old Adult	13	19	32
	<b>40</b>	<b>48</b>	
<b>Total</b>			<b>123</b>

### Chapter Summary

The first two centuries of the Roman principate marked the height of the Empire and a period of relative peace and stability. Roman society was patrilineal and highly stratified along lines of wealth, citizenship, freedom, gender and age, although these boundaries were often fluid. Most Romans worked and lived on the farm. For these Romans, life was harsh and physically demanding. Endemic malnutrition was a daily reality for many among the lower classes. Cities, Rome in particular, served as cultural and political focal points. Life in the city was no less difficult or dangerous. Poverty, crime, poor sanitation and disease posed particularly difficult problems to overcome. Day to day work was physically taxing, particularly if you weren't specialized in a trade. Family life, within the city or on the farm was central to Roman existence. Taken together, the picture of daily life that emerges is one of complexity and heterogeneity. This is particularly true across class lines where it has been shown that elevated social status, as with freedmen and slaves, did not necessarily translate into improved social or physical well-being. The Roman world was geographically diverse and agricultural success likely varied as well (Shotter, 2004). This potentially translated into varied nutritional health (e.g. Farwell and Molleson, 1993; Conheeny, 2000). As a whole, the features of daily life described in this chapter provide a solid foundation to help contextualize the bioarchaeological analyses of the Velia population in Chapter 7.

## Chapter 5 – Methodology

### Sex and Age Determination of the Velia Skeletal Material

#### *Adult Skeletons*

The entire adult skeletal sample at Velia with possible age and sex assignments consists of 115 skeletons. A total of 88 of those 115 adult skeletons (76.5%) were used in this research. The 27 skeletons that were not included were rejected because of poor preservation and/or incomplete skeletons that did not allow age or sex determination, or the preservation of required skeletal elements for analysis. Sample sizes also varied by methodology and required skeletal site, and are summarized in Table (2). Sample sizes for indicators of stress are located in the appropriate section in Chapter 6 (Results).

Age and sex determination are vital for studies of bone loss in the past as bone loss is regarded as primarily an age- and sex-related process. Here, conservative broad age groups were used, as the assignment of precise age estimations of adult skeletons is highly problematic (Jackes, 2000; 2011), particularly in the case of older adults (50+). This improves accuracy in aging while sacrificing some precision. Additionally, the chosen age groups in this study were selected as they reflected important transitional stages in the human life cycle. Specifically, the estimated 18-29 age category captures the development of peak bone mass, the estimated 30-49 age category reflects the important pre-menopausal period in females, and the 50+ age category captures the period of accelerated loss of bone mass with aging driven by the loss of sex steroids in both sexes (Riggs et al., 2008).

#### *Rationale behind selected age categories*

Age determination in adults was assessed with multiple indicators including: degenerative wear of the pubic symphysis (Brooks and Suchey, 1990), auricular surface changes (Lovejoy et al., 1985), morphological changes in the sternal end of ribs (İşcan et al., 1984; 1985), and dental wear (Lovejoy, 1985). Individuals that could be reliably placed within one of the three age groups (indeterminate adult) due to preservation issues were not used. Age was also reassessed in individuals who appeared as outliers in the radiogrammetry, histomorphometry or analyses of trabecular architecture to ensure that the observed values were not due to an error in aging.



## Subadult Skeletons

Sub-adult skeletons were not used in the radiogrammetry or histomorphometry analyses, but only for analyses of trabecular architecture. Radiogrammetry depends on length measurements of complete (epiphyses fused) metacarpals and so subadults cannot be used. The histomorphometry of subadult cortical bone is an expanding research area (see Maggiano et al., 2012), but was not the focus of this research, because the growth process at the tissue level primarily involves modeling, and not remodeling of bone. A small sample was available for analyses of trabecular architecture and this sample is described in Chapter 6. Skeletal growth profiles were assessed from 60 juveniles, aged 3 months to 12 years. The age breakdown is provided in Chapter 6. Sub-adult (juvenile) aging was not conducted by the author, but by Alessa Sperdutti at the Pignorini Museum in Rome. Sub-adult aging was assessed using dental formation and eruption, as well as fusion of long bones (Buikstra and Ubelaker, 1994).

**Table 2** – Sample size by methodology for adult skeletons

<b>Age Group</b>	<b>Females</b>	<b>Male</b>	<b>Total</b>
<i><b>Radiogrammetry</b></i>			
<b>18-29 yrs</b>	7	6	13
<b>30-49 yrs</b>	15	20	35
<b>50+ yrs</b>	10	13	23
<b>Total</b>	<b>32</b>	<b>39</b>	<b>71</b>
<i><b>Histomorphometry</b></i>			
<b>18-29 yrs</b>	7	2	9
<b>30-49 yrs</b>	9	13	22
<b>50+ yrs</b>	10	11	21
<b>Total</b>	<b>26</b>	<b>26</b>	<b>52</b>
<i><b>Analysis of Trabecular Architecture</b></i>			
<b>18-29 yrs</b>	6	4	10
<b>30-49 yrs</b>	7	14	21
<b>50+ yrs</b>	6	10	16
<b>Total</b>	<b>19</b>	<b>28</b>	<b>47</b>

## *Sex Determination of Adult Skeletons*

Sex determination was carried out using standard sex determination methods, with emphasis on pelvic (pubic bone) morphological traits including the ventral arc, sub-pubic angle, sub-pubic concavity and the sciatic notch (Acsádi and Nemeskèri, 1970; Brothwell, 1980; Buikstra and Ubelaker, 1994). Sex-related features of the skull (Buikstra and Ubelaker, 1994) were also examined in order to increase accuracy (Mays, 1998). When the skull and pelvis are combined, accuracy of sex determination can reach 97-98% (Mays, 1998). Concerns over the effects of age on sex (Walker, 1995; 2005) were noted during the sex determination process and accounted for to improve accuracy. Adult individuals with indeterminate sex due to preservation were not utilized. Sex was not assigned to sub-adults, as sexually dimorphic traits do not appear until puberty (White and Folkens, 2005).

Age and sex determination was carried out independently by the author and then verified against database records of age and sex estimations conducted by trained staff at the National Museum of Prehistory and Ethnography “Luigi Pigorini”, where the skeletal remains are currently curated. Any discrepancies in estimates were discussed with the museum staff and a new consensus was reached.

## **Concerns over the Osteological Paradox**

In the nineteen-fifties and sixties there was a shift in biological anthropology to begin to address questions of skeletal health in relation to cultural and environmental factors (see for example Warren, 1951; Livingstone, 1958; Johnston, 1962; Angel, 1969; Johnston, 1969). These early interests can now be seen as the roots of the biocultural approach in bioarchaeology, with a concern with understanding how social, environmental and biological influences interact to shape skeletal morphology. The desire to explore biocultural processes and skeletal variation has been informative for a number of research goals, including our understanding of changing subsistence patterns through time (e.g., Cohen & Armelagos, 1984; Lambert, 1993; Skeckel and Rose, 2002), European contact with the New World (e.g., Verano & Ubelaker, 1992; Larsen & Milner, 1994; Klaus and Tam, 2009), sexual and gendered division of labor (Larsen, 1998; Mays, 1999; Sofaer Derevenski, 2000), diasporas and health (e.g., Blakey, 2001; Armstrong & Fleischman, 2003), and the bioarchaeology of children (Lewis, 2007). Until more recently, there was an assumption that identifying those who were unhealthy in the past would be simple: individuals with pathological lesions were sicker than those without (Wood et al., 1992). This assumption was not held by all (Ortner, 1991), but was fully brought into the minds of all biological anthropologists with the “Osteological Paradox” first articulated by Wood et al. (1992). The problem revolved primarily around selective mortality, hidden heterogeneity in frailty, and demographic non-stationarity (Wood et al., 1992). Individuals we examine are biased because they do not represent the living population; they died for a reason (Wood et al., 1992). Because of this, not all individuals represent a random sample from each age group as well (selective mortality). Further, it is unknown if all individuals were equally susceptible to disease

(morbidity) (Wood et al., 1992). In short, individuals with lesions may in fact represent individuals who were healthier, as they could survive long enough for changes on bone to manifest (Wood et al., 1992). Conversely, those who appear normal could have been more frail and died quickly (selective heterogeneity).

Goodman (1993) and others since then (Wright and Yoder, 2003) have argued that these observations are of concern and need addressing but are not insurmountable. One of the ways the Osteological Paradox can be somewhat mitigated is through the use of multiple indicators in skeletal analyses (Wright and Yoder, 2003). For example, information on diet, risk of pathogen exposure, mortuary contexts and so on can be combined to address questions of hidden heterogeneity and selective mortality. Researchers have offered a number of ways to address these problems, depending on the research question (Goodman, 1993; Storey, 1997; Wright and Chew, 1998; Wright and Yoder, 2003). Historical and cultural contextualization of past populations also plays a fundamental role in addressing problems associated with the Osteological Paradox (Zuckerman and Armelagos, 2011). Following these researchers, multiple indicators of bone remodeling throughout life are used in this project, as well as multiple measures of pathological/physiological stress. Contextual information is also relied on in the interpretive process. This discussion of the Osteological Paradox is addressed again in Chapter 7 in light of the results of the research.

## **Radiogrammetry**

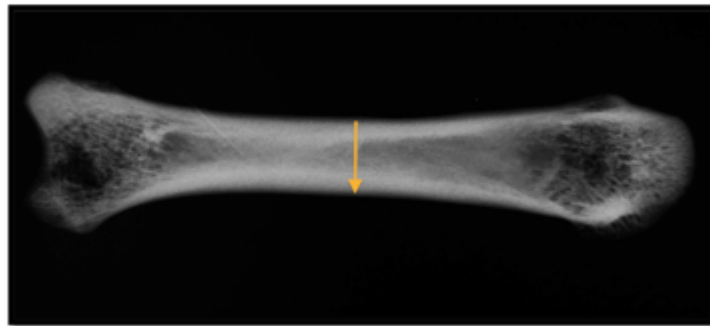
Metacarpal radiogrammetry was developed by clinicians around 50 years ago as a safe and quick way to gauge fracture risk in living patients (Barnett and Nordin, 1960). Shortly after, the method became instrumental in a number of studies that tracked bone growth, and sex and age-related patterns of aging (Virtama and Helelä, 1969; Garn, 1970). Radiogrammetry lost prominence when non-invasive absorptiometric methods gained in popularity in the clinical setting (Mays, 2006). Numerous clinicians have recently shown a renewed interest in the method and are exploring automated uses, particularly as a diagnostic method in developing countries where clinics often do not have access to absorptiometric or advanced imaging facilities (Dey et al., 2000; Montalban et al., 2001; Nielsen, 2001; Rosholm et al., 2001; Reed et al., 2004; Boonen et al., 2005). One clear benefit is that radiogrammetry is a sensitive marker to changes in bone quantity that can be used to track longitudinal changes quite easily (Nielsen, 2001).

Metacarpal radiogrammetry also has long-standing value in bioarchaeology and has been shown to be informative about sex and age-related patterns of bone growth and loss (Mays, 1996, 2000, 2001; 2006; Lazenby, 2002; Ives and Brickley, 2004). Radiogrammetry has been established to be a good proxy for bone status at the spine (Meema and Meindok, 1992; Wishart et al., 1993; Adami et al., 1996; Boonen et al., 2005), forearm (Adami et al., 1996; Dey et al., 2000), hip (Adami et al., 1996; Dey et al., 2000; Boonen et al., 2005), as well as total body mineral content (Mays 2006). Given that the majority of the skeleton is comprised of cortical bone, the study of cortical bone is vital to our understanding of age and sex-related patterns of bone loss in the past and present (Mays, 2006). Additionally, radiogrammetry is very well suited to archaeological purposes as it is non-destructive, rapid, and requires only simple radiographs, which are available near many field locations throughout the world. Haara et al. (2006) have also

noted that radiogrammetry is well suited to inter-group comparison, as the methodology is simple and does not rely on precise machine calibrations or protocols that could confound direct comparisons.

Prior to imaging samples in this study, slits were cut into pieces of large foam and metacarpals were positioned in the antero-posterior position as in for clinical examinations to facilitate comparisons with other data sets (Mays, 1996) (although Ives and Brickley (2004) have noted that positioning has little effect on measurement results). Only individuals with adequate preservation to allow for complete length and thickness measures in the midshaft region of the second metacarpal were used. Radiogrammetric measurements were collected from radiographs taken using a Faxitron™ machine with a kV of 35, mA of three, four minute scan time and Kodak Biomax XAR film (highly sensitive film for biological imaging). Distance from x-ray source to the bone was 40cm. Sliding digital calipers (instrument error: 0.01mm; repeatability: 0.01mm) were used to measure thicknesses from the x-ray films to within 0.1mm. A total of 20 paired bones from left and right sides for both males ( $n = 10$ ) and females ( $n = 10$ ) were tested with Student's *t*-test for the effects of size differences from handedness on all radiogrammetry measures. For both sexes, differences in size (left vs. right) for all measures were not statistically significant ( $p > 0.05$ ) and the use of both right and left sides should not bias results for this population. Consequently, while left sides were predominantly used, right sides were selected when the left was too damaged to maximize sample size as suggested in previous studies (Mays, 1996; Ives and Brickley, 2004). Intraobserver error was also controlled for by paired sample *t*-tests. A random selection of 10 individuals was made 6 weeks after the date the original measurements were taken. No significant difference was found between original and secondary measures ( $p > 0.05$ ).

Measurement protocols were taken according to established standards (Mays, 1996, 2000, 2006; Ives and Brickley, 2004). Total width (TW) was first measured at the midshaft region, which was determined as half of the complete length of the bone. At the same midshaft point, medullary width (MW) was also taken, paying particular attention to potential complications from trabecular bone (Meema and Meema, 1987; Ives and Brickley, 2004). Cortical thickness (CT) was then determined from the formula  $TW - MW$ . Finally, the cortical index (CI) was calculated as  $CT/TW$  to control for body size effects, and multiplied by 100 to reflect the percentage of cortical bone present.



**Figure 8** - Example radiograph of a second metacarpal; Orange line at the approximate location where total width (TW) and medullary width (MW) would be measured.

## Cortical Bone Histomorphometry

Bone loss and turnover can be assessed through a number of histomorphometric (quantitative measures of bone micro-morphology) measures of cortical bone. Mundy (1995) described three characteristic changes in bone morphology and at the microscopic level: 1) thinning and increased resorption of the trabeculae leading to decreased interconnectivity; 2) endosteal bone loss through resorption leading to thinning cortices; 3) reduction in osteoblastic activity leading to an increase in Haversian canal size and intracortical porosity. Meunier (1995: 299) used similar criteria and stressed that only histomorphometric studies can “identify...disturbances in bone remodeling at the basic multicellular unit (BMU) and [basic structural unit] levels because they reflect events occurring at the whole-skeleton”. Although Mundy (1995) stated that increased porosity is due to osteoblastic attenuation, Meunier (1995) argued that osteoporosis is characterized by its heterogeneity. Malluche and Faugere (1986) added that osteoclastic activity is often increased as well. Disturbance in normal mineralization of bone is also common (Malluche and Faugere, 1986; Vajda and Bloebaum, 1999). Clinically speaking, Raubenheimer (2004) suggested that biochemical markers are equivocal and that microscopic techniques should be used whenever possible to provide a differential diagnosis of metabolic bone disease

Although many of the methods available to clinicians are also used by bioarchaeologists, there are complicating factors that limit the use of the full range of clinical tools. The main obstacle that bioarchaeologists face is that the effector cells (BMU) are no longer present in dry bone (Stout and Simmons, 1976; Schultz, 2001). This precludes the possibility of counting osteoclasts directly to gauge their activity level. Additionally, dynamic changes in bone, as Frost (1969) first described by using tetracycline labeling to measure remodeling rates through osteoid formation *in vivo*, cannot be assessed directly. However, Wu et al.'s (1970) landmark study of bone remodeling confirmed that mean annual activation frequency and formation rates (see below) could be accurately estimated from static histomorphometric measures. The assessment of what are essentially dynamic changes in static bone is based on numerous theoretical points (Wu et al., 1970: 206):

- 1) Bone formation occurs in discrete units (BMU). These units are distinct from each other.
- 2) In the human 6<sup>th</sup> rib, a random sample of 100 osteons will yield a nearly constant amount of bone. It then follows that “organ-level osteonal bone formation follows directly and proportionally the number of new BMU created annually”.
- 3) Every osteon remains visible for many years.
- 4) Thus, “*the annual osteonal bone formation rate equals a count of all the osteons previously created, multiplied by the amount of bone in an average osteon, divided by the years of which the osteon creations occurred*” (italics in original).

Although Wu et al.'s (1970) seminal study was a great advancement in assessing dynamic changes in static archaeological bone samples, original equations did not factor in the problem that once osteon population density reaches its asymptote, evidence of previously existing osteons begins to be removed through continued remodeling (Frost, 1987a). Frost (1987a) corrected this problem by developing a scaling operator for Wu et al.'s (1970) equation for total accumulated osteon creations. This scaling factor was able to estimate the number of missing osteons based on the existing osteon population density and its predicted asymptote.

While it is recognized that paleopathologists must make do without pivotal diagnostic features found in the clinical setting, such as cell counts, (Hackett, 1981; Bianco and Ascenzi, 1993), researchers have shown that dynamic measurements are still possible using static bone (Wu et al., 1970; Frost, 1987a). Dynamic measures in static bone do not perfectly match those based on tetracycline labeling, yet they provide accurate estimations that can be of great use to bioarchaeologists who otherwise have no other means available to them (Stout and Paine, 1994; Stout and Lueck, 1995). In fact, the algorithmic method is so accurate that Frost (1987a) and Stout and Paine (1994) have argued that it should be used clinically as well because the activation and formation rates are based on lifelong remodeling, which can be compared to tetracycline labeling in recent remodeling events to detect potential remodeling/metabolic disturbances.

Histomorphometric studies have a long history in bioarchaeology (Richman et al., 1979; Thomson and Gunness-Hey, 1980; Erikson, 1980; Martin and Armelagos, 1985; Burr et al., 1990; Stout and Lueck, 1995; Mulhern, 2000; Cho and Stout, 2003; Cho and Stout, 2011), but are arguably not as common as other methods of investigation due to the destructive nature of the methodology. In the past, a fair amount of inter-study variability could be seen in the histomorphometric variables addressed (Richman et al., 1979; Thomson and Gunness-Hey, 1980; Erikson, 1980; Martin and Armelagos, 1985; Burr et al., 1990) but measures are more recently made with greater standardized (Stout and Lueck, 1995; Mulhern, 2000; Cho and Stout, 2003; Cho and Stout, 2011). The measures used here were chosen to be comparable with other bioarchaeological investigations of bone maintenance and loss Stout and Lueck, 1995; Mulhern, 2000; Cho and Stout, 2003; Cho and Stout, 2011).

### *Thin Sectioning*

Many of the histomorphometric variables described below were based on measurements of specifically the 6<sup>th</sup> rib, but the nature of archaeological material often prevents accurate assessment of rib number (Stout and Lueck, 1995). Rib number could not always be assessed in this research, but midshaft regions were consistently utilized. Stout and Teitelbaum (1976), Stout and Lueck (1995), and Cho and Stout (2003) have demonstrated that the use of the 6<sup>th</sup> rib, while ideal, is not necessary for the application of the histomorphometric measures listed below. Results consistent with expected values based on histomorphometric parameters of the 6<sup>th</sup> rib

were found in all three studies. Consequently, rib samples in this study were selected from rib 5 to rib 8, and this should have no significant effect on remodeling results.

Small 1 cm sections of bone were cut from the midshaft region of ribs using an Isomet® slow-speed saw with a diamond-coated blade. Surface dirt was then carefully removed from the 1 cm pieces of bone when possible (fragile bones were cleaned less thoroughly for fear of damaging them). The small blocks of bone were then placed in a water and ethanol solution for one hour to help clean away the remaining dirt. The 1cm samples were then dehydrated using an ascending ethyl alcohol series (50%, 75% and 100% for 1hr each) to remove excess moisture and to facilitate complete infiltration of the epoxy resin. The thick rib sections were then placed in a vacuum chamber with desiccating salts for 24hrs to dry completely.

Embedding is a vital step in preparing archaeological bone samples as fragile specimens will not be able to withstand the forces caused by grinding and polishing (Stout and Teitelbaum, 1976; Beauchesne and Saunders, 2006). Buehler's Epo-Thin® resin was prepared for embedding following the manufacturer's protocols. The resin is a two-component epoxy system (resin and hardener) that is effective for cold mounting (Li and Risnes, 2004). The Epo-Thin® epoxy was prepared by weight as directed on the bottle. The resin and hardener were mixed in paper cups very slowly to limit the creation of air bubbles for approximately 3 to 5 minutes until all of the hardener was incorporated into the resin (recognized visually by a dissipation of opaque lines created by the hardener and thus a uniform clarity of the resin). The mixed epoxy was then poured very slowly in a thin stream into disposable plastic mounting cups with the thick sections within. The epoxy was poured slowly to reduce the creation of air bubbles, which can hamper visual recognition of bone microstructures and thus impede accurate analysis. Resin was poured into each cup until each sample was completely submerged in the resin. Approximately 5mm of resin was added after the samples were covered to account for the shrinking of the resin when hardened and to make the block slightly larger, which facilitates mounting and cutting in later stages of preparation. The samples were then placed in a Buehler vacuum impregnation chamber and set at 25 mmHg pressure for 15 minutes. Atmospheric pressure was then returned to the chamber to dissipate the bubbles that had risen to the surface of the resin. The pressurizing process was repeated again to ensure that all bubbles had been removed. Finally, the samples are left to harden completely at room temperature for 24hrs.

Once the resin blocks had fully set, the blocks were removed from the mounting cups. Embedded blocks were mounted on to an Isomet® slow-speed saw and thinner sections of approximately 2-3mm were cut from the larger block. The larger embedded blocks were placed aside and stored for future use if additional sections are ever required. The 2-3mm sections were then mounted to glass slides using 2 Ton® Clear Epoxy (Devcon), following the manufacturers protocol. The slides were left to harden 24hrs before final preparation.

The final steps involved grinding the mounted 2-3mm sections down to a thickness of 50-100 microns. Slides were vacuum mounted to a Buehler PetroThin® grinding system and slowly ground down until the desired thickness of 50-100mm was reached. Thin-section thickness was verified by micrometer and microscope to ensure all microstructures could be easily identified and that the proper section thickness had been reached. Prior to the final mounting of cover slips, each slide was cleaned by a 5 second immersion in Xylene to improve visual clarity (pers.comm, Stout, 2009). Immediately after, while the slides were still wet from Xylene immersion, a few (2-3) drops of Permout® mounting medium were then placed over the thin section and then cover-slipped. Special attention was paid to avoid creating air bubbles during this phase, so that visibility of microstructures would not be impeded.

### *Light Microscopy and Digital Imaging Analysis*

Microscopic analysis was conducted using a Leica DM 2500 upright microscope and QImaging Micropublisher 5.0 RTV digital camera. Analysis was conducted under both polarized and plane light. A hilfsobject red 1<sup>st</sup> order quartz compensator (Olympus model U-P521) was also utilized. This compensator works similarly to polarizing lenses (allowing Maltese cross pattern in secondary osteons to be seen), except that the background field is red instead of black. Moreover, collagen bundles appear yellow or blue, depending on the orientation of the specimen in relation to the light source. These features of the compensator often allow for easier visualization of osteons (Schultz, 2001). Specimens with microstructures that were obscured by taphonomic changes under all three visualization methods were discarded from analysis (n = 25). All histomorphometric measures were taken under the 10x objective (100x magnification in total with a 10x eyepiece).

The Bioquant® image analysis software was used to measure histomorphometric measures. Calibration for 100x magnification was based on measures of a standard slide micrometer that adjusted the pixel to length ratio for the 100x magnification. Prior to measurement, visualization began in a random location and afterwards every other field of cortical bone was read. Due to the frequent problem of periosteal degradation in archaeological bone, the goal of this ‘checkerboard’ method is to not rely on a standard anatomical area of analysis within cortical bone (Figure 9) (Robling and Stout, 2000). In this way a methodological standard is preserved while not relying on anatomical areas that are frequently compromised due to taphonomic changes. Sections were analyzed under the “live view” function of Bioquant® so that light polarization and focus could be adjusted to compensate for changing visual quality and slide unevenness, if needed. In each area of cortical bone examined, the total area was manually traced and then area was calculated using the Bioquant® software. All visible osteons, haversian canals and osteon fragments were measured manually as well. For whole osteons and their fragments, a line was drawn along a given reversal (cement) line. Haversian canals were



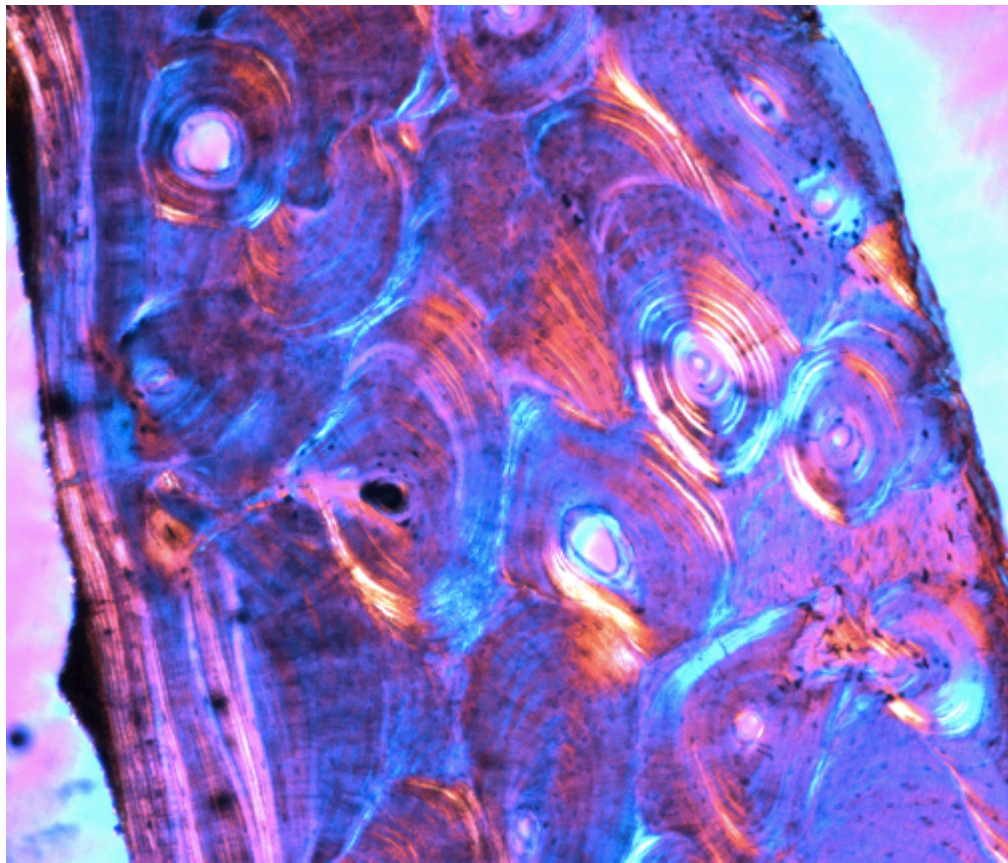
measured by tracing along the outer edge of the canal, where it meets osteonal bone. Bioquant® automatically generates the counts of these structures, in addition to recording the area and perimeter. As measurements were taken, a topographical map (on a separate computer screen) tracked each subsequent measure so that structures would not be double-counted or missed. Measurements were taken in microns, but are presented in Chapter 6 in mm. Figure 10 offers an example of the typical rib microstructure.

### *Histomorphometric Analysis*

Initial histomorphometric analyses produce basic histomorphometric measures, such as the number and area of osteons (see below). Most of the meaningful histomorphometric measures are composites, such as osteon population density (OPD) or mean annual activation frequency ( $\bar{u}_{RC}$ ), and are derived from those original measures. Both basic and composite measures are described in the following sections. Results for composite measures are emphasized in Chapter 6, but Appendix A has a summary of basic measures. Secondary osteons were defined as having 90% of their Haversian canals unremodeled and with intact reversal (cement) lines along their periphery (following Stout and Paine, 1994; Robling and Stout, 2000). Osteon fragments were osteons that had 10% or more of their Haversian canals remodeled (following Stout and Paine, 1994; Robling and Stout, 2000). These percentages are based on visual examination; if there was any doubt that the osteon in question was not complete it was classified as fragmentary. The following is a detailed definition of each basic measure and how it was attained.



**Figure 9** – Alternating field of view pattern used in the histomorphometric analysis of ribs  
(Adapted from Robling and Stout, 2008: 182).



**Figure 10** – Example of a histological section from the Velia population.

## Basic Measures

### **P<sub>i</sub>, P<sub>f</sub>**

P<sub>i</sub> is the total number of intact osteons counted per section, while P<sub>f</sub> is the total number of osteon fragments. The number of osteons here are particularly important, given that at least 25 osteons should be counted for an accurate representation of mean osteon size (Stout and Lueck, 1995). Together, the total number of intact osteons and their fragments informs osteon population density (see below).

### **A<sub>o</sub>, P<sub>o</sub>, A<sub>h</sub>, P<sub>h</sub>**

These four measures represent mean areas and perimeters of osteon and Haversian canals. A<sub>o</sub>, and P<sub>o</sub> are osteon area and perimeter. A<sub>h</sub> and P<sub>h</sub> are Haversian area and perimeter, respectively. Mean osteon area is informative on its own (Pfeiffer, 1998; Stout and Lueck, 1995; Mulhern, 2000; Cho and Stout, 2003; 2011) but also used in conjunction with the other measures to assess mean wall thickness (see below).

## Composite Measures

**Table 3** – List of Composite Histomorphometric Measures

<b>Short-Form</b>	<b>Description</b>
D <sub>h</sub>	(Diameter) Mean Osteonal Cross Sectional
OPD	Osteon Population Density
AOC	Accumulated Osteon Creations
$\bar{U}_{RC}$	Mean Annual Activation Frequency
V <sub>f,r,t</sub>	Mean Annual Bone Formation Rate
$_{net}V_{f,r,t}$	Net Osteonal Remodeling
MWT	Mean Wall Thickness

### **D<sub>h</sub>**

Mean osteonal cross sectional diameter was determined using the formula provided by Stout and Paine (1994: 124):

$$D_h = \sqrt{4(A_o/\pi)}$$

As described above,  $A_o$  is the mean cross sectional area of at least 25 complete secondary osteons for a given specimen. The formula has been modified slightly because of inconsistency in nomenclature between studies. Burr et al., (1990) referred to mean osteonal cross sectional area as  $A_o$ , while Wu et al., (1970) used the symbol  $A_h$ . The calculations for MWT uses the nomenclature  $A_o$ ,  $P_o$ ,  $A_h$  and  $P_h$ , as defined by Burr et al., (1990), while AOC,  $\bar{U}_{RC}$ ,  $V_{f,r,t}$ , and  $net V_{f,r,t}$  use  $A_h$ , as defined by Wu et al., (1970). Thus, changing  $A_h$  to  $A_o$  for this research requires fewer alterations than renaming all of the variables described by Burr et al., (1990) for MWT. To clarify,  $A_o$ , as used in this research is the same  $A_h$  variable used by Wu et al., (1970).

## OPD

OPD is determined by adding  $P_i$  and  $P_f$  for all fields, divided by the total area examined (Wu et al., 1970; Cho and Stout, 2003). As mentioned earlier, every other field is read, and thus the total number of fields read for a given bone will depend on its cortical area, or cross sectional size (Robling and Stout, 2000). Total area is determined by the D7 (total cortical area) array in Bioquant®, which sums all cortical areas measured into a final total area that represents the complete area examined in  $mm^2$ . To obtain OPD, the sum of  $P_i$  and  $P_f$  are divided by the total area examined. This provides a measure in  $\#/ mm^2$ . This is the standard method for assessing OPD (Wu et al., 1970; Frost, 1987a, 1987b; Stout and Paine, 1994; Stout and Lueck, 1995; Robling and Stout, 2000; Cho and Stout, 2003), although this method uses direct measurement of cortical area rather than estimates based on a Merz reticule (Robling and Stout, 2000).

## AOC

AOC, or accumulated osteon creations, is the sum of  $P_i$ ,  $P_f$  and  $P_{missing}$  (described below) for any given OPD (Stout and Paine, 1994). One must account for (estimate) missing osteons ( $P_{missing}$ ) because bone constantly remodels and will eventually produce an asymptote in OPD where secondary osteonal bone occupies the entire cortex (Frost, 1987a, 1987b; Stout and Paine, 1994). Remodeling after the asymptote is reached will remove evidence of previous osteons, and thus a true determination of AOC must take into account these missing osteons. Frost (1987a, 1987b) recognized this fact and developed an algorithm with a scaling operator  $\beta$  to estimate missing osteons. Beta is defined as (Frost, 1987a)

$$\beta = (1-\alpha^x)^{-1} \quad (1)$$

where alpha is OPD “normalized to its predicted asymptote” (Stout and Paine, 1994: 124). Alpha is defined by the equation (Frost, 1987a)

$$\alpha = \text{OPD} (\text{OPD asymptote})^{-1} \quad (2)$$

The  $x$  exponent in equation (1) is 3.5, as empirically determined by Frost (1987a). This is based on models of the relationship between OPD and AOC in clinical trials using tetracycline labeling (Frost, 1987a). The OPD asymptote in equation (2) is defined as

$$\text{OPD asymptote} = k((D_h)^2)^{-1} \quad (3)$$

$(D_h)^2$  is the squared mean osteonal cross sectional diameter and the variable  $k$  is the packing factor “that accounts for the fact that a unit of area of bone can actually contain more intact osteons and their fragments than predicted by a theoretical orthogonal distribution” (Stout and Paine, 1994: 125). In other words, the variable  $k$  accounts for the fact that osteons can be differentially distributed throughout the cortex. It was empirically determined by Frost (1987a) for the human 6<sup>th</sup> rib that  $k$  is 1.7 (see also Stout and Paine, 1994). This result is based on a clinical sample in which all primary lamellar bone had been replaced by osteonal bone for each individual (Stout and Paine, 1994). The maximum OPD in the autopsy sample was 36.25/mm<sup>2</sup>, which was used to determine  $k$  (Stout and Paine, 1994).  $(D_h)^2$  was calculated to be 0.042, which was based on a mean osteonal cross sectional area of 0.037 mm<sup>2</sup> (for the 6<sup>th</sup> rib) in a large clinical sample reported by Wu et al. (1970). Stout and Paine (1994) note that the  $(D_h)^2$  value of 0.042 to determine  $k$  should not be used in equation (3). Rather, a specimen’s own  $D_h$  value, as defined above (Table 9), should be utilized. To summarize, AOC is then defined as

$$\text{AOC} = \beta \cdot \text{OPD} \quad (4)$$

When equations (1) through (3) are put together, the final equation for AOC is represented as follows

$$\text{AOC} = (1 - (\text{OPD} (1.7((D_h)^2)^{-1})^{-1})^{3.5})^{-1} \cdot \text{OPD} \quad (5)$$

### $\bar{U}_{RC}$

The mean activation frequency, or  $\bar{U}_{RC}$ , is the mean number of osteons created annually, per mm<sup>2</sup> of bone (Wu et al., 1970; Frost, 1984a; Stout and Paine, 1994).  $\bar{U}_{RC}$  is calculated as follows

$$\bar{U}_{RC} = \text{AOC} / (\text{chronological age} - 12.5 \text{ years})$$

The ‘effective birth’ of adult compacta (cortical bone) does not equal chronological age because modeling drifts during growth remove bone that was previously present, and thus true ‘adult’

bone is always younger than a person's chronological age (Wu et al., 1970). Wu et al. (1970) determined that the effective birth of adult compacta in the human 6<sup>th</sup> rib occurs around 12.5 years of age. This is the age when most of the adult compacta has formed and will not be removed by further modeling. When calculating  $\bar{U}_{RC}$  it is therefore necessary to subtract 12.5 from the chronological age (determined from estimates based on osteological aging techniques). Individuals in this study are placed into three broad age groups (18-29; 30-49; 50+), and the median of each group is used as chronological the chronological age, as recommended by Stout and Lueck (1995) and Cho and Stout (2003). For example, in the 18-29 age group, the age of 24 is used as chronological age. For individuals in the 50+ group, age 50 is used as chronological age.

### $V_{f,r,t}$

Once  $\bar{U}_{RC}$  has been determined, an individual's bone formation rate, or  $V_{f,r,t}$ , can be calculated.  $V_{f,r,t}$ , measured in  $\text{mm}^2/\text{mm}^2/\text{year}$ , is defined as follows (Stout and Lueck, 1995)

$$V_{f,r,t} = \bar{U}_{RC} \cdot A_o$$

$V_{f,r,t}$  is an estimation of true bone formation rate (Parfitt, 1983). It averages the frequency of osteon creations over mean osteon size, thus approximating the rate of bone formed in a year. Frost (1987a; 1987b) developed the equation based on Wu et al.'s (1970) seminal research. Wu et al. (1970) did not account for missing osteons, and thus Frost (1987a) introduced the algorithm  $\beta$  to account for the missing osteons. Equations (5) through (7) were tested by Stout and Paine (1994) and Stout and Lueck (1995) and the results confirmed Frost's (1987a) position that AOC, bone activation frequency and formation rates could be reliably assessed in archaeological bone.

### $_{net}V_{f,r,t}$

Stout and Paine (1994) suggest using net osteonal remodeling, or  $_{net}V_{f,r,t}$ , if activation frequency cannot be determined. The equation for  $_{net}V_{f,r,t}$  is (Stout and Lueck, 1995; Mulern, 2000).

$$_{net}V_{f,r,t} = AOC \cdot A_o$$

Net osteonal remodeling differs from bone remodeling rate in that activation frequency is not known;  $_{net}V_{f,r,t}$  is a product of total osteon formations and their average size, and thus provides an estimate of the amount of remodeling that has occurred over an individual's lifespan (Stout and Paine, 1994).

## **MWT**

Mean osteon wall thickness is measured in mm using the equation (Burr et al., 1990). Malluche and Faugere (1986) emphasized that the mean wall thickness (MWT) of osteons is an important measure of osteoblast life spans and/or of bone formation rates.

$$\text{MWT} = (A_o - A_h) / (P_o + P_h) \cdot 2$$

### *Macroscopic Analyses (Rib Cross-Sectional Area)*

In addition to histomorphometric measures, the total area (Tt.Ar), endosteal area (En.Ar.) and cortical area (Ct.Ar) of rib cross sections were measured under 8.5x magnification. Bioquant® image analysis software was used for this analysis as well. Prior to measurement, Bioquant® was calibrated to 8.5 magnification using a standardized glass micrometer so that a pixel to length ratio could be established. The thin section slides were placed under a Leica MZ6 dissecting scope with a QImaging Micropublisher 5.0 RTV digital camera attached. The outer area (along periosteal border) was traced and the Bioquant® software then determined the total area (Tt.Ar) for the rib specimen. Endosteal area (En.Ar) was determined the same way, but traced along the margin where rib trabeculae joined the cortical space (endosteal margin). Cortical area (Ct.Ar) was determined afterwards manually by subtracting endosteal area from total area. Finally, relative or percent cortical area was determined by dividing cortical area by total area, and then multiplying by 100 (Ct.Ar/Tt.Ar\*100) (Cho and Stout, 2003).

## **Trabecular Architecture of L4 Vertebrae**

### *Computed Tomography in the Clinical Setting*

Feik et al. (2000: 192) have noted that in studies of bone loss, there has been a “move away from studying global changes, towards examining more localized, i.e., site specific changes that may better predict fracture risk”. In other words, focusing on specific skeletal elements may provide better assessments of bone loss and fragility risk than whole-body scans common in Dual Energy Xray Absorptiometry (DEXA) analysis. Computed Tomography (CT) scanning (and Magnetic Resonance Imaging [MRI]) has been an important part of this shift away from relying on measures of bone quantity alone, specifically Bone Mineral Density (BMD), the prevailing global measure.

CT has been available in the clinical setting biomedicine (e.g. Richardson et al., 1985) for many years. Numerous forms of computed tomography exist. Peripheral quantitative CT (pQCT) and micro-CT ( $\mu$ CT) in particular have become important diagnostic tools more recently as image resolutions have become small enough to distinguish individual trabeculae from surrounding tissue accurately and reliably *in vivo* and *in vitro*. High-resolution pQCT and  $\mu$ CT are also tools for theoretical development of bone biology (e.g. Odgaard and Gundersen, 1993; Genant and Jiang, 2006). For example, Genant and Jiang (2006) have argued that CT technologies have been indispensable in formulating hypotheses about bone quality (e.g. trabecular connectivity, and material properties of bone) and then linking bone quality to overall bone function. Diagnostically, CT has a number of advantages over X-rays assessments of BMD, including DEXA (Damilakis et al., 2007). Boutroy et al., (2005) have shown that high-resolution pQCT to be diagnostically sensitive to changes in BMD *as well as* microarchitecture in patients with and without osteoporotic fracture. The results were also reliable as repeated measurement showed tight groupings of results (Boutroy et al., 2005). While radiography can detect fracture once it has happened (Adami et al., 1992), it is not sensitive enough to accurately gauge premature bone loss beyond large obvious changes.

The distinction between 2-D and 3-D methods of analysis has important biomechanical implications for trabecular bone analysis. Traditional X-rays and histomorphometry provide 2-D images of trabecular bone. CT scanning is both 2-D and 3-D in a way. 3-D data in CT scanning involves the meshing together of many 2-D image “slices” in order to reconstruct the original object (Kazakia and Majumdar, 2006). With CT scans it is thus possible to examine specific slices in 2-D or view 3-D images comprised of many individual slices. The weakness of 2-D images is that they mask the complexity of the trabecular network (Odgaard and Gundersen, 1993; Odgaard, 1997).

The capability of CT to accurately reflect trabecular microstructure has been found in numerous studies (Odgaard, 1997; Gordon et al., 1998; Link et al., 1999; Kazakia and Majumdar, 2006; Damilakis et al., 2007). CT’s ability to detect changes in trabecular microstructure is key as trabecular bone is more metabolically active and is thus more sensitive to incipient bone loss prior to fracture. This is an important point bioarchaeological analyses as well. Specifically, since there were a smaller number of the very elderly in the past, in large part due to high infant mortality rates (Jackes, 2000; Milner et al., 2000), it makes it very hard to detect the frequency of senile or type II osteoporosis in archaeological populations.

The quantitative measurement of trabecular bone directly using histology was traditionally the way to gain insight into trabecular bone turnover. Trabecular histomorphometry was considered more reliable because it was based on measurements taken directly from bone (Rühli et al., 2007). An early but important “verification” study was conducted by Müller et al. (1996) to compare analyses of trabecular morphology with invasive and non-invasive methods Müller et al. (1996) emphasized that BMD was not effective at explaining many of the biological changes in bone with age or pathology, and that the structural properties of trabecular bone had



to be considered. Repeated biopsies for long term observations of patients are not feasible due to their invasive nature, so Müller et al. (1996) wanted to test the ability of pQCT to accurately reflect trabecular bone microstructure so that a noninvasive plan could be developed for patients. Using donated cadavers, Müller et al. (1996) looked at 2D slices of the distal radius using pQCT and compared measurements of bone volume, bone surface fraction and trabecular spacing and thickness to invasive histomorphometric data gathered from the same bone. The results were highly consistent and showed a remarkable overlap in measures obtained from both methodologies (Müller et al., 1996).

A study by MacNeil and Boyd (2007) compared modern 3D high-resolution pQCT for in vivo use with  $\mu$ CT and DEXA in similar test to Müller et al.'s (1996), with  $\mu$ CT replacing the role of trabecular histomorphometry because of its high resolution. MacNeil and Boyd (2007) confirmed that pQCT has the ability to accurately measure BMD and bone qualitative features usually gathered through  $\mu$ CT or actual histomorphometry. This is not to say that pQCT has reached the level of detail that  $\mu$ CT can achieve, but rather that the resolution is “good enough” to be diagnostically meaningful and useful. MacNeil and Boyd (2007) point out that when absolute measures are needed, pQCT may not be the best option. The developments in pQCT analysis have important bioarchaeological applications because gantry (space for specimen) sizes for  $\mu$ CT are still quite small, which sometimes requires a destructive process on larger archaeological bone in order to fit it into the scanner. However, recent developments in both the resolution and gantry size in  $\mu$ CT technology may soon make pQCT seem inadequate. For example, Cooper et al. (2008) recently reported that  $\mu$ CT is reaching resolutions where cortical bone microstructure can be imaged with nearly the same visual quality as standard (invasive) histomorphometry. This is in addition to the well-developed ability to image trabecular bone, as well as the canal structures in cortical bone (Cooper et al., 2008). If these trends continue,  $\mu$ CT will likely overshadow pQCT in bioarchaeological studies in many cases if costs can be kept comparable.

### *Computed Tomography in Bioarchaeology*

Computed tomography (CT) appeared in bioarchaeological research shortly after its clinical development in the early to mid 1970s (e.g. Jungers and Minns, 1979; Wong, 1981). CT has been used in bioarchaeology for paleopathological analysis, investigations of bone biology and early bone loss, biomechanics, and dental anthropology. Biomechanics and paleopathology will be discussed in detail here because they form the dominant areas of research relying on CT. Studies of growth and development using computed tomography are noted as well.

The interpretation of biomechanical data from long bones obtained via cross-sectional geometry is a major research area in bioarchaeology (Ruff, 2000). Biomechanics is the

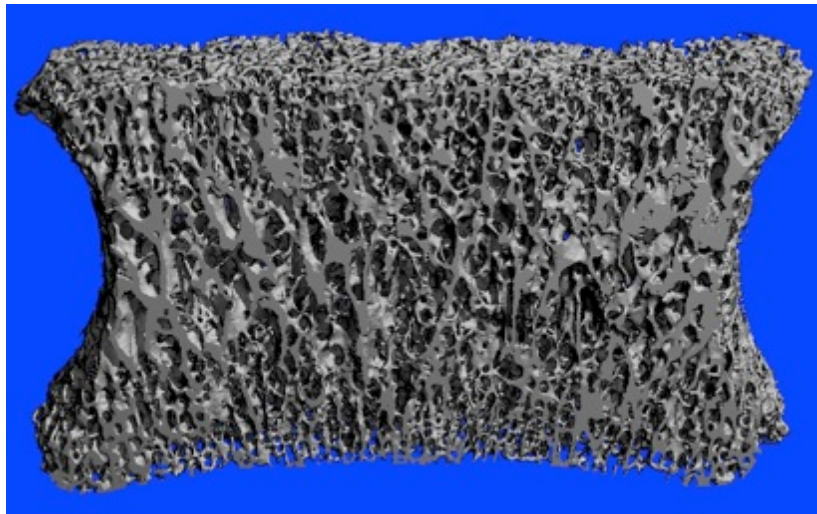
application of engineering principles for the structural analysis of bone (or other biological material) (Wainwright et al., 1982; Robling and Stout, 2003), often to determine their mechanical strengths through geometric space (Ruff, 2000). This area of research is important as it can inform us about growth and development, evolutionary trends, sex differences, and age changes (Ruff, 2000). The effects lifestyle factors, such as activity patterns can also be inferred (Stock and Pfeiffer, 2001) and thus behavioral patterns in the past can potentially be correlated. Cross-sectional geometry is only now almost always conducted noninvasively, but this was not always the case (e.g. Lovejoy and Trinkaus, 1980; Burr et al., 1981; Burr and Piotrowski, 1982; Drusini et al., 2000). Cointry et al. (2004) argue that computed tomography is essential to investigations of bone loss and fragility as bone fragility is fundamentally a biomechanical problem. In other words, the types of questions that must be asked of mechanisms of bone loss are often best answered by the data CT scanning can provide.

The growth and development pathways that ultimately lead to adult morphology are an important part of bioarchaeological inquiry (Saunders, 2000; Bogin, 1999). One aspect of these inquiries is to understand the range and causes of variation in cortical bone growth in long bones, including the degree to which infant and adolescent growth affects adult morphology (Pearson and Lieberman, 2004; Ruff et al., 2006). Noninvasive imaging, including CT, has been an important part of this process (Petit et al, 2005). Paleopathological analyses also rely heavily on CT imaging for diagnostic purposes. Wong (1981) was one of the first to report some uses of CT in paleopathological analysis. In her 1981 article, Wong showed that organs and skeletal structures of natural dissected mummies could be isolated and imaged using CT. CT analysis has now become central to studies of mummified remains (Lynnerup, 2007). Mummies are a powerful source of information as they can inform us about soft-tissue pathology, burial practices, medical practices and cultural practices such as body modification (Lynnerup, 2007).

### *HR-pqCT and the Velia Sample*

The study of vertebral trabecular architecture (cancellous bone) is well established in bioarchaeology (see Brickley and Agarwal, 2003). Vertebrae are one of the primary skeletal regions affected by bone loss due to higher average metabolic activity, and as a result, they are a sensitive marker for remodeling changes that might not be seen in ribs or metacarpals as those regions are composed primarily of thick, less metabolically active cortical bone (Brickley and Agarwal, 2003). Vertebrae were also selected as they can be studied across the life course (Kneissel et al., 1997), providing valuable information on the growth and development of the sample. This study uses only the fourth lumbar vertebra, as it has been the most extensively studied bioarchaeologically (Kneissel et al., 1997; Agarwal et al., 2004), and is more sensitive to structural changes during growth and development (Roschger et al., 2001). This study uses standardized measures of trabecular architecture, including bone volume, degree of anisotropy, trabecular thickness, trabecular separation, trabecular number, connective density and the structural model index (Parfitt et al., 1987). These measures reflect both quantitative and qualitative aspects of trabecular bone.

L4 vertebrae were used in this study and scanned using an HR-pQCT machine (XtremeCT, Scanco Medical AG, Brüttisellen, Switzerland). High-resolution pQCT is able to accurately image microstructural bone features; specifically it is well suited to examine trabecular architecture (MacNeil and Boyd, 2007). Using medical tape, vertebrae were placed on a carbon fiber cast that was later fixed within the gantry of the scanner. Medical foam was used to support the anterior region of the vertebrae to make sure their orientation was kept horizontal and not tilted in either the anterior-posterior or medial-lateral directions. The scanning region of interest was standardized in the scanner's "scout-view" by taking the anterior-posterior distance of vertebrae, dividing by 2, and selecting 4.5mm on either side of the midline of the bone. In this way the central 9mm of trabecular could be examined for each vertebra. For this prescribed location, the scanner captured 1000 projections, which were acquired over 180 degrees with a 200-ms integration time at each angular position. Total scan time was 6 minutes for a total of 220 2-D slices (see Figure 11 for a 3-D reconstruction). The field of view (12.6cm; 3072x3072 matrix) was reconstructed using a modified Feldkamp algorithm, for a nominal voxel thickness of 41 $\mu$ m.



**Figure 11** – 3-D reconstruction of the 220 2-D slices taken from an L4 vertebra

Image analysis was performed on an OpenVMS Alpha-based workstation (HP DS25; Hewlett Packard Corporation, Palo Alto, CA) using Image Processing Language software provided by the scanner manufacturer (IPL v5.08b, Scanco Medical AG). For each vertebra, the cortical shell was manually segmented away from the underlying trabecular bone by tracing the endocortical margin of the cortical shell for every 20 slices of the 220 slices obtained per vertebra. Image analysis using a fixed mineralization threshold to segment bone from marrow was not used, as the range of tissue densities present in the sample varied too greatly, which is not unexpected in archaeological samples. Instead, an automatic histogram based algorithm that determines an "optimal" threshold on a sample-by-sample basis (Ridler, 1978), as well as a light Gaussian filter ( $\sigma=0.5$ , kernel=3) to removed high frequency noise was used. Simple voxel counting was used to determine bone volume fraction (BV/TV). Measures of trabecular number (Tb.N), spacing (Tb.Sp) and thickness (Tb.Th) were assessed directly using a model independent

sphere filling method outlined by Hildebrand and Rügsegger (1997). The degree of anisotropy (DA) was determined by the ratio of major and minor principal components of the MIL ellipsoid (Harrigan and Mann, 1984). The structural model index (SMI) was calculated from a triangular surface representation of 3-D binary data (Hildebrand and Rügsegger, 1997). Finally connective density (Conn.D) was assessed using the Euler number (Odgaard and Gundersen, 1993).

## **Stress Indicators in Adults and Juvenile Skeletons**

Stress markers have long been studied in bioarchaeology (Huss-Ashmore et al., 1982; Porter and Pavitt, 1987; Larsen, 1997; Mays, 1999; Humphrey, 2000; Steckel and Rose, 2002; Cardoso, 2007; Temple, 2008; Klaus and Tam, 2009; Walker et al., 2009) and can be defined as an environmental insult that alters the normal metabolic and physiological function of an individual (Huss-Ashmore et al., 1982). Systemic stress in juveniles (subadults) is emphasized although indicators of stress do occur in adults as well, and both are utilized in this study. Humphrey (2000) has noted that developmental stress makers used on their own are problematic as it is sometimes difficult to distinguish stressed development from normal variation during growth. This issue was discussed at length in Chapter 2, and is mentioned again in Chapter 7. As a remedy, Humphrey (2000) suggests using a combination of markers to strengthen interpretation. Six stress markers have been chosen for this study: *enamel hypoplasia* (Guatelli-Steinberg and Lukacs, 1999; Larsen, 1997; Hillson, 2000; King et al., 2005; Cardoso, 2007; Temple, 2008; Hubbard et al., 2009; Klaus and Tam, 2009), *vertebral neural canal size* (Clark et al., 1986; Porter and Pavitt, 1987; Clark, 1988; Larsen, 1997; Rewekant, 2001), *cribra orbitalia and porotic hyperostosis* (Steckel and Rose, 2002; Ortner, 2003; Walker et al., 2009), and finally *skeletal growth profiles* (Bogin, 1995; 1999; Saunders, 2000, 2008; Humphrey, 2000; 2003; Mays, 1999; Mays et al., 2008; Klaus and Tam, 2009). All of these are non-specific indicators of stress, meaning that they cannot be reliably attributed to a single specific source, but all are intimately related to dietary deficiency and/or pathogen load. While this does limit the scope of the discussion, they are still useful in detecting developmental insult that altered growth and the developmental pathway of the individual (Humphrey, 2000).

## **Subadult Stress**

### *Dental Enamel Hypoplasias*

Dental enamel hypoplasias are a classic skeletal marker in bioarchaeological analyses (Buikstra and Ubelaker, 1994; Guatelli-Steinberg and Lukacs, 1999; Larsen, 1997; Hillson, 2000; King et al., 2005; Cardoso, 2007; Temple, 2008; Hubbard et al., 2009; Klaus and Tam, 2009). Dental enamel hypoplasias can be described as linear grooves or defects that occur during amelogenesis (dental development) (Figure 12) (Goodman and Rose, 1990). Defects can also

occur as pits (Goodman and Rose, 1990). What is particularly useful about dental hypoplasias is that they record growth disturbances in enamel formation that occurred very early in life (Goodman and Rose, 1990; Hillson, 2000). Once a tooth is formed it does not remodel or change, so a record remains as long as the tooth does. The analysis of dental enamel hypoplasias thus offers a way to explore juvenile stress in adults.

Teeth were analyzed under natural light and with a hand-lens and scored according to standards developed by Buikstra and Ubelaker (1994). Only teeth with 50% or more of the crown height were considered (Temple, 2008). Perikymata (which are normal grooves in the enamel) adjacent to dental enamel defects were observed in order to help separate out normal variation from true defects (Skinner et al. 1995; Guatelli-Steinberg, 2003). The anterior dentition and molars are typically the most affected because these teeth have been found to be most sensitive to environmental disturbances and to the formation of dental enamel hypoplasias (Goodman *et al.*, 1980; Goodman & Armelagos, 1985). However, in order to increase sample size, all available teeth were examined following suggestions by Buikstra and Ubelaker (1994). Dental enamel defects were counted as present if the defect could be seen with the naked eye (under a lens) and also be detected with the fingernail. No effort was made to determine the timing of the events (Lovell and Whyte, 1999), as the purpose was to simply gain ascertain the prevalence for the defect in the Velia population. Data were collected for each tooth present, but are presented at the individual level in Chapter 6, in line with the research goals of this methodology. Prevalence of dental enamel hypoplasias was determined by dividing the number of individuals with at least one anterior tooth affected by the number of individuals with at least one anterior tooth present (Temple, 2008).



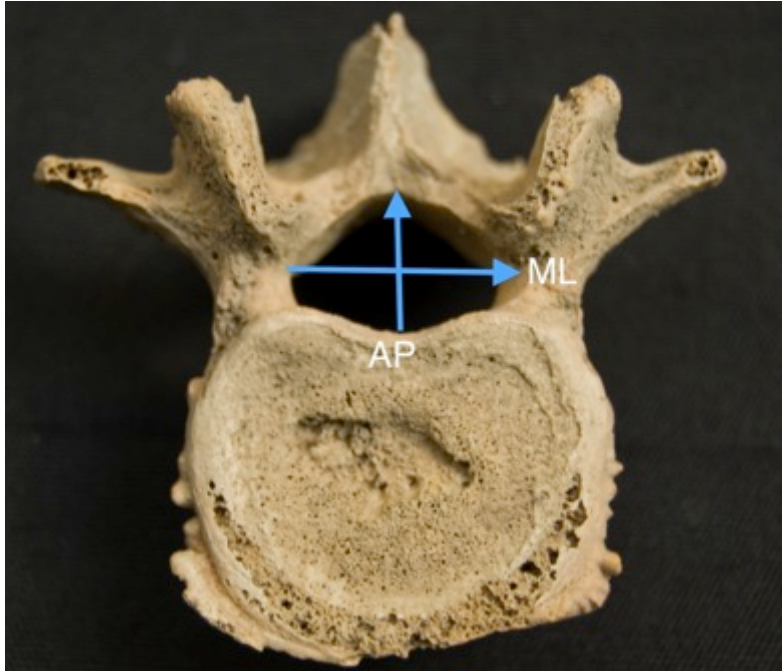
**Figure 12** - An example of linear enamel hypoplasia (a form of dental enamel hypoplasia) from the Urbino population (adapted from Paine et al., 2009: 200). White arrow points to the enamel defect.

### *Vertebral Neural Canal Size*

The size of the vertebral neural canal (VNC) is completed relatively early in life (by age 4-5) and a small canal size has been linked to developmental stress (Porter et al., 1987; Clark et al., 1986; Clark, 1988; Rewekant, 2001). Moreover, once canal size is achieved, it remains very stable with age (Porter et al., 1980). The method is then analogous to dental enamel hypoplasia, as it records stress events early in life and is preserved into adulthood. Clark et al. (1986) reviewed the literature on growth disruption and argued that vertebral canal size should be the most sensitive dental or osseous tissue to developmental stress because of the intimate link to neural and thyrolymphatic growth, which is particularly sensitive to environmental insults early in life. Bioarchaeologically, there are only three published papers using VNC size (Clark et al., 1986; Clark, 1988; Rewekant, 2001). Part of the effort in using VNC in this work was to: a) test the methodology; b) see if VNC sizes were correlated with vertebral wedging, as wedging may weaken vertebral strength and increase the risk of vertebral fractures (Clark et al., 1986); and c) see if the VNC sizes were correlated with bone remodeling measures with each adult age group.

Vertebral measurements were made directly on all available thoracic and lumbar vertebrae for each individual. Only adult individuals were used in the analysis, and only those with vertebral arches intact, so that canal size could be measured (Figure 13). Body heights were only calculated on vertebrae that were well preserved and allowed reliable measurement. Anterior-posterior and medial-lateral distance measures of the vertebral canal size were obtained following guidelines by Clark et al. (1986). Special attention was paid to measure distances from the same landmarks each time. All thoracic and lumbar vertebral canal measures were collapsed into a mean value based on skeletal element (Clark et al., 1986; Clark, 1988). For example, all thoracic anterior-posterior measures of vertebral canal size for an individual were condensed into a mean anterior-posterior size for that person. Clark (1988) has shown alpha coefficients to be high (>0.80) for anterior-posterior and medial-lateral measures of canal size, so the averaging of values is statistically sound. Vertebral body heights were also measured following Clark et al. (1986) by taking height measurements at the anterior and posterior margins, on the annular rings. The degree of wedging was determined by dividing anterior height by posterior height (Clark et al., 1986).





**Figure 13** – Lumbar vertebra showing locations of anterior-posterior (AP) and medial-lateral (ML) measures of canal size. Notice also the presence of a Schmorl's node (depression on the vertebral body) and of osteophytes (small bony spicules) around the edge of the vertebral body.

### *Skeletal Growth Profiles*

Skeletal growth profiles (SGPs) present another means of assessing the relationship between growth and physiological stress (Saunders, 2008). The investigation of SPGs in archaeological populations began with Johnston (1962), who examined the growth of children at Indian Knoll, a Native American site in the United States. This important study ignited interest in the growth of past populations and since then SGPs have been constructed for well over two dozen archaeological populations from across the globe (see Figure 14 for an example) (see Humphrey, 2000 and Saunders, 2008 for excellent summaries). However, growth data must be considered with some caution as the individuals studied are not from a living population, and the possibility exists that they are biased due to selective frailty and hidden heterogeneity (Wood et al., 1992). However, Saunders and Hoppa (1993) have shown that mortality bias is probably small, and that other methodological concerns have a more pronounced bias on the interpretation of SGPs. Some acknowledged inherent biases involved with interpreting SGPs (as well as many indicators of stress), include poor preservation, small samples (particularly after infancy), unknown sex in juveniles, age determination and the cross-sectional nature of bioarchaeological data (Hoppa and Saunders, 1993; Humphrey, 2003; Saunders, 2008).

Sample size ( $n = 60$ ) for the analysis of SGPs in this study were comparable to other published studies (Humphrey, 2003). Most of the individuals used are in the younger ages (3 months to 3.5 years), but this is expected for archaeological populations, as infant mortality was typically high, and survivorship increases afterwards. The Velia population follows this typical mortality curve (see Chapter 4), so there are far fewer individuals past the age group of 3.5 years. Juvenile (subadults) cannot be reliably aged from the skeleton due to the lack of sexual dimorphic features prior to adolescence. As such, differences in growth rates and maturation between juvenile males and females are masked (Bogin, 1999), and must be considered when comparing an archaeological population to a modern group. The cross-sectional nature of bioarchaeological data is also a confounding factor in the analysis of growth as each skeleton represents only a “snap-shot”, or single moment in time for that individual, whereas growth studies using modern populations are typically longitudinal and track individuals through time (Saunders and Hoppa, 1993). As mentioned previously however, the difference between survivors (the living) and non-survivors (skeletal samples) may not be as dramatic (Saunders and Hoppa, 1993) as previously argued (Wood et al., 1992). The ageing of juvenile skeletons does have a real impact on SGPs (Humphrey, 2000; 2003).

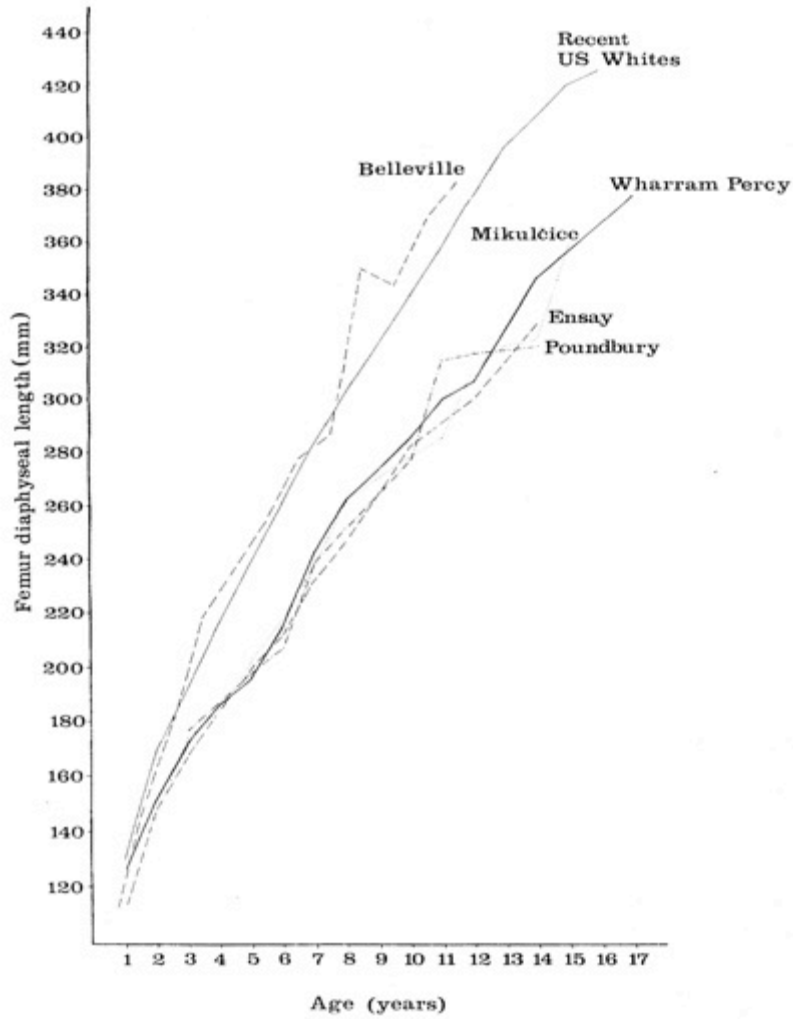
In order to SGPs for the Velia sample, femoral bone lengths for subadults and adults were taken using an osteometric board and following standard protocols (Buikstra and Ubelaker, 1994). Measurements of bone length were taken by Alessandra Sperduti (Pigorini Museum, Rome). Following Humphrey (2000; 2003), data are presented by femoral length and by the percentage of adult size attained. Adult size was determined from the mean of adult long bone lengths (including epiphyses) in each population. Adult length included the epiphyses and no attempt was made to determine diaphyseal length only (Humphrey, 1998). The Velia data was fitted to the modern (Maresh, 1955) curve using a five phased polynomial equation of the form  $a + bx + cx^2 + dx^3 + ex^4 + fx^5$ , where  $x = \text{age}$  and  $a = y \text{ intercept}$  (pers.comm, Humphrey, 2011). Because of differences in growth, two equations were used ensure a proper fit. One equation described the data from birth to 2 years, while the other was used to fit point from 2 to 12 years.

## **Adult Stress**

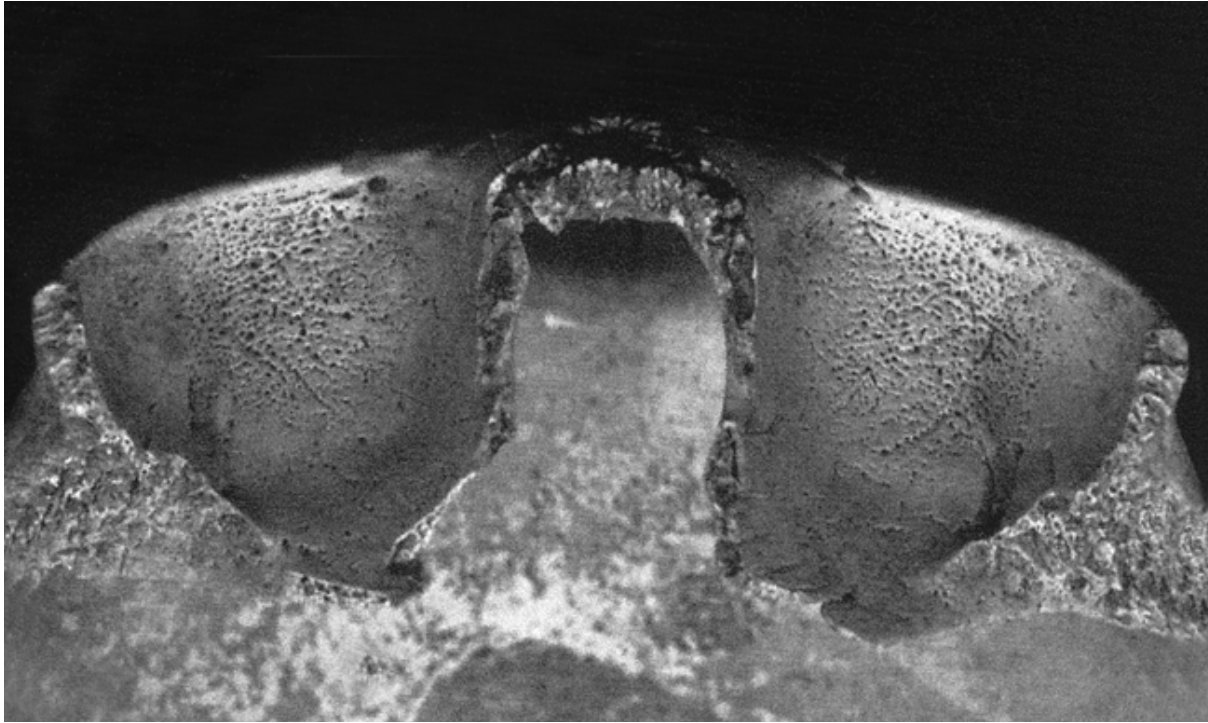
The skeletal markers, cribra orbitalia and porotic hyperostosis, have a complex history in bioarchaeology, predominantly involving the etiology of these symptoms (Walker et al., 2009). The lesions associated with cribra orbitalia appear as porosity and thickening of bone in the orbital roof, most likely as a result of diploe hypertrophy (Figure 15) (Steinbock, 1976). Cribra orbitalia is also usually present bilaterally (Ortner & Putschar, 1985). Originally thought to be caused by iron deficiency anemia, more recent work has show that parasitic load, genetic predispositions, poor nutrition, metabolic imbalance, infectious disease and weaning stress (e.g. diarrhea) can all contribute (Stuart-Macadam, 1991; Larsen, 1997; Aufderheide & Rodriguez-



Martin, 1998; Schultz, 2001; Steckel and Rose, 2002; Ortner, 2003; Blom *et al.*, 2005; Walker *et al.*, 2009). Although it is a non-specific stress marker, cribra orbitalia is considered a robust and useful marker of physiological stress (Walker *et al.*, 2009).



**Figure 14** – Femoral growth in several archaeological populations vs. a modern population (Adapted from Mays, 1999: 295)



**Figure 15** – Example of Cribra orbitalia (pitting/porosity in the orbits) (Adapted from Salavadei et al., 2001: 713)

Porotic hyperostosis presents in a similar manner on the bone as cribra orbitalia, and also has a long history of use in bioarchaeology and a complex etiology, ranging from scurvy, rickets, treponematosi s and more commonly, anemias and dietary deficiencies, particularly limited access to protein (Ortner, 2003; Walker et al., 2009). Although usually assessed separately from cribra orbitalia, many argue that both conditions reflect the same underlying disease process (Mensforth et al., 1978; Stuart-Macadam, 1989; Salvadei et al., 2001; Blom et al., 2005; Keenleyside & Panayotova, 2006). There are others however who dispute this and argue that the two are not always related (Carli-Thiele & Schultz, 1997; Smay & Armelagos, 2000; Rothschild et al., 2004; Wapler et al., 2004; Walker et al., 2009). Nevertheless, porotic hyperostosis remains a very useful indicator of environmental stress, but more work is clearly needed to establish root causes and its relationship with cribra orbitalia.

Assessment of cribra orbitalia in this study was conducted under natural light with a hand-lens. At least one eye orbit had to be present for an individual to be included in the study. Prevalence was determined by dividing the number of individuals with at least one orbit affected with the number of individuals examined. The severity lesions were scored following Stuart-Macadam (1991), and the state of healing was also noted. Lesions were considered unhealed if the borders were sharp and showed no signs of remodeling activity (sclerosing). Porotic

hyperostosis appears quite similar to cribra orbitalia on the bone, with characteristic porosity and hypertrophy of the diploe (Ortner, 2003). Typically the frontal and parietal bones are most affected, but porotic hyperostosis can also affect the occipital bone (Ortner, 2003). Porotic hyperostosis was assessed as absent only in individuals with at least two thirds of the cranial vault present. Individuals with less than two thirds of the cranium present, but with clear evidence for porotic hyperostosis, were counted as well. Prevalence was determined by dividing the total number of individuals affected by total number examined. Assessment of porotic hyperostosis was done under natural light with a hand lens and was scored following Buikstra and Ubelaker (1994).

### *Periostitis*

Periostitis is defined as subperiosteal new bone formation (SPNBF) caused by an inflammatory reaction of the periosteum (outer membrane of bone) (Figure 16) (Ortner, 2003). Ortner (2003) has noted that periostitis is perhaps the most common stress indicator in archaeological populations. This is in large part because periostitis can be caused by local infection, as a response to more widespread infection and from localized trauma to the area (Steinbock, 1976; Ortner, 2003). So for example, the anterior of the tibia (or shin) is very poorly protected and is often affected as a result of trauma or localized infection. Ortner and Putschar (1985) have also noted that periostitis is also related to nutritional stress. Specifically, a higher prevalence of periostitis has been commonly found in cases where both poor nutrition and disease load were common (Ortner and Putschar, 1985). The synergy between nutritional stress and periostitis seems to be related to a lowered immune response (Paine et al., 2009). Much like cribra orbitalia and porotic hyperostosis, the causes of periostitis are ultimately non-specific, but periostitis remains a useful and important bioarchaeological tool in the investigation of general health in the past (Roberts and Machester, 1997).

Periostitis was assessed following Buikstra and Ubelaker (1994). All available upper (humerus, radius, ulna) and lower (femur, tibia, fibula) limbs were examined. At least two thirds of a given long bone diaphysis had to be present to be counted. The sides (left or right) of bone were also recorded to track if one side was more affected than another. The bones were examined under natural light with the aid of a hand lens. Lesion severity was scored using the Ribot and Roberts (1996) model.



**Figure 16** – Periostitis (on the tibia) from the Velia population

### **Paleodietary Reconstructions using Isotopic Analyses**

Craig et al. (2009) have reconstructed diet for the adults of the Velia population. These data are used throughout the discussion (Chapter 7) as they form an important part of biocultural context and in dealing with the osteological paradox (Wright and Yoder, 2003). Readers are referred to the Craig et al. (2009) paper for the methods used to determine diet at Velia. The general findings from the Craig et al. (2009) paper can be found in Chapter 4.

### **Statistical Analyses**

Statistical analyses were conducted using the 0.05 significance level with the JMP 9 statistical software package (SAS Institute Inc.). All data were first examined through descriptive statistics, which include the sample mean, standard error, standard deviation, and normality. Parametric tests, primarily student's T-test and analyses of variance (ANOVA), were whenever possible to compare means and explore significant differences between sex and age. When appropriate (where significance was found), Tukey's Honestly Significant Difference (HSD) post-hoc test was performed after ANOVA to see where the significant differences were located. The Kruskal-Wallis test was utilized when distributions were not normal and a non-parametric equivalent of an ANOVA was needed. Spearman's and Pearson's correlations were used for cross-method analyses. Spearman's was used over Pearson's when normality of the

distribution could not be assumed. Fisher's exact test was used to explore sex differences in the prevalence of stress markers. Fisher's was chosen over Chi-Square as sample sizes were small in these analyses and many of the assumptions held by Chi-Square could not be met.

## Chapter 6 – Results

### Radiogrammetry

The sample distribution (N = 71) for the radiogrammetry measure is outlined in Table 4. All measures were tested for normality and all showed normal distributions. Additionally, the Levene test for equality of variances indicated that variances did not significantly differ between sexes and among age groups, allowing for the use of parametric statistical testing.

Age differences were first explored for each sex separately using one-way ANOVA and Tukey's HSD post-hoc test. Results are summarized in Table 5. Total width (TW) was different between young and old age groups, but was not statistically significant. TW for males increased slightly from young to middle age, but then declined slightly from middle to old age. No significant differences for TW in males were noted. Medullary width (MW) in females expanded with age, with significant differences between young and old females, as well as middle and old age females. In males, MW did not change significantly with age, although statistical significance was nearly reached between the middle and oldest age group ( $p = 0.06$ ), when MW increases substantially. For both sexes, cortical thickness (CT) declined with age, with significant differences occurring between the oldest age group and young and middle aged individuals. Females show significant differences in cortical index (CI) with age. Post-hoc tests indicate significant differences ( $p < 0.05$ ) between the old and both young and middle age groups. No significant difference in CI was observed between the young and middle age group in females ( $p = 0.360$ ). Males also experienced significant differences in CI with age, with post-hoc tests showing this significant difference occurring between old and both young and middle age groups. No significant change was found between young and middle aged males ( $p = 0.904$ ).

**Table 4** - Sample distribution for radiogrammetry measures by age-at-death and sex

Age Group	Females	Male	Total
18-29 yrs	7	6	13
30-49 yrs	15	20	35
50+ yrs	10	13	23
Total	32	39	71

**Table 5** - Age-related cortical bone loss for radiogrammetry measures

	TW		MW		CT		CI	
Age Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD
<i>Females</i>								
18-29 yrs N = 7	7.52	0.38	3.39	0.67	4.13	0.48	55.11	7.52
30-49 yrs N = 15	8.17	0.63	4.13	0.99	4.04	0.77	49.73	10.02
50+ yrs N = 10	8.17	0.69	5.05	0.81	3.12	0.40	38.44	6.02
ANOVA	N.S		18-29 vs. 50+ 30-49 vs. 50+		18-29 vs. 50+ 30-49 vs. 50+		18-29 vs. 50+ 30-49 vs. 50+	
<i>Males</i>								
18-29 yrs N = 6	8.91	0.70	4.20	0.98	4.71	0.72	53.13	9.02
30-49 yrs N = 20	8.98	0.76	4.40	0.87	4.58	0.62	51.24	7.41
50+ yrs N = 13	8.94	0.78	5.26	1.28	3.69	1.03	41.51	12.21
ANOVA	N.S.		N.S		18-29 vs. 50+ 30-49 vs. 50+		18-29 vs. 50+ 30-49 vs. 50+	

TW (Total width); MW (Medullary width); CT (cortical thickness); CI (cortical index); N (Number of individuals). TW, MW, and CW measured in mm. CI is a percentage ( $CW/TW \times 100$ ). Significance measured at the 0.05 level.

Sex differences for each radiogrammetric measure were explored using Student's *t*-tests for each age group and results are summarized in Table 6. TW was significantly larger for males in all age groups. MW was also larger in males for all age groups, but these differences were not statistically significant. CT was also larger in males across age groups, but only reached statistical significance for the 30-49 age group. For the CI measure, females had a higher CI than males in the young age category. In the middle and old age groups, males show a higher CI than females. However, none of these observed differences were statistically significant. Figure 17 graphically illustrates both age and sex-related trends for the CI measure.

**Table 6 - Sex differences in cortical bone for radiogrammetry measures**

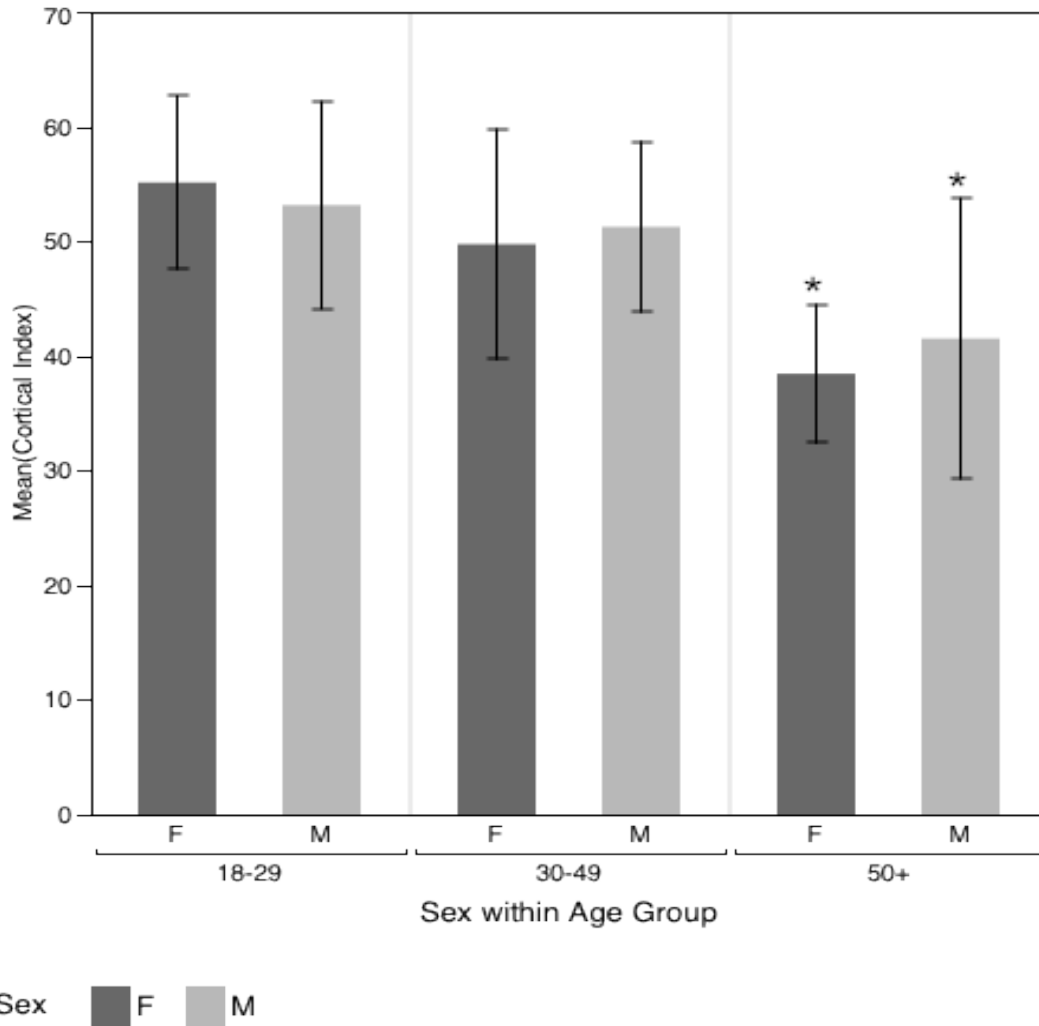
	<b>TW</b>	<b>MW</b>	<b>CT</b>	<b>CI</b>
<b>18-29 yrs</b> <i>Females</i> N = 7 <i>Males</i> N = 6	$p = 0.003^*$	$p = 0.123$	$p = 0.131$	$p = 0.680$
<b>30-49 yrs</b> <i>Females</i> N = 15 <i>Males</i> N = 20	$p = 0.002^*$	$p = 0.407$	$p = 0.035^*$	$p = 0.627$
<b>50+ yrs</b> <i>Females</i> N = 10 <i>Males</i> N = 13	$p = 0.029^*$	$p = 0.637$	$p = 0.186$	$p = 0.439$

TW (Total Width); MW (Medullary width); CT (Cortical Thickness); CI (Cortical index). Significance measured at the 0.05 level. \* Indicates a statistical difference between sexes for the given age group using Student's *t*-test.

Assessing whether or not archaeological bone is “abnormal” or osteoporotic is a difficult task without the clear presence of fragility fractures (Brickley and Ives, 2008). Meema and Meema (1987) have suggested the use of a -2 SD cortical index limit, based on young healthy individuals, to assess abnormal bone with potential elevated fracture risk. This is suited to archaeological skeletons as it bases its dividing line between normal and low bone mass on reference values within the population, as well as being sex specific. This is suited to archaeological skeletons as it bases its dividing line between normal and low bone mass on reference values within the population, as well as being sex specific. To apply this concept to the Velia population, the CI value that falls 2 SD below the young adult mean (for both females and males separately) was used as a “cutoff” point to assess abnormal bone in middle and older aged adults (see Table 7).

As expected, no individuals in the young age group in either sex showed signs of potentially advanced bone loss. In middle age, 13% of females and 10% of males fell 2 SD below their young and sex specific adult means. The most dramatic differences were observed in old age, where 60% of females, and 31% of males could be classified as abnormal under the Meemma and Meema (1987) standard. While the female percentage of individuals with very low mass was twice that of males, in absolute counts, only 2 more females than males had low bone mass.





**Figure 17** – Age and sex differences for the cortical index (CI) measure. Error bars report the standard deviation for each sex/age combination. \* Indicate significant differences with ANOVA

**Table 7** - Age and sex related patterns of low bone mass in the Velia population using the standard set by Meema and Meema (1987)

<b>Age Group</b>	<b>Meema and Meema (1987)</b>
<b>Females</b>	
<b>CI &lt; 2 SD of 18-29 mean</b>	
<b>18-29 yrs</b>	0/7 (0%)
<b>30-49 yrs</b>	2/15 (13%)
<b>50+ yrs</b>	6/10 (60%)
<b>Males</b>	
<b>18-29 yrs</b>	0/6 (0%)
<b>30-49 yrs</b>	2/20 (10%)
<b>50+ yrs</b>	4/13 (31%)

Values indicate number of individuals per age and sex group

### Histomorphometry

The cortical histomorphometry of the ribs from Velia consist of two samples, reflecting both macro- and micro-level analyses (see Chapter 5). The sample distributions for each method are outlined in Table 8.

**Table 8** - Sample distribution for Histomorphometry measures by age-at-death and sex.

<b>Age Group</b>	<b>Females</b>	<b>Male</b>	<b>Total</b>
<i>Histomorphometry (micro)</i>			
18-29 yrs	7	2	9
30-49 yrs	9	13	22
50+ yrs	10	11	21
<b>Total</b>	<b>26</b>	<b>26</b>	<b>52</b>
<i>Histomorphometry (macro)</i>			
18-29 yrs	10	5	15
30-49 yrs	11	22	33
50+ yrs	9	13	22
<b>Total</b>	<b>30</b>	<b>40</b>	<b>70</b>

## *Histomorphometry – Microstructural Changes*

One of the central difficulties in any histomorphometric study is that osteon size can be highly variable, both within skeletal elements and between. For this reason Pfeiffer (1998) recommends that at 50 osteons per individual be used so that any large differences between individuals are detected statistically. Ideally, Pfeiffer (1998) suggests a minimum of 68 osteons should be used to detect a 25% difference in effects related to osteon size and counts between individuals at an alpha of 0.05. However, Stout and Paine (1994) have reported that a sample size of 25 osteons per individual (using the rib) is sufficient to provide a reliable estimate of osteon size and for measures that rely on osteon counts. In the Velia sample, 6 of the 52 individuals used fall below the 50 osteon count. All six individuals represent the lowest percent cortical areas in their age groups, signifying that the main reason fewer osteons were counted is because fewer were available for measure due to a decrease surface area. As a whole, the majority of the sample (71%) has at least 68 osteons counted, 88.5% have at least 50 osteons available, and no osteons fall below the 25 count proposed by Stout and Paine (1994). Ultimately, the osteon counts per individual for the Velia sample are quite robust and so important differences between individuals should be statistically detectable, if present, for all the measures that use osteon size as a factor.

Age differences were first explored using using one-way ANOVA and Tukey's HSD post-hoc test (see Table 9). The measures that are explored in detail in this chapter are On.Ar (mean osteon area), OPD (osteon population density),  $\bar{U}_{rc}$  (mean annual activation frequency),  $V_{f,r,t}$  (mean annual bone formation rate),  $_{net}V_{f,r,t}$  (net osteonal remodeling), and MWT (mean wall thickness). These composite measures (except On.Ar, which is a basic measure) reflect important changes in remodeling over the life course. The basic measures that the composite measures arise from are not discussed in detail in this chapter, but are summarized in Appendix A. All measures of rib cortical microstructure used in this chapter were tested for normality and all showed normal distributions, except for middle and old aged males for the activation frequency measure. Parametric testing was used in all cases except activation frequency in males, where the non-parametric Kruskal-Wallis test was used instead.

Mean osteon size showed an apparent decline across age groups in females, but these differences were not significant. Male mean osteon size was higher in middle age compared to young age, but then decreased slightly from middle to old age. The differences in osteon size in males were not statistically significant. Osteon population density differed significantly in females with age, with the significant difference occurring between the young and old age categories. While male osteon population density also differed with age, it did not do so in a statistically significant way, although statistical significance was nearly reached ( $p = 0.08$ ). Activation frequency showed an apparent decrease with age in females and was significantly different across all age groups. The same pattern was noted for males, where activation frequency differed with age and was significantly different across all ages. In both sexes, bone formation rate showed a negative difference with age and was significantly different between all age groups. Net osteonal remodeling, while also differing slightly with age in both sexes, showed no statistically significant changes. Mean wall thickness declined slightly with age in females and was statistically significant. Mean wall thickness was consistent across age groups in males and was not significantly different with age.

**Table 9** - Age-related cortical bone loss for all measures of cortical histomorphometry

Age Group	On.Ar (mm <sup>2</sup> )		OPD (#/mm <sup>2</sup> )		$\bar{U}_{RC}$ (#/mm <sup>2</sup> /year)		$V_{f,r,t}$ (mm <sup>2</sup> /mm <sup>2</sup> /year)		net $V_{f,r,t}$ (mm <sup>2</sup> /mm <sup>2</sup> )		MWT (mm)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
<b>Females</b>												
18-29 yrs N = 7	0.031	0.005	11.57	2.47	1.69	0.008	0.052	0.008	0.60	0.09	0.07	0.006
30-49 yrs N = 9	0.030	0.005	14.17	2.33	0.83	0.32	0.026	0.014	0.60	0.11	0.07	0.005
50+ yrs N = 10	0.027	0.004	14.93	2.74	0.52	0.005	0.014	0.002	0.54	0.08	0.06	0.005
ANOVA	N.S		18-29 vs. 50+		18-29 vs. 50+ 18-29 vs. 30-49 30-49 vs. 50+		18-29 vs. 50+ 18-29 vs. 30-49 30-49 vs. 50+		N.S		18-29 vs. 50+	
<b>Males</b>												
18-29 yrs N = 2	0.029	0.002	9.10	2.33	1.68	0.007	0.049	0.004	0.56	0.05	0.06	0.002
30-49 yrs N = 13	0.030	0.003	14.39	2.51	0.73	0.01	0.021	0.003	0.58	0.07	0.06	0.005
50+ yrs N = 11	0.028	0.007	14.90	4.01	0.52	0.007	0.015	0.004	0.55	0.14	0.06	0.009
ANOVA	N.S		N.S		N/A		18-29 vs. 50+ 18-29 vs. 30-49 30-49 vs. 50+		N.S		N.S	
Kruskal-Wallis					*							

On.Ar (mean osteon area); OPD (osteon population density);  $\bar{U}_{RC}$  (mean annual activation frequency);  $V_{f,r,t}$  (mean annual bone formation rate); net $V_{f,r,t}$  (net osteonal remodeling); MWT (mean wall thickness). Significance measured at the 0.05 level. \* Indicates a significant difference with age using Kruskal-Wallis tests.

Sex differences for each histomorphometric measure were explored using Student's *t*-tests for each age group and results are summarized in Table 10. For each measure of cortical microstructure, male and female values were extremely close and no sex differences were noted for any of the variables.

**Table 10** - Sex differences in histomorphometric cortical bone measures

	On.Ar (mm <sup>2</sup> )	OPD (#/mm <sup>2</sup> )	$\bar{U}_{RC}$ (#/mm <sup>2</sup> /year)	$V_{f,r,t}$ (mm <sup>2</sup> /mm <sup>2</sup> /year)	$_{net}V_{f,r,t}$ (mm <sup>2</sup> /mm <sup>2</sup> )	MWT (mm)
<b>18-29 yrs</b> Females N = 7 Males N = 2	$p = 0.479$	$p = 0.340$	$p = 0.229$	$p = 0.453$	$p = 0.453$	$p = 0.461$
<b>30-49 yrs</b> Females N = 9 Males N = 13	$p = 0.653$	$p = 0.830$	$p = 0.346$	$p = 0.361$	$p = 0.671$	$p = 0.348$
<b>50+ yrs</b> Females N = 10 Males N = 11	$p = 0.846$	$p = 0.985$	$p = 0.761$	$p = 0.828$	$p = 0.828$	$p = 0.971$

On.Ar (mean osteon area); OPD (osteon population density);  $\bar{U}_{RC}$  (mean annual activation frequency);  $V_{f,r,t}$  (mean annual bone formation rate);  $_{net}V_{f,r,t}$  (net osteonal remodeling); MWT (mean wall thickness). Significance measured at the 0.05 level. \* indicates a statistical difference between sexes for the given age group using Student's *t*-test.

#### *Histomorphometry – Macrostructural Changes*

Age differences were first explored using one-way ANOVA and Tukey's HSD post-hoc test (see Table 11). All measures of rib cortical macrostructure used in this chapter were tested for normality and all showed normal distributions, except for middle-aged males for the Ct.Ar/Tt.Ar (% cortical area) measure. Consequently, parametric testing was used in all cases except for Ct.Ar/Tt.Ar in males, where the non-parametric Kruskal-Wallis test was used instead. Total area (Tt.Ar) in females decreased from young to middle age, and then increased into old age, but none of these changes were statistically significant. Total width decreased steadily in males, but was not statistically significant. Cortical area decreased with age in females and was statistically significant between the young and old age categories. In males, cortical area also declined with age and saw statistically significant changes between the young age group and both middle and old age groups. Endosteal area increased with age in females, but was did not change significantly between age groups. In males, endosteal area increased only slightly with age and was also not significantly different between age groups. Percent cortical area decreased significantly with age in females, with significant differences occurring between young and old age groups. For males, percent cortical bone also declined with age and was significantly different between the young and both middle and old age groups.

Student's *t*-tests were used for each age group to explore sex differences for the macroscopic changes in ribs. Results are summarized in Table 12. For total area, cortical area and endosteal area, mean values for males were consistently larger and a significant sex difference was found for each age group and variable. For percent cortical area, females had greater mean values in young and middle age, while males had a slightly higher mean value in old age. However, no significant sex differences were noted for percent cortical area in any age group.

**Table 11** – Results for adult rib cortical area measures

Age Group	Tt.Ar (mm <sup>2</sup> )		Ct.Ar (mm <sup>2</sup> )		En.Ar (mm <sup>2</sup> )		Ct.Ar/Tt.Ar (%)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
<b>Females</b>								
18-29 yrs N = 10	55.27	16.21	23.39	4.51	31.89	13.15	43.96	7.95
30-49 yrs N = 11	53.92	10.78	19.70	4.98	34.22	10.29	37.48	11.16
50+ yrs N = 9	58.71	8.82	16.88	3.98	41.83	8.51	29.08	6.39
<b>ANOVA</b>	N.S		18-29 vs. 50+		N.S		18-29 vs. 50+	
<b>Males</b>								
18-29 yrs N = 5	86.24	9.72	34.09	7.43	52.15	8.97	39.53	7.68
30-49 yrs N = 22	80.67	17.09	26.16	6.70	54.52	12.16	32.30	4.83
50+ yrs N = 13	77.20	13.75	23.56	5.14	53.64	11.71	30.79	6.16
<b>ANOVA</b>	N.S		18-29 vs. 50+ 18-29 vs. 30-49		N.S		N/A	
<b>Kruskal-Wallis</b>							*	

Tt.Ar (total area); Ct.Ar (cortical area); En.Ar (endosteal area); Ct.Ar/Tr.Ar (percent cortical area). Values for Tr.Ar, Ct.Ar and En.Ar in mm<sup>2</sup>. Ct.Ar/Tt.Ar reported as a percentage. Significance measured at the 0.05 level. \* Indicates a significant difference with age using Kruskal-Wallis tests.

**Table 12** – Sex differences for adult rib cortical area measures

	<b>Tt.Ar (mm<sup>2</sup>)</b>	<b>Ct.Ar (mm<sup>2</sup>)</b>	<b>En.Ar (mm<sup>2</sup>)</b>	<b>Ct.Ar/Tt.Ar (%)</b>
<b>18-29 yrs</b> <i>Females</i> N = 10 <i>Males</i> N = 5	$p = 0.0006^*$	$p = 0.028^*$	$p = 0.0047^*$	$p = 0.328$
<b>30-49 yrs</b> <i>Females</i> N = 11 <i>Males</i> N = 22	$p = 0.0001^*$	$p = 0.0044^*$	$p = 0.0001^*$	$p = 0.167$
<b>50+ yrs</b> <i>Females</i> N = 9 <i>Males</i> N = 13	$p = 0.001^*$	$p = 0.027^*$	$p = 0.013^*$	$p = 0.540$

Tt.Ar (total area); Ct.Ar (cortical area); En.Ar (endosteal area); Ct.Ar/Tr.Ar (percent cortical area). Values for Tr.Ar, Ct.Ar and En.Ar in mm<sup>2</sup>. Ct.Ar/Tt.Ar reported as a percentage. Significance measured at the 0.05 level. \* Indicates a statistical difference between sexes for the given age group using Student's *t*-test.

In order to facilitate comparisons of bone lost between the ribs and the metacarpals, the Meema and Meema (1987) methodology of assessing abnormal bone loss was used for the ribs as well. While the methodology was originally designed for metacarpals, the percent cortical bone measure for the ribs is in essence the same as the cortical index measure in the metacarpals. Both measures assess the amount of cortical bone present and control for body size. Consequently, abnormal bone loss in the ribs was determined by identifying individuals with a percent cortical bone value below -2 SD of young, sex specific healthy individuals. Results are presented alongside those from the radiogrammetry assessment (see Table 13).

Under the Meema and Meema (1987) protocol, two of eleven females in the middle age group and three out of nine females in the oldest age group could be classified as having abnormal bone loss for their respective age group. In contrast no males in the middle age group had signs of abnormal bone loss. However, five out of thirteen males in the old age group were abnormally low, which represents a higher proportion of individuals with abnormal bone loss than females. In comparison, the assessment for metacarpals showed that older females had far worse bone loss than males. Six out of ten females were abnormally low, compared to only four of thirteen for males.

Another approach used to assess bone loss with age was the method suggested by Mays (2006), where individuals in the middle and oldest age groups are normalized as a percentage of young peak adult bone mass. Results for ribs and metacarpals are presented together in Table 14. The results from the Mays (2006) approach reveal that for both sexes, more bone was retained into old age in the metacarpals than in the ribs.

**Table 13** – Age and sex related patterns of low bone mass for ribs using the standard set by Meema and Meema (1987)

<b>Age Group</b>	<b>CI &lt; 2 SD of 18-29 mean</b>	<b>Ct.Ar/Tt.Ar &lt; 2 SD of 18-29 mean</b>
<b><i>Females</i></b>	<b>2<sup>nd</sup> Metacarpal</b>	<b>Rib</b>
<b>18-29 yrs</b>	0/7 (0%)	0/10 (0%)
<b>30-49 yrs</b>	2/15 (13%)	2/11 (18%)
<b>50+ yrs</b>	6/10 (60%)	3/9 (33%)
<b><i>Males</i></b>		
<b>18-29 yrs</b>	0/6 (0%)	0/5 (0%)
<b>30-49 yrs</b>	2/20 (10%)	0/22 (0%)
<b>50+ yrs</b>	4/13 (31%)	5/13 (38%)

Values indicate number of individuals per age and sex group. CI (cortical index); Ct.Ar/Tt.Ar (percent cortical bone).



**Table 14** – Comparison of mean values of CI (2<sup>nd</sup> metacarpal) and Percent Cortical Area (rib) by age groups as measured by percentages of peak bone mass (assessed as age at highest mean for CI and Ct.Ar/Tt.Ar).

Age Group	2 <sup>nd</sup> Metacarpal - CI (Cortical Index)		Rib – Ct.Ar/Tt.Ar (% Cortical Area)	
	Mean	%	Mean	%
<i>Females</i>				
18-29 yrs	55.11 N = 7	100%	43.96 N = 10	100%
30-49 yrs	49.73 N = 15	90.23%	37.48 N = 11	85.26%
50+ yrs	38.43 N = 10	69.73%	29.08 N = 9	66.15%
<i>Males</i>				
18-29 yrs	53.13 N = 6	100%	39.53 N = 5	100%
30-49 yrs	51.24 N = 20	96.44%	32.30 N = 22	81.71%
50+ yrs	41.51 N = 13	78.13%	30.79 N = 13	77.89%

## Trabecular Architecture

The sample distribution (N = 61) for the analysis of trabecular architecture is outlined in Table 15. All measures were tested for normality and all showed normal distributions, except the young male group for BV/TV, the middle age male and female groups for Tb.Th, the old female group for ConnD, and the juvenile 2-6 years group for the SMI measure. In these cases, the non-parametric Kruskal-Wallis test was used to compare means.

**Table 15** - Sample distribution of the Velia skeletal population by age-at-death and sex for the trabecular architecture measures.

Age Group	Females	Male	Total
2-6	Sex Unknown		8
9-16	Sex Unknown		6
<b>Juvenile Total</b>			<b>14</b>
18-29 yrs	6	4	10
30-49 yrs	7	14	21
50+ yrs	6	10	16
<b>Adult Total</b>	<b>19</b>	<b>28</b>	<b>47</b>
<b>Total</b>			<b>61</b>

### *Adult Sample*

Age differences were first explored for each sex separately using one-way ANOVA and Tukey's HSD post-hoc test, as well as the Kruskal-Wallis test when normality assumptions failed. Results are summarized in Table 16. Female bone volume (BV/TV) differed from young to middle age, and then did not change into old age. No age changes for BV/TV in females were statistically significant. Male BV/TV was slightly different between middle and old age groups but no change was statistically significant. For the trabecular number measure (Tb.N), female means declined steadily with age and were significantly different between young and old age. Similarly, male Tb.N declined with age and was also statistically significantly different between the young and old age categories. Trabecular spacing (Tb.Sp) in females increased with age, and was significantly different between the young and old groups. The same pattern was seen in males for Tb.Sp, with spacing increasing with age and a significant difference observed between the youngest and oldest age groups. Trabecular thickness (Tb.Th) in females decreased from young to middle age, but then rose again into old age. Changes to Tb.Th in females with age were not significant however. Male Tb.Th increased steadily with age, but did not reach statistical significance. Connectivity density (Conn.D) decreased with age in females in a statistically significant way, but because normality could not be assumed, the non-parametric

Kruskal-Wallis test was used and post-hoc tests were not available. In males, Conn.D also decline with age, and statistically significant differences were noted between the young age group and the middle and old age categories. The structural model index (SMI) of trabecular bone, a way to three-dimensionally describe trabeculae as either plate- or rod-like, increased with age in females, but this difference was not statistically significant. A similar pattern was observed in males, with SMI increasing with age but not significantly. The final measure, degree of anisotropy (DA), increased with age in both sexes and also did not reach statistical significance.

Sex differences were also explored for the trabecular architecture measures using Student's *t*-tests for each age group. Results for the tests of sex differences are summarized in Table 17. Female BV/TV was higher than that of males in young age, but by middle and old age, male mean values of BV/TV were greater than those of females. The sex differences in BV/TV were not statistically significant however. Tb.N was greater in males for all ages, but was not statistically significantly different. Tb.Sp was higher in females across all age groups, but again, did not reach statistical significance. Female SMI values were also consistently greater than males, as were values from DA across age groups. Both SMI and DA failed statistical significance. Only the Conn.D measure showed a significant sex difference. Mean values of Conn.D were consistently greater in males and were significantly different for young and old age adults.

**Table 16 - Age-related cortical bone loss for all measures of trabecular architecture**

	BV/TV		Tb.N (1/mm)		Tb.Sp (mm)		Tb.Th (mm)		Conn.D (mm <sup>-3</sup> )		SMI		DA	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
<b>Age Group</b>														
<b>Females</b>														
<b>18-29 yrs</b> N = 6	0.37	0.10	1.31	0.18	0.72	0.14	0.33	0.094	3.38	1.17	-0.17	1.18	1.26	0.09
<b>30-49 yrs</b> N = 7	0.28	0.12	1.20	0.17	0.80	0.12	0.26	0.053	3.05	0.66	0.61	1.61	1.35	0.07
<b>50+ yrs</b> N = 6	0.28	0.15	1.03	0.15	0.96	0.14	0.35	0.15	2.07	0.44	0.77	1.51	1.34	0.01
<b>ANOVA</b>	N.S		18-29 vs. 50+		18-29 vs. 50+		N/A		N/A		N.S		N.S	
<b>Kruskal-Wallis</b>							N.S		*					
<b>Males</b>														
<b>18-29 yrs</b> N = 4	0.35	0.03	1.40	0.15	0.66	0.08	0.25	0.003	5.31	1.01	-0.03	0.37	1.22	0.02
<b>30-49 yrs</b> N = 14	0.34	0.10	1.25	0.16	0.76	0.10	0.32	0.095	3.13	0.78	0.18	1.13	1.28	0.08
<b>50+ yrs</b> N = 10	0.32	0.11	1.18	0.15	0.84	0.12	0.33	0.09	2.73	0.78	0.46	1.08	1.31	0.08
<b>ANOVA</b>	N/A		18-29 vs. 50+		18-29 vs. 50+		N/A		18-29 vs. 50+ 18-29 vs. 30-49		N.S		N.S	
<b>Kruskal-Wallis</b>	N.S						N.S							

BV/TV (Bone volume); Tb.N (Trabecular number); Tb.Sp (Trabecular spacing); Tb.Th (Trabecular thickness); Conn.D (Connective density); SMI (Structural model index); DA (Anisotropy - Direction/Orientation of trabeculae). Significance measured at the 0.05 level. \* Indicates a significant difference with age using Kruskal-Wallis tests.

**Table 17** - Sex differences in cortical bone measures

	<b>BV/TV</b>	<b>Tb.N</b> (1/mm)	<b>Tb.Sp</b> (mm)	<b>Tb.Th</b> (mm)	<b>Conn.D</b> (mm <sup>-3</sup> )	<b>SMI</b>	<b>DA</b>
<b>18-29 yrs</b> Females N = 6 Males N = 4	<i>p</i> = 0.632	<i>p</i> = 0.441	<i>p</i> = 0.402	<i>p</i> = 0.071	<i>p</i> = 0.027*	<i>p</i> = 0.800	<i>p</i> = 0.304
<b>30-49 yrs</b> Females N = 7 Males N = 14	<i>p</i> = 0.291	<i>p</i> = 0.572	<i>p</i> = 0.499	<i>p</i> = 0.087	<i>p</i> = 0.808	<i>p</i> = 0.548	<i>p</i> = 0.068
<b>50+ yrs</b> Females N = 6 Males N = 10	<i>p</i> = 0.588	<i>p</i> = 0.081	<i>p</i> = 0.103	<i>p</i> = 0.826	<i>p</i> = 0.049*	<i>p</i> = 0.668	<i>p</i> = 0.451

BV/TV (Bone volume); Tb.N (Trabecular number); Tb.Sp (Trabecular spacing); Tb.Th (Trabecular thickness); Conn.Dens (Connective density); SMI (Structural model index); Anisotropy (Direction/Orientation of trabeculae). Significance measured at the 0.05 level. \* Indicates a statistical difference between sexes for the given age group using Student's t-test.

### *Subadult Sample*

The growth and development of vertebral trabecular architecture in the Velia population was also explored and the descriptive statistics are summarized in Table 18. BV/TV and Tb.N were higher in the 2-6 age group, while Tb.Sp was larger in the older 9-16 age range. Mean values for Tb.Th did not change from the 2-6 to the 9-16 age groups. As with bone volume, Conn.D was higher in the younger 2-6 age group, meaning that bone was more plate-like and that the trabeculae were more randomly oriented. Finally, both SMI and DA were smaller in the 2-6 age group. None of these observations were statistically significant however.

**Table 18** – Subadult summary for all measures of trabecular architecture

	BV/TV		Tb.N (1/mm)		Tb.Sp (mm)		Tb.Th (mm)		Conn.D (mm <sup>-3</sup> )		SMI		DA	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
<b>Age Group</b>														
<b>2-6 yrs n = 8</b>	0.36	0.15	1.52	0.18	0.59	0.09	0.27	0.08	5.27	2.09	-0.17	2.74	1.19	0.04
<b>9-16 yrs n = 6</b>	0.30	0.16	1.37	0.26	0.71	0.17	0.27	0.1	4.63	2.13	0.73	2.03	1.23	0.07
<b>Student's t-test</b>	N.S		N.S		N.S		N.S		N.S		N/A		N.S	
<b>Kruskal- Wallis</b>											N.S			

BV/TV (Bone volume); Tb.N (Trabecular number); Tb.Sp (Trabecular spacing); Tb.Th (Trabecular thickness); Conn.D (Connective density); SMI (Structural model index); DA (Anisotropy – Direction/Orientation of trabeculae). Significance measured at the 0.05 level.

### Cross-Method Analyses

While the independent assessment of radiogrammetry, histomorphometry and trabecular architecture provided many interesting results; these methods were also explored together in individuals that had corresponding available data on at least two of the methods. Table 19 outlines the sample sizes available for the cross-method analyses performed and the results for the cross-method analyses are summarized in Table 20. BV/TV was chosen among the many variables of the analyses of trabecular architecture because it represents the closest proxy to bone mass and was judged most comparable to CI in the metacarpals. Activation frequency ( $\bar{U}_{RC}$ ) in the histomorphometric analysis of ribs was selected because I wanted to test if measures of bone quantity in the metacarpal and lumbar spine were correlated with a more baseline (less biomechanically influenced) remodeling measure at the microstructural level.

**Table 19** – Sample sizes for cross-method analyses

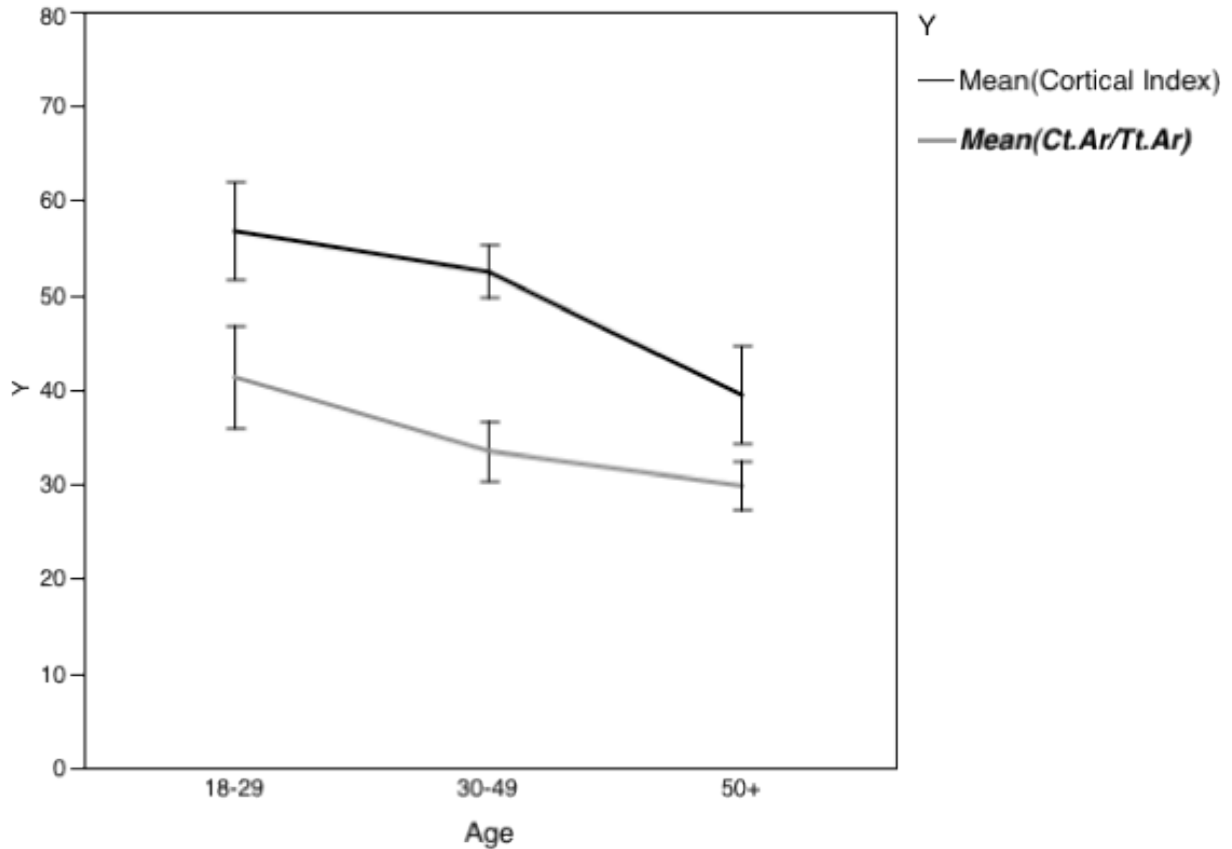
	Female (N)	Male (N)	Total (N)
<b>CI &amp; Ct.Ar/Tt.Ar</b>	21	31	52
<b>CI &amp; BV/TV</b>	18	22	40
<b>CI &amp; <math>\bar{U}_{RC}</math></b>	19	18	37
<b>BV/TV &amp; <math>\bar{U}_{RC}</math></b>	13	10	23

CI (cortical index); Ct.Ar/Tt.Ar (% cortical area); BV/TV (bone volume);  $\bar{U}_{RC}$  (activation frequency)

Cortical index (CI) in metacarpals and the percent cortical bone in ribs (Ct.Ar/Tt.Ar) were compared previously, in the *Radiogrammetry* section of this chapter, but additional results are presented here. Spearman's rank correlation coefficient ( $r_s$ ) was used to test the relationship between CI and Ct.Ar/Tt.Ar. Spearman's  $r_s$  was used instead of Pearson's  $r$  as Ct.Ar/Tt.Ar did not pass normality assumptions. Spearman's  $r_s$  indicates that CI and Ct.Ar/Tt.Ar are positively and significantly correlated ( $r = 0.348$ ,  $p = 0.012$ ). For a natural experiment such as this, this can be classified as a medium effect size (Cohen and Cohen, 1975), signifying an important relationship between the two variables. Although bone loss in the metacarpals is correlated to loss of bone in the ribs, the two trends do show some important differences, as highlighted in Figure 18. Cortical bone is retained longer in the metacarpals, declining most prominently from middle to old age. In contrast, the ribs decline most significantly from young to middle age.

Cortical index and bone volume (BV/TV) are both measures of bone quantity, but reflective of cortical and trabecular bone respectively. CI and BV/TV were compared to see if changes in cortical bone mass correlated with those in trabecular bone. Here, Pearson's correlation coefficient,  $r$ , was used as both CI and BV/TV distributions passed normality. CI and BV/TV are negatively, but poorly correlated and the relationship is not statistically significant ( $r = -0.015$ ,  $p = 0.924$ ). The very poor correlation between cortical and trabecular bone mass in this smaller subset of individuals is not surprising as the independent analyses of CI showed significant age-related bone, while changes in BV/TV did not (see *Radiogrammetry* and *Trabecular Architecture* sections above).

Changes in cortical bone mass (CI) were also contrasted with those of cortical remodeling in the ribs ( $\bar{U}_{RC}$ ). Spearman's  $r_s$  was used as  $\bar{U}_{RC}$  was not normally distributed. Results of Spearman's  $r_s$  indicate that remodeling activity is very strongly and significantly correlated with cortical bone in the metacarpals ( $r = 0.681$ ,  $p = 0.00$ ). Bone formation rate was also highly correlated with CI ( $r = 0.614$ ,  $p = 0.00$ ). BV/TV was also compared to the remodeling measures. Unlike CI, BV/TV was very poorly correlated with  $\bar{U}_{RC}$  and the relationship was not statistically significant ( $r = -0.017$ ,  $p = 0.939$ ). The relationship between BV/TV and bone formation rate ( $V_{f,r,t}$ ) was stronger however, where a mild relationship was shown, although it was not significant ( $r = 0.201$ ,  $p = 0.359$ ).



**Figure 18** – Bone loss with age for CI (cortical index) and Ct.Ar/Tt.Ar (% cortical area). Error bars represent the 95% confidence interval of the mean.

**Table 20** – Results for cross-method analyses

	Spearman's $r$	Pearson's $r$
CI & Ct.Ar/Tt.Ar	$r = 0.348, p = 0.012^*$	N/A
CI & BV/TV	N/A	$r = -0.015, p = 0.924$
CI & $\bar{U}_{RC}$	$r = 0.681, p = 0.000^*$	N/A
BV/TV & $\bar{U}_{RC}$	$r = -0.017, p = 0.939$	N/A

CI (cortical index); Ct.Ar/Tt.Ar (% cortical area); BV/TV (bone volume);  $\bar{U}_{RC}$  (activation frequency)



## Measures of Subadult Stress

### *Vertebral Neural Canal Sizes*

Vertebral neural canal (VNC) sizes in adults ( $N = 67$ ) were used to test three main hypotheses. First, Clark et al. (1986) found a small to moderate ( $r = -0.31$ ;  $p = <0.01$ ) and significant negative correlation between VNC size and vertebral wedging. It was hypothesized here that this same trend would be present for the Velia population. Second, smaller or stunted VNC size has been linked with poor health and increased mortality in adulthood (Clark et al., 1986; Clark, 1988), so it was hypothesized here that smaller VNC sizes would correlate with advanced bone loss in the Velia population. The final hypothesis was to test the relationship between VNC size and environmental stress in adults (determined by cribra orbitalia and porotic hyperostosis).

The summary for VNC and vertebral height measurements are located in Appendix B. Results related to the testing of the hypotheses listed above are presented here. The technical error of measurement (TEM) and coefficient of reliability (Ulijaszek, 1998) were assessed for these measures in ten randomly selected individuals (5 male, 5 female) as they were not reported in the Clark et al. (1986) or Clark (1988) studies and the uncertainty involved in the methodology was unknown. Reliability of the measurements was excellent ( $>0.95$ ), except for thoracic and lumbar anterior-posterior (AP) measures of VNC size. Reliability for thoracic AP was 0.87, and 0.93 for lumbar AP. Ulijaszek (1998) noted that a coefficient of reliability of 0.95 is needed to be confident in the data. This lower reliability in the AP measures suggests that consistent selection of the same anatomical locations is a problem with this methodology. The medial-lateral (transverse in Clark et al., 1986) distance measures passed reliability for both thoracic (0.99) and lumbar (0.98) vertebrae. AP measures of VNC will be explored in the following results for descriptive purposes, but very little weight is placed behind them given the lack of reliability.

Spearman's correlation was used to test the relationship between VNC measures and wedging, as wedging was not normally distributed. Table 21 summarizes the results. With ages combined, the correlation was negative for thoracic vertebrae, as shown by (Clark et al., 1986), but it was very poor and not significant ( $r = -0.080$ ;  $p = 0.522$ ). Given that aging has an effect on wedging independent of VNC, correlations were done by age group as well. Again, the observations of Clark et al. (1986) were not supported. In young adulthood, the correlation was moderate ( $r = 0.417$ ) but positive and non-significant ( $p = 0.138$ ). In both middle and old age groups the correlations are very poor no relationship exists at all. For lumbar vertebrae, a similar trend was noted, with positive, but very poor and non-significant correlations between lumbar VNC and wedging. In all, the findings of Clark et al. (1986) were not detected here. Most correlations were positive, which is counter-intuitive to the idea that smaller VNC sizes would increase wedging. Furthermore, no relationships were statistically significant.

**Table 21** - VNC size vs. Vertebral Wedging

	<b>18-29</b>	<b>30-49</b>	<b>50+</b>	<b>All individuals</b>
<b>T-AP</b>	r = 0.417; p = 0.138	r = 0.049; p = 0.795	r = 0.027; p = 0.904	r = -0.080; p = 0.522
<b>T-ML</b>	r = 0.287; p = 0.319	r = -0.077; p = 0.682	r = 0.062; p = 0.786	r = 0.088; p = 0.480
<b>L-AP</b>	r = 0.014; p = 0.963	r = 0.106; p = 0.511	r = 0.101; p = 0.655	r = 0.021; p = 0.865
<b>L-ML</b>	r = -0.084; p = 0.963	r = 0.193; p = 0.298	r = 0.118; p = 0.599	r = 0.021; p = 0.866

T-AP (Thoracic anterior-posterior); T-ML (Thoracic medial lateral); L-AP (Lumbar anterior-posterior); L-ML (Lumbar medial-lateral); correlations using Spearman's *r*

VNC sizes and stress indicators were examined in two ways. First, Student's *t*-test was used to see if the VNC means were significantly different between the stressed group (at least one indicator of stress) ( $n = 24$ ) and the non-stressed group ( $n = 29$ ). For all VNC measures (AP and ML in both thoracic and lumbar), no significant difference ( $>p = 0.05$ ) was observed. The next test grouped individuals into high and low VNC groups. All individuals above the median were placed in the "high" group, while all those below the median were counted as "low". Thoracic and lumbar ML measures were tested first, because of their reliability. Chi-square was then used to test the stress vs. non-stressed groups. No significant relationships were found between the high/low thoracic ( $X^2 = 0.005$ ;  $p = 0.942$ ) or lumbar ( $X^2 = 2.369$ ;  $p = 0.124$ ) ML measures and the stressed vs. non-stressed groups. Similar non-significant results were found for AP measures. In lumbar vertebrae, but not thoracic, a significant sex difference for medial-lateral VNC size was noted, so the Chi-square was repeated for each sex separately as body size affects the high/low groups for medial-lateral VNC. Again, no differences were found in females ( $X^2 = 0.351$ ,  $p = 0.554$ ) and males ( $X^2 = 0.223$ ;  $p = 0.637$ ) for medial-lateral VNC size and stress group. Individuals that had smaller VNC sizes did not have more evidence for physiological stress than those who had larger VNC sizes. Ultimately the relationship between VNC size and later risk of stress is not simple, as it was not detected here.

VNC size was also tested against the measures of bone maintenance and loss. Cortical index was tested first because of the larger sample size and because it showed the most variability. Table 22 outlines the results of Pearson correlations between cortical index (CI) and VNC sizes. Only young adults are presented as CI index declines with age and is affected by many factors. Overall, the correlations were moderate, but no relationship was significant. In addition, the correlations in young age were all negative, which is counter to what would be expected if small VNC sizes were to have a negative effect on cortical index. These data strongly suggest that no relationship was present between VNC size and cortical index in young adults. The same trends were found for the other two methods of measuring bone maintenance and loss.

**Table 22** – VNC size vs. Cortical Index in young adults

	<b>Correlation</b>
<b>CI vs. T-AP</b>	$r = -0.351; p = 0.263$
<b>CI vs. T-ML</b>	$r = -0.463; p = 0.129$
<b>CI vs. L-AP</b>	$r = -0.467; p = 0.126$
<b>CI vs. L-ML</b>	$r = -0.433; p = 0.159$

CI (cortical index); T-AP (Thoracic anterior-posterior); T-ML (Thoracic medial lateral); L-AP (Lumbar anterior-posterior); L-ML (Lumbar medial-lateral); correlations using Pearson's  $r$

In addition to these tests, wedging was explored by age to see if wedging worsened. ANOVA showed that both thoracic ( $p = 0.804$ ) and lumbar ( $p = 0.374$ ) vertebrae had no significant increases in wedging with age. Body heights were also explored across the age groups. Interestingly, in thoracic vertebrae, both anterior ( $p = 0.035$ ) and posterior ( $p = 0.040$ ) body heights were significantly greater in the 30-49 age group, compared to the 18-29 group. No difference was noted between the 30-49 and 50+ age groups. No significant differences were noted for lumbar vertebrae. Clark et al. (1986) noted that vertebral growth continues until the mid-twenties in modern populations. It is possible here that the significant difference between the young and middle adult groups represents prolonged growth of vertebrae due to earlier stress in life. However, these sample sizes are not very large and this is a cross-sectional observation, so the conclusion that prolonged growth was present is tentative and requires more testing.

### *Dental Enamel Hypoplasias*

The total sample size for the investigation of dental enamel hypoplasias (DEH) was 75 (see Table 23). Ninety individuals were originally examined, but 15 of those had no teeth (or teeth were overly worn) to examine and these individuals were excluded in the DEH analysis. Of the remaining 75 individuals, 37 were female and 38 were male, and so both sexes were equally represented. The prevalence of DEH, defined as having at least one tooth with DEH (see chapter 5), was extremely high. For all individuals combined, the prevalence of DEH was 90.7%. In females, DEH were present in 95.1% of individuals. The prevalence of DEH was lower in males, at 86.8%, but was still quite high. Fisher's Exact test was used to test if these differences were significant and the null that no differences were present could not be rejected ( $p = 0.430$ ). In both sexes, the prevalence of DEH declined slightly with age, although this decline is probably a result of the declining number of available teeth examined with age from ante mortem tooth loss and wear.

**Table 23** – Prevalence of dental enamel hypoplasias in the Velia sample

<b>Age Group</b>	<b>n/N</b>	<b>Prevalence (%)</b>
<b><i>Females</i></b>		
18-29	10/10	100%
30-49	14/15	93.3%
50+	11/12	91.7%
<b>Female Total</b>	<b>35/37</b>	<b>95.10%</b>
<b><i>Males</i></b>		
18-29	7/7	100%
30-49	15/17	88.2%
50+	11/14	78.6%
<b>Male Total</b>	<b>33/38</b>	<b>86.80%</b>
<b>Sample Total</b>	<b>68/75</b>	<b>90.7%</b>

n (individuals with dental lesions); N (all individuals)

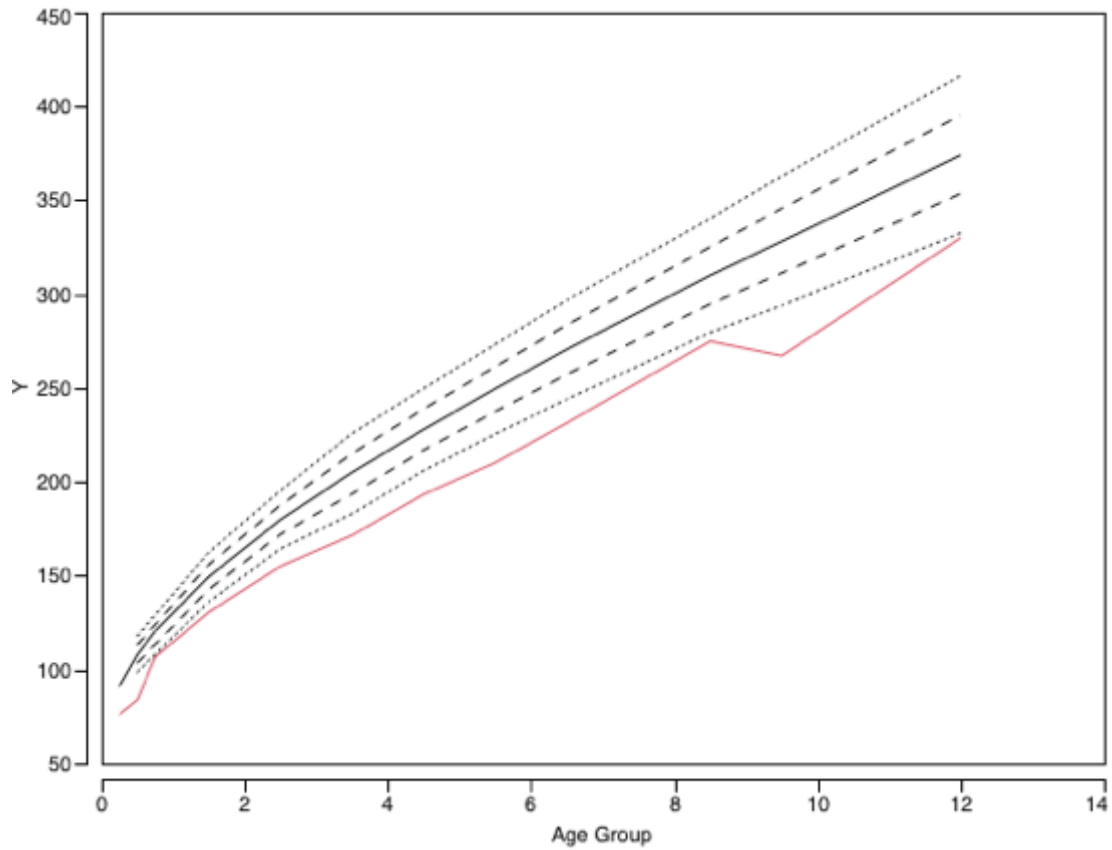
### *Skeletal Growth Profiles*

The reconstruction of skeletal growth profiles in the Velia population was done to provide an additional line of evidence regarding physiological stress during the growth and development period. Skeletal growth profiles (SGPs) were constructed following the protocols laid out by Humphrey (2000; 2003) (see Chapter 5). Modern comparative data are drawn from the studies of Maresh (1955; 1970), who conducted an extensive longitudinal radiographic study of hundreds of Denver children from 2 months of age to 18 years. The Maresh data set (1955; 1970) is commonly used as a modern standard in bioarchaeological studies of SGPs (Humphrey, 2000; 2003). The Maresh (1955; 1970) data that are presented here have been adjusted to correct for radiographic enlargement (Feldesman, 1992) as they are compared to direct measurements of archaeological bone. The mean values by age for Velia are presented in Appendix C. SGPs are presented here in this chapter.

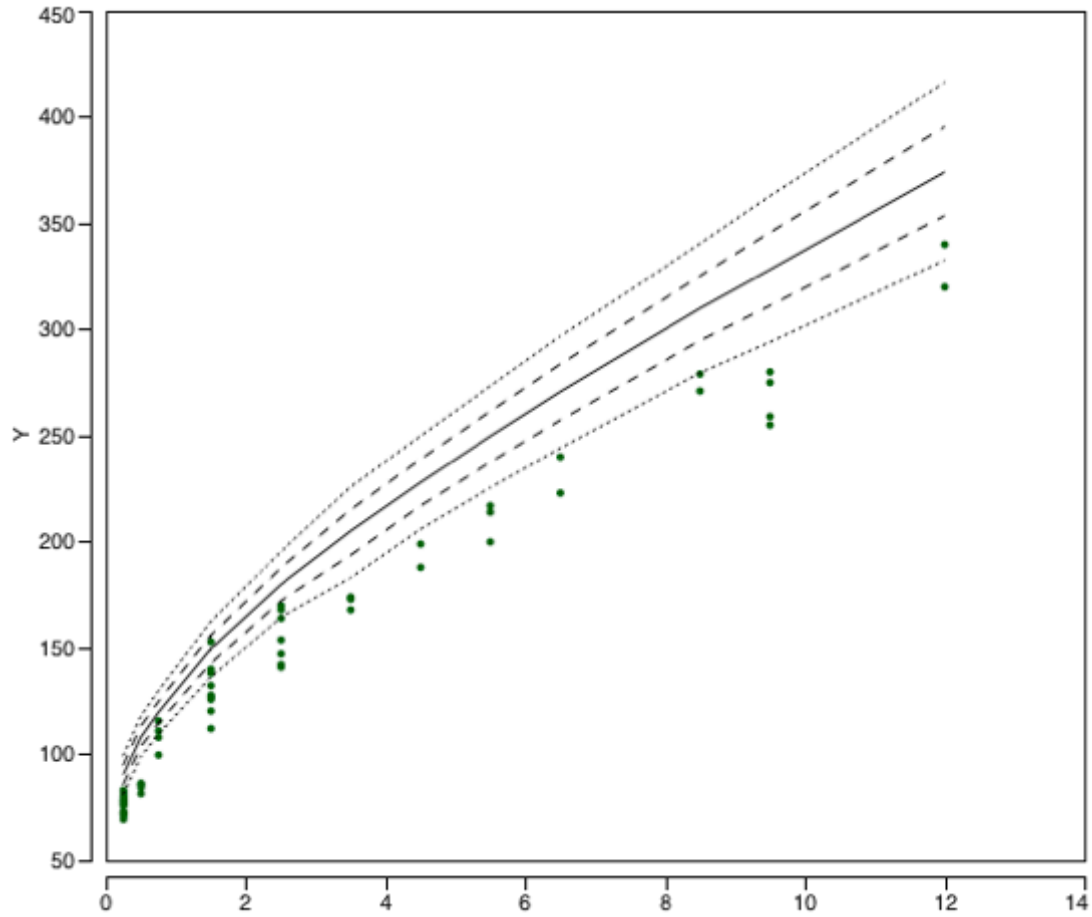
The first comparison drawn between Velia and the Denver sample are between femur diaphysis length and skeletal age (Figure 19). The differences in growth can be immediately seen, as the mean values per age in the Velia sample consistently fall below 2 SD of the Maresh means for each corresponding age group. In Figure 20, all data points for Velia are plotted against the Denver mean and standard deviations and a more nuanced picture emerges. Early in life (3 months and 6 months), all infants at Velia fall below 2SD of the Denver study. From 9 months to 2.5 years, there is more overlap with the Denver sample as some Velian children fall

within -2SD of the Denver mean. However, from 3.5 years to 9.5 years, all individuals are below 2SD of the Denver mean. By age 12, one individual is within 2SD of the Denver mean, while the other lies outside it. As Humphrey (2000) has noted, differences may be better observed by plotting residuals of a line describing the mean size of the children in the Denver study (Figure 21). Once again in this plot, the children of Velia seem to have a marked deficit in growth compared to Denver sample. In the first 2.5 years of life, most individuals are below 2SD of the Denver mean, with only 6 within -2SD of the Denver sample. One additional individual was actually above the Denver sample mean, but below +1SD. From 3.5 years to 12 years, all individuals fall below 2SD of the Denver sample, except for 1 individual in the 12 year olds. Although difficult to quantify, the separation between Velia and the Denver sample appears to worsen with age. Ultimately, there appears to be a marked deficit in growth early in life.

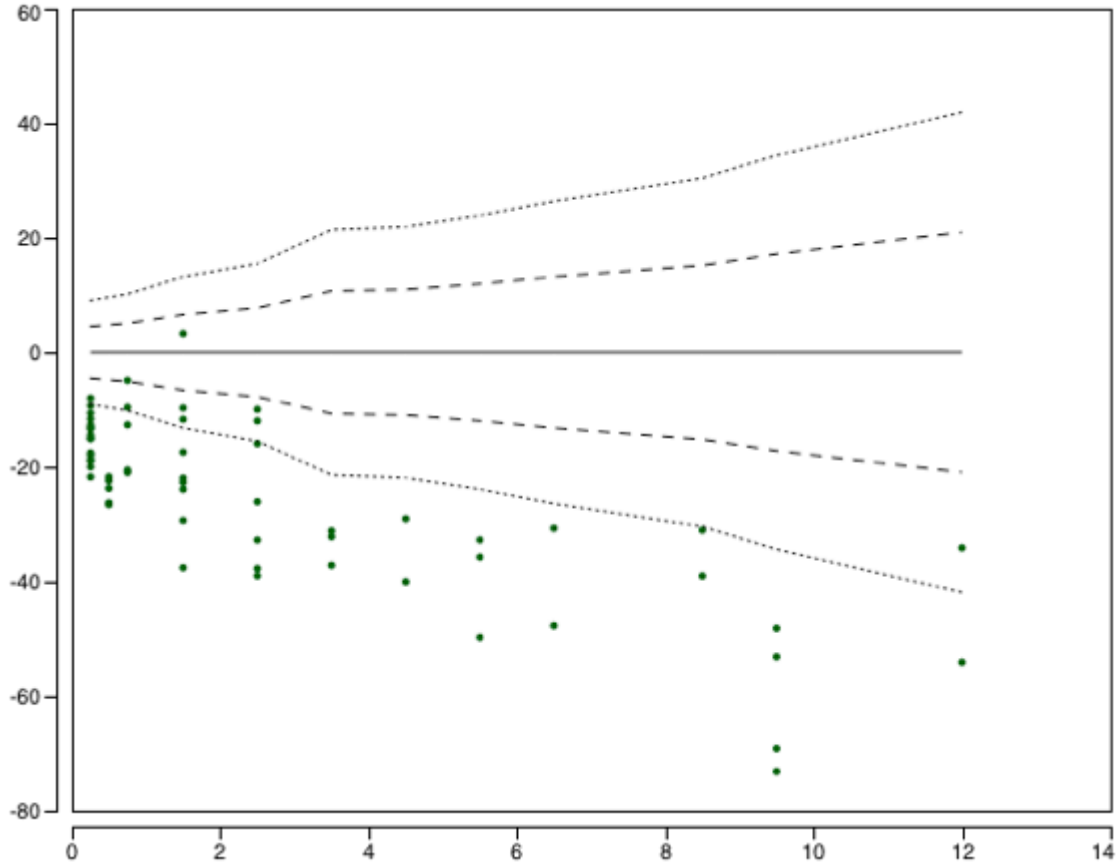
Comparisons were also made using the same growth data, but as percentages of adult stature reached for each age group. Mean adult femur length for the Denver sample was taken from Maresh (1955; 1970), and the Velian mean for adult femur length was calculated as an average of adult femoral lengths using both sexes. Figure 22 plots the Denver and Velia samples as percentages of their own adult means, while Figure 23 plots the residuals of differences in the percentage of adult femur length. Plotted as raw percentages of adult femur length, the Velia population once again shows slower growth, given that for each age group Velians have reached less of their total growth than the Denver sample. Based on mean values, there does not appear to be a tendency for this trend to worsen with age. However, if differences in percentage of adult size are considered (Figure 23), the general slope and trend of the differences do seem to indicate a slight increase in differences with age. Looking at the whole sample from Velia, only 7 Velians are above the Denver mean, while the remaining 53 fall below it. Out of the 53 Velians below the Marsh mean, 24 are below 2SD difference in percent of adult femur length. Early life, from 3 months to 1.5 years, the majority of Velians fall below -1SD of the Denver mean, suggesting that growth retardation began early in life. If the Velia sample as a whole is considered, a total of 58.3% of Velians fall within 2SD of the Denver mean when examining growth as a percent of adult size, leaving a large portion well below the modern standard. Figure 24 provides the same analysis as Figure 23, but uses mean values for Velia rather than individual data points. Here, it can be seen that nearly all individuals fall within 2 SD of the Denver population growth rate. When the SGPs from Velia and the Denver sample are compared as either lengths or percentages of adult length, there is a clear trend indicating that growth was reduced in the Velia population.



**Figure 19** – Femur diaphysis length vs. age. Y axis (femur length); X axis (age group); Solid black line (Denver mean); Dashed lines (+/- 1 SD Denver); Dotted lines (+/- 2 SD Denver); Red line (Velia mean)

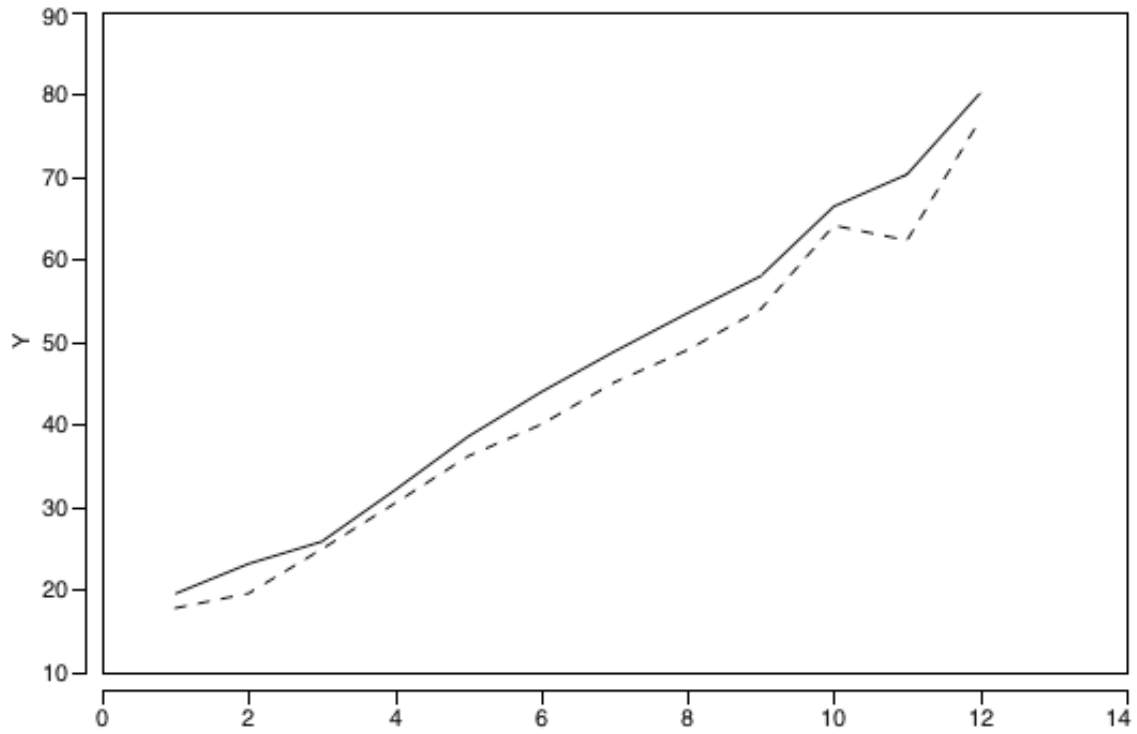


**Figure 20** – Femur diaphysis length vs. age with all individuals from Velia plotted. Y axis (femur length); X axis (age group); Solid black line (Denver Mean); Dashed lines (+/- 1 SD Denver); Dotted lines (+/- 2 SD Denver); Green dots (Individual data points from Velia)

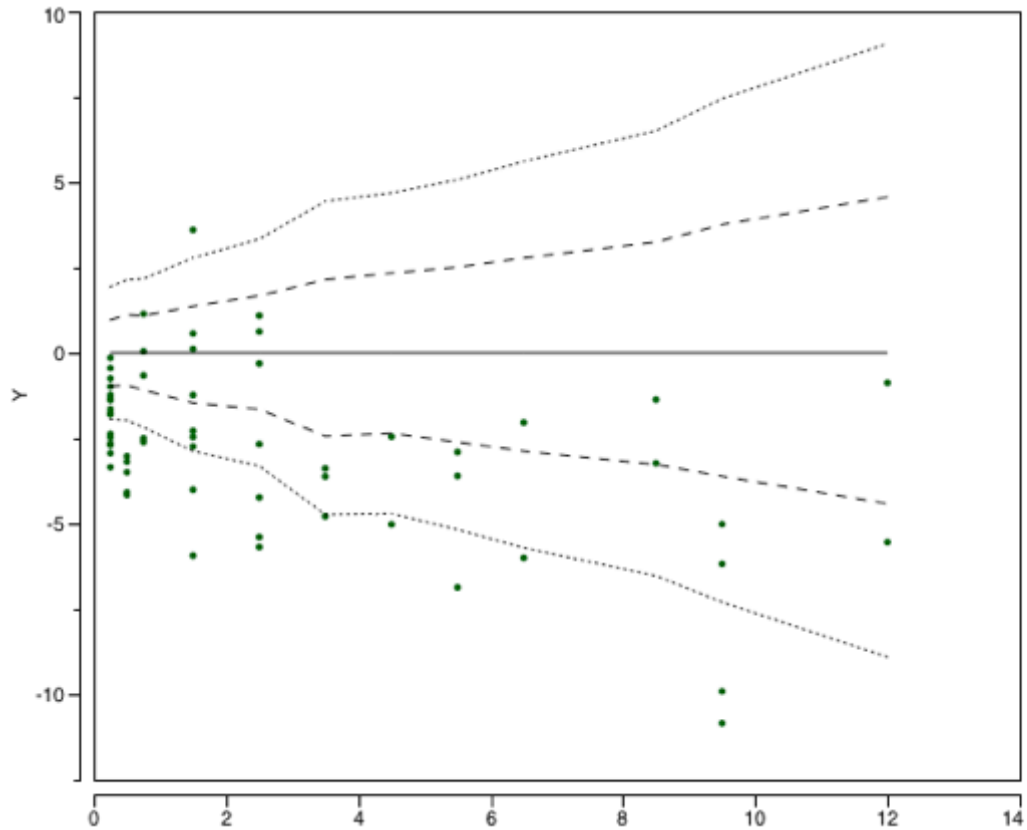


**Figure 21** – Differences in femur diaphysis length from mean value in the Denver sample vs. age. Y axis (Difference in femur length); X axis (age group); Solid black line (Denver Mean); Dashed lines (+/- 1 SD Denver); Dotted lines (+/- 2 SD Denver); Green dots (Individual data points from Velia)

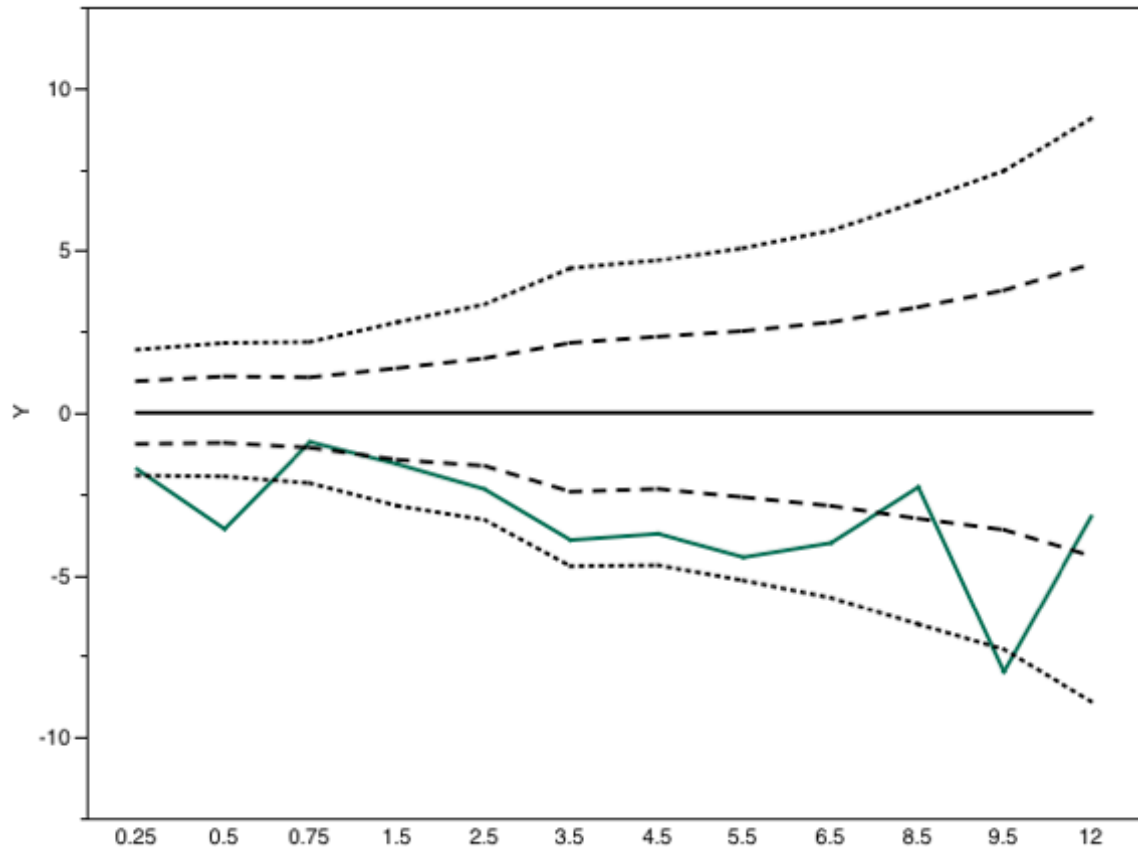




**Figure 22** – Percentage of adult size attained vs. age. Y axis (% of adult size); X axis (age group); Solid black line (Denver Mean); Dashed line (Velia mean)



**Figure 23** - Differences in percentage of adult size from mean value in the Denver sample vs. age for all individuals. Y axis (Difference in % adult size); X axis (age group); Solid black line (Denver Mean); Dashed lines (+/- 1 SD Denver); Dotted lines (+/- 2 SD Denver); Green dots (Individual data points from Velia)



**Figure 24** – Mean differences in percentage of adult size from mean value in the Denver sample vs. age. Y axis (mean difference in % adult size); X axis (age group); Solid black line (Denver Mean); Dashed lines (+/- 1 SD Denver); Dotted lines (+/- 2 SD Denver); Green line (Mean values by age for % difference from Denver mean)

## Measures of Adult Stress and Pathology

### *Porotic Hyperostosis*

A total of 90 adult individuals were examined for porotic hyperostosis and the results are summarized in Table 24 and 25. In 24 individuals, crania were either too fragmentary or not present at all, and in these cases any presence of pathology could not be assessed. From the original 90 individuals examined, 66 were preserved well enough to allow porotic hyperostosis to be examined and recorded. Of the 66 observable crania, 37 (56%) were male and 29 (44%) were females. Neither sex seemed disproportionately affected by poor preservation; twelve individuals in each sex (24.45% of all males and (29.27% of all females) could not be examined.

Males appear to have been more affected with porotic hyperostosis than females (Table 24), although males did compose a slightly larger percent (56%) of the observable sample. Out of the 12 individuals with clear signs of porotic hyperostosis, 3 (25%) were females and 9 (75%) were males. Within each sex, 10% of females and 24% of males had some degree of porotic hyperostosis. Although males had a higher prevalence of porotic hyperostosis than females, Fisher's Exact test fails to reject the hypothesis that there are no significant sex differences with porotic hyperostosis ( $p = 0.203$ ). With the sexes combined, 18% of the adults with observable crania had signs of porotic hyperostosis. If all adults are considered, 13% of the adults had porotic hyperostosis.

Age differences were also explored (Table 25). In females, all affected individuals were in the old age group. In the female old age group, 3 out of 11 (27%) individuals were affected. For males, the distribution of pathological individuals was more spread out. In young males, sample sizes were small, with only 3 individuals with observable crania, of which 2 (66%) showed signs of porotic hyperostosis. For middle-aged males, 6 out of 18 (33%) individuals had clear pathology and in the old age group, only 1 individual (6%) had porotic hyperostosis.

The severity of lesions was also assessed using Ribot and Roberts (1996) and results are in Table 26. None of the observed lesions were active at the time of death. The severity of the lesions was also quite mild, with all 12 cases scored as 1 or 2 (Ribot and Roberts, 1996).

**Table 24** – Summary statistics for prevalence of porotic hyperostosis in adults

	<b>Female</b>	<b>Male</b>	<b>Total</b>
<b>n rejected vs. N examined</b>	12 of 41 (29.27%)	12 of 49 (24.45%)	24 of 90 (26.67%)
<b>n with observable crania</b>	29 (44%)	37 (56%)	66 (100%)
<b>n with pathology vs. N observed</b>	3 of 29 (10.34%)	9 of 37 (24.32%)	12 of 66 (18.18%)
<b>% of pathological individuals</b>	25%	75%	100%

**Table 25** – Age trends of porotic hyperostosis for adults

Female	Age Group		
	18-29 (n = 7)	30-49 (n =11)	50+ (n = 11)
	0	0	3 (27.27%)
Male	Age Group		
	18-29 (n = 3)	30-49 (n =18)	50+ (n = 16)
	2 (66.67%)	6 (33.34%)	1 (6.25%)

**Table 26** - Activity and severity of lesions for porotic hyperostosis

Active (n/N)	Healed (n/N)	Severity $\leq 2$ (n/N)	Severity $\geq 3$ (n/N)
0/12	12/12	12/12	0/12
0%	100%	100%	0%

n (number reflecting corresponding assessment); N (total number with lesions)

### *Cribra Orbitalia*

Cribra orbitalia was investigated independently from porotic hyperostosis and the results are found in tables 27 and 28. A total of 90 individuals were initially observed but only 54 (60%) individuals had at least one intact orbit for analysis. Poor preservation affected sexes nearly equally, with slightly more females (41.46%) lost to taphonomic processes than males (38.78%), although in absolute counts more males were poorly preserved. More males (n = 30) are represented in the observable sample than females (n =24). Males had a higher prevalence of cribra orbitalia, with 13 (43.3%) affected, compared to females who had 9 (37.5%) with clear signs of pathology. However, Fisher’s exact test fails to refute the null hypothesis that no sex differences in pathology are present ( $p = 0.783$ ). Additionally, male prevalence of cribra orbitalia reflects 59% of all recorded cases. If sexes are combined, 22 individuals, or 40.74% of the adults had cribra orbitalia.

Age trends in the prevalence of cribra orbitalia were also examined. In females, cribra orbitalia was distributed roughly equally among age groups (see Table 28). For males, the young age group only had a sample size of 2, severely limiting any conclusions that can be drawn about this age group. The prevalence of cribra orbitalia in the male middle age group was slightly higher than in old age, but as percentages of observable individuals, cribra orbitalia was distributed nearly evenly between middle and old age for males.

As with porotic hyperostosis, cribra orbitalia was assessed for activity of lesions, as well as severity, using the methods outlined by Ribot and Robert (1996) (Table 29). The majority of the cases of cribra orbitalia were of healed lesions (81.8%), with only 4 (18.2%) showing clear signs that the lesions were active at the time of death. Moreover, nearly all observed lesions were mild (90.1%).

**Table 27** - Summary statistics for prevalence of cribra orbitalia in adults

	<b>Female</b>	<b>Male</b>	<b>Total</b>
<b>n rejected vs. N examined</b>	17 of 41 (41.46%)	19 of 49 (38.78%)	36 of 90 (40%)
<b>n with observable crania</b>	24 (44%)	30 (56%)	54 (100%)
<b>n with pathology vs. N observed</b>	9 of 24 (37.5%)	13 of 30 (43.3%)	22 of 54 (40.74%)
<b>% of pathological individuals</b>	41%	59%	100%

**Table 28** - Age trends of cribra orbitalia in adults

<b>Female</b>	<i>Age Group</i>		
	<b>18-29 (n = 7)</b>	<b>30-49 (n =9)</b>	<b>50+ (n = 8)</b>
	3 (42.86%)	3 (33.34%)	3 (37.5%)
<b>Male</b>	<i>Age Group</i>		
	<b>18-29 (n = 2)</b>	<b>30-49 (n =16)</b>	<b>50+ (n = 12)</b>
	1 (50%)	7 (43.75%)	5 (41.67%)

**Table 29** – Activity and severity of lesions for cribra orbitalia

Active (n/N)	Healed (n/N)	Severity $\leq 2$ (n/N)	Severity $\geq 3$ (n/N)
4/22	18/22	20/22	2/22
18.2%	81.8%	90.9%	9.1%

n (number reflecting corresponding assessment); N (total number with lesions)

### *Periostitis*

Periostitis was examined in upper and lower limbs separately. Results for sex and age trends for upper limbs are presented first and are summarize in Tables 30 and 31. Tables 32 and 33 provide detailed results for each upper limb bone and the severity of the remodeling response to infection. Results for periostitis by sex and age in the lower limbs can be found in Tables 34 and 35. Tables 36 and 37 provide a more detailed breakdown of lower limb pathology by bone type and severity of infection.

Upper limb elements (humerus, radius, ulna) were very well preserved, with only 8.9% of available individuals lost to poor preservation. Poor preservation favored males, who had seven of the eight unusable individuals. The remaining sample for study was nearly even. Of the 82

individuals with preserved upper limbs, 40 were female and 42 male. The prevalence of periostitis in upper limbs was remarkably low, as periostitis was only noted in 9.8% of individuals. The distribution of lesions by sex was even at 4 cases per sex. Not surprisingly, Fisher's exact test showed no signs of significant sex differences ( $p = 1.00$ ).

The distribution of periostotic lesions was also considered across age groups for each sex (Table 31). In females, 3 out of the 4 recorded cases were in old age. Periostitis was not found in young females and only 1 case in middle age. For males, there was 1 case in young age, 3 in middle age and none in old age.

Periostitis was also examined to see if either left or right sides were predominantly affected (Table 32). For both males and females, left humeri seemed to be slightly more affected than the right. The same trend was found in radii for females, but not for males where left and right sides were equal. Left and right sides of the ulna were equally affected in females, but one additional right ulna was affected in males. Using both raw counts and percentages, the ulna was the most affected element in females; while in males the humerus and ulna were equally affected (3.9%). The radius was the least affected bone for both sexes.

The severity of lesions was also assessed using the scoring methods outlined by Ribot and Roberts (1996) (Table 33). In females, 92% of lesions were mild (severity 2 or below) and only 8% were moderate (severity 3 and 4). No lesions were severe (severity 5 and 6). The severity of lesions in males was entirely mild, with all bones scoring 2 or below.

**Table 30** - Summary statistics for prevalence of periostitis (upper limbs) in adults

	<b>Female</b>	<b>Male</b>	<b>Total</b>
<b>n rejected vs. N examined</b>	1 of 41 (2.4%)	7 of 49 (14.3%)	8 of 90 (8.9%)
<b>n with observable bones</b>	40 (48.8%)	42 (51.2%)	82 (100%)
<b>n with pathology vs. N observed</b>	4 of 40 (10%)	4 of 42 (9.5%)	8 of 82 (9.8%)
<b>% of pathological individuals</b>	50%	50%	100%

**Table 31** - Age trends of periostitis (upper limbs) in adults

<b>Female</b>	<i>Age Group</i>		
	<b>18-29 (n = 10)</b>	<b>30-49 (n =17)</b>	<b>50+ (n = 13)</b>
	0 (0%)	1 (5.8%)	3 (23.1%)
<b>Male</b>	<i>Age Group</i>		
	<b>18-29 (n = 7)</b>	<b>30-49 (n =18)</b>	<b>50+ (n = 17)</b>
	1 (14.3%)	3 (16.7%)	0 (0%)

**Table 32** – Results for upper limb periostitis by bone

	<b>Humerus</b>		<b>Radius</b>		<b>Ulna</b>	
	<i>Left</i>	<i>Right</i>	<i>Left</i>	<i>Right</i>	<i>Left</i>	<i>Right</i>
<b>Female</b>	3	1	2	1	3	3
n/N (%)	4/80 (5%)		3/78 (3.8%)		6/81 (7.4%)	
<b>Male</b>	2	1	1	1	1	2
n/N (%)	3/77 (3.9%)		2/76 (2.6%)		3/76 (3.9%)	

n (number individual skeletal elements affected); N (total number of skeletal elements examined)

**Table 33** - Results for upper limb periostitis by severity of infection

	<b>Severity 1-2</b>	<b>Severity 3-4</b>	<b>Severity 5-6</b>
<b>Female</b>			
<i>Humerus (n)</i>	3	1	0
<i>Radius (n)</i>	3	0	0
<i>Ulna (n)</i>	6	0	0
<i>Total (%)</i>	92%	8%	0%
<b>Male</b>			
<i>Humerus (n)</i>	3	0	0
<i>Radius (n)</i>	2	0	0
<i>Ulna (n)</i>	3	0	0
<i>Total (%)</i>	100%		

Lower limb elements (femur, tibia, fibula) were examined in the same way as upper limbs. Lower limbs were also very well preserved, with only 5 individuals, or 5.6% of the total adult sample rejected due to poor preservation. The distribution of the remaining individuals slightly favored males (n = 46) over females (n = 39). The prevalence of periostitis in the lower limbs was extremely common, far more so than for the upper limbs, or for porotic hyperostosis or cribra orbitalia in the crania. Overall, with sexes combined, 83.5% of individuals had periostitis in the lower limbs. Nearly all males were affected (89.1%) and about three quarters of females (76.9%) had clear signs of periostitis in the lower limbs. Males represented 58% of all individuals with periostitis and although males were more prone to reactive bone in the lower limbs, Fisher's Exact test indicates that this higher prevalence was not statistically significant ( $p = 0.746$ ).

Examination of periostitis across age groups for each sex reveals that for females the prevalence of periostitis appears to have worsened with age (Table 35). Half of young females had lesions, followed by 81.3% in the middle age group and then finally 92.3% in the old age group. In males, young age marked the period of least stress, although prevalence was still high at 71.4%. Surprisingly, all of the middle aged males showed signs of periostitis. Presence of



periostitis was also high in old age for males, with 83.3% displaying subperiosteal new bone formation.

Analysis by skeletal element reveals some interesting trends (Table 36). Femurs were the least affected for both sexes, with only 17.9% affected in females, and 16.5% in males. Left and right sides were also nearly equally affected for both sexes. Tibiae were by far the most affected skeletal element. In females, 60.5% of all tibiae examined had signs of subperiosteal new bone formation. The prevalence was even higher in males, where 78% of tibiae examined had skeletal lesions. Males showed no bias to either side, with 36 on the left, and 35 on the right affected, but females had a small bias towards the right where 26 were affected compared to 20 on the left side. Infection in fibulae was also quite high. Periostitis was found in 35.6% of female fibulae and 53.8% of male fibulae. A small bias towards the right side was noted in female fibulae, similar to what was observed for tibiae. Once again, male biases towards left or right sides were not present as the left side only had one more case of periostitis than the right.

While the prevalence of subperiosteal new bone formation was high for both sexes, the severity of the reactionary bone tended to be quite low (Table 37). In females, 93% of all elements examined scored a 2 or lower (Ribot and Roberts, 1996). Only 6% scored a “moderate” severity of 3 or 4, only 1 individual (1%) showed signs of very severe reactionary bone. The trend for males differs, in that only 75% of the elements examined scored a 2 or below and 24% of elements scored a 3 or 4. What was similar to females is that only 1 element, a fibula in both cases, scored as 5 or 6.

**Table 34** - Summary statistics for prevalence of periostitis (lower limbs) in adults

	<b>Female</b>	<b>Male</b>	<b>Total</b>
<b>n rejected vs. N examined</b>	2 of 41 (4.9%)	3 of 49 (6.1%)	5 of 90 (5.6%)
<b>n with observable bones</b>	39 (45.9%)	46 (54.1%)	85 (100%)
<b>n with pathology vs. N observed</b>	30 of 39 (76.9%)	41 of 46 (89.1%)	71 of 85 (83.5%)
<b>% of pathological individuals</b>	42.3%	57.7%	100%

**Table 35** – Age trends of periostitis (lower limbs) in adults

<b>Female</b>	<i>Age Group</i>		
	<b>18-29 (n = 10)</b>	<b>30-49 (n = 16)</b>	<b>50+ (n = 13)</b>
	5 (50%)	13 (81.3%)	12 (92.3%)
<b>Male</b>	<i>Age Group</i>		
	<b>18-29 (n = 7)</b>	<b>30-49 (n = 21)</b>	<b>50+ (n = 18)</b>
	5 (71.4%)	21 (100%)	15 (83.3%)

**Table 36** - Results for lower limb periostitis by bone

	<b>Femur</b>		<b>Tibia</b>		<b>Fibula</b>	
	<i>Left</i>	<i>Right</i>	<i>Left</i>	<i>Right</i>	<i>Left</i>	<i>Right</i>
<b>Female</b>	7	7	20	26	11	15
n/N (%)	14/78 (17.9%)		46/76 (60.5%)		26/73 (35.6%)	
<b>Male</b>	7	8	36	35	22	21
n/N (%)	15/91 (16.5%)		71/91 (78%)		43/80 (53.8%)	

n (number individual skeletal elements affected); N (total number of skeletal elements examined)

**Table 37** - Results for upper limb periostitis by severity of infection

	<b>Severity 1-2</b>	<b>Severity 3-4</b>	<b>Severity 5-6</b>
<b>Female</b>			
<i>Femur (n)</i>	14	0	0
<i>Tibia (n)</i>	43	3	0
<i>Fibula (n)</i>	23	2	1
<i>Total (%)</i>	93%	6%	1%
<b>Male</b>			
<i>Femur (n)</i>	13	2	0
<i>Tibia (n)</i>	56	15	0
<i>Fibula (n)</i>	28	14	1
<i>Total (%)</i>	75%	24%	1%

## Chapter 7 – Discussion

The purpose of this chapter is to synthesize and interpret the results of Chapter 6 with the biocultural context of the Roman Imperial period. The skeletal data from Velia is also compared to modern populations where appropriate. The three primary methods, radiogrammetry, cortical histomorphometry and analysis of vertebral trabecular architecture, are explored first individually, and then as a whole. This is followed by a discussion of the stress indicators used and the effect of metabolic stress on bone loss and fragility. To conclude, the limitations of the study are discussed and a summary is provided that contextualizes bone maintenance and health of Velians within the Roman and modern biocultural settings.

### Radiogrammetry

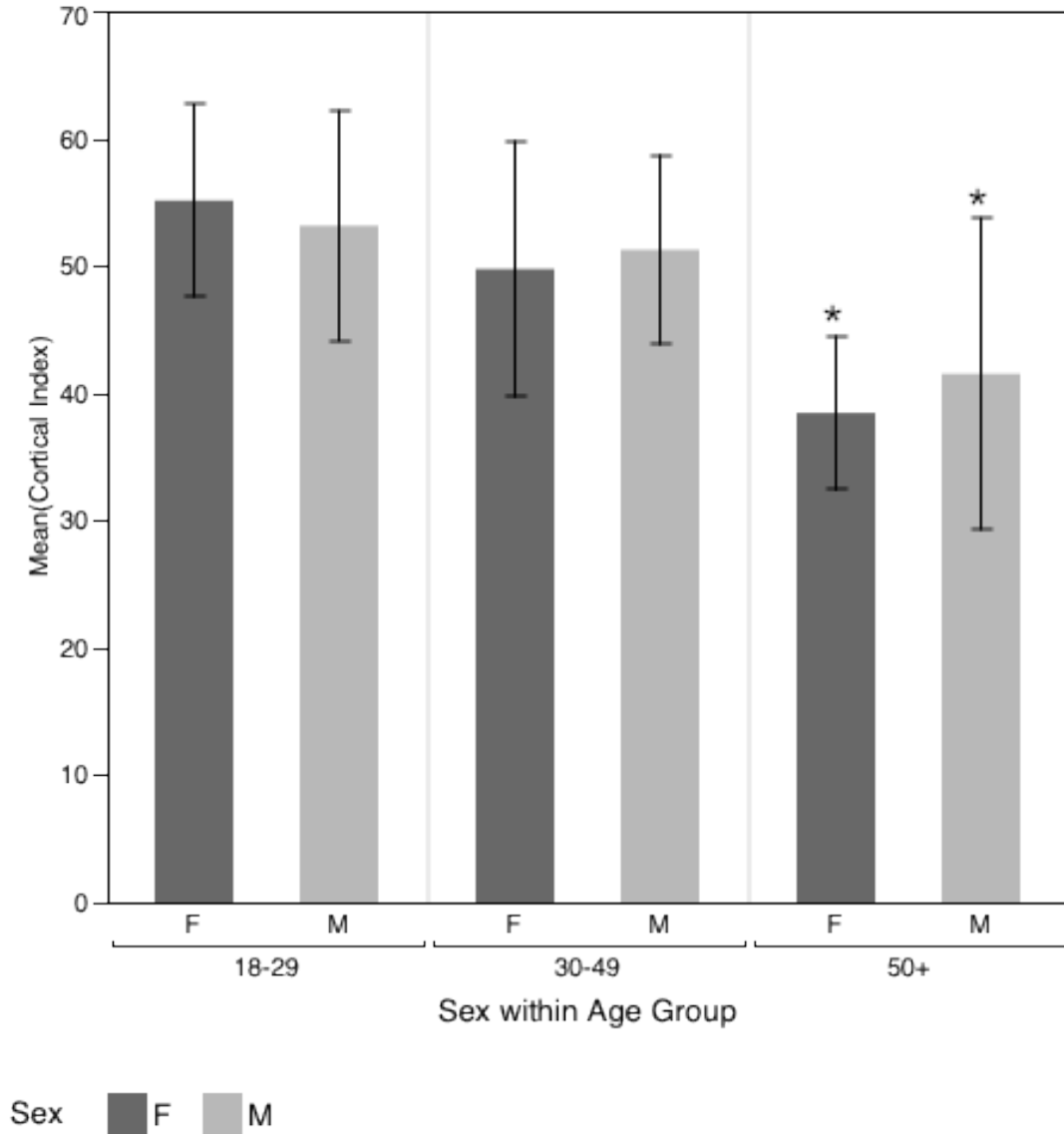
The results of the radiogrammetric analyses have shown that age and sex-related patterns of bone maintenance and loss at Velia generally follow modern population trends, although some notable exceptions are observed. Both females and males showed their highest values in the cortical index (CI) measure in the 18-30 age group, which is consistent with clinical observations on the attainment of peak bone mass (lifelong maximum bone mass) in the sexes (Böttcher et al., 2006). Moreover, females in the Velia population reached a greater peak cortical index than males, which has been noted in modern Italian, German and French groups (Maggio et al., 1997; Böttcher et al., 2006; Szulc et al., 2006), although this difference was small and not statistically significant. Observed higher cortical indices in young age females are likely the result of greater endosteal deposition in anticipation of metabolic requirements for pregnancy and lactation (Martin, 2003), as well as having a generally smaller bone and body size (Böttcher et al., 2006). This is supported by the smaller medullary spaces of young females compared to males of the same age group, although these differences were not statistically significant.

Values of total width (TW) appear to increase slightly with age in females, with greater TW the middle and older age groups, compared to the young age category. TW is a useful measure because it can inform us about periosteal deposition across age groups. This change was small however, and not statistically significant. It should be noted though that Maggio et al. (1997) have shown in a modern Italian sample that metacarpal periosteal expansion does occur in females, so the small increase in TW with age is not surprising. Interestingly, TW in males remained nearly unchanged with age. A large proportion of the males would have experienced difficult manual labor related to the fishing industry at Velia (Crowe et al., 2010) that would have been expected to potentially increase periosteal deposition over time. Although periosteal apposition is generally greater in males, most of the periosteal expansion appears to be in older males, and TW changes very little with age until after 60 (Maggio et al., 1997; Böttcher et al., 2006). These findings could imply that females at Velia only began a very physically demanding daily life in late adolescence and/or young adulthood, explaining the small increase in TW, while males perhaps had high strain-related deposition of bone much earlier and consistently across the life cycle. These data are also cross-sectional in nature, and while sample size is good for the radiogrammetry assessment, there remains the possibility that age-related changes in TW for both sexes are masked by these confounding factors.

Endosteal expansion was explored through the medullary width (MW) measure and is likely the primary cause of cortical thinning in the metacarpal. In the Velia population, medullary width (MW) expanded significantly with age for females, but not males. Standard deviations were slightly larger in males, and this variability may be obscuring observable changes with age. However, based on modern German (Böttcher et al., 2006) and Italian samples (Maggio et al., 1997), large increases in male MW prior to older age (60+) should not be expected, and this is consistent with observations from Velia, as MW expands substantially only in the oldest age group. Moreover, cortical thickness decreased proportionally to increases in medullary width in both sexes. These observed differences across the age groups are supported by clinical studies that have concluded that endosteal expansion outpaces periosteal gains and is the primary agent of cortical thinning with age in both sexes (Maggio et al., 1997; Szulc et al., 2006). It should be noted as well that the MW measures are estimations of true cortical thinning as this is a cross-sectional study and the possibility of group and survival effects exist. Nevertheless, it is argued that these trends at the different bone surfaces are consistent with endosteal expansion as the primary cause of cortical thinning.

Cortical index (CI) values in females decreased steadily across the three age groups (see Figure 25). The gradual drop in CI for Velian females is generally consistent with modern trends that show a steady decline in CI with age, although in modern the decline begins after the age of 45 (Böttcher et al., 2006; Toledo and Jergas, 2006). Due to the large conservative age groups necessary in bioarchaeological studies, some resolution in the timing of age-related bone loss is lost. However, in the Velian females, bone loss appears to begin, in the 30-49 age group, rather than in the 50+ age group alone. This earlier bone loss in the Velia population could be partially explained by biased sampling of older females in the 30-49 age group. However, Toledo and Jergas (2006), using a modern German population, have shown that CI values can drop from ages 25-29 to 30-34, perhaps reflecting accumulated skeletal changes to pregnancy and lactation. The apparent early bone loss in the 30-49 age group at Velia could reflect sampling bias towards older females, as well as those who died prior to their skeletons recovering from the effects of pregnancy and lactation. The importance of reproductive history in bone loss in historical and archaeological populations has been established previously (Agarwal and Stuart-Macadam, 2003; Agarwal et al., 2004; Mays, 2010). Estimated fracture risk was low for this age group as well, with only two out of fifteen individuals displaying CI values two standard deviations below the young adult mean (Meema and Meema, 1987), suggesting that bone loss was not advanced for the 30-49 age group. In contrast, older females had a seemingly high risk of fracture, as six of ten individuals had low bone mass, using the Meema and Meema (1987) standard. However, only one female had clear, observable fragility-related fractures.

In large modern Italian and German samples, males change very little in width, medullary expansion or cortical index until after the age of 65 (Maggio et al., 1997; Böttcher et al., 2006). The same broad pattern was found for males at Velia. In the Velia population, male CI between the middle and oldest age groups is significantly different, but because of the imprecision of aging older individuals, it is impossible to know when bone loss in older males at Velia is occurring. If the Meema and Meema (1987) assessment of fracture risk is taken, few ( $n = 4$ ) males displayed potentially abnormal bone, and only one of these individuals displayed any fragility fractures.



**Figure 25** – Cortical Index by age and sex. Y axis (Cortical Index); X axis (age and sex); \* indicates a statistically significant difference to young (18-29) and middle (30-49) age groups

The most important divergence from clinical patterns of cortical bone loss in the Velia population is that no sex differences were detected for any age group in CI values. This is unexpected as postmenopausal women in modern Western populations show a dramatic decline in CI compared to males (Maggio et al., 1997; Böttcher et al., 2006). However, postmenopausal sex differences in bone loss have not been seen to be necessarily universal in other bioarchaeological studies that have explored age and sex-related trends with measures of both cortical and trabecular bone (Mays, 1996; Lees et al., 1993; Ekenmen et al., 1995; Agarwal et al., 2004; 2009; Holck, 2007; Glencross and Agarwal, 2011). The smaller overall bone size (Böttcher et al., 2006) and steady depletion of endosteal bone with age in females should result in a

significant sex difference in the Velia population in old age, if modern postmenopausal changes are taken as a human universal, but none were observed. Consequently, the lack of sex differences in CI at Velia, particularly in old age, reflects an important difference in bone loss for this population. Exploring the biocultural context is then a critical factor in understanding how bone maintenance and loss varies over the life cycle and between the sexes.

Very few bioarchaeological studies have specifically used radiogrammetry on Roman era skeletal collections. The metacarpal radiogrammetry study by Mays (2006) on the Romano-British settlement of Ancaster, occupied during the 3rd and 4th centuries AD, offers the closest parallel to Velia. The findings from Velia are also comparable to those of Wharram Percy (11-16th centuries) (Mays, 1996) because it also has well-established studies on trabecular architecture (see Trabecular Architecture section in this chapter). The communities at Ancaster and Wharram Percy reflect rural environments, and while Velia was a port city, it too had a substantial agricultural base (Craig et al., 2009). Table 38 summarizes and compares the metacarpal cortical index values for Velia, Ancaster and Wharram Percy. The radiogrammetry data from Ancaster provides valuable information about Roman bone loss, but is somewhat limited in that it only provides data for females, and so potential sex differences are unknown in that population. Following the approach taken by Mays (2006), individuals in the middle and oldest age groups were also normalized as a percentage of young peak adult bone mass (Table 39). As compared to Ancaster, and Velia, Wharram Percy females had much less of a decline in CI in the oldest age group. When normalized as a function of young adult CI, females at Wharram Percy change very little, with values in the oldest age group reflecting 83.83% of those in the youngest, compared to 65.6% for Ancaster and 69.73% for Velia. The normalized values between Velia and Wharram Percy males are very close except in the oldest age group, where the older males at Velia show a greater decline from peak values compared to Wharram Percy. In the Wharram Percy population, peak values were in the middle age group, while at Velia males seem to reach peak bone mass earlier. There appear to be no sex differences in the oldest age group at Wharram Percy, although Mays (1996) does not provide the statistical comparison for this. Both sexes show remarkably consistent cortical indices in old age however, with CI values for females at 41.5% and males at 40.4% (Mays, 1996).

**Table 38** - Summary of cortical index (CI) values for Velia, Wharram Percy (Mays, 1996) and Ancaster (Mays, 2006)

	Wharram Percy		Ancaster		Velia	
Age Group	Mean	SD	Mean	SD	Mean	SD
<i>Females</i>						
<b>18-29 yrs</b>	49.5 N = 15	9.7	51.8 N = 11	10.7	55.11 N = 7	7.52
<b>30-49 yrs</b>	44.4 N = 27	8.4	47 N = 12	5.7	49.73 N = 15	10.02
<b>50+ yrs</b>	41.5 N = 23	7.9	34 N = 16	6.7	38.43 N = 10	6.02
<i>Males</i>						
<b>18-29 yrs</b>	42.9 N = 10	7.8	N/A	N/A	53.13 N = 6	9.02
<b>30-49 yrs</b>	45.4 N = 29	9.0	N/A	N/A	51.24 N = 20	7.41
<b>50+ yrs</b>	40.4 N = 34	7.1	N/A	N/A	41.51 N = 13	12.21

**Table 39** - Comparison of mean values of CI by age groups as measured by percentages of peak bone mass (assessed as age at highest mean for CI) for Velia, Wharram Percy (Mays, 1996), and Ancaster (Mays, 2006).

Age Group	Wharram Percy		Ancaster		Velia	
	Mean	%	Mean	%	Mean	%
<i>Females</i>						
18-29 yrs	49.5 N = 15	100%	51.8 N = 11	100%	55.11 N = 7	100%
30-49 yrs	44.4 N = 27	89.70%	47 N = 12	90.6%	49.73 N = 15	90.23%
50+ yrs	41.5 N = 23	83.83%	34 N = 16	65.6%	38.43 N = 10	69.73%
<i>Males</i>						
18-29 yrs	42.9 N = 10	94.5%	N/A	N/A	53.13 N = 6	100%
30-49 yrs	45.4 N = 29	100%	N/A	N/A	51.24 N = 20	96.44%
50+ yrs	40.4 N = 34	89%	N/A	N/A	41.51 N = 13	78.13%

A number of important observations can be made from these bioarchaeological comparisons. First, the populations of Ancaster and Velia seemed to have achieved higher CI means than Wharram Percy in young and middle aged females. However, the population of Wharram Percy seems to have retained more bone into old age. Males from Velia had consistently higher CI means in all age groups, and yet had around 11% lower bone mass in old age than Wharram Percy males, if compared to their population specific peak bone mass levels. While Velian males had larger and more bone overall, they lost more cortical bone in the transition to old age. For Wharram Percy, fragility fractures were rare, with only four vertebral “typical” osteoporotic fractures in all the females, with no clear evidence of hip or Colles’ fractures (Mays, 1996). At Velia, fractures are also rare, despite seemingly advanced bone loss. Only one female with a vertebral and Colles’ (wrist) fracture and one male with vertebral fractures show clear signs of what appear to be fragility fractures. In contrast, the prevalence of fragility fractures at Ancaster appear much higher, with six out of sixteen older females showing clear signs of fragility fractures, including one hip (femoral) and four Colles’ (wrist) fractures (Mays, 2006). Although the archaeological communities share a number of similarities, this inter-population variability likely reflects important differences in biocultural influences such as diet, reproductive history, and activity that could have created divergent pathways in bone growth and loss between the populations over the lifecourse.



Diet is well-known to be an important factor in maintaining bone health (Cashman, 2007). Diet is particularly important in growing individuals as 90% of peak bone mass on average is reached by the age of 20 in modern populations (Cashman, 2002). Peak bone mass, as interpreted here by the cortical index in the metacarpals, is lower at Velia than we see in modern groups. This finding for Velia is not unexpected as this has been previously been reported for Ancaster (Mays, 2006) and Wharram Percy (Mays, 1996), and it is thought that chronic under-nutrition in many historic populations is to blame. Roman populations such as Velia would be no exception to this as food shortages were common occurrences. Peter Garnsey (1998) has noted that regular, proper nutrition was likely rare in the Roman world and was particularly bad for infants who were weaned on to extremely poor quality foods. The medieval period also experienced swings in food availability, and the peasants of Wharram Percy were likely subjected to periodic food shortages (Mays, 1996). The isotopic dietary profile of all adults at Velia by Craig et al. (2009) shows a diet high in cereals, with a generally low intake of animal or marine protein. The cereals that were consumed were most likely wheat and barley, which are high in vitamin E, iron and calcium, but fairly low in lysine, vitamin A, vitamin B2 (riboflavin), vitamin C, and vitamin D (Garnsey, 1998, 1999; Bisel, 1988; Rickman, 1980). Craig et al. (2009) also note that legumes and olive oil would have been consumed quite often, which would have helped with protein and healthy fat intake, both of which are important in bone health (Cashman, 2007). Legumes also contribute a number of important micronutrients to the diet, such as zinc and calcium (Messina, 1999), which are essential for healthy bone maintenance (Cashman, 2007). The distribution of isotopic signatures in the adult sample revealed that no age bias in diet was noted (Craig et al., 2009). While males did consume more marine protein than females, there is no evidence for completely gendered foodways was not evident, as a number of women ate a significant amount of meat/fish and it was not a strict separation. The lack of clear gendered foodways is somewhat surprising given that it could be assumed males would have more favorable diets based on Roman notions of social worth and prestige between the sexes (Garnsey, 1999). However, the dietary reconstruction at Velia is not an anomaly in the Roman world. Isotopic reconstructions of diet for the larger and mostly contemporary Roman port town of Isola Sacra also showed that while males did seem to have a more varied diet, gendered differences were overall small (Prowse et al., 2005). Much like what was found at Velia, many females at Isola Sacra appear to have consumed more protein than a number of males (Prowse et al., 2005), suggesting that preferential access to food may have been related to elite status rather than sex or gender in port towns. To summarize, under-nutrition at Velia, rather than poor dietary quality, likely contributed to a generally lower peak bone mass compared to living Western populations. Diet was also seemingly not divided strictly along gender lines, and this may help explain in part why no sex differences in cortical index were observed at Velia.

Reproductive behavior could also have influenced bone loss in Roman women. As discussed in Chapter 4, Roman historians have noted that menarche is thought to have occurred in the mid-teens, potentially as late as 16 or 17 (Harlow and Laurence, 2002). The age of marriage for a Roman woman was generally thought to have occurred in the early teens, but new reconsiderations have shown that for non-elite women, marriage occurred in the late teens to early twenties (Kleiner and Matheson, 1996; Harlow and Laurence, 2002). The general consensus among historical reports is that a Roman woman would begin having children shortly after marriage up to her late 30s or even early 40s (Leftkowitz and Fant, 1982). Roman women would typically have had around 5-8 children, with approximately half of those infants surviving

to adulthood (Leftkowitz and Fant, 1982; Garnsey and Saller, 1987; Garnsey, 1998). Estimations of the number of births per woman at Velia agrees with these historical interpretations (pers.comm, Sperdutti, 2011). A similar pattern of menarche and pregnancy has also been suggested for females at Wharram Percy (Mays, 1996; Agarwal et al., 2004). Breastfeeding practices in the Roman world are more difficult to discern. Soranus recommended breastfeeding be done by the mother, but recognized wet-nursing as a viable option as well. A number of Roman scholars have stated that in all likelihood, wet-nursing was probably practiced more by elite women than commoners (Garnsey, 1988; Toner, 2009), as poorer people would not have the financial means to hire a nurse (Toner, 2009). Whether breastfed by the mother or a wet-nurse, weaning would typically start after 3-6 months, usually on to a nutrition poor gruel or pap, and last until 2-2.5 years of age (Garnsey, 1999). This range in weaning years has been supported by a number of bioarchaeological analyses as well (Fuller et al., 2006; Dupras and Tocheri, 2007; Prowse et al., 2008). As a whole, the Roman biocultural practices surrounding reproduction suggest that the hormonal milieu throughout women's lives was considerably different from Western women living today, who typically begin menarche much earlier, have children much later in life, have far fewer children and breastfeed on average less than six months.

The reproductive history of Roman women is then quite similar to women in modern non-industrialized societies today. In these societies the hormonal drop in menopause is greatly mediated by their reproductive history, specifically, later menarche, high parity and long periods of breastfeeding (Agarwal and Stuart-Macadam, 2003). Clinical research has suggested that high parity and extended breast-feeding may have a positive effect on the female skeleton (Cumming and Klineberg, 1993; Michaëlsson et al., 2001). In contrast, recent work in a Mexican mestizo population has shown that longer periods breastfeeding (24-36 months) may act as a risk factor for osteoporosis (Rojano-Mejía et al., 2011). While the work of Rojano-Mejía et al. (2011) highlights that the interplay between reproductive history and osteoporosis risk is not fully understood, the weight of the evidence suggests that reproductive history has no ill effects on bone loss later in life, and may in fact help maintain bone into old age (Lenora et al., 2009). While the exact reproductive histories of women at Velia are not known, they would have been closer to other archaeological populations like Wharram Percy than modern women today. Consequently, women at Velia would have had a reduced lifetime hormonal exposure, with high parity and extended breastfeeding practice, compared to that of Western women today. It is possible, as Agarwal et al. (2004) have argued for the Wharram Percy population, that high parity and extended breastfeeding practices in Roman women could have potentially had an overall positive effect on the skeleton, and could explain why no sex differences are observed in old age at Velia.

Finally, numerous scholars have pointed out that daily life during the Imperial Roman period was likely difficult and physically demanding, at least for non-elite individuals. Agriculture was likely a major source of labor expenditure at Velia and this would have been backbreaking work (see Chapter 4). While the isotopic evidence suggests agriculture was likely a primary source of food, clearly not everyone was involved in this production; historical documents indicate that a substantial variety of occupations were found in Roman cities (Leftkowitz and Fant, 1982; Kleiner and Matheson, 1996; Toner, 2009). Yet despite this apparent diversity of work, a number of Roman scholars have pointed out that most people in Roman cities were probably unskilled labor and would have had to take up physically demanding

jobs even within city walls (Brunt, 1980; Erdkamp, 1999; Toner, 2002, 2009). While urban occupations were certainly organized along gender lines, among commoners, most women would have helped with farming or provided skilled labor in more urban areas in order to survive and help support the family (Scheidel, 1995). Children were often put to work early as well (Dixon, 2001; Redfern, 2007; Sigismund-Nielsen, 2007), and given that subadult skeletons respond more to physical activity with increased periosteal apposition compared to adults (Pearson and Lieberman, 2004; Ruff et al., 2006), childhood labor has important implications for understanding bone growth, maintenance and later loss of bone in Romans.

It seems likely that physically demanding day to day life in the past could have contributed to maintaining bone mass with age and could help explain the lack of sex differences in cortical index in old age at both Velia and other archaeological populations like Wharram Percy and Ancaster. Mays (1996; 2006) has emphasized that the division of labor was likely negligible at Wharram Percy, and that women at Ancaster would have been active in day-to-day work. For the Ancaster metacarpal data, we do not know if sex differences were present. However, fragility fractures were much more common at Ancaster, as compared to Velia and Wharram Percy. It is well established that strenuous physical activity helps mediate bone loss and fragility fractures (Warburton et al., 2006), so it is possible that although women at Ancaster were involved in physically challenging day-to-day activities, it was not sufficient to protect them from fragility fractures.

For all the historical evidence of day-to-day physical activity in the Roman world, Velia shows accelerated bone loss in the oldest age groups, compared to Wharram Percy. While daily physical activity in the Roman world would have been substantial, it may not have been as arduous as the medieval period. It is also important to consider that social roles in the Roman world were quite fluid (Harlow and Laurence, 2002, 2007; Toner, 2009) and arguably more complex than those in medieval populations. This social variability could be reflected in the wider range of cortical bone loss at Velia, as those with advanced bone loss may represent individuals whose occupations did not require strenuous labor. At a port city like Velia, this may have included dozens of trade and service occupations. Even with these cultural differences between these populations, it is hypothesized that physical activity was a significant factor in preventing post-menopausal bone loss in women in the past. Physical activity, while potentially variable among the sexes and social classes, would have been dramatically elevated compared to present day levels and would have still offered some benefit to maintaining bone mass with age. This last point is also reflected by the fact that only one female and one male have clear signs of osteoporotic fractures.

It is important to note here that patterns of bone loss in the Isola Sacra population discussed earlier are quite different than those of Velia, where significant sex differences in cortical remodeling are observed (Cho and Stout, 2011). Unfortunately, in the Cho and Stout (2011) study, only rib and femoral samples were examined for relative cortical area, and these are not directly comparable to radiogrammetry of the second metacarpal. The results of the rib relative cortical area are considered in the *Histomorphometry* section however. Nevertheless, a comparison of the broad trends in age-related bone loss between Isola Sacra and Velia are still important given the overall temporal and lifestyle similarity between the archaeological sites (Craig et al., 2009). Dietary differences between sexes at Isola Sacra were likely minor (Prowse

et al., 2005; Craig et al., 2009). Consequently, factors other than diet were likely responsible for the observed sex differences in old age. The people of Isola Sacra represent those who lived and worked at Portus, which was much larger and more urbanized than Velia (Craig et al., 2009). The people of Portus also seem to have been from a middle class (Waterlow, 1980; Bunson, 1991; Garnsey, 1998), which was somewhat unusual for the Roman period (Toner, 2002; 2009). The people of Velia represent a more working class from a non-elite lifestyle. These socioeconomic differences between the archaeological communities may have translated to important differences in gendered activities between the sites. For example, while it appears many males at Velia participated in the day to day fishing and port operations (Crowe et al., 2010), it is possible that a number of females were involved with agriculture as females did participate in tending the fields in the Roman world (Scheidel, 1995), and Velia relied substantially on its agricultural base (Craig et al., 2009). For the larger, more urbanized Isola Sacra population, while males were also heavily invested in the day-to-day work of ports (Craig et al., 2009), female occupations may have reflected more domestic or commercial enterprises. This would have translated to lower strain on bone throughout life for females of Isola Sacra, and thus greater sex differences later in life (Cho and Stout, 2011).

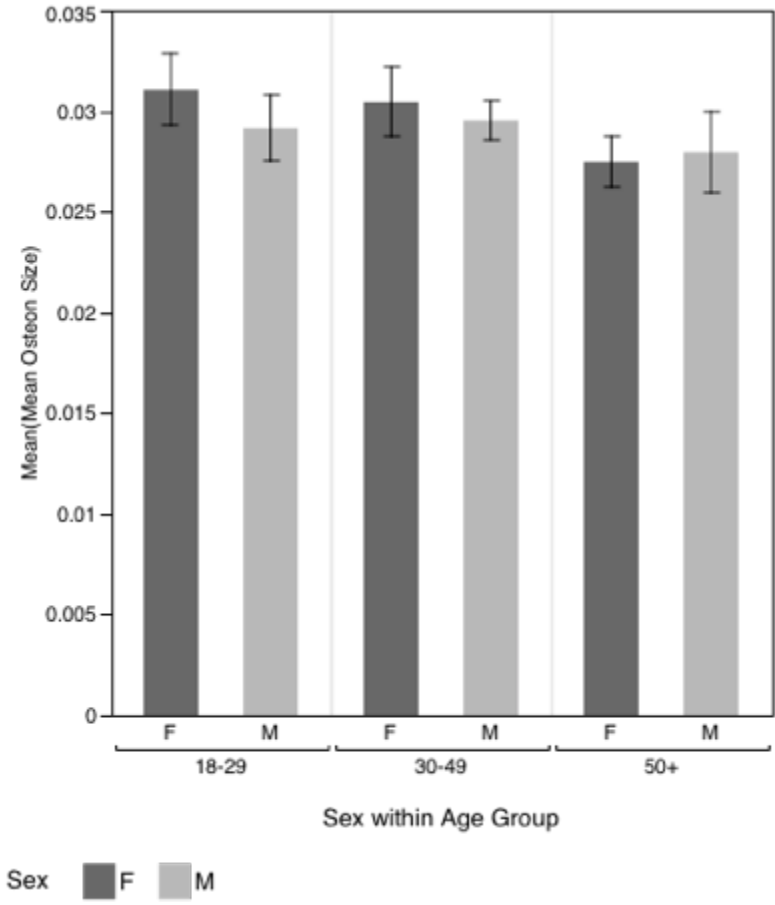
## **Histomorphometry**

### *Microstructural Level*

Osteon size was quite uniform across the three age groups and for each sex (Figure 26). While osteon size did decrease with age in females only, the changes were small and not significant. These results are consistent with a study by Pfeiffer (1998) who showed that osteon size is poorly correlated with sex or age. Pfeiffer (1998) noted that females tend to have smaller osteons, but that in most populations this observation is not significant and cannot be attributed to more than chance. Similarly, differences in osteon size with age are seen in some populations (Pfeiffer, 1998), but most do not support the observation that osteon size changes significantly with age. Finally, the mean osteon size for the Velia population as a whole was  $0.029 \text{ mm}^2$ , which is essentially equal what has been reported for a modern African American population, and to the archaeological population of Isola Sacra, who both have a mean osteon size of  $0.030 \text{ mm}^2$  (Cho and Stout, 2003). It should be noted that the histomorphometric values from Cho and Stout (2003) are used over those from Cho and Stout (2011) because means for age and sex are provided and facilitate comparisons with Velia. Mean values for remodeling are consistent between the studies (Cho and Stout, 2003; Cho and Stout, 2011), except for macro-level analyses of rib endosteal and cortical areas. This is probably due to an increase sample size in the Cho and Stout (2011) study, and so when referring to cortical area, the more recent report was used here. In contrast, a sample from modern European Americans from the Cho and Stout (2003) study had a mean osteon size of  $0.050 \text{ mm}^2$  and was significantly different from both African Americans and the Isola Sacra and Velia populations. Significance of osteon size is difficult to ascertain. Pfeiffer et al. (2006) tested the hypothesis that osteon size was related to biomechanical forces acting on the bone as a result of strenuous physical activity. The study compared femoral osteon sizes against those in the rib in a Later Stone Age population and a British historical population (Pfeiffer et al., 2006). The study found no consistent statistical

relationship between osteon size and physical activity and/or metabolic activity (Pfeiffer et al., 2006). However, van Oers et al (2008) did report that in a modern population smaller osteon size was associated with strain load. More recently, Pfeiffer and Pinto (2012) have noted that the weight of the evidence does suggest a relationship between osteon size and activity, but that this relationship is likely complex and mediated by other factors. Some of these include fluctuations in local and systemic factors such as fluctuations in cytokines, vitamin D, calcitonin, estrogen and parathyroid hormone (Pfeiffer et al., 2006). Newly developed and improved methods (van Oers, 2008; Skedros, 2012) may also help clarify the relationship between osteon size and bone strain in future studies.

Osteon population density, or OPD, increased with age significantly in females, but not for males. A significant increase in OPD with age is expected as osteons accumulated in the cortex through continuous bone remodeling in life. The lack of significant increase in OPD with age in males can be explained by the very small sample size ( $n = 2$ ) in the 18-29 age group. Even with the poor sample size, statistical significance is nearly reached ( $p = 0.08$ ), suggesting that with a larger sample, male OPD would follow expected trends. Moreover, OPD increases most from the second to third decades, continues to increase in mid adulthood, but by age 50 on average, it reaches an asymptote as the cortex begins to dramatically remodel out older osteons (Frost and Wu, 1967; Wu et al., 1970; Stout and Teitelbaum, 1976; Stout and Lueck, 1995). Subsequently, it is not surprising to find only a small, but not significant increase in OPD between the 30-49 and 50+ age groups, especially considering the limitations of skeletal age determination techniques. As seen with osteon size, sex differences were not present for OPD, with age groups either combined or analyzed separately. When OPD is averaged for the whole Velia population, some interesting observations can be made when comparing Velia to other modern and archaeological groups (see Table 40). From these comparisons, the Velia sample has the smallest OPD. The Kulubnarti sample from Sudanese Nubia is the only other population that is close to Velia for the OPD measure.



**Figure 26** – Mean osteon size by age and sex. Y axis (Mean osteon size mm<sup>2</sup>); X axis (age and sex); error bars represent the standard error of the mean.

**Table 40** – Comparisons of histomorphometric values between populations

	On.Ar (mm <sup>2</sup> )	OPD (#/mm <sup>2</sup> )	$\bar{U}_{RC}$ (#/mm <sup>2</sup> /year)	$V_{f,r,t}$ (mm <sup>2</sup> /mm <sup>2</sup> /year)	$netV_{f,r,t}$ (mm <sup>2</sup> /mm <sup>2</sup> )
<b>Velia</b> (~100-200 AD)	0.029 ± 0.005	13.98 ± 3.13	0.827 ± 0.432	0.025 ± 0.015	0.569 ± 0.096
<b>Isola Sacra</b> (Cho and Stout, 2003) (~100-300 AD)	0.030 ± 0.001	20.95 ± 0.81	0.97 ± 0.07	0.030 ± 0.002	0.72 ± 0.05
<b>European American</b> (Cho and Stout, 2003) (20 <sup>th</sup> century)	0.040 ± 0.001	21.02 ± 0.61	1.17 ± 0.09	0.050 ± 0.01	1.01 ± 0.08
<b>African American</b> (Cho and Stout, 2003) (20 <sup>th</sup> century)	0.030 ± 0.001	22.54 ± 0.83	0.79 ± 0.03	0.030 ± 0.001	0.820 ± 0.03
<b>Modern (Mixed)</b> (Stout and Lueck, 1995) (20 <sup>th</sup> Century)	0.040 ± 0.001	18.8 ± 1.07	2.4 ± 0.48	0.102 ± 0.023	0.937 ± 0.091
<b>Ledders</b> (Stout and Lueck, 1995) (~1000 AD)	0.033 ± 0.001	18.6 ± 1.21	2.0 ± 0.57	0.065 ± 0.019	0.702 ± 0.072
<b>Gibson</b> (Stout and Lueck, 1995) (50 BC – 400 AD)	0.035 ± 0.001	17.8 ± 1.22	1.0 ± 0.144	0.038 ± 0.006	0.676 ± 0.046
<b>Windover</b> (Stout and Lueck, 1995) (8120-6900 years B.P.)	0.036 ± 0.001	17.4 ± 0.89	1.1 ± 0.13	0.039 ± 0.005	0.713 ± 0.053
<b>Kulubnarti</b> (Mulhern, 2000) (550-1450 AD)	0.036 ± 0.001	14.24 ± 0.07	0.91 ± 0.09	0.034 ± 0.0003	0.585 ± 0.005

On.Ar (mean osteon area); OPD (osteon population density);  $\bar{U}_{RC}$  (mean annual activation frequency);  $V_{f,r,t}$  (mean annual bone formation rate);  $netV_{f,r,t}$  (net osteonal remodeling); ± (standard deviation)

Explaining the lower mean OPD values at Velia is difficult, but one possible explanation that fits the biocultural context of the Roman period would be that chronic malnutrition might have had an effect. Paine and Brenton (2006) found that in a sample of 20<sup>th</sup> century South African blacks, individuals with general malnutrition had OPD counts that were quite low. The range of OPD values presented by Paine and Brenton (2006) are consistent with those from Velia, which suggests that perhaps malnutrition may have been a factor for many Velians. However, Paine and Brenton (2006) also found that rib osteon size tended to be quite larger in those with malnutrition, compared to healthy controls. The mean osteon size in malnourished

individuals was  $0.044 \text{ mm}^2$  (Paine and Brenton, 2006), well above the  $0.029 \text{ mm}^2$  mean for Velia. However, as mentioned previously, local and systemic factors that contribute to osteon size are complex and not fully understood (Pfeiffer et al., 2006). However, the OPD values for Isola Sacra, a roughly contemporary site to Velia with a generally similar diet (Craig et al., 2009), has OPD values that are much more consistent with other archaeological and modern samples (Cho and Stout, 2003), placing a hypothesis based solely on malnutrition in question.

An alternative hypothesis is that the rib cortices of Velia were thinner and may have biased sampling of osteons and thus affected OPD. Osteon distribution is highly variable (Pfeiffer, 1998) and thinner rib cortices (percent cortical area) may have affected OPD. If percent cortical area is examined, males at Isola Sacra have a larger overall percent cortical area, while the reverse is true for females. Moreover, if the percent cortical area in the rib of the whole sample is considered and compared modern European and African Americans, the hypothesis that percent cortical area might be having an effect on OPD does not hold up well. The mean for Velia for percent cortical area is 34.6%, while the European and African American means are 33% and 35%, respectively (Cho and Stout, 2003). Ultimately the amount of bone present in the ribs at Velia falls well within the range of those from Isola Sacra and modern populations whose OPD values are considerably higher than those of Velia. Subsequently, it is difficult to attribute the OPD values at Velia to sampling error or chronic malnutrition, as there is evidence both for and against either scenario. Given that OPD is intimately tied to activation frequency and bone formation rate, the discussion will now move to discussing those measures and what the significance of those remodeling parameters might mean.

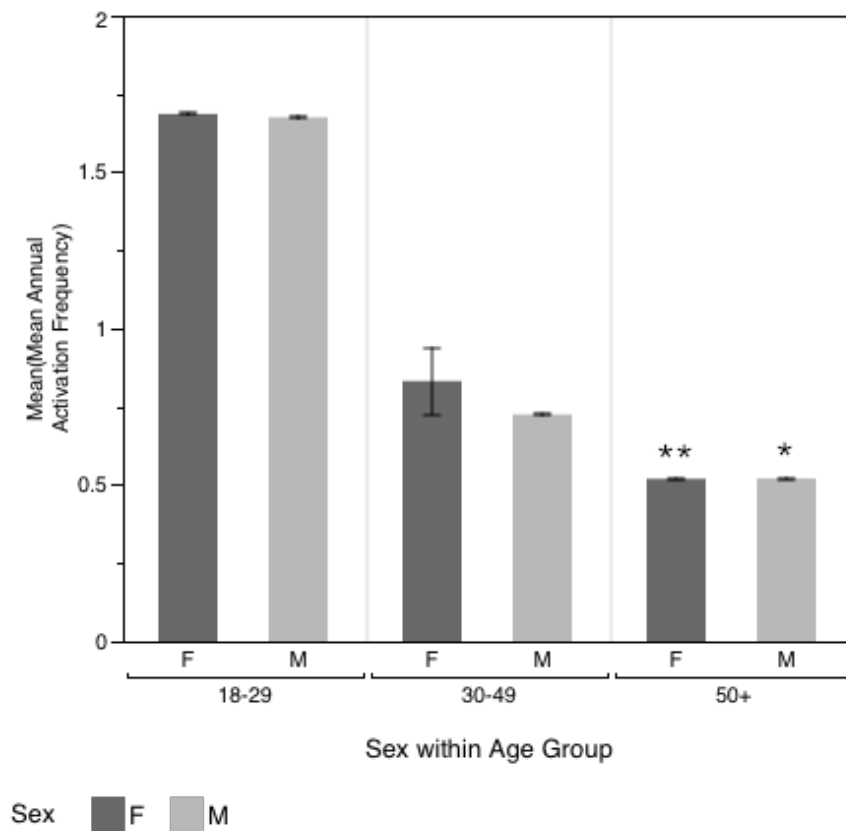
The activation frequency ( $\bar{U}_{RC}$ ) results for Velia closely follow trends published for other archaeological populations (Stout and Lueck, 1995; Mulhern, 2000; Cho and Stout, 2003), in that  $\bar{U}_{RC}$  changes significantly with age in both sexes (Figure 27). This is expected, as activation frequency would only increase in adulthood under unusual metabolically induced demands on the skeleton (Martin and Burr, 1989). However, Cho and Stout (2011) note that hormonal changes from menopause can increase activation frequency in females. For Velia, females in young and middle age created more osteons annually per  $\text{mm}^2$  than males, but this was not statistically significant, as was also noted in the Kulubnarti sample from Sudanese Nubia (Mulhern, 2000). In contrast, the Isola Sacra population did have a significant sex difference in  $\bar{U}_{RC}$ , but only for the 50+ age group and the trend was the same in that female  $\bar{U}_{RC}$  was greater than that for males. Stout and Lueck (1995) did not report sex differences in their study of three Native American populations. When the Velia sample is examined as a whole, activation frequency is second lowest compared to a number of modern and archaeological populations (see Table 40). The closest activation frequency to Velia is from a sample of modern Africa Americans, who have slightly lower activation frequency. The Isola Sacra sample, which is culturally most similar to the population of Velia, has a mean  $\bar{U}_{RC}$  that is substantially higher than Velia.

In order to explore these differences between Velia and Isola Sacra further, Table 41 outlines the mean values for  $\bar{U}_{RC}$  by age and sex. In young age,  $\bar{U}_{RC}$  is higher in both sexes at Velia than at Isola Sacra, and so cortical bone in the ribs was more active at Velia in young age. In middle age, some important differences begin to emerge. Females at Isola Sacra and Velia remain quite closely paced in terms of new osteon creations. In contrast, males in middle age diverge quite substantially, with those from Isola Sacra producing nearly 20% more osteons



annually than those from Velia. In old age, the  $\bar{U}_{RC}$  in females is substantially different, with females from Isola Sacra producing nearly double the amount of new osteons annually than females from Velia. In older males, the differences noted in middle age subside quite a bit as the Isola Sacra males slow down  $\bar{U}_{RC}$  quite substantially and are only just above the  $\bar{U}_{RC}$  produced in males of Velia. From this closer examination, it seems that the differences between the sites are mostly coming from changes in older females, although  $\bar{U}_{RC}$  in middle-aged males differs a fair amount as well. Before investigating some of the potential causes and the significance of these findings, the remaining histomorphometric variables are discussed and then explored as whole.

While activation frequency ( $\bar{U}_{RC}$ ) represents the number of new osteons created annually per  $\text{mm}^2$ , bone formation rate ( $V_{f,r,t}$ ) is a product of  $\bar{U}_{RC}$  and mean osteon size and thus represents the average amount of bone formed annually per  $\text{mm}^2$  in a particular bone.  $V_{f,r,t}$  at Velia decreased significantly with age in both sexes. In both sexes,  $V_{f,r,t}$  changed significantly across every age group (see Figure 28). Significant changes with age were also noted in the archaeological and modern populations found in Table 40. Sex differences in  $V_{f,r,t}$  were also statistically explored and none were noted. This also matches the trends in published reports that find no difference in  $V_{f,r,t}$  between the sexes (Mulher, 2000; Cho and Stout, 2003).



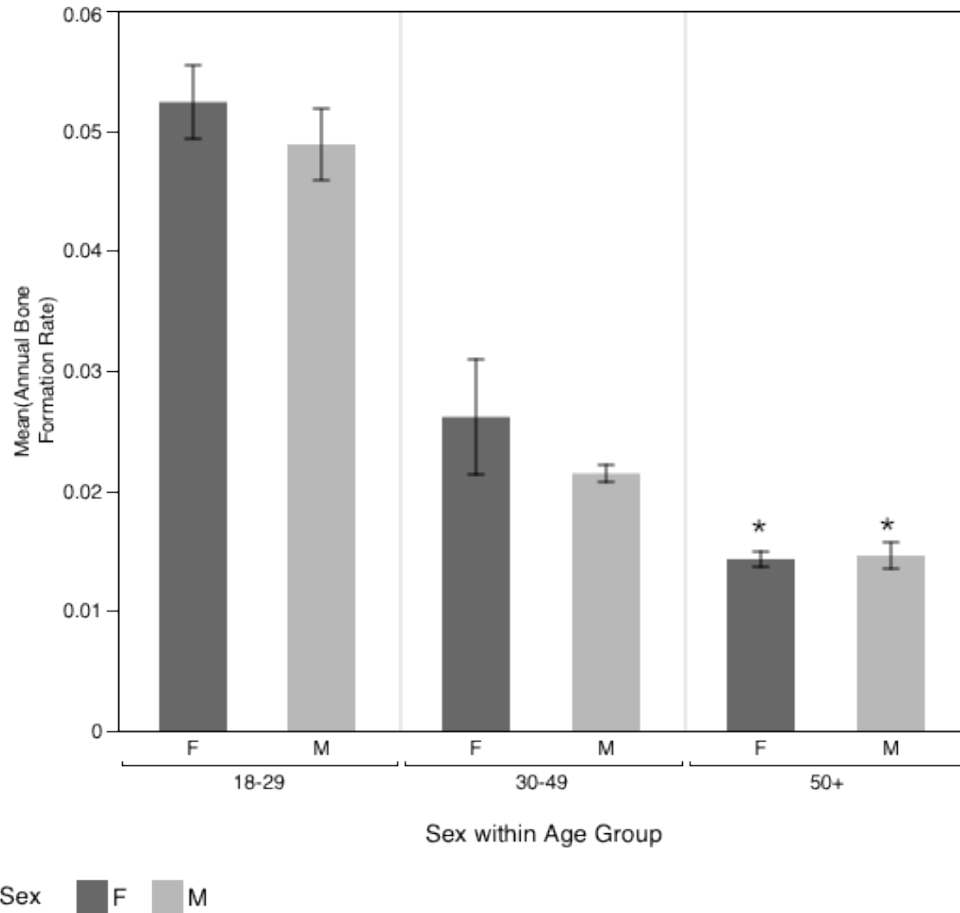
**Figure 27** – Activation frequency by age and sex. Y axis (Mean annual activation frequency  $\#/\text{mm}^2/\text{year}$ ); X axis (age and sex); \* indicate significant differences with age in males (Kruskal-Wallis); \*\* indicates that all three ages are significantly different from each other for females (ANOVA); error bars represent the standard error of the mean.

**Table 41** – Activation frequency ( $\#/mm^2/year$ ) by age and sex for the Velia and Isola Sacra populations

	Isola Sacra		Velia	
Age Group	Mean	SD	Mean	SD
<i>Females</i>				
18-29 yrs	1.5	0.2	1.69	0.008
30-49 yrs	0.9	0.2	0.83	0.32
50+ yrs	0.9	0.2	0.52	0.005
<i>Males</i>				
18-29 yrs	1.3	0.2	1.68	0.007
30-49 yrs	0.9	0.07	0.73	0.01
50+ yrs	0.6	0.1	0.52	0.007

(Values for the 30-49 age group for Isola Sacra are derived from averaging the reported 30-39 and 40-49 age groups)

This is most likely because osteon size differs very little between the sexes on average. Although no significant sex differences were noted for Velia,  $V_{f,r,t}$  was slightly higher in females than in males in both young and middle age. In old age,  $V_{f,r,t}$  in males is just above the female mean. The Velia population as a whole presents a  $V_{f,r,t}$  that is low compared to archaeological and modern groups (Table 40). The mean value for the Velia population is 0.025 ( $mm^2/mm^2/year$ ), while the next closest are from Isola Sacra and modern African Americans who both have a  $V_{f,r,t}$  of 0.030 ( $mm^2/mm^2/year$ ) (Cho and Stout, 2003).



**Figure 28** – Bone formation rate by age and sex. Y axis (Mean annual bone formation rate mm<sup>2</sup>/mm<sup>2</sup>/year); X axis (age and sex); \* indicates that all three ages are significantly different from each other for both sexes (ANOVA); error bars represent the standard error of the mean.

Closer comparisons between Velia and Isola Sacra for bone formation rate present some interesting findings (Table 42). Values from Velia are rounded to reflect the two decimal places reported by Cho and Stout (2003). In females,  $V_{f,r,t}$  is higher at Isola Sacra than Velia in young and old age, but not middle age. The difference is particularly pronounced in young age, where females from Isola Sacra were depositing nearly twice the osteonal bone than females at Velia. For males,  $V_{f,r,t}$  is much more consistent across age groups, with the Isola Sacra males showing more bone being deposited in middle but not young or old age. Young males at Velia had a higher  $V_{f,r,t}$  than those from Isola Sacra and results for old males were equal between the sites.

Net osteonal remodeling ( $_{net}V_{f,r,t}$ ) measures remodeling independent of age and provides an estimate of total remodeling over an individual's life. The Velia population mean for  $_{net}V_{f,r,t}$  (0.569 mm<sup>2</sup>/mm<sup>2</sup>) was the lowest among the groups compared (Table 40), but was extremely close to the value reported for the Kulubnarti population (0.585 mm<sup>2</sup>/mm<sup>2</sup>). Net osteonal remodeling at Isola Sacra (0.720 mm<sup>2</sup>/mm<sup>2</sup>) was elevated compared to Velia, but net osteonal

remodeling in a sample of modern European Americans was nearly twice that of Velia (1.01 mm<sup>2</sup>/mm<sup>2</sup>).

The use of mean wall thickness, or MWT, is common in clinical studies (e.g. Anderson, 1982; Malluche and Faugere, 1986) but infrequent in bioarchaeology. Malluche and Faugere (1986) emphasized that the mean wall thickness (MWT) of osteons is an important measure of osteoblast life spans and/or of bone formation rates. MWT thickness changes significantly with age in females, with statistical difference between old females and the young and middle age groups. This finding is consistent with the observation of Martin and Burr (1989), who found that in modern individuals over 40, osteoblast function and recruitment begins to decline. Interestingly, MWT remained unchanged with age in males, perhaps suggesting that no imbalances in osteoblastic activity were present, or that they were not severe enough to be detected by the methodology. No sex differences in MWT were detected, but females had slightly elevated MWT compared to males in young and middle age. By old age, MWT was equal in both sexes. Archaeologically, MWT is only reported in a Pecos Indian population (Burr et al., 1990), but unfortunately the study is not comparable because the authors used femoral samples. As Pfeiffer et al. (2006) have shown, femoral osteons tend to be larger than those in the rib, and as MWT is partially dependent on osteon size, comparing MWT between ribs and femurs would be misleading.

**Table 42** – Bone formation rate (mm<sup>2</sup>/mm<sup>2</sup>/year) by age and sex for the Velia and Isola Sacra populations

	Isola Sacra		Velia	
Age Group	Mean	SD	Mean	SD
<i>Females</i>				
18-29 yrs	0.1	0.003	0.05	0.008
30-49 yrs	0.03	0.01	0.03	0.014
50+ yrs	0.02	0.004	0.01	0.002
<i>Males</i>				
18-29 yrs	0.04	0.004	0.05	0.004
30-49 yrs	0.03	0.002	0.02	0.003
50+ yrs	0.02	0.002	0.02	0.004

(Values for the 30-49 age group for Isola Sacra are derived from averaging the reported 30-39 and 40-49 age groups)

The remodeling patterns in the Velia sample highlight the uniqueness of the population in many ways. Mean rib osteon sizes were normal, but on the smaller end of what has been reported archaeologically. The density of osteons per mm<sup>2</sup> was also low, including the rate at which they were formed per year. What the results from bone osteon size and formation rate show is that in addition to a lower activation of new osteons per year, less osteonal bone was formed during this turnover. As such, bone remodeling, on average, was reduced compared to a number of modern and archaeological populations, including Isola Sacra, which based on historical and bioarchaeological reconstructions, was likely similar to Velia in many regards. Finally, Haversian canal size did not increase significantly with age ( $p = 0.137$ ) and there are no significant sex differences as well. It is expected that Haversian canal size would increase significantly with age as osteoblast function declines and less bone is filled in after each remodeling event (Mundy, 1995).

There are a number of key implications relating to the remodeling dynamics of the Velia population. Low OPD values for Velia do not pose much of a concern, as lower OPD values are expected in archaeological populations (Mulhern, 2000). Stout and Lueck (1995) have argued that the common observation of lower OPD in archaeological groups may be related to a longer growth period and that skeletal maturity is reached much later than when compared to modern populations. Stout and Lueck (1995) hypothesize that with lower OPD, activation frequency and bone formation rate would be lower as well, given that the age-dependent constant for the ‘effective age of adult bone’ is 12.5 and based on modern samples. For the Velia sample combined, both activation frequency and bone formation rate were indeed low, suggesting that growth was prolonged at Velia, perhaps due to physiological stress during childhood. The high prevalence of dental enamel hypoplasias in adults and the skeletal growth profiles from children aged 3 months to 12 years strongly support a pattern on physiological stress during growth (see *Adult* and *Subadult Stress* sections).

Frost (1987c) and others (Parfitt, 2003; Schoneau et al., 2003; Rauch, 2005; 2007) have also noted the important connection of mechanical usage to bone health, particularly in growth and development. An older ‘effective age of adult bone’ could be a product of extended modeling during growth and development, increasing the amount of bone gained during childhood and adolescents (Mulhern, 2000). This can be seen as an adaptation to a very mechanically active lifestyle (Mulhern, 2000), particularly during important growth years. As Frost (1987c) has shown, increased activity during growth would extend the modeling period, and if physical strains were high for a population, this would change the ‘effective age of adult bone’. High levels of activity at younger ages may also help explain lower remodeling parameters later in life. Parfitt et al. (1997) have argued that the development of more bone early in life could result in reduced levels of fatigue and bone micro-damage later in life, which would require less remodeling to repair micro-cracks and overall lower bone turnover rates.

The remodeling dynamics outlined by Frost (1987c) and Parfitt et al. (1997) mesh very well with what is observed in the Velia population. In young adults (18-29), Velian females (55.27 mm<sup>2</sup>) and males (86.24 mm<sup>2</sup>) had ribs with larger cross sections than those of Isola Sacra females (51.0 mm<sup>2</sup>) and males (85.6 mm<sup>2</sup>). The mean for the whole Velia sample was 69.71 mm<sup>2</sup>, which was higher than Isola Sacra (65.73 mm<sup>2</sup>), as well as samples from modern African (62.83 mm<sup>2</sup>) and European (69.51 mm<sup>2</sup>) American populations (Cho and Stout, 2003). Furthermore, cortical index in the metacarpals was also quite high in young age for both sexes,

substantially surpassing cortical index values from Ancaster and Wharram Percy (see Table ? in *Radiogrammetry* section above). At the tissue level, activation frequency in young age was higher in the Velia sample than at Isola Sacra (see Table 41). Since activation frequency is highest in growing individuals (Frost, 1987c), it seems plausible that a number of the individuals in the 18-29 age group at Velia would be experiencing a longer growth period. The combination of high activation frequency and evidence for high bone mass (in metacarpals) in the 18-30 age group strongly suggests that growth was prolonged in the Velia population, and may also help explain why subsequent remodeling dynamics later in life are reduced, if the observations made by Frost (1987c) and Parfitt (1997) are considered.

Mulhern (2000) has also noted that the minimum effective strain (MES) set point required to repress bone remodeling should also be considered when comparing remodeling dynamics between populations. If strains are repeatedly applied above the MES, activation frequency and bone formation rates are repressed (Frost, 1987c). Conversely, if disuse of bone falls below a set threshold, remodeling rates increase but do not fully replace the bone that is resorbed in order to remove bone that is not being used (Frost, 1987c; 2003). Frost (2003) adds that much of the bone lost during disuse remodeling would occur from trabecular or endocortical regions. This can be seen in the values for activation frequency and rib endosteal area in modern European Americans, who have high values for both (Cho and Stout, 2003). In the Roman context (see Chapter 4), there is substantial historical evidence that substantial day-to-day physical activity would have been the norm for many individuals, including children and adolescents. The prevalence of Schmorl's nodes, which are circular or linear depressions in the vertebrae created by herniated disks (Kelley, 1982; Mann & Murphy, 1990; Ortner & Putchar, 1981), was also very high for Velians. For females, the prevalence was 36%, and for males 73.53%. This is quite substantial, as contemporary Romans from the site of Urbino, who show other extensive pathologies induced from manual labor, commonly had Schmorl's nodes as well (Paine et al., 2009). At Urbino, the prevalence for Schmorl's node in females was 42%, slightly higher than at Velia, but in males it lower, at 38% (Paine et al., 2009). Although not collected for this dissertation, signs of labor-related degenerative joint disease was also quite high at Velia (pers.comm, Bondioli, 2011). What the historical and skeletal data show is that activity, and subsequently repeated strains on the skeleton, was likely quite elevated for the Velia population. This constant strain may have surpassed the MES required to suppress activation frequency and bone formation rates in many individuals, explaining these low remodeling parameters in the Velia population. This may also help explain some of the differences between Velia and Isola Sacra. As mentioned in the *Radiogrammetry* section, the people of Isola Sacra represented an arguably more cosmopolitan, middle class and urban community than Velia. This may have led to a greater diversity of occupations that what would have been found at Velia, many of which would have require less physical labor. The reduced remodeling at Velia may be a sign of these social differences between the communities if physical activity was indeed more demanding for Velians.

While these hypotheses explaining the low remodeling rates in the Velia population have merit, there are some limiting factors. First, the rib is not representative of a more physically active skeletal element like the femur. In order to fully test the role physical activity may have had on bone mass, OPD, osteon size, activation frequency and bone formation rate, future histomorphometric tests should be conducted in femoral and humeral samples and explored to see if the trends observed in the ribs still hold. The rib is subject to continuous low strains at high

frequency, and this should result in higher remodeling rates in the rib than in the femur in order to repair accumulated micro-cracks (Sobelman *et al.*, 2004; Martin *et al.*, 1998). However, Robling and Stout (2003) have observed higher remodeling rates in the femur and hypothesized that high strains were responsible. Cho and Stout (2011) note that sampling methodology, particularly in the femur, can affect results, so there is still some debate as to whether rib remodeling is always more advanced than in the femur. The rib remodeling dynamics from Kulubnarti (Mulhern, 2000) were quite similar to Velia in many regards (see Table 40) and in that study remodeling in the ribs was also compared to femoral remodeling. At Kulubnarti, evidence of increased physical activity through femoral remodeling is consistent with remodeling in the rib, increasing the validity that rib remodeling might act as a good general proxy for general physical activity throughout the body if femoral or humeral thin section are not taken. It is also important to consider that while daily physical activity was undoubtedly high at Velia, it is impossible to assess whether or not activity was substantially lower than in other archaeological populations, all of which also depended on manual labor for survival. For example, the Native American sites of Ledders, Gibson and Windover all represent very active societies, but their remodeling is considerably elevated compared to Velia (Stout and Lueck, 1995). In comparing these sites on the basis of physical activity, it is important to remember that a number of aspects of bone remodeling are still not fully understood (Turner, 1999; Stout and Crowder, 2012) and do not fit the paradigm set out by Frost (1987c). For example, Frost's (1987c) account of remodeling dynamics works very well for load bearing bones, but struggles to account for why non-load bearing bones (e.g. some cranial bones) do not completely remodel out if they are much more "disused" (Turner, 1999). Turner (1999) has argued that MES set points are not universal in the body, but must vary by region. Turner (1999) also argues that bones have cellular memories, so to speak, of earlier life events, so that events during development and growth have life-long effects. Turner (2000) also adds that endocrine and paracrine effects should be explored further as central figures in bone formation and maintenance, whereas Frost (1987c) sees them as secondary. Stout and Crowder (2012) state that a central 'unified theory' of bone remodeling is still needed, but will probably incorporate aspects of Frost's (1987c; 2003) mechanostat hypothesis, Turner's cellular accommodation theory (1999) and the role osteocytes play in altering osteoclast activity in response to strain (Burr, 2002). What this means for archaeological interpretations of remodeling activity is that concrete answers are difficult to produce. For example, while the remodeling dynamics between Velia and the Kulubnarti population may be similar to due a shared high level of physical activity, strenuous exercise was also common in other archaeological populations that show much higher remodeling rates (Stout and Lueck, 1995). Dietary conditions are important (Paine *et al.*, 2009) and also need to be considered. These differences in archaeological populations highlight the need for a better understanding of the interplay between mechanical usage, diet, hormones and the function of bone at the cellular level (Crowder and Stout, 2012).

As mentioned previously, malnutrition at Velia may also help explain reduced remodeling activity, as chronic under nutrition can lower osteon population densities (Paine *et al.*, 2009), although Frost (1987c) has argued that malnutrition can increase remodeling rates if the problem is severe enough. As Garnsey (1998) has noted, starvation was likely not common in the Roman world (although periodic shortages and under-nutrition were), so the findings of Paine *et al.* (2009) are probably more relevant. If starvation, muscle wasting and disuse of the skeleton were common, remodeling increases under these conditions should have been noted for Velia.

Velia was a port city and the possibility exists that this was a mixed population and so the results for the sample may represent the product of individuals with mixed genetic and ethnic backgrounds. However, as mentioned in Chapter 4, the Velia community commonly buried their dead in single internments, which is unusual for the period, and may indicate that the Velians in question were from a small tight-knit ethnically related community (pers.comm, Bondioli, 2011), who may not be genetically diverse. Finally, sample size was moderate as well, and the available sample may have a small bias towards lower remodeling rates.

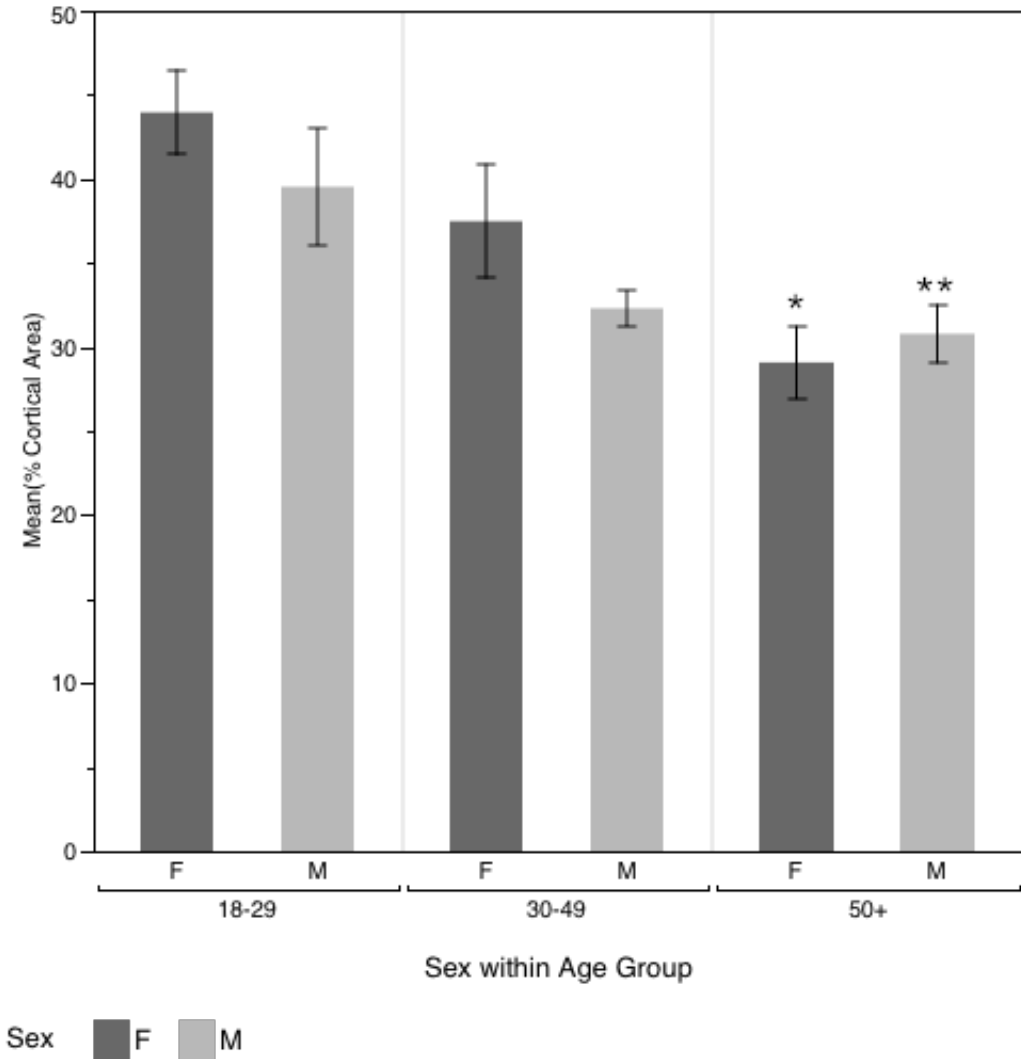
### *Macrostructural Level*

This analysis of intra-cortical remodeling in the Velia population has shown to be quite valuable, but changes at the macroscopic level are informative and important as well. Both sexes had significant decreases in percent cortical area (Ct.Ar/Tt.Ar) with age. Sex differences were present for total area, endosteal area and cortical area, but not for percent cortical area, indicating that the sex differences were related to overall sexual dimorphism, given that when controlled for body size, the amount of cortical bone present in the ribs did not differ significantly (Figure 29). Some of the more interesting factors of percent cortical area in the ribs emerge when compared to the cortical index in the second metacarpal.

In both the rib and the metacarpal, significant decline in percent cortical bone were noted. Between-sex comparisons also yielded similar results, with no sex differences observed. However, closer investigations do reveal some key differences in bone loss between the skeletal regions. Explorations of bone loss using the Meema and Meema (1987) method, which labels individuals as abnormal if their cortical index falls below two standard deviations of the young adult mean, found fairly consistent results between the bone sites across age and sex. The one large exception is for old females, who had twice the amount of individuals with abnormal bone (Meema and Meema, 1987) in the metacarpal versus the rib. At first glance this appears odd, given the more biomechanically involved nature of the metacarpal one would expect ribs to have advanced bone loss over the hand. Looking at the percentage of young bone retained with age (following Mays', 2006 approach), this is in fact what is observed. The second metacarpal retains more bone into old age than the ribs in both sexes and for middle and old aged adults. Furthermore, when using Velian individuals with matching measures only, the observation that bone is retained longer in the metacarpals is supported. Figure 18 in Chapter 6 illustrates that bone loss in the metacarpal occurs predominantly in old age (50+), while in the ribs cortical bone loss occurs earlier (30-49 age range). Interestingly, the values in Table 14 (Chapter 6) suggest that the majority of this difference is coming from middle-aged males, where there is a 15.3% difference between bone lost in the metacarpal versus the ribs. It is unclear why a 15.3% difference in relative cortical bone in the ribs would occur in males between the young and middle age groups. In the metacarpal, percent cortical bone only declined around only 4% in males. There are two outliers in the 30-49 age group for rib percent cortical area in males, but they are actually above the mean and do not contribute to a lower relative cortical area. The males used in this study showed no signs of metabolic bone disorders, so pathology is unlikely. Ultimately, the drastic drop in rib relative cortical area cannot be easily explained beyond



random variation produced through a cross-sectional analysis or potentially through skeletal age-determination bias.



**Figure 29** – Percent cortical area (Ct.Ar/Tt.Ar) by age and sex in the rib. Y axis (mean percent cortical area); X axis (age and sex); \* indicates a significant sex difference between old and young adult in females (ANOVA); \*\* indicates a significant difference with age in males (Kruskal-Wallis); error bars represent the standard error of the mean.

Comparing changes in the amount of relative cortical present between ribs and the second metacarpal is also useful as the ribs represent a more metabolic baseline in the body, while cortical index in the metacarpals is additionally affected by mechanical usage. In the metacarpal, no significant age changes were noted for medullary width in males, while in females a significant change was noted, but only for the old age group. In the ribs, no significant age changes were noted for endosteal area (analogous to medullary width in metacarpals), although endosteal area did increase more in females in old age. These trends at the endosteal envelope suggest that some important differences between males and females may be occurring in post-menopausal years (50+). To explore this further, the percentage of bone retained from middle to old age in both sexes is examined. In the metacarpals, close to 20% of cortical bone is lost between middle and old age in both sexes (see Table 14 in Chapter 6). In contrast, the amount of cortical bone in ribs drops by 19% in females from middle to old age, but only around 4% in males. The drop in the percentage of young adult bone mass retained, as well as increasing endosteal areas in older female ribs suggests that bone loss is advanced in females due to hormonal shifts caused by menopause. However, while medullary width in the second metacarpal changed significantly across age groups in females and not in males, both sexes retained roughly the same amount of that they had in young adulthood. Furthermore, no statistical sexes differences were noted for either cortical index in the metacarpals, or percent cortical area in the ribs at the 50+ age range (or any age group). In metacarpals, the more dramatic increase in medullary width in females seems to be partially offset by a positive change (though not statistically significant) in total bone width across age groups (primarily between young and middle age), which males did not have. Similarly, female total cross sectional area in the ribs increased across age groups, but did not do so in males. Periosteal expansion is expected with age, but primarily in older individuals (Maggio et al., 1997; Böttcher et al., 2006). The fact that metacarpal total bone width in females increased most from 18-29 to 30-49 and that mean total cross sectional area in the ribs increased across age groups, suggests that strenuous activity was present throughout the life course in both sexes and may have helped mediate bone loss, considering the more pronounced endosteal changes in females. As noted in the *Radiogrammetry* section above however, these data are estimations of true cortical thinning and thickness as this is a cross-sectional study and the possibility of group and survival effects exists. Nevertheless, post-menopausal bone loss in females was not worse than in males based on cortical index and percent cortical in ribs, and so some mediating factor, most probably a life of strenuous labor, helped mitigate advanced bone loss in women. Hormonal changes related to reproduction may have also helped retain bone mass with age in females and is an important consideration as well (see *Radiogrammetry* section).

The remodeling dynamics of cortical bone in ribs between Velia and Isola Sacra differ in a few important ways (see above), and this is translated to the macroscopic level as well. In terms of relative (percent) cortical area, males and females from both sites hardly differ. In females, the mean for percent cortical area at Velia is 37.12%, while at Isola Sacra it is 35.52% (Cho and Stout, 2011). For males, the Velia mean is 32.70% and 34.80% for Isola Sacra (Cho and Stout, 2011). It is in endosteal area, cortical area and total area where the sites differ more. Total area in females at Isola Sacra is 49.69 mm<sup>2</sup>, but 55.81 mm<sup>2</sup> at Velia. In males the differences are even more pronounced, with Velians (80.24 mm<sup>2</sup>) producing substantially larger ribs on average than at Isola Sacra (68.31 mm<sup>2</sup>). Mean values for cortical area and endosteal area were also larger at Velia (see Table ?). What these data signify is that ribs were larger at Velia (on average), that more absolute cortical bone was present and that the bone was distributed

farther away from the center of the rib (larger endosteal areas). When controlled for body size, the percentage of cortical bone was higher in females at Velia, but slightly lower in males. One prediction based on the rib remodeling would be that endosteal area would be lower at Velia than at Isola Sacra (on average) because remodeling was less active in the Velian samples. The contrary finding was found, but this seems to have more to do with body size, as Velian ribs were much larger as a whole, and so a larger endosteal area is not surprising. If percent cortical area is considered, which controls for body size, the prediction holds for females at Velia, but not for males, who had less relative cortical bone than males from Isola Sacra. One confounding factor in this analysis is that people from the Isola Sacra necropolis represented individuals from across the Empire (Cho and Stout, 2011), while Velians were probably more local and ethnically similar. Population differences might be confounding this interpretation based on rib size by introducing greater size variability in the Isola Sacra group. Further, Cho and Stout (2011) only present their data as means for grouped males and females, so age-related changes cannot be compared to Velia.

Ultimately the macro-level analysis is consistent with what was observed at the tissue level in the Velia sample. More cortical bone was present in the Velian ribs, but differences in body size may be a confounding factor. As noted previously, endosteal area increased with age for each sex at Velia, but these changes were not statistically significant and occurred in old age, which suggests that the lower remodeling rate at Velia was partially activity induced and helped retain bone mass with increasing age. Comparisons of cortical bone changes between metacarpals and ribs support this as well. In order to better understand the cortical remodeling in both metacarpals and ribs, it would now be useful to examine changes in trabecular bone, as trabecular is more sensitive to physiological demands and this might provide some context about changes in bone throughout the body as a whole across the life course.

## **Trabecular Architecture**

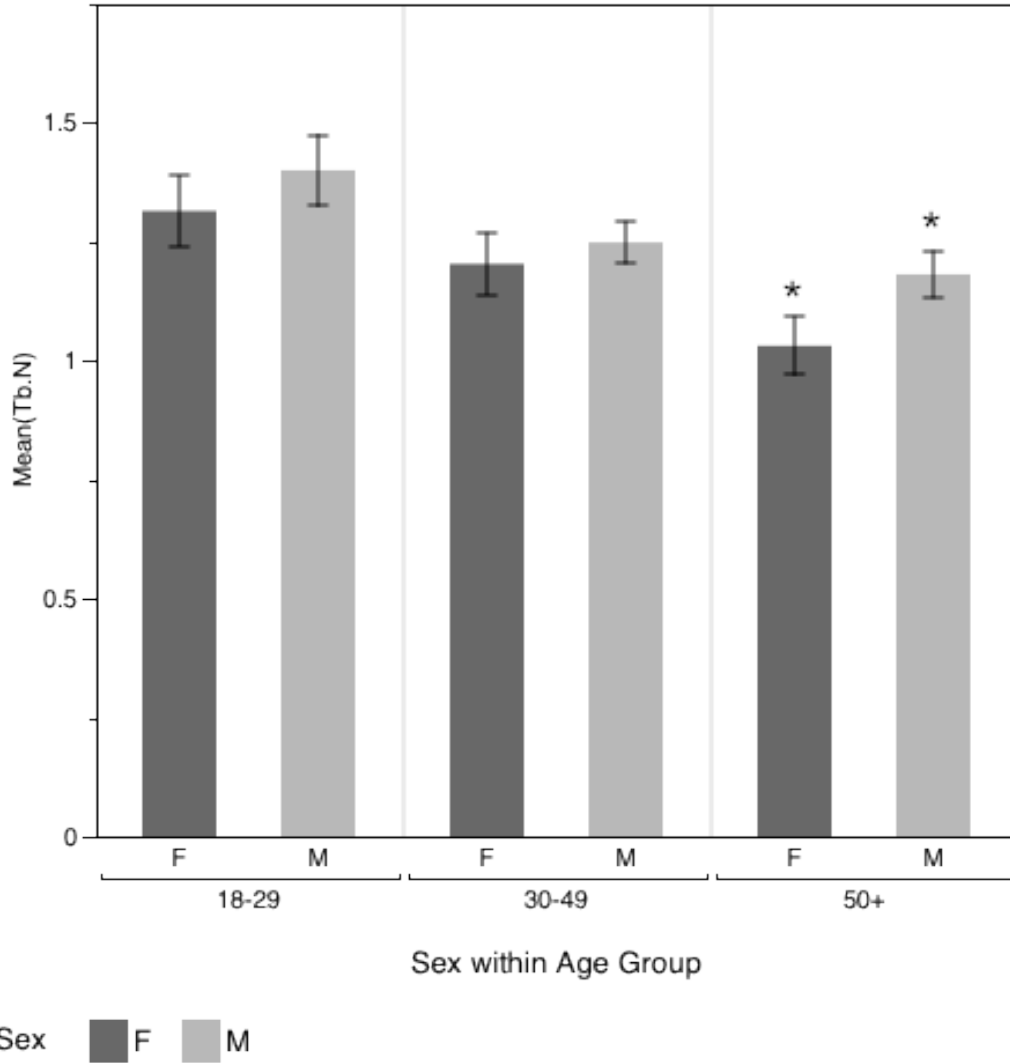
### *Adult Trabecular Bone Maintenance and Loss*

The analysis of trabecular bone in bioarchaeology has become an important part of interpreting bone loss in the past (Agarwal, 2008), in large part due to advancing clinical work that increasingly places emphasis on understanding how the organization and connectedness of trabecular bone affects bone strength and fracture risk (Burr and Turner, 1999; Cooper, 1993; Watts, 2002; Grynopas, 2003). Trabecular bone also has the advantage of being more metabolically sensitive (Brickley and Agarwal, 2003), and thus may provide a picture of bone turnover on a shorter time scale than what can be perceived using cortical bone. Contrasted with the findings from the *Radiogrammetry* and *Histomorphometry* sections above, more complete picture of bone health at Velia can emerge.

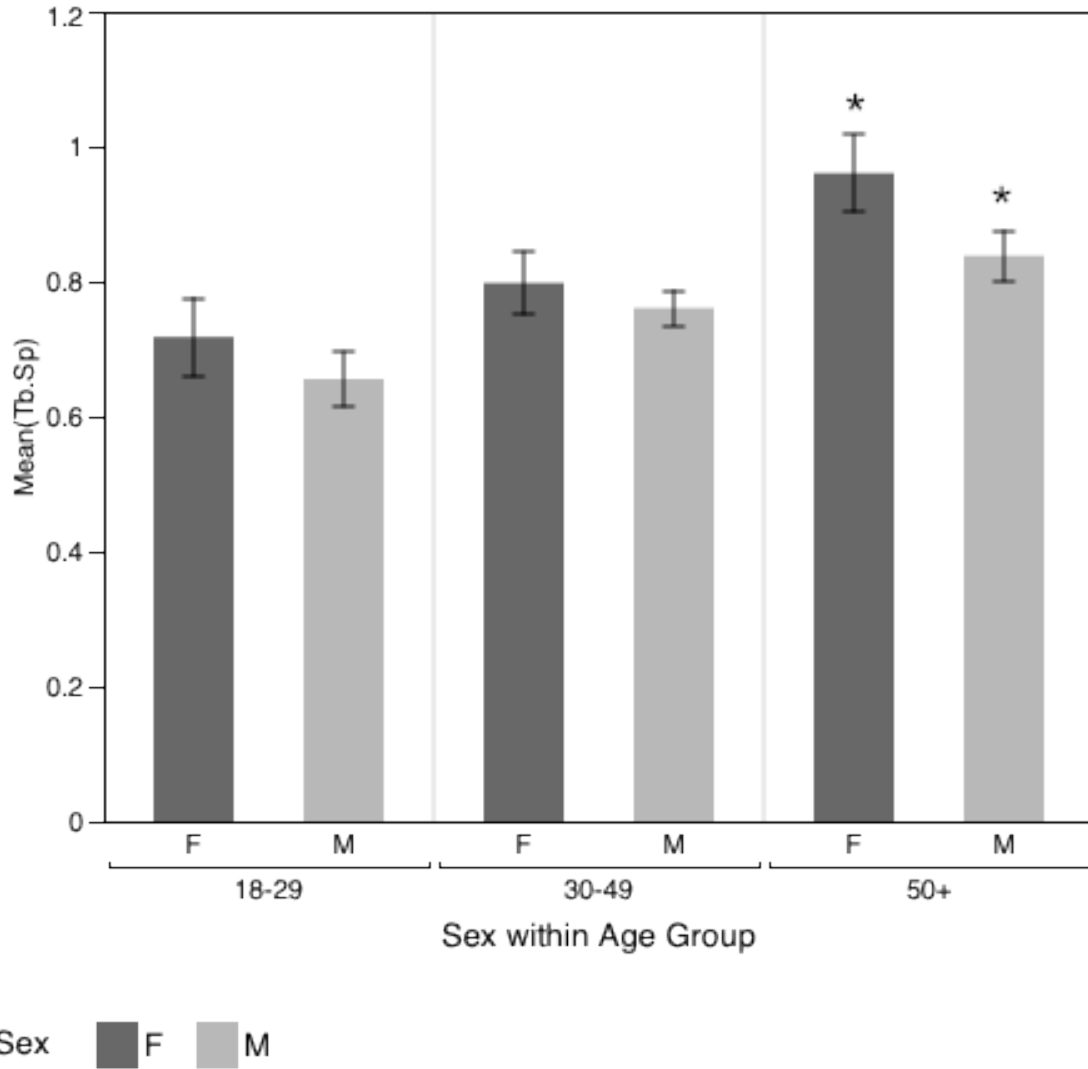
Changes in trabecular bone at Velia are similar to modern populations in many regards, but some trends do show some important diversions. The number of trabeculae (Tb.N) (Figure 30) was significantly different between age groups in both sexes. Conversely, the space between trabeculae (Tb.Sp) (Figure 31) changed positively and significantly with age in both sexes,

showing an age-related loss of Tb.N. This is expected based on findings of modern (Twomey et al., 1983) and archaeological investigations (Agarwal, 2008) as trabecular number and spacing are strongly negatively correlated. The connective density (ConnD) in trabeculae shows an age-related change for both sexes (Figure 32), indicating that as individuals aged, the trabeculae became increasingly disconnected from each other. Sex differences in each age group were not present for either Tb.N or Tb.Sp, but for the ConnD measure, a significant sex difference was found for both young and old age groups. In fact, ConnD is the only measure of trabecular architecture where any significant sex differences were found. The importance of this finding is addressed further below.

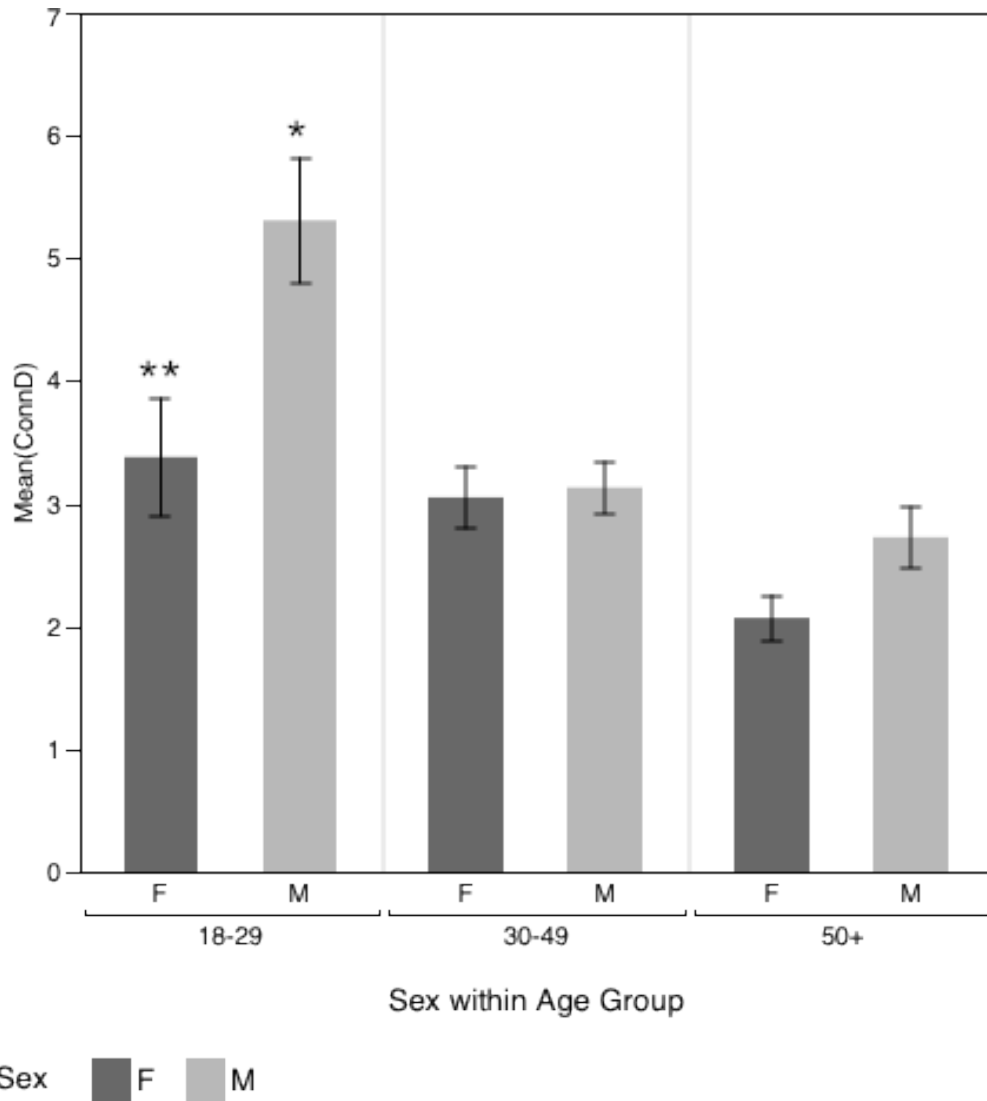
The structural model index, or SMI, measure showed that while trabeculae did shift from a more plate-like to rod-like model of bone, this change was not significant with age (Figure 33). It is difficult to directly compare the SMI findings in the Velia population to those of modern populations, as the methodologies and skeletal sites often differ. However, the role SMI plays in vertebral bone strength has been well identified (Fields et al., 2009; Roux et al., 2010). Fields et al. (2009) found that when measures of bone mass are controlled for, SMI (along with trabecular thickness) is strongly predictive of whole bone vertebral strength. Roux et al. (2010) found that along with bone mineral density, SMI and Tb.Th are strongly predictive of fracture risk. Sornay-Rendu et al. (2009) have also shown that in individuals with a history of fracture, trabecular architecture was significantly different than in controls without fractures. The Sornay-Rendu et al. (2009) study used limb bones, but the point that trabecular architecture contributes to whole bone strength remains. Duan et al. (2001) noted that forward bending motions can create compression forces on vertebrae that are 10-fold than those from standing upright. In the Velian context, manual labor (e.g. dock worker), agriculture and fishing would have necessitated repeated forward bending. Given that parameters of trabecular architecture are strongly related to biomechanical behavior (Fields et al., 2009), the SMI values for Velia suggest activity was high, and that this may have helped retain a more plate-like structure in vertebral trabecular bone with age than we see in modern groups who are far less physically active on average.



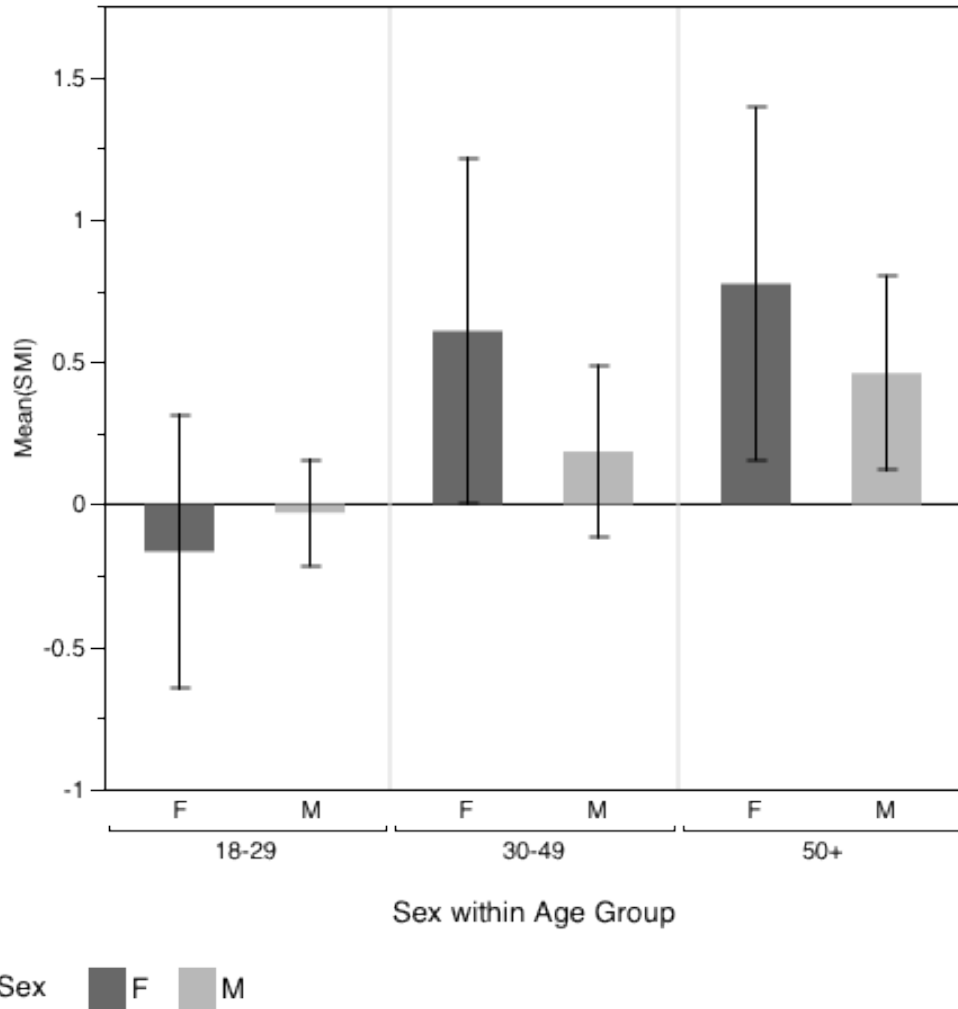
**Figure 30** – Trabecular Number (1/mm) by age and sex. Y axis (trabecular number); X axis (age and sex); \* indicates a significant sex difference between old and young adult in both sexes (ANOVA); error bars represent the standard error of the mean.



**Figure 31** – Trabecular Spacing (mm) by age and sex. Y axis (trabecular spacing); X axis (age and sex); \* indicates a significant sex difference between old and young adult in both sexes (ANOVA); error bars represent the standard error of the mean.

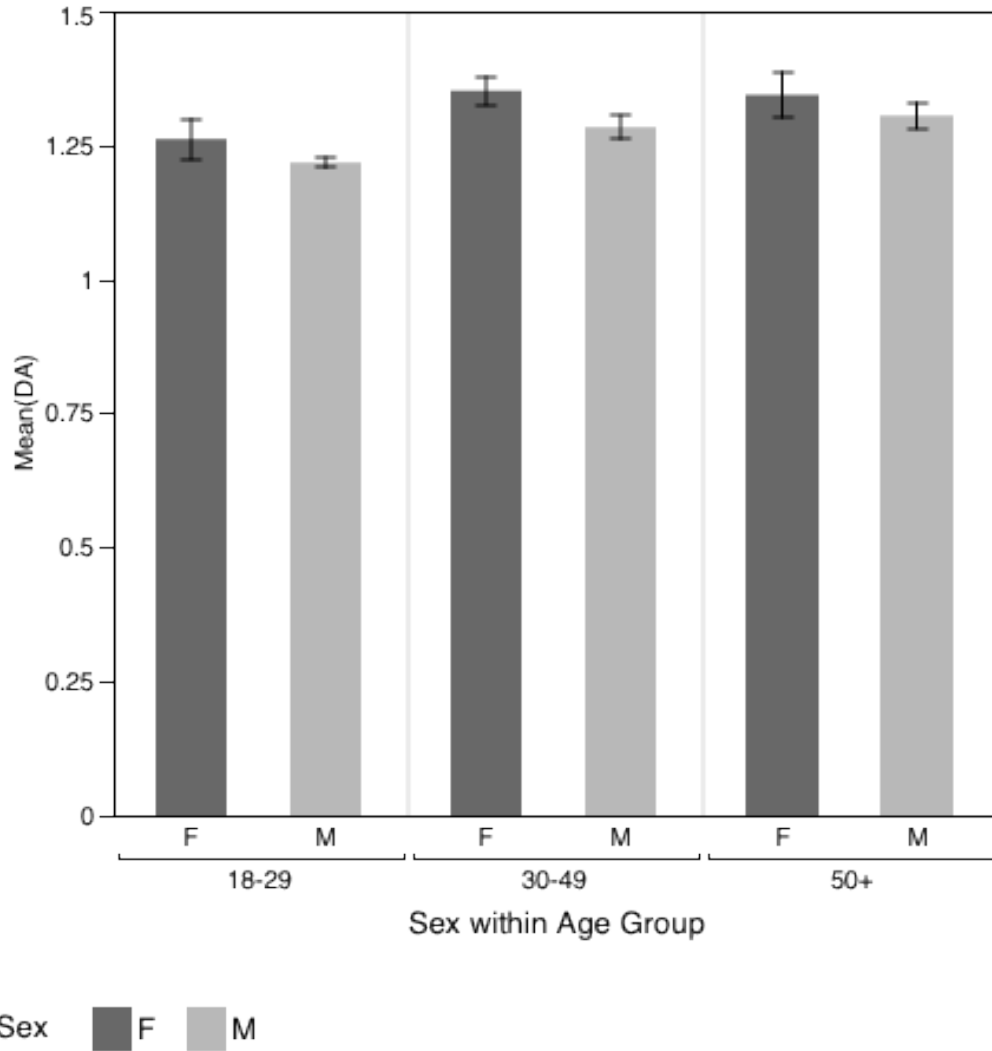


**Figure 32** – Connective Density ( $\text{mm}^{-3}$ ) by age and sex. Y axis (connective density); X axis (age and sex); \* indicates a significant difference between the young and both middle and old age groups in males (ANOVA); \*\* indicates a significant age-related difference in females (Kruskal-Wallis); error bars represent the standard error of the mean.

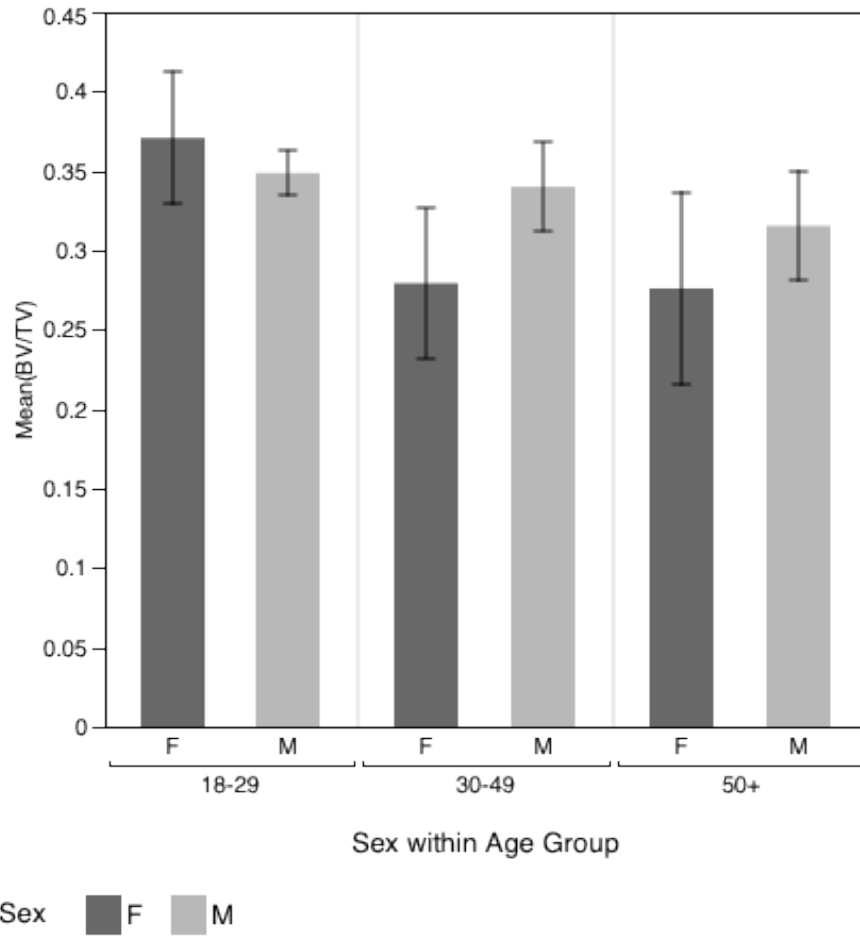


**Figure 33** – Structural Model Index by age and sex. Y axis (structural model index); X axis (age and sex); error bars represent the standard error of the mean.

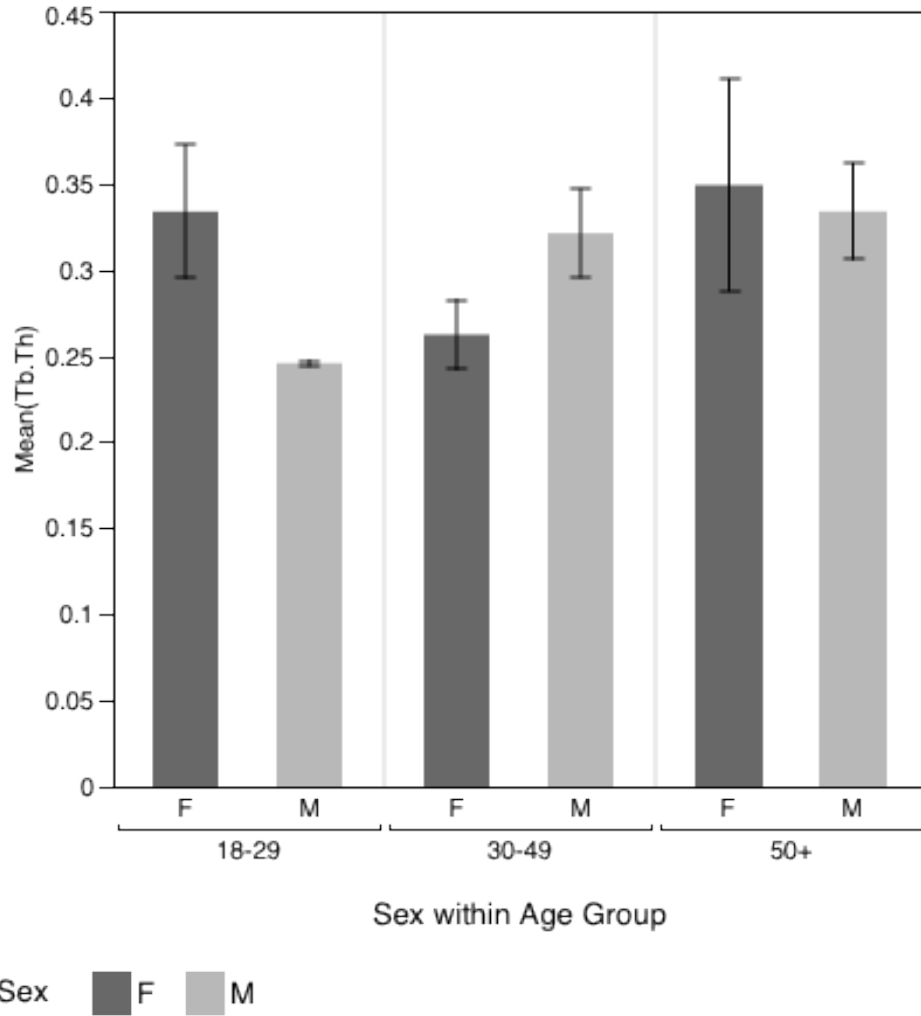




**Figure 34** – Degree of Anisotropy with age and sex. Y axis (degree of anisotropy); X axis (age and sex); error bars represent the standard error of the mean



**Figure 35** – Bone volume by age and sex. Y axis (bone volume); X axis (age and sex); error bars represent the standard error of the mean



**Figure 36** – Trabecular Thickness (mm) by age and sex. Y axis (trabecular thickness - mm); X axis (age and sex); error bars represent the standard error of the mean

Degree of anisotropy (DA) examines the changing direction of trabeculae with age (Njeh et al., 1999), and has also been shown to play a role in bone strength (Fields et al., 2009). As vertebral trabecular bone ages, the organization of trabeculae change from a more isotropic state (equal strength in all directions) to more anisotropic one (Snyder et al., 1993). Anisotropy is considered an important part of bone strength, but DA alone is a poor predictor of biomechanical activity and fracture (Wegrzyn et al., 2010). However, when combined with BV/TV and SMI, the three parameters explained 86% of the biomechanical variability and properties of the L3 vertebrae examined by Wegrzyn et al. (2010). In an early study of anisotropy in the spine, Snyder et al. (1993) hypothesized that vertical trabeculae would be retained over horizontal ones as compressive forces were maximal through the vertical (superior/inferior) plane. Surprisingly, Snyder et al. (1993) found the opposite in the vertebral elements they examined. More recent work has challenged the Snyder et al. (1993) findings and has shown convincingly that vertical trabeculae in vertebrae are preferentially kept over horizontal struts to maintain compressive strength (Nicholson et al., 1997; Thomsen et al. 2002; Wegrzyn et al., 2010). Methodological issues most likely influenced the Snyder et al. (1993) findings as they used small cubes of bone and did not look at trabecular organization across the whole bone. The clinically observed pattern of increasing DA with age was observed in the vertebrae from Velia as well, with DA changing in a positive direction across age groups, although the differences were not significant (Figure 34). The only current bioarchaeological report to examine DA in L4 vertebrae (or any vertebrae) is by Agarwal et al. (2004). In their study of Medieval peasants from Wharram Percy, the authors found that DA increased across the age groups, but like Velia, these differences were not significant. The values for DA, SMI and ConnD cannot be easily compared to published archaeological reports, as no study has yet examined these parameters together in vertebral bone in an archaeological context.

Of all the changes in trabecular architecture at Velia, perhaps the most interesting trend is that for both sexes, bone volume (BV/TV) did not decrease in a statistically significant way with age in either sex (Figure 35). This is unexpected, as clinical studies have show BV/TV to decrease significantly with age, particularly in old age (Bergot et al., 1988; Riggs et al., 2008). BV/TV values did decrease across age groups however, so bone loss was taking place with age, but at an apparently reduced rate. Furthermore, the lack of sex differences, particularly in old age, is another important departure from the modern expectation that females will lose dramatically more bone than males in post-menopausal years (Seeman, 2002; Riggs et al., 2008) Parsing out the causative factors of bone maintenance and loss in archaeological contexts is difficult (Agarwal, 2008) and multiple lines of evidence are required.

A useful first step is to compare the results from Velia to other past populations. Archaeologically, only a handful of studies using lumbar vertebrae are available (Vogel et al., 1990; Kneissel et al., 1994, 1997; Brickley and Howell, 1999; Agarwal et al., 2004), but unfortunately none utilize the full suite of clinically standardized measures of trabecular architecture outlined in this dissertation. Other studies have examined trabecular bone in alternative skeletal elements (tibiae, femora, radii) or with different methods, such as densitometry (Brickley and Howell, 1999; Brickley and Agarwal, 2003; Agarwal, 2008; Agarwal and Grynypas, 2009; Gosman et al., 2009). The report by Agarwal et al. (2004) on the Medieval British site of Wharram Percy (see *Radiogrammetry* section for cortical bone comparisons) presents the closest parallel in terms of the trabecular architecture parameters examined. Kneissel et al. (1997) report on a Medieval Nubian population, but means for BV/TV are provided only in

graphical form and sex and age specific values are not clearly identifiable. Table 43 summarizes BV/TV between Wharram Percy and Velia.

The BV/TV trends for females between the two archaeological sites are remarkably consistent. In males, the comparison between Velia and Wharram Percy is quite different. In particular, the trends differ dramatically by middle and old age, with BV/TV seemingly retained to a much greater extent in Velian males than males from Wharram Percy. Sex differences were also explored for BV/TV at Wharram Percy and none were found (Agarwal et al., 2004). Agarwal et al. (2004) also noted that in their study that for both sexes, significant age-related changes occurred from young to middle age, and then changed very little into old age. This same pattern was noted for females at Velia (although not statistically significant changes were found). In contrast, BV/TV means for males at Velia barely decreased across age groups. The seemingly early age of bone loss at Wharram Percy, followed by stability of bone maintenance into old age, was seen as unusual, as bone loss was expected in old age (Agarwal et al., 2004). However, more recent clinical work has shown that for both sexes, trabecular bone loss in the radius, tibia and lumbar spine begins in young adulthood and decreases substantially prior to the decline in sex steroids in the sixth decade of life (Riggs et al., 2008). Similar findings were found for the lumbar spine in other archaeological populations as well (Vogel et al., 1990; Kneissel et al., 1997). However in the archaeological populations, although trabecular bone volume declined early in life, the loss of trabecular bone was less severe than in modern populations and it 'stabilized' and did not dramatically worsen into post-menopausal years (Vogel et al., 1990; Kneissel et al., 1997). These archaeological findings mesh well with the findings of Riggs et al. (2008) who strongly affirm that the current clinical paradigms for the pathogenesis of osteoporosis are incomplete. In light of the work of Riggs et al. (2008) bioarchaeological investigations are perhaps well poised to explore a suite of biocultural factors that might improve and refine the pathogenesis of osteoporosis.

Agarwal et al. (2004) examine a number of biocultural factors that might explain the patterns in BV/TV they observed at Wharram Percy. The three primary biocultural factors explored were diet, physical activity and reproduction. These biocultural factors are explored in the *Radiogrammetry* section, but are invoked again here briefly. Agarwal et al. (2004) argued that diet was likely not a major factor in the patterns of trabecular bone loss at Wharram Percy as calcium and other nutrients were likely sufficient, although caloric under nutrition may have been common. Physical activity during the Medieval period was undoubtedly high (Mays, 1996; Agarwal et al., 2004) and Agarwal and colleagues hypothesize that it played an important role in preventing a further decline in BV/TV from middle to old age. Finally, reproductive history is considered and is hypothesized to have also mediated age-related bone loss through increased parity and the protective effect of extended periods of breastfeeding (Sowers et al., 1992; Fox et al., 1993; Cumming and Klineberg, 1993; Murphy et al., 1994; Michaëlsson et al., 2001). The majority of the arguments applied to explaining trabecular bone volume at Wharram Percy (Agarwal et al., 2004) are also applicable to Velia (see *Radiogrammetry* section for an extended argument). Analyses comparing cortical bone differences between Velia and Wharram Percy came to a similar conclusion that physical activity and female reproductive histories played an important role in maintaining cortical bone mass in aging. It is argued here that these biocultural factors also protected trabecular bone from excessive decline between the middle and old age groups, which we would expect based on modern observations (NOF, 2011).

**Table 43** – BV/TV for Wharram Percy (Agarwal et al., 2004) and Velia

	<b>BV/TV</b>	
<b>Age Group</b>	Velia	WP
<b><i>Females</i></b>		
<b>18-29 yrs</b>	0.37± 0.042	0.37± 0.024
<b>30-49 yrs</b>	0.28± 0.047	0.27± 0.029
<b>50+ yrs</b>	0.28± 0.061	0.30± 0.020
<b><i>Males</i></b>		
<b>18-29 yrs</b>	0.35± 0.014	0.38± 0.031
<b>30-49 yrs</b>	0.34± 0.028	0.29± 0.020
<b>50+ yrs</b>	0.32± 0.035	0.26± 0.024

BV/TV (Bone volume); WP (Wharram Percy); ± (refers to standard error of the mean). (Comparison is used to examine general trends; values themselves cannot be directly compared, as the methodologies between the studies were not the same).

One area where the Velia and Wharram Percy populations differ, and where these biocultural explanations may fall short, is in the change observed in BV/TV with age in males. Hormonal changes would not have likely affected males dramatically in the 30-49 age range at either Velia or Wharram Percy. Rather, the seemingly higher retention of trabecular bone volume with age may do the particular day-to-day activities of males in both communities. Males from Wharram Percy would have been primarily involved in agricultural labor (Mays, 1996). Agricultural work was important at Velia as well (Craig et al., 2009), but social complexity and occupations during the Roman period (Aldrete, 2004; Toner, 2002; 2009) were arguably more varied. At Velia, many males would have been engaged as fishermen and dockworkers (Crowe et al., 2010) and perhaps these activities contributed to higher strains on the axial skeleton and spine that could have mediated bone loss. As mentioned previously, prevalence of Schmorl's nodes in males was high (nearly 74%) and evidence for degenerative joint wear in the axial and appendicular regions of the skeleton is also substantial (pers.comm, Sperduti, 2011). Subsequently, a hypothesis that physical activity in males was greater than at Velia is plausible, but tentative only, as evidence from cortical bone loss in the metacarpals provides a contradictory scenario (see *Radiogrammetry* section).

The differences in connective density between the sites also call into question the similarity of biocultural influences between Velia and Wharram Percy. At Wharram Percy, no sex differences in connective density were noted (Agarwal et al., 2004). In the Velia sample, sex differences in connective density were noted for young and old aged adults. Agarwal et al. (2004) hypothesized that reproductive history at Wharram Percy was a significant factor in mediating a loss of trabecular connectivity. Reproductive patterns would have been very similar at Velia (see *Radiogrammetry* section), but connectivity was significantly different between the sexes. One important distinction that should be noted is that the analysis by Agarwal et al. (2004) was two-dimensional (from thick sections of bone), while the analysis of vertebrae at Velia was three-dimensional and from CT. This is an important difference. The methodological weakness of 2-D images is that they mask the complexity of the trabecular network. Odgaard (1997) argued that most 2-D analyses of trabecular bone used variations of a model by Parfitt et al. (1983). The Parfitt model emphasized the thickness and amount of trabeculae over their orientation. The model has contributed to knowledge of normal vs. pathological levels of trabecular thickness, but it did not consider that loss may occur in other forms, such as reduced connectivity or anisotropy (Odgaard, 1997). Kleerekoper et al. (1985) have shown that trabecular architecture, independent of mass, is an important part of trabecular bone strength. If the hypotheses being tested involve aspects of bone quality, such as connectivity, 3D reconstructions are superior because 2D methods have failed to assess how well trabeculae are distributed through three-dimensional space, a key feature of bone quality in trabecular bone (Odgaard and Gundersen, 1993). Furthermore, the strut analysis by Agarwal et al. 2004 is not directly comparable to the ConnD measure used in this dissertation. Unfortunately, because of these methodological differences, comparing the connectedness of trabeculae between the sites greatly inhibits a more conclusive interpretation.

Another important consideration in the analysis of BV/TV for the Velia population is the fact that apparent trabecular thickness (Tb.Th) seems to have changed positively across age groups, particularly in males (Figure 36). The trends for Tb.N, Tb.Sp and ConnD suggest that BV/TV would decline significantly with age. However, the apparent general trend for Tb.Th to go up across age groups indicates that while trabeculae were being lost with age, the remaining trabeculae were thickened to compensate for this loss in numbers. This finding is not without precedent in clinical observations (Mosekilde, 1988; Nicholson et al., 1997; Roschger et al., 2001; Thomsen et al., 2002; Wegryzn et al., 2010). Nicholson et al. (1997) observed that with increasing DA, the remaining trabeculae often thickened, probably to compensate for the loss of horizontal struts. The mechanism by which trabecular thickening might happen is still not fully understood, but the fact that vertically oriented trabeculae appear to thicken more commonly (Nicholson et al., 1997) is suggestive the biomechanical forces are at least partly responsible. Nicholson et al. (1997) have argued that thickening could occur through a repair process, microcalluses are accumulated over time.

Bioarchaeological reports also provide some insight about how Tb.Th changes with age. Agarwal et al. (2004) noted that Tb.Th decreased with increasing age at Wharram Percy, but the authors note that the methodology employed may not have been sensitive enough to accurately assess the thickness of trabeculae. In a Medieval Nubian population, Kneissel et al. (1997) noted also that Tb.Th consistently decreased across age groups, but only in males. In females, Tb.Th was higher in the oldest age group (50+) than in middle age (Kneissel et al., 1997). In both the Agarwal et al. (2004) and Kneissel et al. (1997) studies, the apparent changes in Tb.Th were

small not statistically significant however. It should also be noted that both studies used the L4 vertebra and that the other trends for Tb.Sp and Tb.N in the Agarwal et al. (2004) and Kneissel et al. (1997) studies were consistent with findings from Velia. However, the methodologies employed in all three studies differ, and this may be confounding accurate comparisons. Conservatively speaking, the difference in Tb.Th (and other measures) trends between Velia and the studies of Agarwal et al. (2004) and Kneissel et al. (1994; 1997) suggest that Tb.Th may in fact be variable in archaeological populations and that more work using consistent methods on the same skeletal elements is required.

To conclude this section, it is important to also note that the small and non-significant changes in BV/TV, positive changes in Tb.Th with increasing age and non-significant differences in SMI with age all support the cortical histomorphometric findings (see *Histomorphometry* section). The low remodeling rates in the rib were suggestive of strains that commonly exceeded the MES for the Velia population. Based on the cortical remodeling dynamics, one would expect evidence of high biomechanical strain in other areas of the body. In the trabecular bone examined here, the general trend was for retention of bone volume across age groups. Connective density was lost with age, and was significantly different between the sexes, but SMI changed very little with age, and Tb.Th increased, indicating that biomechanical activity had a role to play in maintaining bone strength. Together, these data on trabecular structure provide another compelling line of evidence that supports the findings in the *Histomorphometry* and *Radiogrammetry* sections.

### *Subadult Trabecular Bone Growth*

In addition to the adult patterns of bone maintenance and loss described above, subadult trabecular bone growth was explored at Velia as well. Sample size was small for all subadults (N = 14), as neonates and children under 2 were excluded from the analysis. The sample was divided into two age groups, one representing individuals from 2-6 years, and the other from 9-16 years. These groups were chosen in order to contrast the slower steady growth in younger age to the faster paced growth that comes with the onset of sex hormones (Bogin, 1999).

All of the measures of trabecular architecture examined in adults were used in the analysis of subadults as well. Although differences were noted between the two age groups, no change was statistically significant, most likely due to small sample size and large standard deviations. In the youngest subadult age group, BV/TV, Tb.N and ConnD were all higher than in the older age group. Conversely, Tb.Sp, SMI and DA were lower in the 2-6 years age group. Trabecular thickness did not differ between the age groups. Only one study to date examines the growth of the L4 vertebra in an archaeological sample (Kneissel et al., 1997). The results from Velia for Tb.Sp and Tb. N are consistent with those from Kneissel et al. (1997). The two populations differ in terms of BV/TV and Tb.Th (ConnD, SMI and DA were not examined by Kneissel et al., 1997). The Medieval Nubian sample (Kneissel et al., 1997), mean values for BV/TV and Tb.Th are higher in the second decade of life than in the first. The contrary was seen for BV/TV at Velia, and Tb.Th was not different. In studies of tibiae (Gosman and Ketcham, 2009) and femora (Ryan and Krovitz, 2006), it was found that BV/TV was high at birth and in early life, declined prior to puberty, and then rose again during puberty before beginning age-



related bone loss. The patterns of trabecular bone change in femora and tibiae early in life (Ryan and Krovitz, 1996; Gosman and Ketcham, 2009) are not entirely consistent with the observations of Kneissel et al. (1997), since the Nubian study failed to detect a decrease in BV/TV prior to puberty. Gosman and Ketcham (2009) argued that the drop and then subsequent increase in BV/TV (as well as changes in related structural parameters) coincides with changing ambulatory behavior (walking). The authors argue that the advent of regular walking/running plays an important part of the reorganization of trabecular bone properties in the tibia (Gosman and Ketcham, 2009).

The discrepancies in trabecular bone growth between vertebral (Kneissel et al., 1997) and limb (Ryan and Krovitz, 2006; Gosman and Ketcham, 2009) elements may be behavioral. Trabeculae in limb bones, particularly lower limbs, may develop differently than in vertebrae, where biomechanical forces operate differently. Unfortunately, very little is known clinically about the development of trabecular structure, particularly in vertebrae, but also limb elements, due to concerns over radiation in *in vivo* studies (Burrows et al., 2010). A number of studies have looked at bone mineral density in growing skeletons, but BMD cannot account for subtle changes in morphology, structure and geometry that accompany the rapidly growing skeleton (Burrows et al., 2010). Roschger et al. (2001) explored developing trabecular architecture in L4 vertebrae in cadaver samples, but utilized 2D methods. In the Roschger et al. (2001) sample, bone volume increased steadily with age from 15 weeks post-conception to the end of adolescence, before beginning to decline in adulthood. Trabecular thickness increased as well, but did not decline with age (Roschger et al., 2001). What these findings (Kneisse et al., 1997; Roschget et al., 2001; Ryan and Krovitz, 2006; Gosman and Ketcham, 2009) suggest is that the development of vertebral trabecular bone operates through different mechanisms than in the limbs, and that these differences are probably biomechanically related. Studies of trabecular structure development are emerging (Kirmani et al., 2009; Burrows et al., 2010) and intra-skeletal differences in trabecular development will likely be identified better and explained in coming years.

The differences between the Nubian population (Kneissel et al., 1997) and Velia are also not easily explained. Sample size in the Velia subadult sample is problematic, and most certainly the largest confounding factor. For example, in the 9-16 years age group, three individuals have very low BV/TV (<0.20). One of these individuals (aged 16-18) could have been a reproductive female, potentially explaining some transitional loss in BV/TV (Sowers et al., 1993, 1995; Affinito et al., 1996; Lopez et al., 1996; Sowers, 1996), but the other two were 12 to 13 years old. Assuming marriage was in the late teens to early twenties (Kleiner and Matheson, 1996; Harlow and Laurence, 2002), the two 12-13 year olds would likely not have been reproductive, although the possibility still remains. Moreover, all three could be males, as sex determination is not possible in subadults, so explaining the very low BV/TV values in these three individuals is speculative only. The remaining three individuals in the 9-16 age range have BV/TV values that are quite high, and would support the observation of Kneissel et al. (1997) that BV/TV in vertebra steadily increases until young adulthood and then declines.

## Cross-Method Analyses

The use of multiple methods in this study was chosen to deliberately investigate different types of bone, whose biomechanical and metabolic properties differ (see Chapters 2 and 3). This was done to explore changes in bone maintenance and health across the life course so that a more complete and thorough examination of bone health in the past could be achieved. The previous three sections have shown that radiogrammetry, histomorphometry and the analysis of trabecular architecture can be successfully integrated to create a more holistic reconstruction of bone health in an archaeological population. In this section, these methods are explored again, but only in individuals that have data for at least two of the methods. This section then concludes with a summary of how the three principle methods (radiogrammetry, histomorphometry, analysis of trabecular architecture) are informative about the life course of bone in the Velia population.

### *Cross-Method Correlations*

Bone maintenance and loss in the second metacarpals and in the ribs ( $n = 52$ ) were positively and significantly correlated ( $r = 0.348$ ,  $p = 0.012$ ). The strength of this relationship can be described as moderate for this type of study (Cohen and Cohen, 1975). As noted in the previous sections, both methods show significant bone loss with age, and this loss occurs earlier in the ribs than in the second metacarpals. The difference in timing is probably due to biomechanical differences throughout life. While macro-level analyses between ribs and second metacarpals showed a moderate, but important relationship, tissue level changes (activation frequency) in the rib were even more positively correlated ( $r = 0.681$ ,  $p = 0.000$ ) with cortical index in the metacarpal ( $n = 37$ ). This relationship is close no doubt because both methods showed significant age-related changes. The reason why activation frequency is more positively correlated with metacarpal cortical index than percent cortical area in the ribs is not certain, given that activation frequency and percent cortical area follow a very similar trend of greater decline between the young and middle age groups, than between the middle and old groups (as cortical index does). Exploring the relationship between remodeling activity (activation frequency) in the ribs and cortical index in the metacarpal is difficult because they come from two distinct areas with varying biomechanical strains.

Trabecular bone volume was also compared to CI in the metacarpal in order to see if changes in trabecular bone quantity tracked with those from cortical bone. Results from Pearson correlations indicate that the relationship is essentially non-existent ( $r = -0.015$ ,  $p = 0.924$ ) and was not statistically significant. The relationship was also negative, which is not expected, as bone quantity in both types of bone follow a very similar general trend with age, followed by a decrease from early to late adulthood. Sample size was small here however ( $n = 40$ ), which introduces greater error. Also, BV/TV changed little with age, while CI decreased significantly across age groups. With these caveats in mind, it does bear noting that trabecular and cortical bone quantity do seem to operate fairly independently in this sample, particularly since CI dropped significantly with age while BV/TV did not. The fact that they did not track (even mildly), and that trabecular bone is quicker to respond to mechanical and metabolic demands,

seems to support the argument made previously that biomechanical activity played a central role in mediating age-related bone loss.

A final comparison was made between activation frequency and changes in BV/TV in the lumbar vertebrae ( $n = 23$ ). Again, BV/TV tracked poorly here with changes in cortical ( $r = -0.017$ ,  $p = 0.939$ ) bone, but this time at the tissue level. Sample sizes here very small and this comparison is very tentative only. The poor correlation is can be explained by trends observed in each independent and much large sample. BV/TV changed very little with age, while remodeling activity slowed significantly across age groups. Furthermore, remodeling in cortical bone operates independently from that of trabecular bone, in large part because of significant difference in surface area and metabolic activity (Martin and Burr, 1989). In the end, this poor correlation is expected based on these fundamental differences in the bone tissues

### *The Life Course Approach from the Use of Multiple Methods*

By using three methods of investigating bone loss in one archaeological population, a life course approach (see Chapter 2) can be implemented to explore how bone maintenance and loss occurred over the life cycle. What facilitates the development of a life course approach is not only the use of multiple methods, but the fact that the methods reflect different types of bone and regions over the skeleton with different bio-historical trajectories. I argue here that the analysis of trabecular architecture can be interpreted as representing more short terms changes in bone, given the high metabolic activity and great sensitivity to biomechanical strain (Barak et al., 2011). Remodeling in the ribs is interpreted at the mid-range level, as intra-cortical remodeling is slower than in cancellous bone (Compston, 1999), but can react more quickly at the tissue level than larger morphological changes at the whole-bone level. Finally, radiogrammetry of the second metacarpal is seen as representing longer-term changes in cortical bone, as whole-bone morphology is a product of years of interplay between dietary, hormonal, biomechanical and lifestyle factors (Martin and Burr, 1989; Ruff et a., 2006).

The characterization of long-term bone maintenance and loss at Velia can be summarized as mostly typical and what we expect based on modern (Maggio et al., 1997; Böttcher et al., 2006; Szulc et al., 2006) and archaeological (Mays, 1996; 2006) expectations. Broadly speaking, bone quantity is highest in young adulthood and then declines significantly with age. One fundamental difference between the trends at Velia and in modern populations is that no sex differences are observed in CI in the 50+ age group (or in any). Diet and reproductive history were explored as potential biocultural influences (see *Radiogrammetry* section), but the weight of the evidence suggests that physical activity throughout life played the most important mediating role in the prevention of bone loss with age, and particularly in females. There are a number of lines of evidence for this conclusion, beginning with the CI data.

Cortical index in young adults at Velia is higher than has been observed in Romano-British (Mays, 2006) and Medieval British (Mays, 1996) populations. The CI in the young adult age group reflects changes in cortical bone that have accrued over adolescents, a key period in the formation of peak bone mass in adulthood (refs). It was argued in the *Radiogrammetry* section that dietary deficits and reproductive history were generally similar between the sites, so

it is probable that physical activity during adolescent years formed a significant role in the attainment of higher cortical indices. Compared to modern populations, young adult means for CI are lower than a modern Finnish population (Virtama and Helelä, 1969), but higher than another modern German sample (Böttcher et al., 2006). Typically, peak bone mass, and thus a measure such as CI, would be expected to be lower in the past because of common nutritional deficiencies (Mays, 1996), but clearly this cannot be always assumed based on the comparison of the young adult means from Velia to modern populations. Nutrition was undoubtedly poorer in the Roman context Velia was part of (Garnsey, 1998), so an argument for the crucial role that physical activity played during adolescence is strengthened by the observation that CI at Velia in young adults surpassed those of a modern European population (Böttcher et al., 2006). These arguments are supported by the mid and short term remodeling in ribs and vertebrae as well. Percent cortical area (and overall size) was very high in young adult ribs, and indicators of remodeling activity were typically lower than other archaeological and modern populations (Cho and Stout, 2003). Bone volume (BV/TV) in both males and females at young age was higher at Velia than in a modern normative study (Compston, 1999). Together, this suite of measures implies a longer period growth, but also a significant effect from high strains induced from physical activity (Frost, 1987c).

Important differences between the skeletal sites begin to emerge when middle and old aged adults are considered. In the second metacarpal, bone loss begins to occur, typically earlier than is observed in modern populations (Virtama and Helelä, 1969; Maggio et al., 1997; Böttcher et al., 2006; Szulc et al., 2006). This would suggest muscular disuse (Frost, 1987c), but lines of evidence from other skeletal sites suggest otherwise. Remodeling activity remains low in middle age, which probably meant that biomechanical strains were elevated (see arguments in *Histomorphometry* section for details). Further, measures of trabecular bone, particular BV/TV, SMI and Tb.Th also indicated that vertebrae were being substantially loaded. It should also be noted that the CI for males barely changed between young and middle age, and this does not support an argument that activity levels declined. Therefore, the drop in CI into middle age, occurring primarily in females, while not severe, might be better explained by other biocultural factors such as temporary bone loss due to pregnancy and lactation. For example, many in this age group may have died during pregnancy and/or lactation. Moreover, bone loss with pregnancy and lactation has been noted in other archaeological studies but it is considered to be transitional and temporary (Agarwal et al., 2004), although Mays (2010) argues that this may not always be the case.

Cortical index in old adults was significantly different than in young and middle aged adults for both sexes. Interpreted in light of the shorter and mid-level remodeling, activity seems to have remained relatively high and this change in CI should be seen as caused by other factors. BV/TV did not change significantly with age and the trends observed in analyses of trabecular architecture, such as increasing trabecular thickness, suggest that day-to-day activities remained strenuous into old age. Remodeling activity in the ribs was low in old age, but this is expected and normal to some extent (Mulhern, 2000), so histomorphometric analyses become less useful here. Ultimately what the changes in cortical index show is that even if physical activity was high and sustained throughout life, hormonal changes during the aging process cannot be fully mediated and still have a large effect on bone loss in some areas of the skeleton.

To summarize, the analysis of trabecular architecture throughout adulthood showed a consistent trend indicating that biomechanical activity played an important role in maintaining bone mass with age. Unfortunately the small sample of subadults really prevents a meaningful examination of childhood and adolescent growth in the vertebrae, although the high bone volume in young adulthood implies that strains were high in adolescents. Rib remodeling activity also supported an argument for long period of growth and for the substantial influence of mechanical strain in reducing remodeling activity throughout life. The longer-term picture offered by the metacarpals indicates that activity during adolescent growth was substantial. Significant bone loss still occurred with age (as seen by changes in CI), but the consistent lack of sex differences between the methods is important, and reaffirms the argument that physical activity throughout the life course played a key role in preventing greater bone loss in females, which we so commonly observed in modern populations. Moreover, the very low prevalence of fragility fractures is also strongly supportive of the role physical activity played in maintaining bone strength throughout life.

Individually, the three primary methods used in this study provide useful information on bone maintenance and loss. When juxtaposed, the combination of methods provides a reconstruction of bone health that is much more compelling than any single method can provide. Moreover, the use of multiple lines of skeletal evidence can incorporate the complexity and heterogeneity of bone of a living material and so changes in bone that occur at different rates can be explored, broadening what can be said about bone health over the life course. The life course approach can also be investigated by considering the role of physiological stress during childhood, and what role stress may have had on bone health later in life.

### **Physiological Stress in Subadult and Adults**

Stress markers have long been studied in physical anthropology and more recently in bioarchaeology (Huss-Ashmore et al., 1982; Porter et al., 1987; Larsen, 1997; Mays, 1999; Humphrey, 2000; Cardoso, 2007; Temple, 2008; Klaus and Tam, 2009; Walker et al., 2009) and can be defined as an environmental insult that alters the normal metabolic and physiological function of an individual (Huss-Ashmore et al., 1982). Systemic stress in subadults is emphasized in this project, although it does occur in adults as well (Huss-Ashmore et al., 1982). Humphrey (2000) has noted that developmental stress makers used on their own are problematic because it is sometimes difficult to distinguish stressed development from normal variation during growth. As a remedy, Humphrey (2000) suggests using a combination of methods to strengthen interpretation. In the investigation of subadult stress, *Dental Enamel Hypoplasia (DEH)* (Guatelli-Steinberg and Lukacs, 1999; Larsen, 1997; Hillson, 2000; King et al., 2005; Cardoso, 2007; Temple, 2008; Hubbard et al., 2009; Klaus and Tam, 2009), *Vertebral Neural Canal (VNC) Size* (Clark et al., 1985; 1986; Porter et al., 1987; Clark, 1988; Larsen, 1997; Rewekant, 2001), and *Skeletal Growth Profiles (SGP)* based on femoral growth (Bogin, 1995; 1999; Saunders, 2000, 2008; Humphrey, 2000; Mays, 1999; Mays et al., 2008; Klaus and Tam, 2009) are examined. In the adults, *Cribra Orbitalia* and *Porotic Hyperostosis* (Steckel and Rose, 2002; Ortner, 2003; Walker et al., 2009), as well as *Periostitis* are considered (Mensforth et al., 1978; Ortner & Putschar, 1985; Larsen, 1997; Ortner, 2003). All of these are non-specific indicators of stress, meaning that they cannot be reliably attributed to a single specific source,

such as dietary deficiency or pathogen load. However, the non-specificity allows us to gauge the level of physiological insults endured.

### *Subadult Stress*

Dental enamel hypoplasias (DEH) are defects in tooth enamel that are formed during the formation of the tooth and are a classic marker with a long history of use in bioarchaeology (Goodman & Armelagos, 1985; Goodman & Rose, 1990; Ten Cate, 1994; Hillson, 1996; Cardoso, 2007). Once a tooth is formed, it does not remodel like bone, so a record remains as long as the tooth does (Hillson, 2000). What is particularly useful about hypoplasias is that they can be examined in adults because they record growth disturbances in enamel formation that occurred very early in life (Goodman & Armelagos, 1985; Goodman & Rose, 1990; Hillson, 2000). DEHs thus provide one important line of evidence that can help link physiological events that occurred early in life, with bone remodeling in adulthood. This can help broaden what can be said about bone health throughout the life course.

DEHs in the Velia sample ( $n = 75$  for DEH evaluation) were very high (see Figure ?). Approximately 90% of individuals showed signs of DEH (at least one tooth affected). If sexes are separated, 95.1% of females had some DEH, compared to 86.8% of males. This difference was not significant however. DEH was higher in younger adults, but this is most likely because more teeth are generally observable in younger adults archaeologically; tooth wear and ante mortem tooth loss increase with age, and thus sample sizes become more limited.

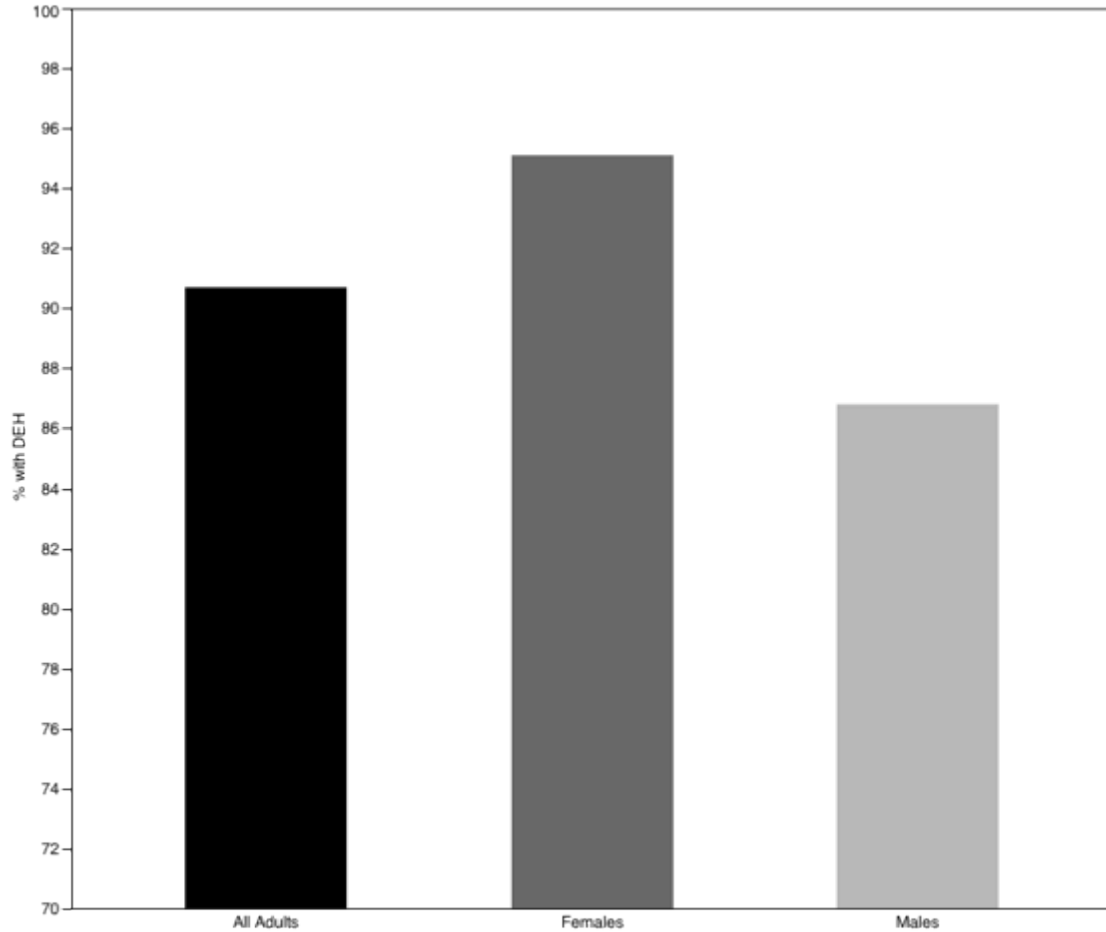
The prevalence DEH at Velia is quite high, even for an archaeological population (Steckel and Rose, 2002). However, Goodman and Martin (2002) have noted that in modern developing countries, the prevalence of enamel defects often exceeds 50% of all individuals. This is because socioeconomic status is highly correlated with the formation of DEH during development (Goodman and Martin, 2002). There is evidence to suggest that the most sensitive period for DEHs to occur is the period between 18 and 36 months (Goodman et al. 1987), and this thought to be related to the weaning process (Goodman and Martin, 2002). The 18 to 36 month period is commonly when weaning took place in the Roman world (Garnsey, 1999; Fuller et al., 2006; Dupras and Tocheri, 2007; Prowse et al., 2008). The weaning period was probably quite stressful for children, as they seem to have been weaned onto very poor quality foods (Garnsey, 1999). Further, protein-calorie deficiency seems to be strongly associated with the development of dental hypoplasias (Malville, 1997). The very high prevalence of DEHs at Velia seems to support the historical descriptions of poor childhood diets very well. Although DEHs are not solely indicators of dietary stress (Armelagos et al., 2009), diet certainly plays a major role (Goodman and Martin, 2002). As discussed in Chapter 4, the Roman world was a difficult, and pathogen loads were probably high. The combination of poor infant diets, weaning stress and high potential pathogen load all seem to have greatly affected the dental development of Velian children.

While the prevalence of DEH is high for Velia, it is not unique in the Roman context. Paine et al. (2009) reported that the prevalence of DEH for the Urbino population was 100% for both sexes. At another Roman Imperial site (Quadrella) in Molise, Italy (Bonfiglioli et al., 2003) DEH prevalence was nearly identical to what was observed for Velia. At Quadrella, 92% of

males and 100% of females had evidence of DEHs. The overall prevalence for Quadrella was 95%, essentially the same as Velia. Very high rates of DEH were also recorded for the population of Vallerano, which was located close to Rome and also dates to the Imperial Roman period (Cuccina et al., 2006). The prevalence of DEH has also been assessed for the Isola Sacra population (Manzi et al., 1989; 1999). Interestingly, while still quite high, the overall prevalence of DEH at Isola Sacra is 81.03% (Manzi et al., 1989), roughly 14% lower than at Velia.

Overall, the available data on DEHs in the Imperial Roman context strongly indicate that hypoplasias were extremely common (Paine et al., 2009). All of the sites listed above, except Isola Sacra, represent non-elite individuals. Although the people of Isola Sacra were not really from the Roman elite, they represent a more middle class population (Manzi et al., 1997). The lower prevalence of DEHs at Isola Sacra suggests that childhood may have been less stressful than at Velia and other non-elite sites (Manzi et al., 1999; Bonfiglioli et al., 2003; Cuccina et al., 2006; Paine et al., 2009), perhaps because socio-economic status was seemingly better. However, this is a tentative conclusion as the etiologies of DEHs are complex (Goodman and Martin, 2002) and the prevalence for DEHs at Isola Sacra was still very high.

A recent study has shown that DEHs are strongly associated with an earlier age at death (Armelagos et al., 2009), and supports hypotheses that stress early in life will have a negative on health in adulthood (see Chapter 2). The correlation between age-at-death and DEHs was not assessed for Velia, as the original focus was comparing bone health in adulthood to early stress, rather than mortality profiles. However, given that the prevalence of DEHs are so high at Velia, it is not possible to reliably test if bone health was better or worse in those without and without evidence for DEHs. Furthermore, Temple (2008) has recently argued that while DEHs are good indicators of general stress, they fail to predict what the impact of that stress might have on adult phenotypic variation. The predictive limitations of DEHs (Temple, 2008) might be surpassed however, by using a combination of methods. Armelagos et al. (2009) suggested using vertebral canal size in conjunction with DEHs to improve what could be said about the relationship between development and health in adulthood. DEHs and vertebral canal sizes are explored together in the following section. Additionally, future work on the Velia sample will examine the relationship between DEHs and mortality (following Armelagos et al., 2009) in order to better understand what role DEHs might have played in limiting adult health.



**Figure 37** – Percentage of individuals with dental enamel hypoplasias by sex

Vertebral neural canal (VNC) sizes were explored in light of their relationship with increased morbidity and earlier risk of death later in life (Porter and Pavitt, 1987; Clark et al., 1986; Clark, 1988; Rewekant, 2001). Three hypotheses were explored for the Velia population. It was first hypothesized that VNC size would negatively and significantly correlated with vertebral wedging. Clark et al. (1986) note that increased wedging would place individuals at greater risk for osteoporotic (fragility) fractures in the vertebrae. If small or stunted VNC sizes were correlated with wedging, this could help demonstrate the relationship between early childhood development and increased morbidity (specifically osteoporosis) later in life (Cooper et al., 2006). The correlations for the Velia population were extremely weak and non-significant, diverging greatly from what was observed in the Native American Dickson Mound (950-1300 AD) population (Clark et al., 1986). Furthermore, vertebral wedging did not increase significantly with age in the Velia population, suggesting also that VNC size had little long term negative effects on wedging, or that other factors prevented wedging with age. Ultimately, the hypothesis that VNC size has an effect on wedging cannot be supported in the Velia population.

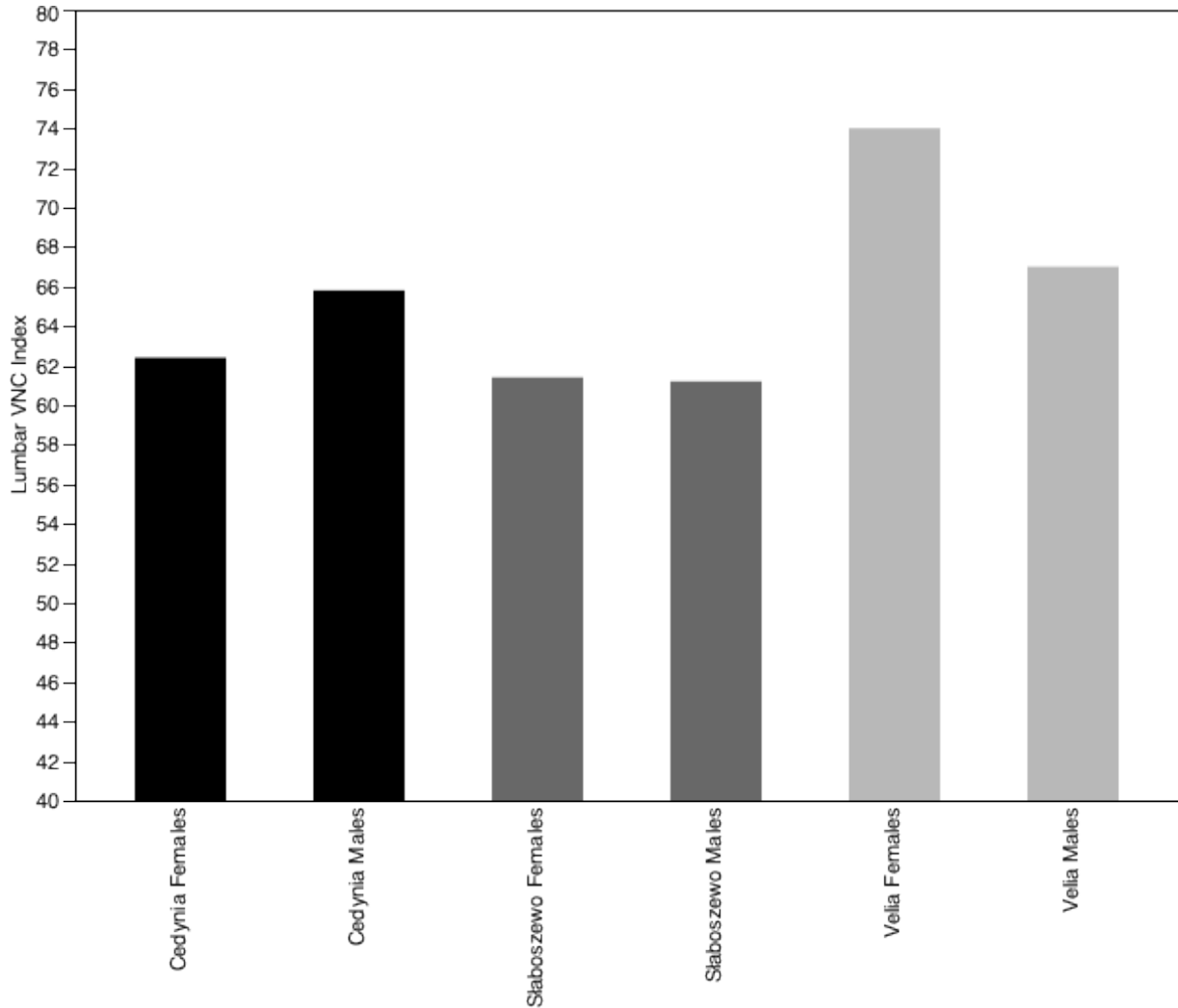


The second hypothesis examined the relationship between VNC size and stress. It was hypothesized that individuals with smaller VNC size would show more evidence of stress. Chi-square and *t*-test analyses could not support this hypothesis. There is no convincing evidence to suggest that individuals with higher developmental stress (smaller VNC) had increased incidences of stress (cribra orbitalia or porotic hyperostosis). The final hypothesis examined the relationship between VNC size and measures of bone maintenance. Again, no clear and consistent pattern could be established. One important problem was that the relationships were negative, which is not what would be predicted if small VNC size had a negative effect on bone maintenance later in life. Second, none of the correlations were statistically significant.

The study of VNC sizes and vertebral body heights showed promise, particularly the work of Clark et al. (1986) and Clark (1988). This was a under utilized method analogous to DEH that could potentially help clarify the relationship between early life stress and bone remodeling later in life. Unfortunately, none of the results for the Velia population support the findings of Clark et al. (1986) or Clark (1988). Furthermore, the anterior-posterior (AP) measure of VNC size was not reliable statistically for Velia, and may not have been for the Indian Knoll studies as well (this was not reported) (Clark et al., 1986; Clark, 1988). It is possible that the problems in reliability for the method are why nearly no bioarchaeological study has explored it since the 1980s. One exception is a study by Rewekant (2001), who used AP and ML canal measures to form an index (AP/ML), but he did not establish if the measures were reliable. In the Rewekant (2001) study, two ethnically similar, but socioeconomically different Medieval Polish populations were compared to assess if evidence of physiological stress (lower socioeconomic status) translated into worse health throughout the life course. The VNC index used by Rewekant (2001) found no differences between females of the two communities, but a significant difference was noted for males. In males, VNC indices were generally higher in the community with higher socioeconomic status (Rewekant, 2001). The differences between Indian Knoll (Clark et al., 1986), the Polish Medieval sites (Rewekant, 2001) and Velia for measures of VNC are hard to explain.

The methodology itself is problematic, but there is more to it than that. Sample size ( $n = 90$ ) was larger at Indian Knoll, but not dramatically so. However, sample size for the Rewekant (2001) study was much larger ( $n = 219$  and  $n = 145$  for the two sites). The Indian Knoll population differs substantially socioeconomically from Velia, and these divergent biocultural contexts may be responsible for the disagreement between the studies. However, as Clark et al. (1986) pointed out, the causes of smaller VNC are not important, much like other stress markers. If VNC size has a consistent effect on adult health, it should be noted in any archaeological population. To explore this further, the lumbar VNC index used by Rewekant (2001) was used and Figure 38 highlights the differences in results between the studies. Lumbar VNC indices were higher at Velia, suggesting that infant stress, although substantial based on the high prevalence of DEH and the SGPs (see below), was not worse than these Medieval Polish populations. It is possible that a certain threshold is needed before VNC size affects adult morbidity. The medial-lateral (transverse in Clark, 1988) measures were larger for Velia for both sexes than either pre-Mississippian or Mississippian cultural periods at Indian Knoll (these data are not available for the Rewekant, 2001 study). The anterior-posterior sizes were larger in the Indian Knoll group, but this measure is unreliable. Perhaps VNC sizes at Velia were large enough to not affect health. A large part of the Clark et al. (1986) and Clark (1988) studies was to see if small VNC could predict earlier age at death, which it did. This was not done for Velia,

but could be explored in future work to see if the pattern still holds. Ultimately, this method needs more exploration in different cultural contexts to see if the reliability is a widespread problem and to see if VNC size has an effect on adult health in other populations.



**Figure 38** – Lumbar VNC Index (AP/ML) for the Velia, Cedylnia (higher status) and Slaboszewo (lower status) populations (Rewekant, 2001). Values for the Cedylnia and Slaboszewo populations are estimates taken from a graph provided by Rewekant (2001: 439), as exact means are not provided.

Skeletal growth profiles (SGPs) present another means of assessing the relationship between growth and physiological stress (Humphrey, 2000; 2003; Saunders, 2008). The SGPs produced for the Velia population (Chapter 6) are highly suggestive of slowed growth during the first 12 years of life. When mean femoral length is plotted against age for both the Velia and Denver populations, mean femur lengths at Velia consistently fall below 2 SD of the Denver means (Figure 19). When individuals, rather than group means from Velia are plotted on the Denver curve, a more refined picture emerges. Early in life (before 2.5 years) there is some overlap between the populations. From ages 3.5 to 9.5 however, no individuals from Velia fall within 2 SD of the Denver mean (Figure 20). Humphrey (2000) has argued that differences between the Denver population and archaeological groups may be better observed by plotting residuals of a line describing the mean size of the children in the Denver study. In the plot of the residuals, the children of Velia seem to have a marked deficit in growth compared to Denver sample. Early in life (up to 2.5 years), most individuals are below 2SD of the Denver mean, with only 6 within -2SD of the Denver sample. Only one individual was actually above the Denver sample mean, but below +1SD. From 3.5 years to 12 years, all individuals fall below 2SD of the Denver sample, except for 1 individual in the 12 year category. Although difficult to quantify, the separation between Velia and the Denver sample appears to worsen with age based on the slope of the residuals.

Humphrey (2000; 2003) has suggested that exploring femoral growth as a percentage of adult stature is quite useful because it shifts emphasis from absolute lengths, to a focus on the rate of growth towards completed adult stature. When plotted raw percentages of adult femur length, the Velia population once again shows slower growth, given that for each age group Velians have reached less of their total growth than the Denver sample and based on the mean values, there does not appear to be a tendency for this trend to worsen with age. When the residuals of percent adult length are considered for all individuals, a similar trend to the residuals plot of mean femur length emerges. Prior to 2.5 years, there is more overlap with the Denver study than beginning at 3.5 years and onwards to 12 years. Finally, if the residual means for % difference in adult size attained are considered against the Denver data, the means for the Velian children fell within 2SD of the Denver growth rate (Figure 24). Humphrey (2003) provides the same data as Figure 24 for 11 archaeological populations and the mean values of the residuals for % difference of adult size attained from Velia are fully consistent with most of the archaeological populations reported, as most of them fell below the Denver mean and were within 2 SD. It should be noted however that the slopes and trajectories of each population, including Velia, vary considerably and reflect the particular genetic, cultural, and environmental factors in each population that might influence growth (Humphrey, 2003). Specific comparisons of the Velia growth trajectories to those in other archaeological populations are beyond the scope of this study, but the overall the growth rates at Velia, if explored as the percentage of adult size attained by age, do not differ appreciably from many other archaeological populations (Humphrey, 2003), most likely because in many of these groups, infant stress from weaning and pathogens was high, and growth suffered.

These results are enticing and at first they strongly imply that growth was retarded for the Velia population. There seems to be a shift around 2.5 to 3.5 years of age, where the Velia population seems to shift away from the Denver one to an even greater degree. The children of Velia were shorter for their age than the modern Denver sample, and the rate at which they

attained adult size was also slower. This suggests that the growth period may have been extended. This is only a tentative argument based on the SGPs, given that older adolescents were not used, due to small sample size. What the data from the SGPs do support is a hypothesis for substantial infant stress. However, caution is warranted as growth data face a number of methodological concerns (Humphrey, 2000; 2003; Saunders, 2008) (see Chapter 5). Nevertheless, the SGPs, in conjunction with the data on dental enamel hypoplasias, considered in light of the biocultural context of the Roman period, present strong support for the evidence of considerable stress during the weaning period.

### *Adult Stress*

The etiology of cribra orbitalia and porotic hyperostosis is complex and the lesions present themselves as responses to a suite of factors, including genes, dietary stress, infectious disease, and parasitic infection (Aufderheide and Rodriguez-Martin, 1998; Larsen *et al.*, 2002; Steckel and Rose, 2002; Ortner, 2003; Walker *et al.*, 2009). Although the specific causes of the lesions can be rarely determined, there exists a general bioarchaeological consensus that these lesions represent a poor quality of life (Cohen & Armelagos, 1984; Larsen, 1997; Larsen *et al.*, 2002).

The prevalence of porotic hyperostosis ( $n = 66$ ) in adults was 18.18% for all adults. If the sexes are considered separately, males displayed more lesions (24.32%) than females (10.34%) and represent 75% of all affected individuals. The severity of porotic hyperostosis (Ribot and Robert, 1996) was low and all lesions were nearly fully healed. The prevalence of cribra orbitalia ( $n = 54$ ) was more pronounced, with 40.74% of the observable orbits in adults showing typical lesions. Sex differences for cribra orbitalia were less pronounced, with 37.5% of females and 43.3% of males displaying orbital lesions. Males represented 59% and females 41% of all individuals affected with orbital lesions. Goodman and Martin (2002) have stated that cribra orbitalia and porotic hyperostosis could be combined and examined together. When the two assessments are combined for Velia, 32 of 69 (46.38%) individuals have at least one lesion in the orbit or on the cranial vault. Lesions that occur in both were not counted twice. Difference in the prevalence of lesions between the sexes is fairly pronounced. The prevalence in females was 35.48% and 55.26% in males. Furthermore, females have 11 of the 32 lesions (34.37%), while males have the remaining 21 (65.63%).

The prevalence of cranial lesions was fairly high for Velia, and is comparable to what was observed for the Imperial Roman skeletal populations of Urbino (Paine *et al.*, 2009), Vallerano (Cuccina *et al.*, 2006), Lucus Feroniae (Salvadei *et al.*, 2001) and Ravenna Area and Rimini (Facchini *et al.*, 2004). All of these sites represent various classes of non-elite individuals in the Roman world. All of these sites, except Lucus Feroniae (Salvadei *et al.*, 2001) have adult frequencies of cranial lesions between 40-50%, with sexes combined. The frequency of cranial lesions at Lucus Feroniae (Salvadei *et al.*, 2001) is lower in adults (19.4%), but when juveniles are included, rises to 49.5%. Another exception is for the Vallerano site (Cuccina *et al.*, 2006), where females had a prevalence of cribra orbitalia of 85.7%, but sample size ( $n = 26$ ) was quite small. At Velia, males were more affected than females, but this difference was not significant. The same pattern was noted for Urbino (Paine *et al.*, 2009). At Ravenna Area and Rimini

(Facchini et al., 2004) and at Lucus Feroniae (Salvadei et al., 2001), some females had higher frequencies of cranial lesions than males, but none were significant.

What the overall patterns at Velia, as well as other Imperial Roman sites (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006; Paine et al., 2009), indicate is that cranial lesions (porotic hyperostosis and cribra orbitalia) were very common in the Roman world. Numerous reasons have been offered that explain these high frequencies of cranial lesions in the Roman context. Iron deficiency anemia is the most common explanation for cranial lesions (Walker et al., 2009) throughout all bioarchaeological studies, and has been used as an explanation in the Roman context as well (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006; Paine et al., 2009). As Facchini et al. (2004) point out, the typical Roman diet is poor in iron, particularly during weaning when animal products are rarely consumed. However, Stuart-Macadam (1992) and Walker et al. (2009) have argued convincingly that the central premise of iron deficiency anemia is flawed. When the human body is deficient in iron, red blood count is actually restricted, not increased, and so marrow spaces should not expand (creating lesions) due to iron deficiency anemia (Walker et al., 2009). Rather, the mechanisms that produce the typical cranial lesions of porotic hyperostosis and cribra orbitalia are better explained by vitamin B<sub>12</sub> deficiency and through reduced intestinal absorption of nutrients from unsanitary living conditions (Walker et al., 2009). Vegetarians are commonly deficient in vitamin B<sub>12</sub> (Walker et al., 2009), as most sources of B<sub>12</sub> are from animal origins. The typical Roman diet (Garnsey, 1998) is largely vegetarian, although not strictly so. Periods of famine or severe food shortage can worsen any existing deficiencies, particularly in infants (Lindstrom and Berhanu, 2000), which probably largely explain the high prevalence of cranial lesions (even healed or healing ones) in Roman children (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006). In adults, the B<sub>12</sub> hypothesis (Walker et al., 2009) also applies in the Roman context, since animal products did not typically form a key role in the non-elite diet (Garnsey, 1998). Furthermore, living conditions in Roman cities were probably not sanitary (see Chapter 4) and may have contributed to poor absorption of nutrients due to parasitic infections (Stuart-Macadam, 1992; Walker et al., 2009). If vitamin B<sub>12</sub> intake was already low or barely adequate, diarrhea and intestinal malabsorption caused by parasites could have easily further reduced the amount of available B<sub>12</sub> in the body (Walker et al., 2009). It should be noted however that a very recent study has placed some doubt on the hypotheses of Walker et al. (2009). Oxenham and Cavill (2011) have argued that Walker et al. (2009) have misread the clinical literature and that iron-deficiency anemia remains a possible source for cribra orbitalia and porotic hyperostosis. Nevertheless, the contributions by Stuart-Macadam (1992) and Walker et al. (2009) regarding vitamin deficiency and general living conditions remain. Clearly though, more work is needed to clarify how the various anemias related to cribra orbitalia and porotic hyperostosis (Oxenham and Cavill, 2011).

In summary, the common explanation given for the high prevalence of cranial lesions due to cribra orbitalia and porotic hyperostosis in the Roman world have been poor diet and unsanitary living conditions, including parasite load and infectious disease (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006). Dietary causes of cranial lesions are typically attributed to iron deficiency, but a deficiency in vitamin B<sub>12</sub> and general pathogen load is much more consistent with physiological and skeletal changes observed clinically (Walker et al., 2009). The biocultural context outlined for other Imperial Roman sites (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006; Paine et al., 2009) is fully consistent with what we

know about Velia (see Chapter 4) and the explanations provided by those authors for the observed cranial lesions apply to Velia as well, including the important contribution by Walker et al. (2009). Although juveniles were not examined for cranial lesions in this study, it can be argued based on previous studies (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006) and on the high prevalence of lesions in adults at Velia, that general nutritional and environmental stress was high throughout the life course.

Periostitis is common in archaeological populations (Ortner, 2003). Periostitis is defined as subperiosteal new bone formation (SPNBF) caused by an inflammatory reaction of the periosteum (outer membrane of bone) (Ortner, 2003). Periostitis is a non-specific stress indicator and can be caused by localized infection, secondary responses to infection and trauma to the area (Steinbock, 1976; Ortner, 2003). In addition, periostitis is also strongly associated with nutritional deficiency and a higher prevalence of periostitis has been commonly found in cases where both poor nutrition and disease load were common as well (Ortner and Putschar, 1985). The synergistic relationship between nutritional deficiency and periostitis is probably in large part caused by a lowered immune response due to nutritional stress (Paine et al., 2009). Although the causes are non-specific, periostitis has become a useful and important tool bioarchaeologically to investigate general health in the past (Roberts and Machester, 1997).

Periostitis in Velians was fairly low in the upper limbs, but quite high in the lower limbs. In the upper limbs, periostitis was present in only 9.8% of individuals and essentially equally distributed between the sexes. In contrast, nearly 84% of adults had periostitis on lower limb bones. The prevalence of periostitis in males (89.1%) was higher than in females (76.9%), but this difference was not statistically significant. In both upper and lower limbs, neither side of the body was significantly affected over the other. In order to help distinguish potential localized trauma, which would theoretically affect only one side, the percentage of cases that had both sides affected were examined. Only individuals with both sides present were considered to avoid bias from missing elements. In females, 73% of cases were bilateral and in males 58% were bilateral. This difference was not significant however ( $X^2 = 0.760, p = 0.384$ ). The higher number of individuals with only one side affected in males vs. females may signify important behavioral differences between the sexes, perhaps related to occupational roles. For example, the fishing and dock work that many males participated in (Crowe et al., 2009) may have placed males at risk for localized trauma, particularly in the lower leg (the tibia is most commonly affected). This is speculative of course, as the causes of periostitis have a number of sources and cannot be easily parsed out (Ortner 2003). In addition, trauma that could have affected one leg may just as easily have struck both. Future work on the Velia sample, particularly with regards to trauma, may help clarify how occupation was related to localized vs. more systemic periostitis.

The prevalence of periostitis at Velia seems to be higher than in other Imperial Roman sites. In the Urbino population, total prevalence of periostitis was 31% (Paine et al., 2009). Urbino males had a higher prevalence at 41%, compared to 20% in females, but this difference was not statistically significant (Paine et al., 2009). In the Quadrella population, total tibial periostitis was higher, at 63.4% (Belcastro et al., 2007), which is more similar to Velia than Urbino. No sex differences were noted in tibial periostitis for the Quadrella population, but males (63.6%) had a slightly higher prevalence than females (61.1%).

Lead poisoning is another potential cause of periostitis and has been applied often in the Roman context as they often utilized lead and pewter dishware, as well as lead acetate (Facchini

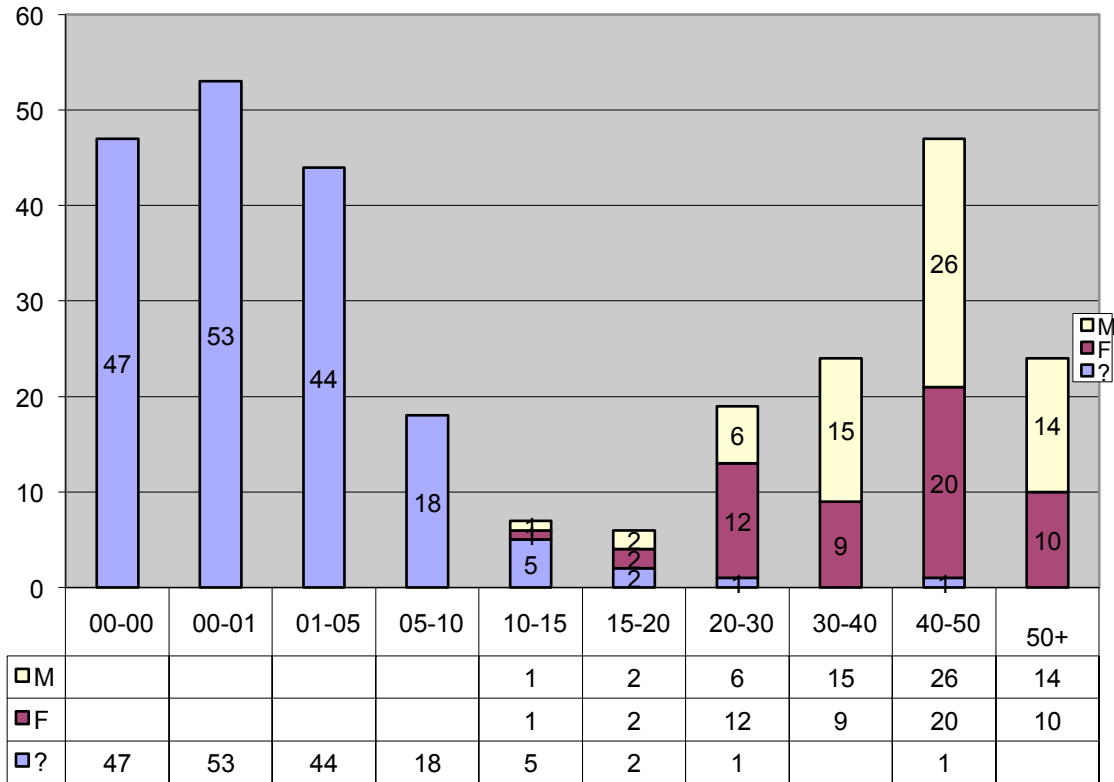
et al., 2004). Interestingly, Paine et al. (2007) found no statistical difference in the amount of lead in bone between individuals with and without periostitis. An older study by Grandjean (1988) also found no statistical difference in lead content between individuals with and without cribra orbitalia. Paine et al. (2007) cautioned that although no statistical difference in lead content was found between individuals with and without lesions, the possibility remains that lead still contributed to the development of lesions in at least some people. Hence, while lead poisoning may have caused some lesions in Velians, statistical evidence from previous studies on other populations does not support a strong correlation between lead intake and non-specific lesions in crania or long bones (Grandjean, 1998; Paine et al., 2007). Future work at Velia might test this hypothesis further however by testing for lead content in individuals with and without lesions at the time of death.

In summary, neither Belcastro et al. (2007) or Paine et al. (2009) place much emphasis on interpreting the patterns of periostitis. This is understandable as the etiologies of the lesions are complex and can rarely be clarified in archaeological populations (Larsen, 2002). In some cases, patterning can be useful. For example, the diagnosis of leprosy is bolstered by the presence of bilateral periostitis in the tibiae (Belcastro et al., 2007). In the majority of cases however, little can be said about periostitis other than stating the prevalence for a given population in order to track changes through time and in different biocultural contexts (Larsen, 2002). The very common occurrence of periostitis at Velia supports previous arguments for nutritional stress and potentially high parasitic and pathogen loads. Trauma may have played a substantial role in a number of individuals at Velia, probably to greater extent in males.

## **Study Limitations and Future Directions**

In a bioarchaeological investigation such as this one, there are a number of methodological limitations that should be considered in the interpretive process. Some of these have been addressed throughout the dissertation, but are summarized again here for clarity. All bioarchaeological studies are cross-sectional in nature, and thus reflect, in any particular measure of bone, a snapshot in time. Cross-sectional studies also assumed that each individual has the same variability in measures of bone loss (Cho and Stout, 2011). Longitudinal studies are ideal for studying age-related bone loss, but this is a luxury not available to the bioarchaeologist. Larger sample sizes can help mitigate some of the limitations of cross-sectional studies, but poor preservation, differential burial and excavation techniques all operate to limit sample sizes in bioarchaeology (Jackes, 2011). Sample size varied for this study, depending on the analysis in question, but was quite good overall for a bioarchaeological investigation. Concerns over sample size were noted throughout where appropriate. Furthermore, the mortality profile for the Velia population shows that differential preservation or excavation was not a large biasing factor, given that many young infants were recovered and that the mortality profile as a whole follows an ideal U-shaped distribution (Figure 39) (Chamberlain, 2000).





**Figure 39** – Mortality Profile for the Velia population (courtesy of Dr. Luca Bondioli, Pignori Museum, Rome).

Selective mortality and frailty (Wood et al., 1992) are potential confounding factors in this project because it may not be known if a) all individuals had the same susceptibility to environmental stress and b) some individuals who experienced environmental stress in development may not have markers of those stress events. The research design of this project, primarily through the use of multiple lines of evidence (Larsen, 2002), was selected in part to minimize these effects. The use of multiple lines of evidence removes some of the potential bias given that a convergence of data from different measures along a single path partially controls for some of the effects of selective mortality and frailty (Wood et al., 1992; Wright and Yoder, 2003; Milner et al., 2008). Thirdly, a good understanding of a population’s diet and weaning practices can also control for factors of selective mortality and frailty (Wright and Yoder, 2003). Dietary profiles, via stable isotope analysis, are available for this population (Craig et al., 2009) and were used to show that no age or sex groups showed any clear sign of distinct dietary profiles that may have increases their frailty. The age and process of weaning in the Roman context was discussed as well. Weaning and subsequent diet over the life course are important as they may reveal age specific periods of stress, brought on by weaning or other periods of dietary transition. The dietary and weaning considerations were supplemented with detailed cultural information of Roman daily life gathered from analyses of historical documents as well as the particular archaeology of Velia and of the Imperial Roman period in general. Culture has an important role in determining hidden heterogeneity and its effects on selective mortality (Wright



and Yoder, 2003) and it is argued that the biocultural emphasis of this project helped alleviate some of the concerns raised by Wood et al. (1992).

It should also be acknowledged that the time period of the Velia necropolis spans the 1<sup>st</sup> and 2<sup>nd</sup> centuries A.D., which may introduce further variability as biosocial factors may have changed over time. However, the community of Velia examined here most likely represents a close-knit subgroup based on burial styles and archaeological assessment (Bondioli, 2011, personal communication). Roman society was also very complex (Garnsey, 1999; Toner, 2002; 2009; Aldrete, 2004), including the full range of gendered occupations. It is then very difficult to say what any individual may have been doing throughout life in specific terms, and we must rely on general knowledge of age and gender related activities. Further, life expectancy in the past may be considered a confounding factor in investigations of age-related disease processes, but it should be noted that low life expectancy in the historic period was primarily related to high infant mortality. For those who survived infancy in the Imperial Roman period, many would have lived past 50 to experience age-related bone loss (Harlow and Laurence, 2002).

Perhaps the most significant bias that bioarchaeology faces today is in the assessment of age-determination (Jackes, 2011). Our methods are still inaccurate, particularly for individuals past 50 years of age (Jackes, 2011). Bias can enter in a number of ways, including biases within original reference populations, preservation, and the fact that some skeletal indicators may change in usefulness between populations (Jackes, 2011). In order to increase accuracy, at the cost of precision, broad age categories were used in this study, but many of the biases Jackes (2011) highlights remain and are fully acknowledged. Furthermore, in a study of bone loss such as this, it is recognized also that much of the changes we see clinically occur after the age of 50, and so those subtle changes are lost when older individuals are grouped into a 50+ category. However, an attempt to create more refined age groups past 50 years would be deeply flawed based on available methods (Jackes, 2011). Consequently, some of the age-related changes that occur in older individuals may be washed out, but the changes that occur in Type I (post-menopausal) osteoporosis should be visible to bioarchaeological testing.

Finally, not much is known regarding the specific archaeological contexts of each burial (pers.comm, Bondioli, 2011). If this information becomes available, it would be interesting to see if mortuary patterns correlated with measures of bone health. However, based on current knowledge of the Velia sample, there is no expectation that large status or social differences would be manifested in burial styles. No association was noted for sex or age with tomb type (Craig et al., 2009) and the population seems to be very tight knit (pers.comm, Bondioli, 2011). Dietary patterns were also distributed essentially evenly across the group (Craig et al., 2009) and sex differences were not pronounced. While the existing lines of evidence all suggest that social differences would have no measurable effect on measures on bone maintenance, it is something worth testing further if the opportunity arises.

## Summary

The primary goal of this study was to provide a comprehensive bioarchaeological investigation of the Velia population using a multi-method, life course approach to the study of bone loss in the past. A number of secondary goals were also explored. Specifically, the life course perspective used in this study was supplemented with examinations of skeletal growth profiles and both juvenile and adult stress markers.

The multi-method approach revealed some important findings regarding bone health at Velia. First, the Velia population was similar to modern populations in many ways. Patterns of cortical bone deposition followed expected differences that result from the divergent hormonal pathways between the sexes. For example, males had more periosteal deposition, while females had smaller endosteal areas from the influence of estrogen during growth and development. In addition, the radiogrammetry and histomorphometry analyses showed significant age-related changes. Many of the changes in the measures of trabecular architecture were also consistent with modern populations.

A number of observations distinguish the patterns described for Velia. For example, trabecular bone volume did not decrease significantly with age, which is expected clinically. In addition, only one female and one male with clear signs of osteoporosis related fragility fractures could be identified. But perhaps the most meaningful observation has been that no statistically significant sex differences were recorded, except for one measure of trabecular bone (ConnD). This is an important finding as it adds to existing bioarchaeological observations that sex differences in the past should not be taken as a priori assumptions (Agarwal, 2008; Agarwal, 2011). This research highlights the fact that bone loss is complex and can defy expectations across dramatically different biocultural contexts, like we see between Velia and modern populations.

Velia also differed from other Imperial Roman sites, particularly Isola Sacra. The pathological and stress markers at Velia were very consistent with other non-elite Imperial Roman sites. Unfortunately, many of these studies do not have data on age and sex related bone loss, so it is not known yet how those similar biocultural contexts may have translated into patterns of bone loss between the sites. The information on bone loss that is available for Isola Sacra, a more middle-class population, shows that patterns of bone loss differed from Velia. Cortical bone remodeling in the ribs was advanced at Isola Sacra compared to Velia, although changes in cortical quantity were more similar. Analyses of trabecular architecture for Isola Sacra are still pending and radiogrammetry work is expected as well. It is hoped that a more complete comparison of bone maintenance and health between the sites may help explain how the slightly different biocultural contexts may have contributed to different patterns of bone loss and what that might mean for bioarchaeological investigation of other populations. Much more work is needed on Roman period skeletons in general (Bondioli and Macchiarelli, 1999; Paine et al., 2009) and this research has contributed substantially towards that goal.

The multi-method approach used to investigate bone loss in the past has made two substantial achievements. The use of multiple methods can overcome some of the deficiencies inherent in bioarchaeological analyses, including biases from poor preservation and small sizes. Reconstructions of bone loss in the past will stand on much firmer ground if multiple lines of

evidence can be juxtaposed and play off of each other. Second, the multi-method approach also helped contextualize changes in bone over the life course and was interpreted in light of historical and archaeological reconstructions of Roman biocultural contexts.

Overall, the pattern of bone maintenance and loss at Velia mesh very well with what historical reconstructions of Roman life. Dietaries deficiencies in the Roman world (Garnsey, 1998) were mirrored in the high prevalence of stress markers throughout the skeleton and across the life course. It seems nutritional stress may have extended to beyond the difficult weaning years. The stress markers also highlighted the negative impact of living in close and unsanitary quarters, as parasites and pathogens contributed to the high incidences of stress markers and pathology. This fits very well with biocultural contexts outlined by numerous Roman scholars (Garnsey, 1998; 1999; Aldrete, 2004; Toner, 2002) and in other Imperial Roman populations (Salvadei et al., 2001; Facchini et al., 2004; Cuccina et al., 2006; Paine et al., 2009). It is unclear however, if stress early in life had any measurable effect on bone remodeling parameters in adulthood.

Reproductive history of Roman women has been reviewed by a number of Roman scholars (Leftkowitz and Fant, 1982; Garnsey and Saller, 1987; Garnsey, 1998; Harlow and Laurence, 2002). The general pattern of high parity and extended periods of breastfeeding in the past has been hypothesized to protect women (Agarwal et al., 2004) from more severe bone loss observed in modern populations. The life course approach used in the research supports this hypothesis. However, the weight of the skeletal evidence suggests that physical activity was probably the most important biocultural factor that was responsible for the observed differences between Velia and modern populations. All three primary methods provided compelling evidence to suggest that day-to-day life was strenuous and that this mediated bone loss with age, particularly in females. Reproductive history and physical activity in all likelihood played a significant role in limiting observable sex differences with age.

# Conclusion

## Future Directions

This dissertation has provided a comprehensive analysis of bone health in the Velia population, but new avenues can be pursued to improve our knowledge of this archaeological community. An important new direction would be to explore the cross-sectional geometry of lower and upper limbs to further explore the role physical activity played in maintaining bone health. It was hypothesized in this research that the predominant biocultural factor that helped protect against bone loss was physical activity. Cross-sectional geometry is well poised to explore bone strength in more biomechanically active bones (such as the femur and humerus) (Ruff, 2008). The health of juveniles is also an area where more refined would be useful for the Velia population. Future work should explore stress markers (such as cribra orbitalia, porotic hyperostosis and periostitis) in conjunction with skeletal growth profiles. This approach has been shown to provide a much more detailed reconstructions of general health status in childhood, as well as improving interpretations of skeletal growth profiles themselves (Wheeler, 2009). Finally, this research has focused on a life course perspective to investigate bone loss, but the life course perspective can be implemented to explore a more social bioarchaeology (Agarwal and Glencross, 2011). For example, aging should be explored as a social process (Sofaer, 2011) in the Velia community to better understand what aging meant on a social level in Roman society. The bioarchaeology of childhood (Lewis, 2007) is also an important emerging research focus and one that would benefit from life course approaches.

## Contributions to Bioarchaeology

This dissertation contributes to biological anthropology by advancing the dialogue on life course approaches and the study of bone health in the past. Specifically, this research has demonstrated that life course approaches should incorporate a developmental plasticity perspective to better understand how early life experiences shape adult skeletal morphology and variation. This dissertation also explored the link between environmental stress in development and bone remodeling in adulthood. The effects of early life stress in the Velia population did not seem to dramatically advance bone loss in adulthood. Although the growth period was physiologically stressful for juveniles at Velia, patterns of bone maintenance and loss in adults reflect patterns that are no worse than modern populations. In fact, bone loss in females is arguably less severe at Velia than for modern populations. One significant line of evidence for this is the observation that no sex differences were noted for cortical index of the metacarpal, bone volume (BV/TV) of L4 vertebrae and for rib remodeling measures. Females today are at far greater risk of osteoporosis than males (NOF, 2011) and this pattern was not seen for the Velia population. The life course perspective used in this work helped to establish that reproductive history, and life-long strenuous physical activity in particular, were likely responsible for key differences observed between Velia and modern populations. Consequently, this research has helped demonstrate the modern pattern of post-menopausal bone loss in females is situated in

modern cultural contexts and that this pattern should not be expected for other populations with vastly different cultures.

This project also made substantial methodological contributions to bioarchaeology by demonstrating the utility of using multiple methods to examine both developmental stress and bone maintenance and loss in adulthood. Studies using single methods to assess bone loss cannot reflect the complexity and heterogeneity of bone throughout the body. This dissertation has demonstrated the utility of selecting multiple methods that reflect both cortical and trabecular bone, in multiple areas throughout the skeleton. In this study, all lines of evidence supported each other, but this should not be assumed for other populations. Ultimately, the use of multiple methods to explore tissue-level changes, interpreted within a biocultural framework, greatly expands interpretive possibilities (Wright and Yoder, 2003).

Lastly, the bioarchaeology of ancient Rome is also advanced with this project. There has been some excellent recent work on the bioarchaeology of the Roman world (Dupras et al., 2001; Salvadei et al., 2001; Facchini et al., 2004; Prowse et al., 2005; Cuccina et al., 2006; Belcastro et al., 2007; Cho and Stout, 2011) but Killgrove (2005) and Paine et al. (2009) have argued that compared to the archaeological and historical information available on Roman life, bioarchaeology still has much to contribute. This project contributes meaningfully to Roman bioarchaeology by providing a comprehensive examination of bone maintenance and loss in adults and frames this analysis within life course and biocultural perspective.

## References

- Abelow, BJ., Holford, TR and Insogna, KL. 1992. Cross-Cultural Association Between Dietary Animal Protein and Hip Fracture: A Hypothesis. *Calcified Tissue International* 50: 14-18.
- Acsádi G and Nemeskéri J. 1970. *History of human life-span and mortality*. Akadémiai Kiadó: Budapest.
- Adkins, L and Adkins, RA. 1994. *Handbook to Life in Ancient Rome*. New York: Facts on File.
- Adami, S., Gatti, D., Rossini, M., Adamoli, A., James, G., Girardello, S and Zamberlan, N. 1992. The Radiological Assessment of Vertebral Osteoporosis. *Bone* 13: s33-s36.
- Adami S, Zamberlan N, Gatti G, Zanfisi C, Braga V, and Broggin M. 1996. Computed radiographic absorptiometry and morphometry in the assessment of post-menopausal bone loss. *Osteoporosis International* 6: 8–13.
- Affinito P, Tommaselli GA, di Carlo C, Guida F, and Nappi C. 1996. Changes in bone mineral density and calcium metabolism in breastfeeding women: a one year follow-up study. *Journal of Clinical Endocrinology and Metabolism* 81: 2314–2318.
- Agarwal SC. 2008. Light and Broken Bones: Examining and Interpreting Bone Loss and Osteoporosis in Past Populations. In *Biological Anthropology of the Human Skeleton* 2nd Edition, Katzenberg MA and Saunders SC (eds.). Wiley-Liss: New York; 387-410.
- Agarwal, SC. 2011. The past of sex, gender, and health: bioarchaeology of the aging skeleton. *American Anthropologist*. In press.
- Agarwal SC and Beauchesne P. 2011. It is Not Carved in Bone: Development and Plasticity of the Aged Skeleton. In *Social Bioarchaeology*, Agarwal SC and Glencross B (eds.). Wiley-Blackwell: New York; 312-332.
- Agarwal SC, Dumitriu M, Tomlinson GA, and Grynepas MD. 2004. Medieval trabecular bone architecture: The influence of age, sex, and lifestyle. *American Journal of Physical Anthropology* 124: 33-44.
- Agarwal, SC and Grynepas, MD. 1996. Bone Quantity and Quality in Past Populations. *Anatomical Record* 246: 423-432.
- Agarwal SC, and Grynepas M. 2009. Measuring and interpreting age-related loss of vertebral bone mineral density in a medieval population. *American Journal of Physical Anthropology* 139: 244-252.
- Agarwal SC, Stuart-Macadam P. 2003. An Evolutionary and Biocultural Approach to Understanding the Effects of Reproductive Factors on the Female Skeleton. In *Bone Loss and*

- Osteoporosis: An Anthropological Perspective, Agarwal SC and Stout SD (eds.). Kluwer Academic/Plenum Publishers: New York; 105-120.
- Alcock, SE and Osborne, R. 2007. Eds., *Classical Archaeology*. Blackwell Publishing.
- Aldrete, GA. 2004. *Daily Life in the Roman City: Rome, Pompeii, and Ostia*. Connecticut: Greenwood Press.
- Anderson, C. 1982. *Manual for the Examination of Bone*. Boca Raton: CRC Press
- Angel, L.A. (1969). The bases of paleodemography. *American Journal of Physical Anthropology* 30: 427-438.
- Arendt, JF. 1997. Adaptive Intrinsic Growth Rates: An Integration Across Taxa. *The Quarterly Review of Biology* 72(2): 149-177.
- Armelagos, G.J., Mielke, J.H., Owen, K.H. and Van Gerven, D.P. 1972. Bone growth and development in prehistoric populations from Sudanese Nubia. *Journal of Human Evolution* 1: 89-119.
- Armelagos GJ and VanGerven, DP. 2003. A Century of Skeletal Biology and Paleopathology: Contrasts, Contradictions, and Conflicts. *American Anthropologist* 105(1): 53-64.
- Armelagos, GJ, and KN Harper. 2005. Genomics at the Origins of Agriculture, Part Two. *Evolutionary Anthropology* 14:109-121.
- Armelagos, GJ, Goodman, AH., Harper, KN., and Blakey, ML. 2009. Enamel hypoplasia and early mortality: Bioarchaeological support for the Barker hypothesis. *Evolutionary Anthropology* 18: 261-271.
- Armstrong, D.V. and Fleischman, M.L. 2003. House-yard burials of enslaved laborers in eighteenth-century Jamaica. *International Journal of Historical Archaeology* 7: 33- 65.
- Aspray, TJ., Prentice, A., Cole, TJ., Sawo, Y., Reeve, J., and Francis, RM. 1996. Low bone mineral content is common but osteoporotic fractures are rare in elderly rural Gambian women. *Journal of Bone and Mineral Research* 11(7): 1019-1025.
- Aufderheide, A.C. and Rodríguez-Martín, C. 1998. *The Cambridge Encyclopedia of Human Paleopathology*. Cambridge, Cambridge University Press.
- Baker, PT. 1984. The Adaptive Limits of Human Populations. *Man* 19(1): 1-14.
- Baker, PT., Hanna, JM and Baker, TS. 1986. *The Changing Samoans: Behaviour and Health in Transition*. Oxford: Oxford University Press.

- Barnett E and Nordin BEC. 1960. The radiological diagnosis of osteoporosis: A new approach. *Clinical Radiology* 11: 166-174.
- Barak, MM., Lieberman, DE. and Hublin, JJ. 2011. A Wolff in sheep's clothing: Trabecular bone adaptation in response to changes in joint loading orientation. *Bone* 49: 1141-1151.
- Barker, D. 1998. *Mothers, Babies and Health Later in Life*, 2<sup>nd</sup> ed. Edinburgh: Churchill Livingstone.
- Barker, D. 2001. Fetal and infant origins of adult disease. *Monatsschrift Kinderheilkunde* (Suppl. 1) 149: s2-s6.
- Barrett, AR and Blakely, ML. 2011. Life histories of enslaved Africans in colonial New York: A bioarchaeological study of the New York African Burial Ground. . In *Social Bioarchaeology*, Agarwal SC and Glencross B (eds.). Wiley-Blackwell: New York; 212-251.
- Bathurst, RR. 2005. Archaeological evidence of intestinal parasites from coastal shell middens. *Journal of Archaeological Science* 32: 115-123.
- Baxter, J. H. 1875 *Statistics, Medical and Anthropological, of over a Million Recruits*. Washington, DC: Government Printing Office.
- Beall, CM and Steegmann Jr, AT. 2000. Human Adaptation to Climate: Temperature, Ultraviolet Radiation, and Altitude. In: Stinson, S., Bogin, B., Huss-Ashmore, R and O'Rourke, D, eds., *Human Biology: An Evolutionary and Biocultural Perspective*. New York: Wiley-Liss. 163-224.
- Beauchesne, P. and Saunders, S. 2006. A test of the revised Frost's rapid manual method for the preparation of bone thin sections. *International Journal of Osteoarchaeology* 16 (1): 82-87.
- Becker, DJ., Killgore, ML., and Morrissey, MA. 2010. The social burden of osteoporosis. *Current Rheumatology Reports* 12: 186-191.
- Ben-Schlomo, Y., and D. Kuh 2002 A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges, and Interdisciplinary Perspectives. *International Journal of Epidemiology* 31:1-9.
- Bencivenga Trillmich C. 1990. Elea: problems of the relation between city and territory. In: *Greek colonists and native populations: proceedings of the First Australian Congress of Classical Archaeology* held in honour of Emeritus Professor A.D. Trendall, Sydney, 9-14 July 1985, Descoeudres JP (ed.). Clarendon Press: Oxford; 365-371.
- Bengston, VL and Allen, KR. 1993. The life course perspective applied to families over time. In: Boss, PG., Doherty, WJ., LaRossa, WR., Scham, WR., Steinmetz, SK., eds. *Sourcebook of Families, Theories, Methods: A Contextual Approach*. New York: Plenum Press. 469-498.



- Bennike, P., Bohr, H and Toft, T. 1993. Determination of mineral content and organic matrix in bone samples using dual photon absorptiometry. *International Journal of Anthropology* 8: 111-116.
- Bergot C, Laval-Jeantet AM, Preteux F, and Meunier A. 1988. Measurement of anisotropic vertebral trabecular bone loss during aging by quantitative image analysis. *Calcified Tissue International* 43:143– 149.
- Bentley, RA., Tayles, N., Higham, C., Macpherson, C., and Atkinson, TC. 2007. Shifting gender relations at Khok Phanom Di, Thailand: Isotopic evidence from the skeletons. *Current Anthropology* 48:301-314.
- Bianco, P and Ascenzi, A. 1993. Paleohistology of Human Bone Remains: A Critical Evaluation and Example of its Use. In Grupe, G and Garland, AN, eds., *Histology of Ancient Human Bone: Methods and Diagnosis*. Berlin: Springer-Verlag. 157-170.
- Birnbaum, E. 1992. Osteoporosis: A Summary of Recent Literature. *Chronic Diseases in Canada* 13(5): 89-95.
- Bisel S. 1988. Nutrition in first century Herculaneum. *Anthropologie*, 26: 61-66.
- Bisel, S and Bisel, JF. 2002. Health and nutrition at Herculaneum: an examination of human skeletal remains. In: Jashemski, WE and Meyer, FG, eds., *The Natural History of Pompeii*. Cambridge: Cambridge University Press. 471-475.
- Blakely, RL, ed. 1977. *Biocultural adaptation in prehistoric America, Southern Anthropological Society Proceedings, No. 11*. Athens: The University of Georgia Press.
- Blakey, M. 2001. Bioarchaeology of the African diaspora in the Americas: Its origin and scope. *Annual Review of Anthropology* 30: 387-422.
- Blom DE, Buikstra JE, Keng L, Tomczak PD, Shore- man E, and Stevens-Tuttle D. 2005. Anaemia and child- hood mortality: latitudinal patterning along the coast of Pre-Columbian Peru. *American Journal of Physical Anthropology* 127: 152–169.
- Boas, F. 1912. Changes in the Bodily Form of Descendants of Immigrants. *American Anthropologist* 14(3): 530-562.
- Bogin, B and Rios, L. 2003. Rapid morphological change in living humans: implications for modern human origins. *Comparative Biochemistry and Physiology Part A* 136: 71-84.
- Bogin, B. 1995 Plasticity in the Growth of Mayan Refugee Children Living in the United States. In: *Human Variability and Plasticity*. C. G. N. Mascie-Taylor, and B. Bogin, eds. Pp. 46–74. Cambridge: Cambridge University Press.
- Bogin, B. 1999. *Patterns of Human Growth*. Cambridge: Cambridge University Press.

- Boldsen, J.L. 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 (1): 1-8.
- Bonjour, JP., Chevalley, T., Ammann, P., Slosman, D and Rizzoli, R. 2001. Gain in bone mineral mass in prepubertal girls 3.5 years after discontinuation of calcium supplementation: a follow up study. *Lancet* 358: 1208-1212.
- Bonjour, J., Chevalley, T and Ferrari, S. 2007. Gene-Environment Interactions in the Skeletal Response to Nutrition and Exercise during Growth. Daly R, Petit M., eds, *Optimizing Bone Mass and Strength. The Role of Physical Activity and Nutrition during Growth. Med Sport Sci.* Basel, Karger, 51: 64-80.
- Bondioli L, Macchiarelli R. 1999. The "Isola Sacra project." In: 1, Rossi PF, Bondioli L, Geusa G, Macchiarelli R (eds). *Digital Archives of Human Paleobiology E-LISA* Sas: Milan.
- Bonfiglioli B., Brasil P. and Belcastro M.G. 2003. Dento-alveolar lesions and nutritional habits of a Rome Imperial age population (1st-4th c. AD): Quadrelli (Molise, Italy). *Homo*, 54:35- 56.
- Bonnick, SL. 2002. Current controversies in bone densitometry. *Current Opinion in Rheumatology* 14: 416-420.
- Boldsen JL. 1998. Body proportions in a medieval village population: effects of early childhood episodes of ill health. *Annals of Human Biology* 25: 309-317.
- Boonen S, Nijs J, Borghs H, Peeters H, Vanderschueren D, and Luyten FP. 2005. Identifying postmenopausal women with osteoporosis by calcaneal ultrasound, metacarpal digital radiogrammetry and phalangeal radiographic absorbtometry: a comparative study. *Osteoporosis International* 16: 93–100.
- Böttcher J, Pfeil A, Schafer ML, Petrovitch A, Seidl BE, Mentzel HJ, Lehmann G, Malich A, Heyne JP, Hein G, Wolf G, and Kaiser WA. 2006. Normative data for digital x-ray radiogrammetry from a female and male german group. *Journal of Clinical Densitometry* 9(3): 341-350.
- Boutroy, S., Bouxsein, ML., Munoz, F and Delmas, PD. 2005. In Vivo Assessment of Trabecular Bone Microarchitecture by High-Resolution Quantitative Computed Tomography. *Journal of Clinical Endocrinology and Metabolism* 90(12): 6508-6515.
- Bouxsein ML and Karasik D. 2006. Bone geometry and skeletal fragility. *Current Osteoporosis Reports* 2: 49-56.
- Bowditch, H. P. 1879 *The Growth of Children, a Supplementary Investigation*, pp. 25-62. Boston, State Board of Health of Massachusetts.

- Brickley, M. 1998. *Age-related bone loss and osteoporosis in archaeological bone: A study of two London collections, Redcross Way and Farrington Street*. Doctoral thesis, University of London, London.
- Brickley M and Agarwal SC. 2003. Techniques for the Investigation of Age-Related Bone Loss and Osteoporosis in Archaeological Bone. In: Agarwal SC and Stout SD (eds.), *Bone Loss and Osteoporosis: An Anthropological Perspective*. Kluwer Academic/Plenum Publishers: New York; 157-172.
- Brickley M, Ives R. 2008. *The Bioarchaeology of Metabolic Bone Disease*. Elsevier: London.
- Bridges PS. 1991. Skeletal evidence of changes in subsistence activities between the Archaic and Mississippian time periods in northwestern Alabama. In: Powell ML, Bridges PS, Mires AMW, editors. *What Mean These Bones: Studies in Southeastern Bioarchaeology*. Tuscaloosa: University of Alabama Press. pp. 89 – 101.
- Brooks S and Suchey JM. 1990. Skeletal age determination based on the os pubis, a comparison of the Acsádi-Nemeskéri and Suchey-Brooks methods. *Human Evolution* 5: 227–238.
- Brothwell DR. 1981. *Digging up bones*. Oxford University Press: London.
- Brothwell, D. 1988. Foodstuffs, cooking, and drugs. In: Grant, M and Kitzinger, R., eds. *Civilization of the Ancient Mediterranean*. New York: Charles Scribner's Sons. 247-261.
- Brunader, R and Shelton, DK. 2002. Radiologic Bone Assessment in the Evaluation of Osteoporosis. *American Family Physician* 65(7): 1357-1364.
- Brunt PA. 1980. Free Labour and Public Works at Rome. *The Journal of Roman Studies* 70: 81-100.
- Buikstra JE, and Ubelaker DH. 1994. *Standards for data collection from human skeletal remains*. Arkansas Archaeological Survey Research Series No. 44: Fayetteville, Arkansas.
- Bunson M. 1991. *A Dictionary of the Roman Empire*. Oxford University Press, New York.
- Burr, DB, Piotrowski, G and Miller, G. 1981. Structural Strength of the Macaque Femur. *American Journal of Physical Anthropology* 54: 305-319.
- Burr, DB and Piotrowski, G. 1982. How do trabeculae affect the calculation of structural properties of bone? *American Journal of Physical Anthropology* 57: 341-352.
- Burr, DB., Ruff, CB and Thompson, DD. 1990. Patterns of Skeletal Histologic Changes Through Time: Comparison of an Archaic Native American Population with Modern Populations. *Anatomical Record* 226: 307-313.

- Burr, DB and Turner, CH. 1999. Biomechanical measurement in age-related bone loss. In: Rosen, CJ, Glowacki, J and Bilizekian, JP, eds. *The Aging Skeleton*. San Diego: Academic Press: 301-311
- Burr, DB. 2002. Targeted and non-targeted remodeling. *Bone* 30(1): 2-4.
- Burr, DB. 2004. Bone Quality: Understanding what matters. *Journal of Musculoskeletal and Neuronal Interactions* 4(2): 184-186.
- Cameron, N., and E. W. Demerath 2002 Critical Periods in Human Growth and their Relationship to Diseases of Aging. *American Journal of Physical Anthropology* 119:159–184.
- Cameron, N. 2006. Growth Patterns in Adverse Environments. *American Journal of Physical Anthropology* 129: 30. Abstract.
- Capasso, L. 2007. Infectious Diseases and Eating Habits at Herculaneum (1<sup>st</sup> Century AD, Southern Italy). *International Journal of Osteoarchaeology* 17: 350-357.
- Carballido-Gamio, J and Majumdar, S. 2006. Clinical utility of microarchitecture measurements of trabecular bone. *Current Osteoporosis Reports* 4: 64-70.
- Cardoso, H. 2007. Environmental Effects on Skeletal Versus Dental Development: Using a Documented Subadult Skeletal Sample to Test a Basic Assumption in Human Osteological Research. *American Journal of Physical Anthropology* 132: 223-233.
- Carli-Thiele P and Schultz M. 1997. Microscopic differential diagnosis of so called cribra orbitalia - a contribution to the etiology of orbital porotic hyperostosis. *American Journal of Physical Anthropology* (Suppl 24): 88.
- Cashman K. 2002. Calcium intake, calcium bioavailability and bone health. *British Journal of Nutrition*. 87: S169–77.
- Cashman K. 2007. Diet, nutrition, and bone health. *The Journal of Nutrition* 137: 2507S-2512S.
- Casson, L. 1998. *Everyday Life in Ancient Rome*. Baltimore and London: John Hopkins University Press.
- Center, J and Eisman, J. 1997. The Epidemiology and Pathogenesis of Osteoporosis. *Ballieres Clinical Endocrinology and Metabolism* 11 (1): 23-62.
- Chamberlain, A. 2000. Problems and Prospects in Paleodemography. In: Cox, M and Mays, S., eds., *Human Osteology in Archaeology and Forensic Science*. Cambridge: University Press. 101-116.
- Cho, H and Stout, SD. 2003. Bone Remodeling and Age-Associated Bone Loss in the Past: A Histomorphometric Analysis of the Imperial Roman Skeletal Population of Isola Sacra. In:

Agarwal, SC and Stout , SD, eds., *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 207-228.

Cho H, Stout SD. 2011. Age-associated bone loss and intraskeletal variability in the Imperial Romans. *Journal of Anthropological Sciences* 89: 1-17.

Christodoulou, C and Cooper, C. 2003. What is osteoporosis? *Postgraduate Medical Journal* 79: 133-138.

Clark G. 1988. New method for assessing changes in growth and sexual dimorphism in paleoepidemiology. *American Journal of Physical Anthropology* 77(1): 105-116.

Clark, G., Hall, N., Armelagos, G., Borkan, G, Panjabi, M., and Wetzel, F. 1986. Poor growth prior to early childhood: Decreased health and life-span in the adult. *American Journal of Physical Anthropology* 70:145-160.

Cohen, M.N. & Armelagos, G.J., Eds. 1984. *Paleopathology at the Origins of Agriculture*. New York, Academic Press.

Country, GR., Capozza, RF (ex-aequo)., Negri, AL., Roldán, EJA and Ferretti, JL. 2004. Biomechanical background for a noninvasive assessment of bone strength and muscle-bone interactions. *Journal of Musculoskeletal and Neuronal Interactions* 4(1): 1-11.

Compston J. 1999. Histomorphometric Manifestations of Age-Related Bone Loss. In *The Aging Skeleton*, Rosen CJ, Glowacki J, Bilezikian JP (eds.). Academic Press: San Diego; 251-262.

Conheaney, J. 2000. Inhumation Burials. In: Barber, B and Bowsher, D. *The Eastern Cemetery of Roman London*, pp. 277-295. London: Museum of London Archaeological Service, monograph 4.

Connolly, P and Dodge, H. 1998. *The Ancient City: Life in Classical Athens and Rome*. Oxford: Oxford University Press.

Cooper, C. Campion, G and Melton III, LJ. 1992: Hip Fractures in the Elderly: A World-Wide Projection. *Osteoporosis International* 2: 285-289.

Cooper, C. 1993. The epidemiology of fragility fractures: is there a role for bone quality? *Calcified Tissue International* 53(Suppl 1): S23-S26.

Cooper, C., M. I. D. Cawley, and A. Bhalla 1995. Childhood Growth, Physical Activity and Peak Bone Mass in Women. *Journal of Bone and Mineral Research* 10:940-947.

Cooper, C., Atkinson, EJ., Hensrud, DD., Wahner, HW., O'Fallon, WM., Riggs, RL and Melton III, LJ. 1996. Dietary Protein Intake and Bone Loss in Women. *Calcified Tissue International* 58: 320-325.

Cooper, C., Fall, C., Egger, P., Hobbs, R., Eastell, R and Barker, D. 1997. Growth in infancy and bone mass in later life. *Annals of the Rheumatic Diseases* 56: 17-21.

Cooper C, Eriksson JG, Forsen T, Osmond C, Tuomilehto J, and Barker DJP. 2001. Maternal height, childhood growth and risk of hip fracture in later life: a longitudinal study. *Osteoporosis International* 12(8): 623-629.

Cooper C, Westlake S, Harvey N, Javaid K, Dennison E, and Hanson M. 2006. Review: developmental origins of osteoporotic fracture. *Osteoporosis International* 17 (3): 337-347.

Cooper, DML., Clement, JG., Thomas, CDL., Hallgrímsson, B., Turinsky, AL., Sensen, CW and Goldman, HM. 2008. Advances in high resolution imaging and the emerging application of 3D cortical bone histomorphometry in interpreting health. *American Journal of Physical Anthropology* 135: 81. Abstract.

Coulston, J and Dodge, H. 2000. Introduction: the archaeology and topography of Rome. In: Coulston, J and Dodge, H., eds. *Ancient Rome*. Oxford: Oxbow Books. 1-15.

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

Creighton, M. 2000. *A Primer History of Rome*. Nashville: Wimbledon Publishing.

Crowder, C and Stout, S. 2012. *Bone Histology: An Anthropological Perspective*. Boca Raton: CRC Press.

Crowe F, Sperduti A, O'Connell TC, Craig OE, Kirsanow K, Germoni P, Macchiarelli R, Garnsey P, and Bondioli L. 2010. Water-related occupations and diet in two Roman coastal communities (Italy, first to third century AD): Correlation between stable carbon and nitrogen isotope values and auricular exostosis prevalence. *American Journal of Physical Anthropology* 142: 355-366.

Cuccina, A., Vargiu, R., Mancinelli, D., Ricci, R., Santandrea, E., Catalano, P and Coppa, A. 2006. The Necropolis of Vallerano (Rome, 2<sup>nd</sup>-3<sup>rd</sup> Century AD): An Anthropological Perspective on the Ancient Romans in the Suburbium. *International Journal of Osteoarchaeology* 16: 104-117.

Cummings R and Klineberg R. 1993. Breast-Feeding and Other Reproductive Factors and the Risk of Hip-Fractures in Elderly Women. *International Journal of Epidemiology* 22: 684-691.

Cummings RG, Klineberg RJ. 1994. Case-control study of dairy product consumption and risk of hip fracture. *American Journal of Epidemiology* 139:S2.

- Cummings SR, Nevitt MC, Browner WS, Stone, K., Fox, K., Ensrud, KE., Cauley, J., Black, D., and Vogt, TM. 1995. Risk factors for hip fracture in white women. *New England Journal of Medicine* 332: 767–73.
- Cummings, SR., Bates, D and Black, DM. 2002. Clinical Use of Bone Densitometry. *Journal of the American Medical Association* 268(15): 1889-1897.
- Cummings, C. 2008. Dietary Practices in Roman Britain: The evidence from carbon and nitrogen stable isotopes. *American Journal of Physical Anthropology* 135: 85. Abstract.
- Currey, J. 1984. The mechanical adaptations of bone. In: Currey, J., ed. *The mechanical adaptations of bones*. Princeton: Princeton University Press. 88-97.
- Damilakis, J., Maris, TG and Karantanas, AH. 2007. An update on the assessment of osteoporosis using radiologic techniques. *European Radiology* 17: 1591-1602.
- Daniels, ED., Pettifor, JM., Schnitzler, CM., Russell, SW., and Patel, DN. 1995. Ethnic differences in bone density in female South African nurses. *Journal of Bone and Mineral Research* 10(3): 359-367.
- Dawson-Hughes B. 1991. Calcium supplementation and bone loss: a review of controlled clinical trails. *American Journal of Clinical Nutrition* 54:274S–280S.
- Dennison, E. M., H. E. Syddall, S. Rodriguez, A. Voroppanov, I. N. Day, and C. Cooper, 2004 Polymorphism in the Growth Hormone Gene, Weight in Infancy, and Adult Bone Mass. *Journal of Clinical Endocrinology and Metabolism* 89:4898–4903.
- Dennison, E. M., H. E. Syddall, A. Sayer, H. Gilbody, and C. Cooper 2005 Birth Weight and Weight at 1 Year are Independent Determinants of Bone Mass in the Seventh Decade: The Hertfordshire Cohort Study. *Pediatric Research* 5:582–586.
- Dempster, DW. 2002. Bone Remodeling. In: Coe, FL and Favus, MJ, eds., *Disorders of Bone and Mineral Metabolism*. Philadelphia, London : Lippincott Williams & Wilkins. 315-343.
- Dempster, DW. 2011. Osteoporosis and the burden of osteoporosis-related fractures. *The American Journal of Managed Care* 17(6): s164-s169.
- Dequeker J, Ortner DJ, Stix AL, Cheng X-G, Brys P, and Boonen S. 1997. Hip fracture and osteoporosis in a XIIth dynasty female skeleton from Lisht, Upper Egypt. *Journal of Bone and Mineral Research* 12:881–888.
- DeWitte, SN. 2010. Sex differentials in frailty in Medieval England. *American Journal of Physical Anthropology* 143: 285-297.

- Dey A, McCloskey EV, Taube T, Cox R, Pande KC, Ashford RU, Forster M, de Takats D, Kanis JA. 2000. Metacarpal morphometry using a semi-automated technique in the assessment of osteoporosis and vertebral fracture risk. *Osteoporosis International* 11: 953–958.
- Dixon S. 2001. *Reading Roman Women*. Duckworth: London.
- Djurić-Srejić, M, and C Roberts. 2001. Palaeopathological evidence of infectious disease in skeletal populations from later medieval Serbia. *International Journal of Historical Archaeology* 11(5):311-320.
- Dobzhansky, T. 1957 *Evolution, Genetics and Man*. New York: John Wiley.
- Dressler, WW. 1995. Modeling Biocultural Interactions: Examples from Studies of Stress and Cardiovascular Disease. *Yearbook of Physical Anthropology* 38: 27-56.
- Dressler, WW. 2005. What's Cultural about Biocultural Research? *Ethos* 33(1): 20-45.
- Drusini, AG., Bredariol, S., Carrara, N., and Bonati, MR. 2000. Cortical Bone Dynamics and Age-related Osteopenia in a Longobard Archaeological Sample from Three Graveyards in the Veneto Region (Northeast Italy). *International Journal of Osteoarchaeology* 10: 268-279.
- Duan Y, Seeman E, Turner CH. 2001. The biomechanical basis of vertebral body fragility in men and women. *Journal of Bone Mineral Research*. 12: 2276– 2283.
- Dufour, DL. 2006. Biocultural Approaches in Human Biology. *American Journal of Human Biology* 18: 1-9.
- Duncan, E., L. Cardon, J. Sinsheimer, J. Wass, and M. Brown 2003 Site and Gender Specificity of Inheritance of Bone Mineral Density. *Journal of Bone and Mineral Research* 18:1531–1538.
- Dupont, F. 1993. *Daily Life in Ancient Rome*. Oxford: Blackwell.
- Dupras T and Tocheri M. 2007. Reconstructing infant weaning histories at Roman period Kellis, Egypt using stable isotope analysis of dentition. *American Journal of Physical Anthropology*. 134: 63-74.
- Durkheim, E. 1951. *Suicide*. Translation J Spaulding and G Simpson. New York: Free Press.
- Eisman, JA. 1999. Genetics of osteoporosis. *Endocrine Reviews* 20(6): 788-804.
- Ekenman I, Eriksson SA, and Lindgren JU. 1995. Bone density in medieval skeletons. *Calcified Tissue International* 56: 355–358.
- Elders PJM, Lips P, Netelenbos JC, Van Ginkel FC, Khoe E, Van Der Vijgh WJF, and Van Der Stelt PF. 1994. Long-term effect of calcium supplementation of bone loss in perimenopausal women. *Journal of Bone and Mineral Research* 9:963–970.



Elder, G., Johnson, MK., and Crosnoe, R. 2003. The emergence and development of life course theory. In: Lee, BJ., Mortimer, JT., Shanahan, MJ., eds. *Handbook of the Life Course*. New York: Plenum Press. 3-19.

Ellison, P. 2005 Evolutionary Perspectives on the Fetal Origins Hypothesis. *American Journal of Human Biology* 17:113–118.

Ellison, PT. 2006. Pathology, Constraint and Adaptation: how can we tell them apart? *American Journal of Physical Anthropology* 129: 31. Abstract.

Englund, U., Nordstrom, P., Nilsson, J., Bucht, G., Bjornstig, U., Hallmans, G., Svensson, O., and Pettersson, U. 2011. Physical activity in middle-aged women and hip fracture risk: the UFO study. *Osteoporosis International* 22: 499-505.

Erdkamp P. 1999. Agriculture, Underemployment and the Cost of Rural Labour in the Roma World. *The Classical Quarterly* 49(2): 566-572.

Eriksen, MF. 1976. Cortical Bone Loss with Age in Three Native American Populations. *American Journal of Physical Anthropology* 45: 443-452.

Eriksen, MF. 1980. Patterns of Microscopic Bone Remodeling in Three Aboriginal American Populations. In: Browman, DL, ed., *Early Native Americans: Prehistoric Demography, Economy and Technology*, 239-270. The Hague: Mouton Publishers.

Erickson, GM., Catanese III, J and Keaveny, TM. 2002. Evolution of the Biomechanical Material Properties of the Femur. *The Anatomical Record* 268: 115-124.

Facchini, F., Rastelli, E., and Brasili, P. 2004. Cribra orbitalia and cribra cranii in Roman skeletal remains from the Ravenna Area and Rimini (I-IV century AD). *International Journal of Osteoarchaeology* 14: 126-136.

Farwell, DE and Molleson, T. 1993. *Poundbury Vol. 2: The Cemeteries*. Dorset Natural History and Archaeological Society: Monograph Series 11.

Fausto-Sterling, A. 2005. The Bare Bones of Sex: Part 1- Sex and Gender. *Journal of Women in Culture and Society* 30(2): 1491-1527.

Feldblum, PJ., Zhang, J., Rich, LE., Fortney, JA and Talmage, RV. 1992. Lactation history and bone mineral density among perimenopausal women. *Epidemiology* 3: 327-251.

Feldsman, MR. 1992. Femur/Stature ratio and estimates of stature in children. *American Journal of Physical Anthropology* 87: 447-459.

- Felsenberg, D and Boonen, S. 2005. The bone quality framework: Determinants of bone strength and their interrelationships, and implications for osteoporosis management. *Clinical Therapeutics* 27(1): 1-11.
- Ferretti, JL. Capozza, RF., Cointry, JR., Capiglioni, R., Roldan, EJA and Zanchetta, JR. 2000. Densitometric and tomographic analyses of musculoskeletal interaction in bone. *Journal of Musculoskeletal and Neuronal Interactions* 1: 31-34.
- Ferretti, JL., Cointry, JR., Capozza, RF and Frost, H. 2003. Bone mass, bone strength, muscle-bone interactions, osteopenias and osteoporoses. *Mechanisms of Aging and Development* 124: 269-279.
- Feskanich, D., Willet, WC., Stampfer, MJ and Colditz, GA. 1996. Protein Consumption and Bone Fractures in Women. *American Journal of Epidemiology* 143 (5): 472-479.
- Fiammenghi CA. 2003. La Necropoli di Elea-Velia: qualche osservazione preliminare. In Elea-Velia. Le Nuove ricerche, Quaderni del Centro Studi Magna Grecia 1 (ed.). Pozzuoli: Italy; p 49–61.
- Fields, AJ., Eswaran, SK., Jekir, MG., and Keaveney, TM. 2009. Role of trabecular microarchitecture in whole-vertebral body biomechanical behavior. *Journal of Bone and Mineral Research* 24: 1523-1530.
- Fitzgerald, C., Saunders, SR., Bondioli, L and Macchiarelli, R. 2006. Health of Infants in an Imperial Skeletal Roman Sample: Perspective from dental microstructure. *American Journal of Physical Anthropology* 130: 179-189.
- Foldes AJ, Moscovici A, Popovtzer MM, Mogle P, Urman D, and Zias J. 1995. Extreme osteoporosis in a sixth century skeleton from the Negev Desert. *International Journal of Osteoarchaeology* 5:157–162.
- Formicola, V and Giannecchini, M. 1999. Evolutionary trends of Stature in Upper Paleolithic and Mesolithic Europe. *Journal of Human Evolution* 36: 319-333.
- Fox KM, Magaziner J, Sherwin R, Scott JC, Plato CC, Nevitt M, and Cummings S. 1993. Reproductive correlates of bone mass in elderly women. Study of Osteoporotic Fractures Research Group. *Journal of Bone Mineral Research* 8: 901–908.
- Foxhall, L and Forbes, HA. 1982. Sitometria: The role of grain as a staple food in classical antiquity. *Chiron* 12: 41-90.
- Fuller, BT., Molleson, T., Harris, DA., Gilmour, LT and Hedges, REM. 2006. Isotopic Evidence for Breastfeeding and Possible Adult Dietary Differences from Late/Sub-Roman Britain. *American Journal of Physical Anthropology* 129: 45-54.

- Frost, H. 1969. Tetracycline based histological analysis of bone remodeling. *Calcified Tissue International* 3: 211-237.
- Frost HM. 1987a. Secondary osteon populations: an algorithm for determining mean bone tissue age. *Yearbook of Physical Anthropology* 30:221–238.
- Frost, H. 1987b. Secondary Osteon Populations: An Algorithm for Estimating the Missing Osteons. *Yearbook of Physical Anthropology* 30: 239-254.
- Frost HM. 1987c. Bone “mass” and the “mechanostat”: a proposal. *Anatomical Record* 219:1–9.
- Frost, H. 1996. Perspectives: A proposed general model of the mechanostat (suggestions from a new paradigm). *Anatomical Record* 244: 139-147.
- Frost, H. 1997. On our age-related bone loss: insights from a new paradigm. *Journal of Bone Mineral Research* 12:1539–1546.
- Frost, H. 2000. Muscle, bone, and the Utah paradigm: A 1999 overview. *Medicine and Science in Sports and Exercise*, 32(5): 911-917.
- Frost, H. 2001. From Wolff’s Law to the Utah Paradigm: Insights About Bone Physiology and Its Clinical Applications. *Anatomical Record* 262: 398-419.
- Frost, H. 2003. On Changing Views about Age-Related Bone Loss. In: Agarwal, SC and Stout, SD, eds., *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 19-32.
- Gafni, RI., Welse, M., Robrecht, DT et al. 2001. Catch-up growth is associated with delayed senescence of the growth plate in rabbits. *Pediatric Research* 50: 618-623.
- Gafni, RI and Baron, J. 2007. Childhood Bone Mass Acquisition and Peak Bone Mass May Not Be Important Determinants of Bone Mass in Late Adulthood. *Pediatrics* 119: s131-s136.
- Gale, C. R., C. N. Martyn, S. Kellingray, R. Eastell, and C. Cooper 2001 Intrauterine Programming of Adult Body Composition. *Journal of Clinical Endocrinology and Metabolism* 86:267–272.
- Garn S. 1970. *The earlier gain and the later loss of cortical bone, in nutritional perspective*. Thomas: Springfield, Ill.
- Garnsey P, and Saller, R. 1987. *The Roman Empire: Economy, Society and Culture*. University of California Press: Berkeley.
- Garnsey P. 1988. *Famine and Food Supply in the Graeco-Roman World: Responses to Risk and Crisis*. Cambridge University Press: Cambridge.

Garnsey, P. 1998. *Cities, Peasants and Food in Classical Antiquity*. Cambridge University Press: Cambridge.

Garnsey P. 1999. *Food and Society in Classical Antiquity*. Cambridge University Press: Cambridge.

Geller, P. 2008. Conceiving sex: Fomenting a feminist bioarchaeology. *Journal of Social Archaeology* 8(1):113-138.

Genant, HK and Jiang, Y. 2006. Advanced Imaging Assessment of Bone Quality. *Annals of the New York Academy of Sciences* 1068: 410-428.

Glencross, B., Agarwal, SC., Beauchesne, P and Larsen, CS. 2008. Bone fracture patterns and cortical bone loss in an Anatolian Neolithic population. *American Journal of Physical Anthropology* 135: 104. Abstract.

Glencross B and Agarwal SC. 2011. An investigation of cortical bone loss and fracture patterns in the neolithic community of Çatalhöyük, Turkey using metacarpal radiogrammetry. *Journal of Archaeological Science* 38: 513-521.

Glencross, B. 2011. Skeletal injury across the life course: towards understanding social agency. In *Social Bioarchaeology*, Agarwal SC and Glencross B (eds.). Wiley-Blackwell: New York; 390-409.

Gluckman, P and Hanson, M. 2004. *The Fetal Matrix: Evolution, Development and Disease*. Cambridge: Cambridge University Press.

Goldstein, M. S. 1943 *Demographic and Bodily Changes in Descendants of Mexican Immigrants*. Austin: University of Texas, Institute of Latin American Studies.

González-Reimers, E., Velasco-Vázquez, J., Arnay-de-la-Rosa, M., Santolaria-Fernández, F., Gómez-Rodríguez, MA and Machado-Calvo, M. 2002. Double-Energy X-Ray Absorptiometry in the Diagnosis of Osteopenia in Ancient Skeletal Remains. *American Journal of Physical Anthropology* 118: 134-145.

González-Reimers, E., Mas-Pasqual, MA., Arnay-de-la-Rosa, M., Velasco-Vázquez, J., Santolaria-Fernández, F and Machado-Calvo, M. 2004. Noninvasive estimation of bone mass in ancient vertebrae. *American Journal of Physical Anthropology* 125: 121-131.

Goodman, A.H. (1993). On the interpretation of health from skeletal remains. *Current Anthropology* 34, 281-288.

Goodman, A.H. 1996. Early life stresses and adult health: insights from dental enamel development. In (C.J.K. Henry & S.J. Ulijaszek, Eds.) *Long-term Consequences of Early*

*Environment: Growth Development and the Lifespan Developmental Perspective*. Society for the Study of Human Biology Series No. 37. New York, Cambridge University Press, pp. 163-183.

Goodman, A.H., Armelagos, G.J., and Rose, J.C. 1980. Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Human Biology* 52: 515-528.

Goodman, AH and 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 and Rose, JC. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yearbook of Physical Anthropology* 33: 59-110.

Goodman, A and Leatherman, TL. 1998. *Building a New Biocultural Synthesis*. Ann Arbor: University of Michigan Press.

Goodman A.H. and Martin D.L. 2002. Reconstructing health problems from skeletal remains. In R.H. Steckel & J.C. Rose (eds): *The Backbone of History: health and nutrition in the Western Hemisphere*. Cambridge University Press: Cambridge. 11-60.

Gould, SJ and Lewontin, RC. 1979. The spandrels of San Marco and the Panglossian paradigm: a critique of the adaptationist programme. *Proceedings of the Royal Society of London* 205: 581-598.

Gordon, CL., Lang, TF., Augat, P and Genant, HK. 1998. Image-Based Assessment of Spinal Trabecular Bone Structure from High-Resolution CT Images. *Osteoporosis International* 8: 317-325.

Gosman JH, and Ketcham RA. 2009. Patterns in ontogeny of human trabecular bone from SunWatch Village in the Prehistoric Ohio Valley: General features of microarchitectural change. *American Journal of Physical Anthropology* 138: 318-332.

Grandjean P. 1988. Ancient skeletons as silent witnesses of lead exposures in the past. *CRC Critical Reviews in Toxicology* 19(1): 17.

Gray, R. 2001 Selfish Genes or Developmental Systems? In. S. R. Krimbas, D. Paul, and J. Beatty, eds. : *Thinking about Evolution: Historical, Philosophical, and Political Perspectives*. Cambridge: Cambridge University Press. 184-207.

Greco E. 1975. Velia e Palinuro: problemi di topografia antica. *Mélanges de l'École française de Rome, Antiquité* 87: 81-142.

Griffiths P E., and R. Gray 1994 Developmental Systems and Evolutionary Explanations. *Journal of Philosophy* 91:277-304.

Grynepas, MD. 2003. The Role of Bone Quality on Bone Loss and Fragility. In: Agarwal, SC and Stout, SD, eds., *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 33-44.

Guatelli-Steinberg D, and Lukacs J. 1999. Interpreting sex differences in enamel hypoplasia in human and non-human primates: developmental, environmental and cultural considerations. *Yearbook of Physical Anthropology* 42:73-126.

Gurevitch, O and Slavin, S. 2006. The hematological etiology of osteoporosis. *Medical Hypothesis* 67: 729-735.

Haara M, Heliövaara M, Impivaara O, Arokoski JPA, Manninen P, Knekt P, Kärkkäinen A, Reunanen A, Aromaa A, Kröger H. 2006. Low metacarpal index predicts hip fracture: A prospective population study of 3,561 subjects with 15 years of follow-up. *Acta Orthopaedica* 77: 9-14.

Hackett, CJ. 1981. Microscopical Focal Destruction (Tunnels) in Exhumed Human Bones. *Medicine, Science and the Law* 21(4): 243-265.

Hall, J and Merrifield, R. 1986. *Roman London*. London: Her Majesty's Stationery Office & The Museum of London.

Hall, B. 2005. *Bones and Cartilage: Developmental and Evolutionary Skeletal Biology*. San Diego: Elsevier Academic Press.

Hallgrímsson, B., K. Willmore, and B.Hall 2002 Canalization, Developmental Stability, and Morphological Integration in Primate Limbs. Dedicated to the memory of Nancy Hong. *American Journal of Physical Anthropology* 119:131–158.

Hanna, J., Little, MA and Austin, DM. 1989. Climatic Physiology. In: Little, MA and Haas, JD., eds., *Human Population Biology: A Transdisciplinary Science*. Oxford: Oxford University Press. 132-151.

Harlow M, Laurence R. 2002. *Growing Up and Growing Old in Ancient Rome: A life course approach*. Routledge: London.

Harlow M, Laurence R. 2007. Age and Ageing in the Roman Empire, *Journal of Roman Archaeology* Supplementary No. 65. *Journal of Roman Archaeology*: Portsmouth.

Heckster, O. 2006. The Roman Empire. In: Bispham, E., Harrison, T and Sparkes, BA. *The Edinburgh Companion to Ancient Greece and Rome*. Edinburgh: Edinburgh University Press. 108-113.

Hennerberg, M and Hennerberg, RJ. 2002. Reconstructing medical knowledge in ancient Pompeii from the hard evidence of bones and teeth. In: Renn, J and Castagnetti, G, eds., *Homo*

*Faber: Studies on Nature, Technology, and Science at the time of Pompeii*. Rome: “L’Erma” di Bretschneider. 169-187.

Henry JP and Cassel, JC. 1969. Psychological Factors in Essential Hypertension: Recent Epidemiologic and Animal Experimental Evidence. *American Journal of Epidemiology* 90(3): 171-200.

Hermansen, G. 1981. *Ostia: Aspects of Roman City Life*. Edmonton: University of Alberta Press.

Hildebrand T and Rueggegger P. 1997. A new method for the model-independent assessment of thickness in three-dimensional images. *Journal of microscopy*. 185(1): 67–75.

Hillson, S. 1996. *Dental Anthropology*. Cambridge, Cambridge University Press.

Hillson, S. 2000. Dental Pathology. In Katzenburg, MA and Saunders, SR, eds., *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss. 301-340.

Hind, K and Burrows, M. 2007. Weight-bearing exercise and bone mineral accrual in children and adolescents: A review of controlled trials. *Bone* 40(1): 14-27.

Holck P. 2007. Bone mineral densities in the prehistoric, Viking-Age and medieval populations of Norway. *International Journal of Osteoarchaeology*. 17: 199-206.

Hollimon, SE. 2011. Sex and gender in bioarchaeological research: Theory, method, and interpretation. . In *Social Bioarchaeology*, Agarwal SC and Glencross B (eds.). Wiley-Blackwell: New York; 149-182.

Hope, VM and Marshall, E. 2000. *Death and Disease in the Ancient City*. London: Routledge.

Hoppa, R and Fitzgerald, C. 1999. *Human Growth in the Past: Studies from Bones and Teeth*. Cambridge: Cambridge University Press.

Hubbard A, Guatelli-Steinberg D, and Sciulli PW. 2009. Under restrictive conditions, can the widths of linear enamel hypoplasias be used as relative indicators of stress episode duration? *American Journal of Physical Anthropology* 138(2): 177-189.

Hudelmaier, M., Kuhn, V., Lochmüller, EM., Well, H., Priemel, M., Link, TM and Eckstein, F. 2004. Can geometry-based parameters from pQCT and material parameters from quantitative ultrasound (QUS) improve the prediction of radialbone strength over that by bone mass (DXA)? *Osteoporosis International* **15**: 375-381.

Hui, SL., Slemenda, CW., and Johnston, CC. 1988. Age and bone mass as predictors of fracture in a prospective study. *Journal of Clinical Investigation* 81(6): 1804-1809.

Humphrey, LT. 2000. Growth Studies of Past Populations: An Overview and an Example. In: Cox, M and Mays, S., eds., *Human Osteology in Archaeology and Forensic Science*,

23-38. Cambridge: University Press.

Humphrey, L. 2003. Linear growth variation in the archaeological record. In (J.L. Thompson, G.E. Krovitz & A.J. Nelson, Eds.) *Patterns of Growth in the Genus Homo*. Cambridge, Cambridge University Press. 144-169.

Huss-Ashmore, R., Goodman, AH., and Armelagos, GJ. 1982. Nutritional inference from paleopathology. *Advances in Archaeological Method and Theory* 5: 395-474.

Ingold, T. 1998. From complementary to obviation: on dissolving the boundaries between social and biological anthropology, archaeology and psychology. *Zeitschrift fur Ethnologie* 123: 21-52.

İşcan M, Loth S, and Wright R. 1984. Age estimation from the rib by phase analysis: white males. *Journal of Forensic Sciences* 29: 1094–1104.

İşcan M, Loth S, and Wright R. 1985. Age estimation from the rib by phase analysis: white females. *Journal of Forensic Sciences* 30: 853–863.

Ives R and Brickley M. 2004. A procedural guide to metacarpal radiogrammetry in archaeology. *International Journal of Osteoarchaeology* 14: 7-17.

Ives R and Brickley M. 2005. Metacarpal radiogrammetry: a useful indicator of bone loss throughout the skeleton? *Journal of Archaeological Science* 32: 1552-1559.

Jackes, M. 2000. Building the bases for paleodemographic analyses: Adult age determination. In: Katzenberg MA, Saunders, SR, eds., *Biological Anthropology of the Human Skeleton*. Wiley-Liss: New York; 417-466.

Jackes, M. 2011. Representativeness and Bias in Archaeological Skeletal Samples. In: Agarwal SC, Glencross BA (eds.). *Social Bioarchaeology*. Wiley-Blackwell: Chichester; 107-246.

Jackson, R. 1988. *Doctors and Diseases in the Roman Empire*. London: British Museum Press.

Jansen, M. 1920. On bone formation: its relation to tension and pressure. University of Manchester Med. Sr. #16. Manchester, 114.

Järvinen, TLN., Kannus, P., and Sievänen, H. 2007. Bone quality: Emperor's New Clothes. *Journal of Musculoskeletal and Neuronal Interactions* 8(1): 2-9.

Javaid M and Cooper C. 2002. Prenatal and childhood influences on osteoporosis. *Best Practice and Research Clinical Endocrinology and Metabolism* 16(2): 349-367.

Javaid M, Lekamwasam S, Clark J, Dennison E, Syddall H, Loveridge N, Reeve J, Beck T, and Cooper C. 2006. Infant growth influences proximal femoral geometry in adulthood. *Journal of Bone and Mineral Research* 21: 508-512.



- Jiang JX., Siller-Jackson, AJ and Burra, S. 2007. Roles of gap junctions and hemichannels in bone cell functions and in signal transmission of mechanical stress. *Frontiers in Bioscience* 12: 1450-1262.
- Johnston, F.E. (1962). Growth of the long bones of infants and young children at Indian Knoll. *American Journal of Physical Anthropology* 20, 249-254.
- Johnston, F.E. (1969). Approaches to the study of developmental variability in human skeletal populations. *American Journal of Physical Anthropology* 31, 335-341
- Judd MA and Roberts CA. 1999. Fracture trauma in a medieval British farming village. *American Journal of Physical Anthropology* 109: 229-243.
- Jungers, WL and Minns, RJ. 1979. Computed Tomography and Biomechanical Analyses of Fossil Long Bones. *American Journal of Physical Anthropology* 50: 285-290.
- Kamm, K., E. Thelen, and J. L. Jensen 1990 A Dynamical Systems Approach to Motor Development. *Physical Therapy* 70:763–775.
- Kanis JA. 1994. Osteoporosis. Oxford: Blackwell Science.
- Kasl, S. V., and L. Berkman 1983. Health Consequences of the Experience of Migration. *Annual Review of Public Health* 4: 69–90.
- Kazakia, GJ and Majumdar, S. 2006. New imaging technologies in the diagnosis of osteoporosis. *Reviews of Endocrine and Metabolism Disorders* 7: 67-74.
- Keenleyside, A and Panayotova, K. 2006. Cribra Orbitalia and Porotic Hyperostosis in a Greek Colonial Population (5<sup>th</sup> to 3<sup>rd</sup> Centuries BC) from the Black Sea. *International Journal of Osteoarchaeology* 16: 373-384.
- Kehoe T. 2006. Bone quality: A perspective from the food and drug administration. *Current Osteoporosis Reports*. 4(2): 76-79.
- Kelley M.A 1982. Intervertebral osteochondrosis in ancient and modern populations. *American Journal of Physical Anthropology*. 59: 271-280.
- Kemkes-Grottenthaler, A. 2005. The Short Die Young: The Interrelationship between Stature and Longevity – Evidence from Skeletal Remains. *American Journal of Physical Anthropology* 128: 340-347.
- Key, LL and Bell, NH. 1999. Racial Determinants of Peak Bone Mass. In: Rosen, GJ., Glowacki, J and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 127-135.

Khosla, S., Amin, S., and Orwoll, E. 2008. Osteoporosis in men. *Endocrine Reviews* 29: 441-464.

Killgrove, K. 2008. Slums or suburbs? Health status of a population from Imperial Rome. *American Journal of Physical Anthropology* 135: 128. Abstract.

King T, Humphrey LT, and Hillson S. 2005. Linear enamel hypoplasias as indicators of systemic physiological stress: Evidence from two known age-at-death and sex populations from postmedieval London. *American Journal of Physical Anthropology* 128(3):547-559.

Koch, JC. 1917. Laws of bone architecture. *American Journal of Anatomy* 21: 177.

Klaus, HD and Tam, ME. 2009. Contact in the Andes: Bioarchaeology of systemic stress in colonial Morrope, Peru. *American Journal of Physical Anthropology* 138: 356-368.

Kleerekoper, M., Villaneuva, AR., Stanciu, J., Rao, DS and Parfitt, AM. 1985. The Role of Three-Dimensional Trabecular Microstructure in the Pathogenesis of Vertebral Compression Fractures. *Calcified Tissue International* 37: 594-597.

Kleiner DE and Matheson, SB. 1996. *I, Claudia: Women in Ancient Rome*. Yale University Art Gallery: New Haven.

Kneissel, M., Boyd, A., Hahn, M., Teschler-Nicola, M., Kalchhauser, G and Plenk Jr., H. 1994. Age- and Sex- dependent cancellous bone changes in a 4000y BP population. *Bone* 15: 539-545.

Kneissel M, Roschger P, Steiner W, Schamall D, Kalchauser G, Boyde A, Teschler-Nicola M. 1997. Cancellous Bone Structure in the Growing and Aging Lumbar Spine in a Historic Nubian Population. *Calcified Tissue International* 61: 95-100.

Knudson, KJ and Stojanowski, CM. 2008. New dimensions in bioarchaeology: recent contributions to the study of human social identities. *Journal of Archaeological Research* 16(4): 397-432.

Knüssel, C. 2000 Bone Adaptation and its Relationship to Physical Activity in the Past. In: M. Cox, and S. Mays, eds. *Human Osteology in Archaeology and Forensic Science*. Cambridge: Cambridge University Press. 381–401.

Kreiger, N., Kelsey, JL., Holford, TR and O'Connor, T. 1982. An epidemiological study of hip fracture in post-menopausal women. *American Journal of Epidemiology* 116: 141-148.

Krieger, N. 2001 Theories for Social Epidemiology in the 21st Century: An Ecosocial Perspective. *International Journal of Epidemiology* 30:668–677.

Krieger, N. 2005 Embodiment: A Conceptual Glossary for Epidemiology. *Journal of Epidemiology and Community Health* 59:350–355.

Kuh, D and Ben-Schlomo, Y. 1997. *A Life Course Approach to Chronic Disease Epidemiology*. Oxford: Oxford University Press.

Kuzawa, C. 2006. Life history perspectives on growth, productivity and adult physiology and function. *American Journal of Physical Anthropology* 129: 31. Abstract.

Lambert, PM. 1993. Health in the prehistoric populations of the Santa Barbara Channel Islands. *American Antiquity* 58(3): 509-522.

Larsen, CS. 1997. *Bioarchaeology: Interpreting Behavior from the Human Skeleton*. Cambridge: Cambridge University Press.

Larsen, CS. 1998. Gender, Health and Activity in foragers and farmers in the American Southeast: Implications for social organization in the Georgia Bight. In: Grauer, A and Stuart-Macadam, P, eds., *Sex and Gender in Paleopathological Perspective*. Cambridge: Cambridge University Press. 165-189.

Larsen, CS. 2002. Bioarchaeology: The lives and lifestyles of past people. *Journal of Archaeological Science* 10(2): 119-166.

Larsen, C.S. & Milner, G.R., Eds. (1994). *In the Wake of Contact: Biological Responses to Conquest*. New York, Wiley-Liss.

Lasker, G. W. 1969 Human Biological Adaptability. *Science* 166:1480–1486.

Lasker, G. W. 1976. *Physical Anthropology, 2nd edition*. New York: Holt Rinehart and Winston.

Lasker, G. W., and Evans, F. G. 1961 Age, Environment and Migration: Further Anthropometric Findings on Migrant and Non-Migrant Mexicans. *American Journal of Physical Anthropology* 19:203–211.

Laurence, R. 2005. Health and the life course at Herculaneum and Pompeii. In: King, H, Ed., *Health in Antiquity*. London: Routledge. 83-96.

Lazenby RA. 2002. Circumferential variation in human second metacarpal cortical thickness: sex, age, and mechanical factors. *Anatomical Record* 267: 154-158.

LeBoff, M and Glowacki, J. 1999. Sex Steroids, Bone and Aging. In: Rosen, CJ., Glowacki, J., and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 159-174.

Lees B, Molleson T, Arnett TR, Stevenson JC. 1993. Differences in proximal femur bone density over two centuries. *The Lancet* 341: 673–675.

Leftkowitz MR, Fant MB. 1982. *Women's Life in Greece and Rome*. London: Duckworth.

- Lenora, J., Lekamwasam, S and Karlsson, MK. 2009. Effects of multiparity and prolonged breast-feeding on maternal bone mineral density: a community-based cross-sectional study. *BMC Women's Health* 9: 19.
- Lewis, N and Reinhold, M. 1966. *Roman Civilization: Sourcebook II, The Empire*. New York: Columbia University Press.
- Lewis, ME. 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(3): 211-223.
- Lewis, ME and Gowland, R. 2007. Brief and Precarious Lives: Infant Mortality in Contrasting Sites from Medieval and Postmedieval England (A.D. 850-1859). *American Journal of Physical Anthropology* 134(1): 117-129.
- Lewis, ME. 2007. *The bioarchaeology of children: Perspectives from biological and forensic anthropology*. Cambridge: Cambridge University Press
- Lewontin, R. C. 2001 Gene, Organism and Environment. In: S. Oyama, P. E. Griffiths, and R. D. Gray, eds. *Cycles of Contingency: Developmental Systems and Evolution*. Cambridge; MIT Press. 59–66.
- Link, TM., Majumdar, S., Grampp, S., Guglielmi, G., van Kuijk, C., Imhof, H., Glueer, C and Adams, JE. 1999. Imaging of trabecular bone structure in osteoporosis. *European Radiology* 9: 1781-1788.
- Little, MA and Haas, JD. 1989. *Human Population Biology: A Transdisciplinary Science*. Oxford: Oxford University Press.
- Livingstone, F. 1958. Anthropological implications of sickle cell gene distribution in West Africa. *American Anthropologist* 60: 533-562.
- Lloyd, T and Cusatis DC. 1999. Nutritional Determinants of Peak Bone Mass. In: Rosen, CJ., Glowacki, J., and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 95-104.
- Locke, M. 2004. The Structure of Long Bones in Mammals. *Journal of Morphology* 262 (2): 546-565.
- Lopez JM, Gonzalez G, Reyes V, Campino C, and Diaz S. 1996. Bone turnover and density in healthy women during breastfeeding and after weaning. *Osteoporosis International* 6: 153–159.
- Lovejoy, O and Trinkaus, E. 1980. Strength and Robusticity of the Neandertal Tibia. *American Journal of Physical Anthropology* 53: 465-470.

- Lovejoy CO. 1985. Dental wear in the Libben population: its functional pattern and role in the determination of adult skeletal age at death. *American Journal of Physical Anthropology* 68: 47–56.
- Lovejoy, CO, RP Mensforth, and GJ Armelagos. 1982. Five decades of skeletal biology as reflected in the American Journal of Physical Anthropology. In: F. Spencer, ed. *A History of American Physical Anthropology 1930-1980*. New York: Academic Press. 329-336.
- Lovejoy CO, Meindl RS, Pryzbeck TR, Mensforth RP. 1985. Chronological metamorphosis of the auricular surface of the ilium: a new method for the determination of adult skeletal age at death. *American Journal of Physical Anthropology* 68: 15–28.
- Lovejoy, OC., McCollum, MA., Reno, PL and Rosenman, BA. 2003. Developmental Biology and Human Evolution. *Annual Review of Anthropology* 32: 85-109.
- Lovell, N.C. and Whyte, I. (1999). Patterns of dental enamel defects at ancient Mendes, Egypt. *American Journal of Physical Anthropology* **110**, 69-80.
- Lynnerup, N. 2007. Mummies. *Yearbook of Physical Anthropology* 50: 162-190.
- Mace, R. 1999. Evolutionary ecology of human life history. *Animal Behaviour* 59: 1-10.
- Mackay, CS. 2004. *Ancient Rome: A Military and Political History*. Cambridge: Cambridge University Press.
- MacNeil, JA and Boyd, SK. 2007. Accuracy of high-resolution peripheral quantitative computed tomography for measurement of bone quality. *Medical Engineering and Physics* 29: 1096-1105.
- Macho, GA., Abel, RL., and Schutkowski, H. 2005. Age changes in Bone microstructure: Do they occur uniformly? *International Journal of Osteoarchaeology* 15: 421-430.
- Maggiano, CM. 2012. Making the Mold: A microstructural perspective on bone modeling during growth and mechanical adaptation. In: Crowder, C and Stout, S, eds., *Bone Histology: An Anthropological Perspective*. Boca Raton: CRC Press. 45-90.
- Maggio D, Pacifici R, Cherubini A, Simonelli G, Luchetti M, Asia M, Cucinotta D, Adami S, Senin U. 1997. Age-related cortical bone loss at the metacarpal. *Calcified Tissue International* 60 94-97.
- Makovey, J., Nguyen, TV., Naganathan, V., Wark, JD and Sambrook, PN. 2007. Genetic Effects of Bone loss in Peri- and Postmenopausal Women: A Longitudinal Twin Study. *Journal of Bone and Mineral Research* 22(11): 1173-1780.
- Malluche, HH and Faugere, MC. 1986. *Atlas of Mineralized Bone Histology*. Basel: Karger.
- Malville N.J. 1997. Enamel hypoplasia in ancestral Puebloan populations from southwestern

- Colorado: 1. Permanent dentition. *American Journal Physical Anthropology* 102: 351-367.
- Mann RW and Murphy S. 1990. *Regional Atlas of Bone Disease*. Springfield: Charles C Thomas Publishers.
- Manzi G, Censi L, Sperduti A, and Passarello P. 1989. Linee di Harris e ipoplasia dello smalto nei resti scheletrici delle popolazioni umane di Isola Sacra e Lucus Feroniae (Roma, I-III sec. dC.). *Riv Antropol* 67: 129–148.
- Manzi G, Santandrea E, and Passarello P. 1997. Dental size and shape in the Roman Imperial Age: two examples from the area of Rome. *American Journal of Physical Anthropology* 102: 469–479.
- Manzi G., Salvadei L., Vienna A. and Passarello P. 1999. Discontinuity of life conditions at the transition from the Roman Imperial age and early Middle Ages: Example from central Italy evaluated by pathological dental alveolar lesions. *American Journal of Human Biology* 11: 327-341.
- Maresh, M.M. 1955. Linear growth of long bones of extremities from infancy through adolescence. *American Journal of Diseases of Children* **89**, 725-742.
- Maresh, M.M. 1970. Measurements from roentgenograms. In (R.W. McCammon, Ed.) *Human Growth and Development*. Springfield, Charles C. Thomas, pp. 157-200.
- Margerison, BJ and Knüsel, CJ. 2002. Paleodemographic Comparison and a Catastrophic and an Attritional Death Assemblage. *American Journal of Physical Anthropology* 119: 134-143.
- Mascie-Taylor, C. G. N. 1984 The Interaction Between Geographical and Social Mobility. In: A. J. Boyce, ed. *Migrants and Mobility*. London: Taylor and Francis. 161–178.
- Mathisen, RW. 2003. *People, Personal Expression, and Social Relations in Late Antiquity, Volume I*. Ann Arbor: University of Michigan Press.
- Martin DL, Armelagos GJ. 1979. Morphometrics of compact bone: an example from Sudanese Nubia. *American Journal Physical Anthropology* 51:571–578.
- Martin DL. 1981. Microstructural examination: possibilities for skeletal analysis. In: Martin DL, and Bumstead MP, eds. *Biocultural Adaptation: Comprehensive Approaches to Skeletal Analysis*. Research Reports No. 20, Department of Anthropology. Amherst: University of Massachusetts. 96–107.
- Martin, D. L., G. J. Armelagos, A. H. Goodman, and D. P. Van Gerven, 1984 The Effects of Socioeconomic Change in Prehistoric Africa: Sudanese Nubia as a Case Study. In: M. N. Cohen, and G. J. Armelagos, eds. *Paleopathology at the Origins of Agriculture*. New York: Academic Press. 193–214.

- Martin, D. L., A. H. Goodman, and G. J. Armelagos 1985 Skeletal Pathologies as Indicators of Quality and Quantity of Diet. In: R. I. Gilbert, and J. H. Mielke, eds. *The Analysis of Prehistoric Diets*. New York: Academic Press. 227–279.
- Martin, DL and Armelagos, GJ. 1985. Skeletal Remodeling and Mineralization as Indicators of Health: an Example from Prehistoric Sudanese Nubia. *Journal of Human Evolution* 14: 527-537.
- Martin, RB and Burr, DB. 1989. *Structure, Function and Adaptation of Compact Bone*. New York: Raven Press
- Martin R.B., Burr D.B. and Sharkey N.A. 1998. *Skeletal Tissue Mechanics*. Springer-Verlag, New York.
- Martin B. 2003. Functional Adaptation and Fragility of the Skelton. In: Agarwal SC, Stout SD (eds.). *Bone Loss and Osteoporosis: An Anthropological Perspective*. Kluwer Academic/Plenum Publishers: New York; 3-17.
- Marzano A. 2007. Fish salting versus fish breeding: the case of Roman Italy. *British Archaeological Reports International Series* 1686: 301–313.
- Mays S. 1996. Age-dependent cortical bone loss in a mediaeval population. *International Journal of Osteoarchaeology* 6: 144–154.
- Mays, S. 1998. Osteoporosis in earlier human populations. *Journal of Clinical Densitometry* 2: 71-78.
- Mays S. 1999. Linear and appositional long bone growth in earlier human populations: a case study from Medieval England. *Cambridge Studies in Biological and Evolutionary Anthropology* 25: 290-312.
- Mays S. 2000. Age-Dependent Cortical Bone Loss in Women from the 18<sup>th</sup> and Early 19<sup>th</sup> Century London. *American Journal of Physical Anthropology* 112: 349-361.
- Mays S. 2001. Effects of Age and Occupation on Cortical Bone in a Group of 18<sup>th</sup>-19<sup>th</sup> Century British Men. *American Journal of Physical Anthropology* 116: 34-44.
- Mays S. 2006. Age-Related Cortical Bone Loss in Women from a 3<sup>rd</sup>-4<sup>th</sup> Century AD Population from England. *American Journal of Physical Anthropology* 129: 518-528.
- Mays S, Lees B, and Stevenson JC. 1998. Age-dependent bone loss in the femur in a mediaeval population. *International Journal of Osteoarchaeology* 8: 97–106.
- Mays S, Turner-Walker G, and Syversen U. 2006. Osteoporosis in a Population from Medieval Norway. *American Journal of Physical Anthropology* 131: 343-351.



- Mays, S. Brickley, M., and Ives, R. 2008. Growth in an English population from the industrial revolution. *American Journal of Physical Anthropology* 136: 85-92.
- Mays, S. 2010. The effects of infant feeding practices on infant and maternal health in a Medieval community. *Childhood in the Past: An International Journal* 3(1): 63-78.
- Mazess, RB and Mather, W. 1975. Bone Mineral Content in Canadian Eskimos. *Human Biology* 47 (1): 45-63.
- McDade, TW. 2005. The Ecologies of Human Immune Function. *Annual Review of Anthropology* 34: 495-521.
- McDade, TW., Reyes-Garcia, V., Tanner, S., Huanca, T and Leonard, WR. 2008. Maintenance versus Growth: Investigating the Costs of Immune Activation Among Children in Lowland Bolivia. *American Journal of Physical Anthropology* 136(4): 478-484.
- McEwan JM, Mays S, and Blake GM. 2005. The relationship of bone mineral density and other growth parameters to stress indicators in a medieval juvenile population. *International Journal of Osteoarchaeology* 15: 155-163.
- Meema EH, Meema S. 1987. Postmenopausal osteoporosis: Simple screening method for diagnosis before structural failure. *Radiology* 164: 405-410.
- Meema EH, Meindok H., 1992. Advantages of peripheral radiogrammetry over dual photon absorptiometry of the spine in the assessment of prevalence of osteoporotic vertebral fractures in women. *Journal of Bone and Mineral Research* 7: 897-903.
- Meiggs, R. 1960. *Roman Ostia*. Oxford: Oxford University Press.
- Mellor, R. 2006. *Augustus and the Creation of the Roman Empire: A Brief History with Documents*. New York: Palgrave Macmillan.
- Melton, LJ., Kan, SH., Frye, MA., Wahner, HW., O'Fallon, WM., and Riggs, BL. 1989. Epidemiology of vertebral fractures in women. *American Journal of Epidemiology* 129(5): 1000-1011.
- Melton, L. 1995. How Many Women Have Osteoporosis Now? *Journal of Bone and Mineral Research* 10: 175-177.
- Mensforth R, Lovejoy CO, Lallo H, and Armelagos G. 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.
- Messina, M. 1999. Legumes and soybeans: overview of their nutritional profiles and health effects. *American Journal of Clinical Nutrition*. 70 (3): 439S-450.



- Meunier, PJ. 1995. Bone Histomorphometry. In: Riggs, L and Melton III, LJ., eds., *Osteoporosis: Etiology, Diagnosis, and Management*. Philadelphia: Lippincott-Raven. 299-318.
- Michaëlsson K, Baron J, Farahmand B, and Ljunghall S. 2001. Influence of parity and lactation on hip fracture risk. *American Journal of Epidemiology* 153: 1166-1172.
- Mielke, JH., Armelagos, GJ and Van Gerven, DP. 1972. Trabecular involution in femoral heads of a prehistoric (X-Group) population from Sudanese Nubia. *American Journal of Physical Anthropology* 36(1): 39-44.
- Miller, M. 2005. Hypothesis: Fetal movement influences fetal and infant bone strength. *Medical Hypotheses* 65: 880-886.
- Milner, GR., Wood, J and Boldsen, JL. 2000. Paleodemography. In Katzenburg, MA and Saunders, SR, eds., *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss. 467-497.
- Montalban SJ, Rico LH, Cortes PJ, Pedrera ZJD. 2001. Cortical bone mass and risk factors for osteoporosis among postmenopausal women in our environment. *Revisita Clinica Espanola* 201: 16–20.
- Morel JP. 1999. Hyélè revue à la lumière de Massalia. In: Krinzinger F, Tocco G (eds.). *Neue Forschungen in Velia*. Vienna:Osterreichische Akademie der Wissenschaften.11-22.
- Mosekilde L. 1988. Age-related changes in vertebral trabecular bone architecture – Assessed by a new method. *Bone*. 9: 247–250.
- Mosekilde, L. 1990. Sex differences in age-related changes in vertebral body size, density, and biomechanical competence in normal individuals. *Bone* 11(2): 67-73.
- Mulhern D. 2000. Rib remodeling dynamics in a skeletal population from Kulubnarti, Nubia. *American Journal of Physical Anthropology* 111:519-530.
- Müller, R., Hahn, M., Vogel, M., Delling, G and Rügsegger, P. 1996. Morphometric Analysis of Noninvasively Assess Bone Biopsies: Comparison of High-Resolution Computed Tomography and Histologic Sections. *Bone* 18(3): 215-220.
- Mundy, G. 1995. *Bone Remodeling and Its Disorders*. London: Martin Dunitz
- Murphy S, Khaw KT, May H, and Compston JE. 1994. Parity and bone mineral density in middle-aged women. *Osteoporosis International* 4:162–166.
- Murphy, NM and Carroll, P. 2003. The effect of physical activity and its interaction with nutrition on bone health. *Proceedings of the Nutrition Society* 62: 829-838.

- Nazarian, A., Muller, J., Zurakowski, D., Müller, R and Synder, BD. 2007. Densitometric, morphometric and mechanical distribution in the human proximal femur. *Journal of Biomechanics* 40: 2573-2579.
- Nelson, DA and Villa, ML. 1999. Racial/Ethnic Influences on Risk of Osteoporosis. In: Rosen, GJ., Glowacki, J and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 237-250.
- Nelson, A. J., and J. L. Thompson 1999 Growth and Development in Neandertals and Other Fossil Hominids: Implications for the Evolution of Hominid Ontogeny. In: R. D. Hoppa, and C. M. Fitzgerald, eds. : *Human Growth in the Past: Studies from Bones and Teeth* . Cambridge: Cambridge University Press. 88–110.
- Nelson, DA and Villa, ML. 2003. Ethnic Differences in Bone Mass and Bone Architecture. In: Agarwal, SC and Stout , SD, eds., *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 47-62.
- Nelson, DA., Sauer, NJ and Agarwal, SC. 2003. Evolutionary Aspects of Bone Health. *Clinical Reviews in Bone and Mineral Metabolism* 1(3): 1-11.
- (NOF) National Osteoporosis Foundation; c2011 (cited 2011 Dec 15). Available from: <http://www.nof.org/node/40>
- Nielsen SP. 2001. The metacarpal index revisited: a brief over- view. *Journal of Clinical Densitometry* 4: 199–207.
- Njeh, CF., Cheng, XG., Elliot, JM., Meunier, PJ. 1999. Bone, bone diseases, and bone quality. In: Njeh, CF., Hans, D., Fuerst, T., Gluer, CC., and Genant, HK, eds. *Quantitative Ultrasound: Assessment of Osteoporosis and Bone Status*. London: Martin Dunitz: 1-20.
- Nyati, LH., Norris, S., Cameron, N and Pettifor, JM. 2006. Effect of Ethnicity and Sex on the Growth of the Axial and Appendicular Skeleton of Children Living in a Developing Country. *American Journal of Physical Anthropology* 130: 135-141.
- Nystrom, KC. 2006. Late Chachapoya population structure prior to Inka conquest. *American Journal of Physical Anthropology* 131:334-342.
- Odgaard, A and Gundersen, HJG. 1993. Quantification of connectivity in cancellous bone, with special emphasis on 3D reconstructions. *Bone* 14: 173-182.
- Odgaard, A. 1997. Three Dimensional Methods for Quantification of Cancellous Bone Architecture. *Bone* 20(4): 315-328.
- Ortner DJ. and Putcshar WG. 1981. *Identification of Pathological Condition in Human Skeletal Remains*. Smithsonian Institution Press, Washington D.C.

Ortner, DJ. and Putschar, WG. 1985. *Identification of Pathological Conditions in Human Skeletal Remains*. Washington, D.C., Smithsonian Institution Press

Ortner, D.J. (1991). Theoretical and Methodological Issues in Paleopathology. In: Ortner, DJ and Aufderheide, AC., eds, *Human Paleopathology: Current Synthesis and Future Options*. Washington, D.C., Smithsonian Institution Press. 5-11.

Ortner, DJ. 2003. *Identification of Pathological Conditions in Human Skeletal Remains*. San Diego: Academic Press.

Ortner, DJ, and H Schutkowski. 2008. Ecology, culture and disease in past populations. In: H. Schutkowski, ed. *Between Biology and Culture*. Cambridge: Cambridge University Press. 105-129.

Orwoll, E. 1999. Androgens. In: Rosen, GJ., Glowacki, J and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 521-540.

Orwoll, ES., Belknap, JK., and Klein, RF. 2001. Gender specificity in the genetic determinants of peak bone mass. *Journal of Bone and Mineral Research* 16(1): 1962-1971.

Oxenham, MF and Cavill, I. 2011. Porotic hyperostosis and cribra orbitalia: the erythropoietic response to iron-deficiency anaemia. *Anthropological Science* 118(3): 199-200.

Oyama, S. 2000a. *Evolution's Eye: A Systems View of the Biology-Culture Divide*. Durham: Duke University Press.

Oyama, S. 2000b. *The Ontogeny of Information: Developmental Systems and Evolution*. Durham: Duke University Press.

Paine, RR and Brenton, BP. 2006. Dietary health does affect histological age assessment: An evaluation of the Stout and Paine (1992) age estimation equation using secondary osteons from the rib. *Journal of Forensic Science* 51(3): 489-492.

Paine R.R., Vargiu R., Coppa A., Morselli C. and Schneider E.E. 2007. A health assessment of high status Christian burials recovered from the Roman-Byzantine archaeological site of Elaiussa Sebaste, Turkey. *HOMO*, 58: 173-190.

Paine, RR., Vargiu, R., Signoretti, C., and Coppa, A. 2009. A health assessment for Imperial Roman burials recovered from the necropolis of San Donato and Bivio CH, Urbino, Italy. *Journal of Anthropological Sciences* 87: 193-210.

Parfitt, AM., Matthews, CHE., Villaneuva, AR., Kleerekoper, A., Frame, B and Rao, DS. 1983. Relationships between surface, volume and thickness of iliac trabecular bone in aging and in osteoporosis. *Journal of Clinical Investigation* 72: 1396-1409.

Parfitt AM, Han ZH, Palnitkar S, Rao DS, Shih MS, and Nelson D. 1997. Effects of ethnicity and age or menopause on osteoblast function, bone mineralization, and osteoid accumulation in iliac bone. *Journal of Bone Mineral Research* 12:1864–1873.

Parfitt, AM. 2003. New Concepts of Bone Remodeling: A Unified Spatial and Temporal Model with Physiologic and Pathophysiologic Implications. In: Agarwal, SC and Stout, SD, eds. *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 3-17.

Pasco JA, Sanders KM, Hoekstra FM, Henry MJ, Nicholson GC, and Kotowicz MA. 2005. The human cost of fracture. *Osteoporosis International*. 16(12): 2046-2052.

Patel, DN., Pettifor, JM., Becker, PJ., Grieve, C and Leschner, K. 1992. The effect of Ethnic group on appendicular bone mass in children. *Journal of Bone and Mineral Research* 7(3): 263-272.

Patterson, J. 2000. Living and Dying in the city of Rome: houses and tombs In: Coulston, J and Dodge, H., eds. *Ancient Rome*. Oxford: Oxbow Books. 259-289.

Pearson, O and Lieberman, DE. 2004. The Aging of Wolff's "Law": Ontogeny and Responses to Mechanical Loading in Cortical Bone. *Yearbook of Physical Anthropology* 47: 63-99.

Peck J, and Stout S. 2007. Intraskkeletal variability in bone mass. *American Journal of Physical Anthropology* 132: 89-97.

Peel, N and Eastell, R. 1995. ABC of rheumatology: osteoporosis. *British Medical Journal* 15, 310 (6985): 989-992.

Petit, MA., Beck, TJ and Kontulainen, SA. 2005. Examining the developing bone: How do we measure and how do we do it? *Journal of Musculoskeletal and Neuronal Interactions* 5(3): 213-224.

Pfeiffer SK and Lazenby RA. 1994. Low bone mass in past and present aboriginal populations. In: Draper HH, editor. *Advances in Nutritional Research*. Vol. 9. New York: Plenum Press. 35–51.

Pfeiffer, SK. 1998. Variability in osteon size in recent human populations. *American Journal of Physical Anthropology* 106: 219-227.

Pfeiffer, SK., Crowder, C., Harrington, L., and Brown, M. 2006. Secondary osteon and Haversian canal dimensions as behavioral indicators. *American Journal of Physical Anthropology* 131(4): 460-468.

Pfeiffer, SK and Pinto, D. 2012. Histological examination of human bone in archaeological contexts. In: Crowder, C and Stout, S, eds., *Bone Histology: An Anthropological Perspective*. Boca Raton: CRC Press. 297-312.

Porter, RW, Hibbert, C, and Wellman, P. 1980. Backache and the lumbar spinal canal. *Spine* 5: 99-105.

Porter, RW and Pavitt, D. 1987. The Vertebral Canal: 1. Nutrition and Development, an Archaeological Study. *Spine* 12(9): 901-906.

Poulsen LW, Qvesel D, Brixen K, Vesterby A, Boldsen JL. 2001. Low bone mineral density in the femoral neck of medieval women: a result of multiparity? *Bone* 28(4):454-458.

Prentice, A., Laskey, MA., Shaw, J., Cole, TJ., and Fraser, DR. 1990. Bone mineral content of Gambian and British children age 0-36 months. *Bone and Mineral* 10: 211-224.

Prentice A. 2007. Diet, nutrition and the prevention of osteoporosis. *Public Health Nutrition* 7: 227-243.

Pritchard, D. J. 1995 Plasticity in Early Development. In: C. G. N. Mascie-Taylor, and B. Bogin, eds. *Human Variability and Plasticity*. Cambridge: Cambridge University Press. 18–45.

Prowse, T. Schwarcz, HP., Saunder, S., Macchiarelli, R and Bondioli, L. 2004. Isotopic paleodiet studies of skeletons from the Imperial Roman-age cemetery of Isola Sacra, Rome, Italy. *Journal of Archaeological Science* 31: 259-272.

Prowse T, Schwarcz H, Saunders S, Macchiarelli R, and Bondioli L. 2005. Isotopic evidence for age-related variation in diet from Isola Sacra, Italy. *American Journal of Physical Anthropology* 128:2-13.

Prowse T, Saunders SR, Schwarcz H, Garnsey P, Macchiarelli R, Bondioli L. 2008. Isotopic and dental evidence for infant and young child feeding practices in an imperial Roman skeletal sample. *American Journal of Physical Anthropology* 137: 294-308.

Purcell, N. 2007. Urban Places and Central Spaces: The Roman World. . In: Alcock, SE and Osborne, R., eds. *Classical Archaeology*. Blackwell Publishing. 182-202.

Räisänen, U., Bekkers, M., Boddington, P., Sarangi, S and Clarke, A. 2006. The causation of disease – The practical and ethical consequences of competing explanations. *Medicine, Health Care and Philosophy* 9: 293-306.

Raisz, L and Seeman, E. 2001. Causes of Age-related bone loss and bone fragility: An alternative view. *Journal of Bone and Mineral Research* 16(1): 1948-1952.

Raubenheimer, E. 2004. Histopathologic Changes in Metabolic Bone Disease. *Advances in Anatomical Pathology* 11(1): 38-48.

Rauch F, Bailey D, Baxter-Jones A, Mirwald R, and Faulkner R. 2004. The “muscle-bone unit” during the pubertal growth spurt. *Bone* 34: 771-775.

Rauch, F. 2005. Bone Growth in Length and Width: The Yin and Yang of Bone Stability. *Journal of Musculoskeletal Neuronal Interactions* 5(3): 194-201.

Rauch, F. 2007. Bone Accrual in Children: Adding Substance to Surfaces. *Pediatrics* 119: s137-s140.

Rawson, B. 1986. The Roman Family. In: Rawson, B., ed. *The Family in Ancient Rome: New Perspectives*. New York: Cornell University Press.

Reave, H and Sherman, PW. 1993. Adaptation and the Goals of Evolutionary Research. *The Quarterly Review of Biology* 68 (1): 1-32.

Redfern, R. 2007. The influence of culture upon childhood: and osteological study of Iron Age and Romano-British Dorset. In: Harlow M, Laurence, R (eds.). *Age and Ageing in the Roman Empire, Journal of Roman Archaeology, Supplement No. 65*. Journal of Roman Archaeology: Portsmouth; 171-194.

Reed MR, Murray JRD, Abdy SE, Francis RM, McCaskie AW. 2004. The use of digital X-ray radiogrammetry and peripheral dual X-ray absorptiometry in patients attending fracture clinic after distal forearm fracture. *Bone* 34: 716– 719.

Reginster JY and Burlet N. 2006. Osteoporosis: A still increasing prevalence. *Bone* 38:S4-S9.

Reinhard, K. 1992. Parasitology as an interpretive tool in archaeology. *American Antiquity* 52: 231-245.

Rewekant A. 1994. Aging in prehistoric and contemporary human populations: comparative analysis of the process. *Variability and Evolution* 4: 57-65.

Rewekant A. 2001. Do environmental disturbances of an individual's growth and development influence the later bone involution processes? A study of two medieval populations. *International Journal of Osteoarchaeology* 11: 433-443.

Ribot, I. and Roberts, C. 1996. A study of non-specific stress indicators and skeletal growth in two mediaeval subadult populations. *Journal of Archaeological Science* 23, 67-79.

Richardson, ML., Genant, HK., Cann, C., Ettinger, B., Gordan, GS., Kolb, FO and Reiser, UJ. 1985. Assessment of Metabolic Bone Disease by Quantitative Computed Tomography. *Clinical Orthopedics and Related Research* 195: 224-238.

- Richman, EA., Ortner, DJ and Schuller-Ellis, FP. 1979. Differences in Intracortical Bone Remodeling in Three Aboriginal American Populations: Possible Dietary Factors. *Calcified Tissue International* 28: 209-214.
- Rickman, G. 1980. *The corn supply of ancient Rome*. Oxford: Oxford University Press.
- Rickman, G. 1988. The archaeology and history of Roman ports. *The International Journal of Nautical Archaeology and Underwater Exploration* 17(3): 257-267.
- Ridler TW. 1978. Picture Thresholding Using an Iterative Selection Method. *IEEE Transactions on Systems, Man, and Cybernetics* 8(8): 630-632.
- Riggs, LB and Melton, JL .1983. Evidence for two distinct syndromes for involuntional osteoporosis. *The American Journal of Medicine* 75(6): 899-901.
- Riggs, LB., Khosla, S., and Melton, LJ, 1998. A unitary model for involuntional osteoporosis: Estrogen deficiency causes both type I and type II osteoporosis in postmenopausal women and contributes to bone loss in aging men. *Journal of Bone and Mineral Research* 13(5): 763-773.
- Riggs, LB and Melton, JL. 2002. Bone Turnover Matters: The Raloxifene Treatment Paradox of Dramatic Decreases in Vertebral Fractures Without Commensurate Increases in Bone Density. *Journal of Bone and Mineral Research* 17(1): 11-14.
- Riggs, LB., Khosla, S and Melton, JL. 2002. Sex Steroids and the Construction and Conservation of the Adult Skeleton. *Endocrine Reviews* 23(3): 279-302.
- Riggs BL, Melton LJ, Robb RA, Camp JJ, Atkinson EJ, McDaniel L, Amin S, Rouleau PA, and Khosla S. 2008. A population- based assessment of rates of bone loss at multiple skeletal sites: evidence for substantial trabecular bone loss in young adult women and men. *Journal of Bone Mineral Research* 23:205–214.
- Rittweger, J. 2006. Can exercise prevent osteoporosis? *Journal of Musculoskeletal and Neuronal Interactions* 6(2): 162-166.
- Rizzoli, R., Boonen, S., Brandi, ML., Burlet, N., Delmas, P., and Reginster, JY. 2008. The role of calcium and Vitamin D in the management of osteoporosis. *Bone* 42: 246-249.
- Roberts, D. F., and D. Bainbridge 1963 Nilotic Physique. *American Journal of Physical Anthropology* 21:341–370.
- Roberts, D. F. 1977 Physique and Environment in the Northern Nilotes. *Mitteilungen der Anthropologischen Gesellschaft in Wien* 107: 161–168.
- Roberts C, and Wakely J. 1992. Microscopical findings associated with a diagnosis of osteoporosis in paleopathology. *International Journal Osteoarchaeology* 2:23–30.

- Roberts, C and Manchester, K. 1997. *The Archaeology of Disease*. New Jersey: Cornell University Press.
- Roberts, D. F. 1995 The Pervasiveness of Plasticity. In: C. G. N. Mascie-Taylor, and B. Bogin, eds. *Human Variability and Plasticity*. Cambridge: Cambridge University Press. 1–17.
- Robert, J., B. Hall, and W. Olson 2001 Bridging the Gap between Developmental Systems Theory and Evolutionary Developmental Biology. *Bioessays* 23:954–962.
- Robling, AG and Stout, SD. 2000. Histomorphometry of Human Cortical Bone: Applications to Age Estimation. In: Katzenburg, MA and Saunders, SR, eds., *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss. 187-214.
- Robling, AG and Stout, SD. 2003. Histomorphology, Geometry, and Mechanical Loading in Past Populations. In: Agarwal, SC and Stout, SD, eds., *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 189-206.
- Robling, AG and Stout, SD. 2008. Histomorphometry of Human Cortical Bone: Applications to Age Estimation. In: Katzenburg, MA and Saunders, SR, eds. *Biological Anthropology of the Human Skeleton, 2<sup>nd</sup> edition*. New York: Wiley-Liss. 149-182.
- Rojano-Mejía D, Aguilar-Madrid G, López-Medina G, Cortes-Espinosa L, Hernández-Chiu M, Canto-Cetina T, Vergara-López A, Coral-Vázquez R, Canto P. 2011. Risk factors and impact on bone mineral density in postmenopausal Mexican mestizo women. *Menopause: The Journal of The North American Menopause Society* 18(3): 302-306.
- Roschger, P., Grabner, B.M., Rinnerthaler, S., Tesch, W., Kneissel, M., Berzlanovich, A., Klaushofer, K., and Fratz, P. 2001. Structural Development of the Mineralized Tissue in the Human L4 Vertebral Body. *Journal of Structural Biology*. 136: 126-136.
- Ross, PD., Santora, A and Yates, AJ. 1999. Epidemiology and Consequences of Osteoporotic Fractures. In: Rosen, GJ., Glowacki, J and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 339-347.
- Roughead, ZK., Johnson, LK., Lykken, GI and Hunt, JR. 2003. Controlled High Meat Diets Do Not Affect Calcium Retention or Indices of Bone Status in Healthy Postmenopausal Women. *Journal of Nutrition* 133: 1020-1026.
- Rouleau PA, Khosla S. 2008. A Population-Based Assessment of Rates of Bone Loss at Multiple Skeletal Sites: Evidence for Substantial Trabecular Bone Loss in Young Adult Women and Men. *Journal of Bone and Mineral Research* 23: 205-214.
- Roux, W. 1885. Beiträge zur Morphologie der funktionellen anpassung. 3. Beschreibung und Erläuterung einer knöchernen Kniegelenkankylose. *Arch. Anat. Physiol. Anat. Abt.* 9: 120-158.



Roux, JP., Wegrzyn, J., Arlot, ME., Guyen, O., Delmas, PD., Chapurlat, R., and Bouxsein, ML. 2010. Contribution of trabecular and cortical components to biomechanical behavior of human vertebrae: an ex vivo study. *Journal of Bone and Mineral Research* 25(2): 356-361.

Rosholm A, Hyldstrup L, Baeksgaard L, Grunkin M, and Thodberg HH. 2001. Estimation of bone mineral density by digital X- ray radiogrammetry: theoretical background and clinical testing. *Osteoporosis International* 12: 961–969.

Rothschild, BM and Martin, LD. 1993. *Paleopathology, Diseases in the Fossil Record*. London: CRC Press.

Rothschild BM, Rühli FJ, Sebes J, Naples V, and Billard M. 2004. Relationship between porotic hyperostosis and cribra orbitalia? *Paleobios* 13: 4–7.

Ruff, C., A. Walker, and E. Trinkaus 1994 Postcranial Robusticity in Homo. III: Ontogeny. *American Journal of Physical Anthropology* 93:35–54.

Ruff, C. 2000. Biomechanical Analyses of Archaeological Human Skeletons. . In: Katzenburg, MA and Saunders, SR, eds. *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss. 71-102.

Ruff, C. 2005. Mechanical determinants of bone form: Insights from skeletal remains. *Journal of Musculoskeletal and Neuronal Interactions* 5(3): 202-212.

Ruff, C., Holt, B and Trinkaus, E. 2006. Who’s Afraid of the Big Bad Wolff?: “Wolff’s Law” and Bone Functional Adaptation. *American Journal of Physical Anthropology* 129: 484-498.

Ruff, C. 2008. Biomechanical Analyses of Archaeological Human Skeletons. . In: Katzenburg, MA and Saunders, SR, eds. *Biological Anthropology of the Human Skeleton, 2<sup>nd</sup> edition*. New York: Wiley-Liss. 183-206.

Rühli, FJ., Kuhn, G., Evison, R., Müller, R and Schultz, M. 2007. Diagnostic Value of Micro-CT in Comparison With Histology in the Qualitative Assessment of Historical Human Skull Bone Pathologies. *American Journal of Physical Anthropology* 133: 1099-1111.

Runciman, WG. 1983. Capitalism without classes: the case of classical Rome. *The British Journal of Sociology* 34(2): 157-181.

Ryan, TM and Krovitz, GE. 2006. Trabecular bone ontogeny in the human proximal femur. *Journal of Human Evolution* 51: 591-602.

Saggese, G., Baroncelli, GI and Bertelloni, S. 2002. Puberty and bone development. *Best Practice and Research Clinical Endocrinology and Metabolism* 16(1): 53—64.

Sah, AP., Thornhill, TS., LeBoff, MS and Glowacki, J. 2007. Correction of plain radiographic indices of the hip with quantitative bone mineral density. *Osteoporosis International* 18: 1119-1126.

Saller, RP and Shaw, B. 1984. Tomstones and Roman Family Relations in the Principate: Civilians, Soldiers and Slaves. *The Journal of Roman Studies* 74: 124-156.

Sattenspiel, L. 2000. Epidemiology of Human Disease. In: Stinson, S., Bogin, B., Huss-Ashmore, R and O'Rourke, D, eds., *Human Biology: An Evolutionary and Biocultural Perspective*, 225-272. New York: Wiley-Liss.

Salvadei L, Ricci F, Manzi G. 2001. Porotic hyperostosis as a marker of health and nutritional conditions during childhood: studies at the transition between imperial Rome and the early middle ages. *American Journal of Human Biology* 13: 709–717.

Saunders, S.R. and Hoppa, R.D. (1993). Growth deficit in survivors and non-survivors: biological mortality bias in subadult skeletal samples. *Yearbook of Physical Anthropology* 36: 127-152.

Saunders, SR. 2000. Subadult Skeletons and Growth-Related Studies. In Katzenburg, MA and Saunders, SR, eds. *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss. 135-162.

Saunders, S.R. 2008. Juvenile Skeletons and Growth-Related Studies. In: Katzenburg, MA and Saunders, SR, eds. *Biological Anthropology of the Human Skeleton, second edition*. New York: Wiley-Liss. 117-148.

Saxon, LK and Turner, CH. 2005. Estrogen receptor  $\beta$ : the antimechanostat? *Bone* 36: 185-192.

Scheidel W. 1995. The Most Silent Women of Greece and Rome: Rural Labour and Women's Life in the Ancient World. *Greece & Rome* 42: 202-217.

Schell, L. M. 1995 Human Biological Adaptability with Special Emphasis on Plasticity: History, Development and Problems for Future Research. In: C. G. N. Mascie-Taylor, and B. Bogin, eds. *Human Variability and Plasticity*. Cambridge: Cambridge University Press. 213–237.

Schell, LM. 2006. What does growth mean? Biomedical and adaptionist perspectives. *American Journal of Physical Anthropology* 129: 31. Abstract.

Scheuer, L. and Black, S. 2000. *Developmental Juvenile Osteology*. San Diego, Academic Press.

Scheuer, L and Black, S. 2004. *The Juvenile Skeleton*. Sand Diego: Academic Press.

Schmeidt G. 1970. Contributo alla ricostruzione della situazione geotopografica di Velia nell' Antichita. *Parola Passato* 25: 65– 92.

- Schmitt NM, Schmitt J, and Dören M. 2009. The role of physical activity in the prevention of osteoporosis in postmenopausal women—An update. *Maturitas* 63: 34-38.
- Schoenau, E., Fricke, O and Rauch, F. 2003. The regulation of bone development as a biological system. *Homo* 54(2): 113-118.
- Schoenau, E. 2005. From mechanostat theory to development of the “Functional Muscle-Bone-Unit”. *Journal of Musculoskeletal and Neuronal Interactions* 5(3): 232-238.
- Schultz, M. 2001. Paleohistopathology of Bone: A New Approach to the Study of Ancient Diseases. *Yearbook of Physical Anthropology* 44: 106-147.
- Schultz, M. 2003. Differential Diagnoses of Intravital and Postmortem Bone Loss at the Micro-Level. In: Agarwal, SC and Stout, SD, eds., *Bone Loss and Osteoporosis: An Anthropological Perspective*. New York: Kluwer Academic/Plenum Publishers. 173-188.
- Schwartz, RN. 1998. *The Roman Empire: A Concise History of the First Two Centuries*. Maryland: University Press of America.
- Schwartz, L., Maitournam, H., Stolz, C., Steayert, JM., Ho Ba Tho, MC and Halphen, B. 2003. Growth and cellular differentiation: a physic-biochemical conundrum? The example of the hand. *Medical Hypotheses* 61(1): 45-51.
- Scullard, HH. 1980. *A History of the Roman World 753-146 BC*. London and New York: Methuen & Co.
- Shakespeare, T. 1999. Commentary: Observations on Disability and Archaeology. *Archaeological Review from Cambridge* 15(2):99-101.
- Shotter, D. 2004. *Roman Britain 2<sup>nd</sup> Edition*. London and New York: Routledge.
- Shotter D. 2005. *The Fall of the Roman Republic 2<sup>nd</sup> edition*. Oxford: Routledge.
- Seeman E. 1996. The effects of tobacco and alcohol use on bone. In: Marcus R, Feldman D, Kelsey J, eds. *Osteoporosis*. San Diego: Academic Press: 577–97.
- Seeman, E. 1997. From Density to Structure: Growing Up and Growing Old on the Surfaces of Bone. *Journal of Bone and Mineral Research* 12(4): 509-521.
- Seeman, E. 1999. Genetic Determinants of the Population Variance in Bone Mineral Density. In: Rosen, GJ., Glowacki, J and Bilezikian, JP, eds. *The Aging Skeleton*. San Diego: Academic Press. 77-94.
- Seeman E. 2002. Pathogenesis of bone fragility in women and men. *The Lancet* 359: 1841-1850.

- Segal, DA, and SJ Yanagisako. 2005. *Unwrapping the Sacred Bundle: Reflections on the Disciplining of Anthropology*. Durham: Duke University Press.
- Shaffer, JR., Kammerer, CM., Bruder, JM. Cole, SA, Dyer, TD and Almasy., L 2008. Genetic influences on bone loss in the San Antonio Family Osteoporosis study. *Osteoporosis International* 19: 1759–1767.
- Shahtaheri, SM., Aaron, J., Johnson, D and Purdie, DW. 1999. Changes in trabecular bone architecture in women during pregnancy. *British Journal of Obstetrics and Gynaecology* 106: 432-438.
- Shapiro, H. L. 1939 *Migration and Environment*. New York: Oxford University Press.
- Sievänen, H., Kannus, P., and Jarvinen, TLN. 2007. Bone quality: An empty term. *PLoS Medicine* 4(3): 0407-0410.
- Sievert, LL. 2006. *Menopause: A Biocultural Perspective*. Rutgers University Press.
- Singh, M., Nagrath, AR and Maini, PS. 1970. Changes in the trabecular pattern of the upper end of the femur as an index of osteoporosis. *Journal of Bone and Joint Surgery British Volume* 52a: 457-467.
- Sigismund- Nielsen, H. 2007. Children for profit and pleasure. In: Harlow M, Laurence R (eds.). *Age and Ageing in the Roman Empire, Journal of Roman Archaeology, Supplementart No. 65*, Journal of Roman Archaeology: Portsmouth; 37-54.
- Sinclair, D and Dangerfield, P. 1998. *Human Growth after Birth*. Oxford: Oxford University Press.
- Singer, M. 1989. The Limitations of Medical Ecology: The Concept of Adaptation in the Context of Social Stratification and Social Transformation. *Medical Anthropology* 10: 223-234.
- Singer, M. 1992. The Application of Theory in Medical Anthropology: An Introduction. *Medical Anthropology* 14: 1-8.
- Skedros, JG. 2012. Interpreting load history in limb-bone diaphyses: important considerations and their biomechanical foundations. In: Crowder, C and Stout, S, eds, *Bone Histology: An Anthropological Perspective*. Boca Raton: CRC Press. 153-220.
- Skerry, TM. 2006. One Mechanostat or Many? Modifications of the site-specific response of bone to mechanical loading by nature and nurture. *Journal of Musculoskeletal and Neuronal Interactions* 6(2): 122-127.
- Skinner MF, Dupras TL, and Moya-Sola S. 1995. Periodicity of enamel hypoplasia among Miocene Dryopithecus from Spain. *Journal of Paleopathological Monographs* 7:197–222.

- Slemenda, CW., Peacock, M., Hul, S., Zhou, L and Johnston, CC. 1997. Reduced rates of skeletal remodeling are associated with increased bone mineral density during development of peak skeletal mass. *Journal of Bone and Mineral Research* 12: 676-682.
- Smay DB, and Armelagos GJ. 2000. Histologic examination of apparent stages of healing in cribra orbitalia: a new method using silicone casting material. *American Journal of Physical Anthropology* (Suppl 30): 284
- Sobelman O.S., Gibeling J.C., Stover S.M., Hazelwood S.J., Yeh O.C., Shelton D.R. and Martin R.B. 2004. Do microcracks decrease or increase fatigue resistance in cortical bone? *Journal of Biomechanics.*, 37:1 295-1303.
- Sofaer Deverenski, J. 2000. Sex differences in activity-related osseous change in the spine and the gendered division of labor at Ensay and Wharram Percy, UK. *American Journal of Physical Anthropology* 111: 333-354.
- Sofaer, J. 2006. Gender, bioarchaeology, and human ontogeny. In: Gowland, R and Knüssel, C., eds. *Social Hierarchy of Funerary Remains*. Oxford: Oxbow. 155-167.
- Sofaer, J. 2011. Towards a social bioarchaeology of age. In: Agarwal SC and Glencross B (eds.). *Social Bioarchaeology*. Wiley-Blackwell: New York. 285-311.
- Soloman, L. 1979. Bone density in aging Caucasian and African populations. *Lancet* 2: 1326-1330.
- Sornay-Rendu, E., Boutroy, S., Munoz, F., and Bouxsein, ML. 2009. Cortical and trabecular architecture are altered in postmenopausal women with fractures. *Osteoporosis International* 20: 1291-1297.
- Sowers M, Clark MK, Hollis B, Wallace RB, and Jannausch M. 1992. Radial bone mineral density in pre- and perimenopausal women: a prospective study of rates and risk factors for loss. *Journal of Bone Mineral Research* 7: 647-657.
- Sowers MR, and Galuska DA. 1993. Epidemiology of bone mass in premenopausal women. *Epidemiological Reviews* 15:374-398.
- Sowers M, Eyre D, Hollis BW, Randolph JF, Shapiro B, Jannausch ML, and Crutchfield M. 1995. Biochemical markers of bone turnover in lactating and nonlactating postpartum women. *Journal of Clinical Endocrinology and Metabolism* 80: 2210-2216.
- Sowers M. 1996. Pregnancy and lactation as risk factors for subsequent bone loss and osteoporosis. *Journal of Bone Mineral Research* 11: 1052-1060.

- Spencer, H., Kramer, L., DeBartolo, M., Norris, C and Osis, D. 1983. Further studies of the effect of a high protein diet as meat on calcium metabolism. *American Journal of Clinical Nutrition* 37: 924-929.
- Stambaugh, JE. 1988. *The Ancient Roman City*. Baltimore and London: Johns Hopkins University Press.
- Steckel, RH and Rose, JC. 2002. *The Backbone of History: Health and nutrition in the Western hemisphere*. Cambridge: Cambridge University Press.
- Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation: Bone Disease in Ancient Human Populations*. Springfield, Charles C. Thomas.
- Stewart, TL and Ralston, SH. 2000. Role of genetic factors in the pathogenesis of osteoporosis. *Journal of Endocrinology* 166: 215-245.
- Stevenson JC, Lees B, Devenport M, Cust MP, and Ganger KF. 1989. Determinants of bone density in normal women: risk factors for future osteoporosis? *British Medical Journal [Clinical Research]* 298: 924–928.
- Stini, WA. 1995. Osteoporosis in Biocultural Perspective. *Annual Review of Anthropology* 24: 397-421.
- Stinson, A., Bogin, B., Huss-Ashmore, R and O'Rourke, D. 2000. *Human Biology: An Evolutionary and Biocultural Perspective*. New York: Wiley Liss.
- Stock, J and Pfeiffer, S. 2001. Linking Structural Variability in Long Bone Diaphyses to Habitual Behaviors: Foragers from the Southern African Later Stone Age and the Andaman Islands. *American Journal of Physical Anthropology* 115: 337-348.
- Stojanowski, CM, and JE Buikstra. 2005. Research Trends in Human Osteology: A Content Analysis of Papers Published in the *American Journal of Physical Anthropology*. *American Journal of Physical Anthropology* 128:98-109.
- Stojanowski, CM, and MA Schillaci. 2006. Phenotypic approaches for understanding patterns of intracemetery biological variation. *Yearbook of Physical Anthropology* 131:49-88.
- Storey, R. 1992. Preindustrial Urban Lifestyle and Health. In: Huss-Ashmore, R., Schall, J., and Hediger, M. (eds.) *Health and Lifestyle Change, MASCA Research Papers in Science and Archaeology* 9. Philadelphia: University Museum of Archaeology and Anthropology, University of Pennsylvania. 33–42.

Storey, R. (1997). Individual frailty, children of privilege, and stress in Late Classic Copán. In (S.L. Whittington & D.M. Reed, Eds.) *Bones of the Maya: Studies of Ancient Skeletons*. Washington, D.C.: Smithsonian Institution Press, pp. 116-137

Stout, SD and Paine, RE. 1994. Brief Communication: Bone Remodeling Rates: A Test of an Algorithm for Estimating Missing Osteons. *American Journal of Physical Anthropology* 93: 123-129.

Stout, SD and Teitelbaum, SL. 1976. Histological Analysis of Undecalcified Thin Sections of Archaeological Bone. *American Journal of Physical Anthropology* 44: 263-270.

Stout, SD and Simmons, DJ. 1979. Use of Histology in Ancient Bone Research. *Yearbook of Physical Anthropology* 22: 228-249.

Stout, SD and Lueck, R. 1995. Bone Remodeling Rates and Skeletal Maturation in Three Archaeological Skeletal Populations. *American Journal of Physical Anthropology* 98: 161-171.

Stuart-Macadam P. 1989. Porotic hyperostosis: relationship between orbital and vault lesions. *American Journal of Physical Anthropology* 80: 187-193.

Stuart-Macadam, P. (1991). Anaemia in Roman Britain: Poundbury Camp. In: Bush, H and Zvelebil, M, eds, *Health in Past Societies: Biocultural Interpretations of Human Skeletal Remains in Archaeological Contexts*. BAR International Series 567. Oxford, Oxbow Books. 101-14.

Stuart-Macadam PL.1992. Porotic hyperostosis: a new perspective. *American Journal of Physical Anthropology* 87: 39-47.

Szulc P, Seeman E, Duboeuf F, Sornay-Rendu E, and Delmas P. 2006. Bone fragility: failure of periosteal apposition to compensate for increased endocortical resorption in postmenopausal women. *Journal of Bone and Mineral Research* 21(12): 1856-1863.

Temple DH. 2008. What can variation in stature reveal about environmental differences between prehistoric Jomon foragers? Understanding the impact of systemic stress on developmental stability. *American Journal of Human Biology* 20(4): 431-439.

Ten Cate, A.R. (1994). *Oral Histology: Development, Structure, and Function*. 4<sup>th</sup> Edition. St. Louis, Mosby.

Thomsen JS, Ebbesen EN, and Mosekilde L. 2002. Age-related differences between thinning of horizontal and vertical trabeculae in human lumbar bone as assessed by a new computerized method. *Bone*. 31: 136-142.

Thompson, DD and Gunness-Hey, M. 1981. Bone Mineral-Osteon Analysis of Yupik-Inupiaq Skeletons. *American Journal of Physical Anthropology* 55: 1-7.



- Tingay, GIF and Badcock, J. 1989. *These Were the Romans*, 2<sup>nd</sup> edition. Cheltenham: Stanley Thornes and Hulton.
- Toledo VAM, Jergas M. 2006. Age-related changes in cortical bone mass: data from a German female group. *European Radiology* 16(4): 811-817.
- Toner, J. 2002. *Rethinking Roman History*. Cambridge: Oleander Press.
- Toner, J. 2009. *Popular Culture in Ancient Rome*. Cambridge: Polity.
- Turner-Walker G, Syverson U, and Mays S. 2001. The archaeology of osteoporosis. *Journal of European Archaeology* 4: 263-268.
- Turner, CH. 1999. Toward a Mathematical Description of Bone Biology: The Principle of Cellular Accomodation. *Calcified Tissue International* 65: 466-471.
- Turner, CH. 2000. Toward a Mathematical Description of Bone Biology: The Principle of Cellular Accomodation. *Calcified Tissue International* 67: 185-187.
- Twomey L, Taylor J, Furniss B. 1983. Age changes in the bone density and structure of the lumbar vertebral column. *Journal of Anatomy* 136:15–25.
- Ulijaszek, S.J. (1998). Measurement error. In (S.J. Ulijaszek, F.E. Johnston, & M.A. Preece, Eds.) *The Cambridge Encyclopedia of Growth and Development*. Cambridge, Cambridge University Press, pp. 28.
- Ulrich, D., van Rietbergen, B., Laib, A and Rügsegger, P. 1999. The Ability of Three-Dimensional Structural Indices to Reflect Mechanical Aspects of Trabecular Bone. *Bone* 25(1): 55-60.
- US Department of Health and Human Services. 2004. Bone health and osteoporosis: a report of the surgeon general. Rockville, MD: US Department of Health and Human Services, Office of the Surgeon General.
- Van Gerven, D. P., J. R. Hummert, and D. B. Burr. 1985. Cortical Bone Maintenance and Geometry of the Tibia in Prehistoric Children from Nibia's Batn el Hajar. *American Journal of Physical Anthropology* 66:272–280.
- Van Oers, RFM., Ruimerman, R., van Rietbergen, B., Hilbers, PAJ., and Huiskes, R. 2008. Relating osteon diameter to strain. *Bone* 43: 476-482.
- Vajda, EG and Bloebaum, RD. 1999. Age-Related Hypermineralization in the Female Proximal Femur. *The Anatomical Record* 255: 202-211.



- Verano, J.W. & Ubelaker, D.H., Eds. (1992). *Disease and Demography in the Americas*. Washington, D.C., Smithsonian Institution Press.
- Vieth, R. 2005. The role of vitamin D in the prevention of osteoporosis. *Annals of Medicine* 37: 278-285.
- Virtama P and Helelä T. 1969. Radiographic measurements of cortical bone: variation in a normal population between 1 and 90 years of age. *Acta Radiologica Supplement* 293, 1-268.
- Vogel M, Hahn M, Caselitz P, Woggan J, Pompesius-Kempa M, and Delling G. 1990. Comparison of trabecular bone structure in man today and an ancient population in Western Germany. In: Takahashi HE, ed, *Bone morphometry*. Tokyo, Japan: Nishimura Co. 220–223.
- Walker, PL. 1995. Problems of Preservation and Sexism in Sexing: Some lessons from historical collections for paleodemographers. In: Saunders, SR and Herring, A, eds., *Grave Reflections*. Toronto: Canadian Scholars' Press. 31-47.
- Walker, PL. 2005. Greater Sciatic Notch Morphology: Sex, age and population differences. *American Journal of Physical Anthropology* 127: 385-391.
- Walker, PL., Bathurst, RR., Richman, R., Gjerdrum, T., and Andrushko, A. 2009. The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis. *American Journal of Physical Anthropology* 139(2): 109-125.
- Wall, JC., Chatterji, SK., and Jeffrey, JW. 1979. Age-related changes in the density and tensile strength of human femoral cortical bone. *Calcified Tissue International* 27(2): 105-108.
- Wang, O., Nicholson, P., Suuriniemi, M., Lyytika IA., Helkala, E., Alen, M., Suominen, H and Cheng, S. 2004. Relationship of Sex Hormones to Bone Geometric Properties and Mineral Density in Early Pubertal Girls. *Journal of Endocrinology and Metabolism* 89(4): 1698-1703.
- Wapler U, Crubezy E, Schultz M. 2004. Is cribra orbitalia synonymous with anaemia? Analysis and interpretation of cranial pathology in Sudan. *American Journal of Physical Anthropology* 123: 333–339.
- Warburton D, Nicol C, Bredin S. 2006. Health benefits of physical activity: the evidence. *Canadian Medical Association Journal* 174: 801-809.
- Ward JA, Lord SR, Williams P, Anstey K, Zivanovic E. 1995. Physiologic health and lifestyle factors associated with neck bone density in older women. *Bone* 16: 373s-378s.
- Warren, KB. ed. 1951. *Origin and Evolution of Man*. New York: Long Island Biological Association.

- Warren, MP. 1999. Hormonal Influences on the Establishment of Peak Bone Mass. In: Rosen, GJ., Glowacki, J and Bilezikian, JP, eds., *The Aging Skeleton*. San Diego: Academic Press. 115-126.
- Waterlow J.C. 1989. Diet of the classical period of Greece and Rome. *European Journal of Clinical Nutrition*. 43: 3-12.
- Watts, NB. 2002. Bone quality: Getting closer to a definition. *Journal of Bone and Mineral Research* 117(7): 1148-1150.
- Weaver DS. 1998. Osteoporosis in the bioarchaeology of women. In: Grauer A, Stuart-Macadam P, editors. *Sex and Gender in Paleopathological Perspective*. Cambridge: Cambridge University Press. 27–46.
- Wegrzyn, J., Roux, JP., Arlot, ME., Boutroy, S., Vilayphiou, N., Guyen, O., Delmas, PD., Chapurlat, R., and Bouxsein, ML. 2010. Role of trabecular microarchitecture and its heterogeneity parameters in the mechanical behavior of ex vivo L3 vertebrae. *Journal of Bone and Mineral Research* 25 (11): 2324-2331.
- Weitzmann, MN and Pacifici, R. 2006. Estrogen deficiency and bone loss: and inflammatory tale. *Journal of Clinical Investigation* 116(5): 1186-1194.
- Wheeler, S. 2009. *Bioarchaeology of Infancy and Childhood at the Kellis 2 Cemetery, Dakhleh Oasis, Egypt*. PhD Dissertation. London, Ontario: University of Western Ontario.
- White, KS. 1976. Food requirements and food supply in classical times in relation to the various classes. *Progress in Food and Nutrition Science* 2: 143-191.
- White, KD. 1988. Farming and animal husbandry. In: Grant, M and Kitzinger, R., eds. *Civilization of the Ancient Mediterranean*. New York: Charles Scribner's Sons. 211-246.
- White, TD and Folkens, PA. 2005. *The Human Bone Manual*. Burlington: Elsevier Academic Press.
- Wiley, AS. 1992. Adaptation and the Biomedical Paradigm in Medical Anthropology: A Critical Review. *Medical Anthropology Quarterly* 6(3): 216-236.
- Wiley, AS. 1993. Evolution, Adaptation and the Role of Biocultural Medical Anthropology. *Medical Anthropology Quarterly* 7(2): 192-199.
- Wishart JM, Horowitz H, Bochner M, Need AG, Nordin BEC. 1993. Relationships between metacarpal morphometry, fore- arm and vertebral bone density and fractures in postmenopausal women. *British Journal of Radiology* 66: 435–440.

- Witt, JM., Balen, HV., Kamp, GA and Oostdijk, W. 2004. Benefit of postponing normal puberty for improving final height. *European Journal of Endocrinology* **151**: s41-s45.
- Wizemann, T and Pardue, ML. eds. 2001. *Exploring the Biological Contributions to Human Health: Does Sex Matter?* National Academy Press: Washington, D.C.
- Wohl, GR., Loehrke, L., Watkins, BA., Zernicke, RF. 1998. Effects of High-Fat Diet on Mature Bone Mineral Content, Structure, and Mechanical Properties. *Calcified Tissue International* **63**: 74-79.
- Wolff, J. 1870. Über die innere Architecture des Knochen und ihre Bedeutung für die Frage von Knochenwachstum. *Virchows Arch. Pathol. Anat.* **50**: 324-341.
- Wong, PA. 1981. Computed Tomography in Paleopathology: Technique and Cast Study. *American Journal of Physical Anthropology* **55**: 101-110.
- Wood, J.W., Milner, G.R., Harpending, H.C., and Weiss, K.M. 1992. The osteological paradox: problems of inferring prehistoric health from skeletal samples. *Current Anthropology* **33**: 343-370.
- Worthman, CM. 1995. Hormones, Sex, and Gender. *Annual Review of Anthropology* **24**: 593-616.
- Worthman, CM and Kuzara, J. 2005. Life history and the early Origins of health differentials. *American Journal of Physical Anthropology* **17**: 95-112.
- Wright, LE. and Chew, F. 1998. Porotic hyperostosis and paleoepidemiology: a forensic perspective on anemia among the ancient Maya. *American Anthropologist* **100**: 924- 939.
- Wright, LE, and CJ Yoder. 2003. Recent Progress in Bioarchaeology: Approaches to the Osteological Paradox. *Journal of Archaeological Research* **11**(1):44-70.
- Wu, K., Schubeck, KE., Frost, H and Villanueva, A. 1970. Haversian Bone Formation Rates Determined by a New Method in a Mastodon, and in Human Diabetes Mellitus and Osteoporosis. *Calcified Tissue Research* **6**: 204-219.
- Young, R. L., and A. V. Badyaev 2007 Evolution of Ontogeny: Linking Epigenetic Remodeling and Genetic Adaptation in Skeletal Structures. *Integrative and Comparative Biology* **47**:234–244.
- Zuckerman, MK and Armelagos, GJ. 2011. The origins of biocultural dimensions in bioarchaeology. In: Agarwal SC and Glencross B (eds.). *Social Bioarchaeology*. Wiley-Blackwell: New York. 15-43.

## Appendix A – Basic Histomorphometry Measures

	<b>P<sub>i</sub></b>	<b>P<sub>f</sub></b>	<b>A<sub>o</sub> (mm<sup>2</sup>)</b>	<b>A<sub>p</sub> (mm)</b>	<b>P<sub>o</sub> (mm<sup>2</sup>)</b>	<b>P<sub>h</sub> (mm)</b>
<b><i>Females</i></b>						
18-29 <i>n</i> = 7	73.43 ± 28.62	34.86 ± 15.69	0.031 ± 0.005	0.71 ± 0.06	0.0018 ± 0.0003	0.17 ± 0.02
30-49 <i>n</i> = 9	70.89 ± 10.49	43.56 ± 10.52	0.030 ± 0.005	0.71 ± 0.07	0.0019 ± 0.0005	0.17 ± 0.03
50+ <i>n</i> = 10	64.4 ± 25.11	39.5 ± 15.40	0.027 ± 0.004	0.66 ± 0.05	0.0023 ± 0.0007	0.19 ± 0.03
<b><i>Males</i></b>						
18-29 <i>n</i> = 2	102.5 ± 61.52	29 ± 9.90	0.029 ± 0.002	0.68 ± 0.04	0.0018 ± 0.0007	0.18 ± 0.002
30-49 <i>n</i> = 13	100.23 ± 32.88	47.23 ± 20.59	0.030 ± 0.003	0.69 ± 0.04	0.0020 ± 0.0006	0.18 ± 0.03
50+ <i>n</i> = 11	90.82 ± 35.25	47.27 ± 23.21	0.028 ± 0.007	0.68 ± 0.07	0.0023 ± 0.001	0.18 ± 0.03

P<sub>i</sub> (number of intact osteons); P<sub>f</sub> (number of osteon fragments); A<sub>o</sub> (mean osteon area); A<sub>p</sub> (mean osteon perimeter); P<sub>o</sub> (mean Haversian canal area); P<sub>h</sub> (mean Haversian canal perimeter); ± (1 standard deviation)

## Appendix B – Vertebral Neural Canal Measures

	T-AP	T-ML	L-AP	L-ML	T-ABH	T-PBH	L-ABH	L-PBH
<i>Females</i>								
18-29 <i>n</i> = 9	15.05 ± 1.55	17.69 ± 1.55	16.67 ± 2.24	21.52 ± 2.26	18.08 ± 1.98	19.90 ± 3.03	27.37 ± 2.22	26.36 ± 1.32
30-49 <i>n</i> = 12	15.51 ± 1.05	17.63 ± 1.10	16.58 ± 1.21	22.18 ± 1.33	19.93 ± 1.83	22.21 ± 2.14	26.53 ± 1.36	26.8 ± 0.82
50+ <i>n</i> = 9	14.96 ± 0.97	16.72 ± 1.65	15.18 ± 1.04	22.44 ± 1.86	18.42 ± 2.02	20.15 ± 2.16	26.24 ± 0.74	26.02 ± 1.45
<i>Males</i>								
18-29 <i>n</i> = 5	15.08 ± 0.83	17.43 ± 1.05	15.33 ± 0.85	21.50 ± 1.13	20.23 ± 1.10	21.84 ± 1.25	26.87 ± 1.79	27.42 ± 1.62
30-49 <i>n</i> = 19	15.18 ± 0.97	17.47 ± 1.41	15.93 ± 1.58	23.54 ± 1.46	20.37 ± 1.0	22.14 ± 1.27	27.54 ± 1.36	27.36 ± 1.71
50+ <i>n</i> = 13	15.57 ± 1.30	18.00 ± 1.47	16.03 ± 1.39	23.33 ± 1.21	19.77 ± 1.39	21.74 ± 1.38	26.62 ± 1.44	27.21 ± 1.64

T-AP (mean thoracic anterior-posterior distance); T-ML (mean thoracic medial-lateral distance); L-AP (mean lumbar anterior-posterior distance); L-ML (mean lumbar medial-lateral distance); T-ABH (mean thoracic anterior body height); T-PBH (mean thoracic posterior body height); L-ABH (mean lumbar anterior body height); L-PBH (mean lumbar posterior body height); ± (1 standard deviation)

## Appendix C – Juvenile Femoral Lengths

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<b>Age Group</b>	<b>N</b>	<b>Mean (mm)</b>	<b>SD (mm)</b>
0.25	16	76.31	4.00
0.5	5	83.93	2.21
0.75	5	106.88	6.98
1.5	9	130.70	11.93
2.5	7	155.17	12.24
3.5	3	171.67	3.21
4.5	2	193.50	7.78
5.5	3	210.33	9.07
6.5	2	231.50	12.02
8.5	2	275.00	5.66
9.5	4	267.25	12.12
11-13	2	330.00	14.14
<b>Total N =</b>	<b>60</b>		

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