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BONE MINERAL DENSITY VARIATION: DEMOGRAPHIC, LIFESTYLE, AND SOCIOECONOMIC FACTORS IN URBAN DETROIT

By

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ABSTRACT

BONE MINERAL DENSITY VARIATION: DEMOGRAPHIC, LIFESTYLE, AND SOCIOECONOMIC FACTORS IN URBAN DETROIT

By

Mary Schultz Megyesi

The ubiquitous use of racial categories in bone density and osteoporosis research has contributed to widely held erroneous assumptions about inherent racial differences in bone mineral density (BMD). Racial concepts in the BMD literature are often not defined and ambiguous, frequently leading to conclusions about biological or inherent racial differences in BMD variation. In addition, racial variables are often treated as singular variables instead of complex proxies for several attributes that can affect the skeleton, such as diet, behavior, environment, and socioeconomic status.

The concept of race, as understood in anthropology, is inconsistent with presumptions of biological differences. Anthropological views on the concept of race, however, are not monolith, and other scientific disciplines do not necessarily subscribe to similar notions. In fact, clinical and medical research quite often uses racial categories as convenient shortcuts for biological differences between populations. This common practice frequently results in interpretations of racial differences in BMD that default to biological explanations, without carefully measuring other social or environmental covariates. Such flaws in the interpretation of racial BMD differences obscures rather than clarifies the underlying causes of BMD variation.

This dissertation addresses this problem by examining social, economic, and lifestyle variation in bone density more directly, without assuming that racial variables contribute meaningful biological information. This analysis explores intra-group BMD

variation in a sample of African-American participants from Detroit, Michigan. Systematic racial differences in BMD commonly attributed to biological factors may be more effectively captured by demographic (age, sex, and body size), lifestyle (diet, physical activity and smoking), and socioeconomic status (income, education, occupation and other social features). In limiting this investigation of BMD to one, traditionally understudied group, several pitfalls associated with racial categories are avoided and advantages are gained. Investigation of social, economic, demographic, and behavioral characteristics important to bone mass can be examined directly without resorting to ambiguous notions of inherent racial differences. In addition, the complex relationship between race and factors that can affect BMD can be clarified.

This analysis uncovered previously unrecognized correlates with BMD and revealed that the relationship between variables associated with economic strain and BMD is likely to be discordant. The complex relationship between socioeconomic status and BMD may not be suited to analyses that use composite socioeconomic status scores. This analysis determined that body size was one of the most important factors to BMD differences. Systematic body size differences between populations may be one of the primary causes for racial BMD differences seen in the U.S.

Many anthropologists and public health experts advocate moving away from presumptions of racial biological difference and towards explanations based in social and environmental causes. This analysis represents one facet of that initiative which considers bone mass and bone density.

This dissertation is dedicated to Dr. William T. Schultz,
a great scientist, naturalist, and father, who gave me a cow bone (humerus of *B. taurus*)
for show and tell, caught crickets for my pet praying mantis, and instilled in me a respect
and wonder for the natural world that has fueled my pursuits in anthropology and beyond.
I love you Dad,
and thanks.

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KEY TO ABBREVIATIONS

BMC	Bone Mineral Content
BMD	Bone Mineral Density
BMI	Body Mass Index
BMAD	Bone Mineral Apparent Density
CDC	Centers for Disease Control and Prevention
CUAAH	Center for Urban African-American Health
DXA	Dual Energy X-ray Absorptiometry
EXCEL	Exploring Changes in Experiences and Lifestyles (CUAAH study)
FN	Femoral Neck bone density
NIH	National Institutes of Health
ONOSS	Obesity, Nitric Oxide, Oxidative Stress and Salt Sensitivity (CUAAH study)
SES	Socioeconomic Status
TH	Total Hip bone density
WBBMC	Whole Body Bone Mineral Content
WHLS	Women's Healthy Lifestyle (CUAAH study)
ZFN	Z-score of the Femoral Neck
ZTH	Z-score of the Total Hip

Introduction

Anthropology and the social science disciplines have long argued that racial classifications are social constructs and not based on biological or genetic attributes (Gravlee, 2009;Keita et al., 2004;Weiss, 1998). Most social scientists agree that health research using racial classifications needs to be cautious about what information these variables capture (Goodman, 2000;Templeton, 2002;Winker, 2006;Wolf, 1994). Racial identities are quite complex and often interrelated with several attributes that can influence health. In medical contexts, however, racial classifications are frequently used as if they are synonymous with biological variation (Bamshad et al., 2004;Collins, 2004;Tang et al., 2005).

Recently, there has been a great deal of attention on the controversy surrounding the interpretation and use of racial variables in genetic, epidemiological, medical, and clinical research. Several authors have expressed concern over the increasing number of scientific publications that interpret racial differences in health and disease as biological or genetic in origin (Comstock et al., 2004;Frank, 2007;Jones et al., 1991;Shanawani et al., 2006). Reducing racial categories to biological meanings often occurs in these studies because racial categories are not treated with the same rigor as environmental or behavioral variables. Racial concepts are quite commonly ambiguous and vague, and racial classifications are treated as if they are obvious and self-evident when in fact quite the opposite is true.

Racial categories are routinely used in public health, epidemiological, and clinical studies to define groups, base comparisons, and report findings. Numerous authors have noted that racial categories in such studies are typically poorly defined, applied inconsistently, and are associated with unclear concepts that make them unsuited for use as variables in scientific studies (Braun, 2002;Dressler et al., 2005;Hunt & Megyesi, 2008b;Shields et al., 2005). Unfortunately, lack of careful attention to the way in which racial variables are defined and applied can often lead to erroneous conclusions about biological or inherent racial difference. In many studies, interpretations of racial difference in health or disease are driven by underlying notions about innate racial differences and not the evidence or data collected.

The problems associated with ambiguous notions of racial variables in the medical and clinical literature are regularly discussed, yet racial categories continue to be used in numerous studies without addressing these issues. One particular area where the use of racial categories is quite common is in bone density and osteoporosis research. Interpretations of racial categories in bone density and osteoporosis research routinely invoke biological, inherent, and genetic explanations, however, there is very little critical discourse about these issues (Fausto-Sterling, 2008). This dissertation examines the use of racial categories in bone density research and addresses how analyses could be constructed to limit erroneous interpretations about inherent racial differences in bone mass.

Racial differences in bone density between white and black Americans are well documented, but not well understood. If we reject biological presumptions of racial differences in bone density, what else could these differences be attributed to? There is

extensive evidence that a variety of non-biological factors are influential to bone density in adults. These include, but are not limited to: body size, diet, calcium intake, sex, and physical activity. In addition, socioeconomic status has been widely recognized as being one of the most important determinants of health, but this relationship is only rarely studied in bone density research (see: Fausto-Sterling, 2008;Krieger et al., 2003;Leslie & Lentle, 2006). Could these factors contribute to what is often presumed to be 'inherent' racial differences in bone density?

In Chapter One I examine the history of the concept of race in anthropology in order to provide a background on the genesis of the concept. Skeletal analyses played a key role in the transformation of the meaning of the race concept in anthropology over the last 400 years. Currently, bone density is frequently invoked as an example of inherent racial differences, much like other skeletal measures were in the past. Chapter 2 reviews how racial categories have often retained biological meanings in medical and clinical contexts in contrast to anthropology. Bone density research has primarily progressed over the last 40 years within the medical field, and racial differences are frequently interpreted as innate or biological.

The way in which interpretations of racial difference in bone density are generated requires an in depth analysis of the bone density and osteoporosis literature. Chapter 3 is a literature review examining how racial variables are conceptualized, applied, and interpreted in bone density and osteoporosis studies. Unclear racial concepts, inconsistent methodology, and interpretations based on *a priori* assumptions of racial differences frequently drive the analysis of bone density. Under these circumstances, racial differences in bone density are quite suspect and should be

investigated without presumptions of inherent racial bone density difference. Chapter 4 addresses the question raised from the literature review: If "racial" differences cannot be biological, then what else could be contributing to the pattern of bone density variation seen in the U.S.? Various environmental, behavioral, demographic, and economic factors can affect the skeleton and contribute to variation in bone density.

The analysis of this dissertation investigates the contribution of several demographic, lifestyle, and socioeconomic attributes to variation in bone density in a sample of African-American participants from Detroit, Michigan. Details of this sample used in this analysis are discussed in Chapter 5. By limiting this investigation to one racial group, several drawbacks associated with the use of racial categories are avoided and other advantages are gained. Consideration of the contribution of non-biological factors to bone density variation is explored without resorting to ambiguous notions of inherent racial differences. Presumptions of inherent racial differences, which plague the interpretations of many studies comparing bone density between racial groups, are eliminated in a study of intra-racial variation.

The final two chapters of this dissertation present the results of my analysis and the discussion and conclusions. I argue in the final chapter of this dissertation that based on the results of the analysis, that much of the variation in bone density in this sample can be attributed to non-biological factors. In particular, systematic racial differences in bone density seen in the U.S. may stem not from 'inherent' racial differences, but from population-based differences in body size. This analysis identified several new socioeconomic variables that are associated with bone density. Careful consideration of both body size and socioeconomic status are warranted for future investigations. These

attributes may help clarify the causes of inter-racial bone density differences documented in the U.S.

Chapter 1

The "Skull Doctors" and the History of Biological Race

When scientists first began to investigate human origins and human variation during the age of exploration and European expansion, they initiated the specialty of skeletal biology and played a significant role in the development of physical anthropology (Baker, 1998;Brace, 1982;Smedley, 1999). The "skull doctors" (a term used by Michael Blakey, see: (Blakey, 1987;Blakey, 1996) included anatomists and other scientists of the 18th and 19th centuries who measured cranial capacity and documented skeletal features in order to compare various human groups, or 'races'. Explanations and interpretations of human skeletal variation studied during this time formed the beginning of scientific inquiry of 'race' and 'racial' groups. This was a transformative era in history as both the science of skeletal biology and the concept of race arose from similar avenues of exploration. Many prominent scientists throughout the 18th to 20th century pursued physical anthropology, while at the same time influenced and shaped future notions of race.

Therefore, the history of the "skull doctors" is the history of both skeletal biology, and the concept of race, as they are intimately entwined. Interpretations based on skeletal analyses contributed to the transformation of the term 'race' from a general categorizing term, to a term synonymous with biology, to its current position as a socio-cultural construct in anthropology. This chapter chronicles how skeletal analysis and the use of

various bone measures were catalysts for the changing definitions of the concept of race in physical anthropology.

Etymology of 'Race' and the Age of Discovery

During the 16th century, words such as *razza* in Italian and *reazza* in Spanish were used as general categorizing terms similar to *type*, *kind*, or *breed,* in reference to both groups of people and animal breeding stocks (Smedley, 1999:37-41). The Italian, Spanish, Portuguese, Dutch, French, German, and English often used an idea of *razza* or *reazza* to describe and refer to different human groups as they established colonial empires in Asia, Africa, and the Americas (Smedley, 1999). Over time the term was anglicized to 'race' and was often used by European explorers to describe and refer to different groups encountered in the Americas. Prior to this, religion and language were the most important criteria of identity in the empires of the ancient world such as Egypt, Greece, and Rome, and people were incorporated regardless of physical traits and variations (Graves, 2001:17).

The concept of race experienced a remarkable transformation between the 16th and 19th centuries. The prevailing definition in the 19th and early 20th centuries was one synonymous with human biological variation, which stands in pointed contrast to its use at the inception in the 16th century as a general grouping term. During this transformation, race became a core construct in the emerging discipline of physical anthropology via skeletal analysis, especially of the cranium, by the "skull doctors" of Europe and the American Colonies.

Physical characteristics become increasingly important to describe different 'varieties' or 'types' of people encountered during European colonial expansion during the 16th through 18th centuries. This was a pivotal difference in how human variation was conceptualized. Prior to extensive travel by boat in the 16th century, differences between human groups were encountered gradually (Brace, 2005). The expansion of the slave trade between Africa and North America during the 17th through 19th centuries further propelled the definition of race as distinguishing types of people through physical characteristics. The compelling physical contrast between European colonialists and West Africans cemented the idea of racial 'types' in the minds of many early American scientists (Graves, 2001;Smedley, 1999;Smedley & Smedley, 2005). Slavery in the American colonies introduced an entirely different system that was lifelong, hereditary, and based on 'racial' traits such as skin color. The slave trade in North America helped solidify the race concept as a way of physically distinguishing and classifying human populations along a graded scale (Baker, 1998;Gould, 1996;Graves, 2001;Smedley, 1999).

A widely accepted paradigm in Western thought since the time of Aristotle was the idea of the "Great Chain of Being." It described all natural objects arranged in an upward progression of complexity with humans at the top closest to God (Gould, 1983;Lovejoy, 1936). It was thought to represent the natural order of biblical creation, where types of living organisms occupied static 'rungs' or 'links' on a chain. Human diversity encountered during this time was assumed to represent distinct 'racial' types that occupied different positions relative to God. Further entrenchment of the race concept as a signifier of the natural order of human hierarchy in both physical and

behavioral characteristics occurred in scientific publications and research throughout the 18th and 19th centuries.

This is evident in the taxonomic classification of racial types first seen in *Systema Naturae*, published by Carolus Linnaeus in several editions from 1735 to 1770 (Linnaeus, 1956). *Systema Naturae* or 'system of nature' describes Linnaeus's ideas about the natural hierarchy and classification of all living things. Linnaeus describes four subspecies of humans: *Africanus*, cunning, passive, inattentive, and governed by impulse; *Americanus*, stubborn, prone to anger, and governed by traditions; *Asiaticus*, severe, conceited, stingy, and governed by opinion; and *Europeaus*, changeable, clever, inventive, and governed by laws (Linnaeus, 1956:20-22). Physical and behavioral characteristics are linked together, and the classifications imply a natural hierarchy with Europeans ranked at the top.

The roots of biological anthropology are entwined with the concept of the "Great Chain of Being" as scientists during the 18th and 19th century began studying skeletal traits in order to place human groups on this presumed natural scale. Dutch anatomist Pieter Camper (1722-1789) was an early predecessor of biological anthropology and introduced a method to distinguish between apes and humans using the facial profile, or facial angle, of a line drawn from the forehead to upper lip and measuring the angle of that line with a line from the ear opening to the juncture of the upper lip and nose.

Camper noted that the facial angle of Europeans and Ancient Greek sculptures approached 90 degrees, and less steep facial angles corresponded with Africans, Chinese, and apes along a continuum, which was assumed to be evidence of innate ranking within the Great Chain (Camper, 1794). Camper's work stands as an early example of the study

of human skeletal morphology and its adherence to the putative idea of natural hierarchy. Camper's example was repeated throughout the following 200 years to justify differences in worth between different human groups using various physical characteristics (Brace, 2005).

The idea of the Great Chain of Being embodied a widespread and implicit assumption about a ranked system of human diversity through the 19th century (Lovejoy, 1936). An associated school of thought that was influential in the 18th and 19th century (and beyond) was polygeny, from the Greek root for "many beginnings." Polygeny postulated that races represented separate human lineages created as fixed, unchanging types (Gould, 1983). The idea first gained traction during the Renaissance, which Brace (2005:37) suggests was a result of explorers traveling long distances by boat and encountering people very different from themselves. It was difficult for these early explorers to believe that such diversity arose since the time of the biblical creation. The idea that perhaps there was more than one "original pair" of humans to account for the variations in customs and physical traits encountered during this time was suggested but was also considered blasphemy in the 16th century. By the late 1700s, however, the idea of separately created lineages of human groups was more accepted by many scientists in both Europe and America (Smedley 1999:231).

The beginning of physical anthropology as a discipline in the US is closely associated with both the popular folk notion of polygeny, and the entrenched paradigm of the Great Chain of Being. These flawed credos were held by many distinguished scientists who influenced the course and landscape of the concept of race in American physical anthropology, such as Louis Agassiz, John Bachman, and Josiah Nott (Baker,

1998:16;Graves, 2001:44). Another leading figure was Samuel Morton (1799-1851), an American scientist from Philadelphia who created over a dozen cranial measurements that are used today by osteologists (Morton, 1839). Morton recognized 5 "races" Caucasian, Mongolian, Malay, American, and Ethiopian (Morton, 1839:5-6). These categories followed the descriptions of Blumenbach, a German scientist who described 5 "varieties" of mankind in his doctoral dissertation in 1775, and is perhaps best known for inventing the word "Caucasian" to describe people of European origin (Blumenbach, 1969). Unlike Blumenbach, who proposed that the 5 "varieties" of mankind only differed from each other "by degree" and blended into each other; Morton, a polygenist, offered that races were categorically distinct and unrelated (Morton, 1839:3).

Morton searched for evidence that races were separate species based on the average cranial capacity of different races, and concluded that Europeans had the largest cranial capacity, followed by Asians, Native Americans, and Africans with the smallest cranial capacity (Morton, 1839:260). These data were used to support the interpretation that human races represented separate species, and that these species were naturally ranked in a hierarchy with Europeans representing the pinnacle. Morton was a renowned scientist in his day, and is a good example of how the idea that races represented separate biological species was propagated through the study of skeletal traits (Hume, 2008).

A striking transformation occurred from the 16th to the 19th century where the concept of race morphed from a general categorizing expression to a core conceptual framework for understanding human biological variation. Biological race was viewed as a legitimate and scientifically sanctioned avenue of studying human skeletal variation by the mid 19th century (Baker, 1998). This stands in marked contrast to the designation of

the early terms "*razza*" or "*reazza*" to mean "type" or "kind." This brings us to an important juncture in the genesis of both the concept of race and the discipline of physical anthropology. The next 100 years of scientific research would revolutionize the natural sciences and see the development of modern theories about human origins and variation. Despite this, the notion of biological race lingered in physical anthropology, and as will be discussed in later chapters, lingers still in genetics, public health, and clinical research.

Anthropology in Transition and the Tenacity of Biological Race

There was one serious intellectual flaw that was largely responsible for the prevailing ideas of polygeny, the Great Chain of Being, and their resulting influence on the concept of biological race. The continued acceptance of biblical creation and related calculation of the age of the earth as only approximately 5-6 thousand years old plagued scientists through the 1850s. Until the revolutionary work of Charles Darwin, Charles Lyell, Alfred Russell Wallace and others, most scientists were working under a flawed archetype (Eiseley, 1968;Gould, 1996). Darwin's (1859) monumental work, *On the Origin of Species*, had, and continues to have, lasting implications for all of the natural sciences. The influence of Darwin on the emerging discipline of anthropology is treated in detail in numerous texts (see: Eiseley, 1958;Mayr, 1982;Montagu, 1964;Shipman, 1994;Smedley, 1999) and can be summarized as offering scientific explanations such as natural selection and 'deep' geological time for phenomena that polygeny and the Great Chain of Being had previously described. Consequently, these folk paradigms largely

lost support. However, the notion of biological race had (and still has, as we shall discover) tenacity, particularly within the discipline of anthropology.

Many post-Darwin anthropologists were slow to reverse positions on the idea of biological race. As a result, a racialized perception of inherent human variation pervaded anthropology until the 1960s (Armelagos & Goodman, 1998;Baker, 1998). The transition in anthropology that eventually rejected biological race took time.

In the late 19th and early 20th century, Franz Boas (1858-1942) challenged the notion that races were fixed, natural groupings of people arranged hierarchically. His work as one of the founders of the modern discipline of anthropology created the basis for much of the current anthropological perspective of race that goes against the earlier typological and hierarchical modes of race (Baker, 1998;Boas, 1940;Caspari, 2003). Boas demonstrated that cranial traits, such as cranial capacity and cranial index, long cited as evidence for superior European intellect, were actually very plastic and varied from one generation to the next (Boas, 1911). His research in skeletal morphology demonstrated that racial characteristics described by Linnaeus, Camper, Morton, and others represented an arbitrary hierarchy that erroneously linked physical characteristics such as cranial capacity and morphology, with culture or behavior (Gravlee et al., 2003).

Boas grounded his racial philosophy in osteological methods and bone morphology to debunk long-held notions of fixed racial groups. Following Boas were other influential anthropologists and humanists who helped reverse the course of the discipline away from viewing race as a biologically valid grouping strategy. The work of Ashley Montagu (1905-1999) and Frank Livingstone (1928-2005) was instrumental in addressing the concept of race and its applicability to human populations. Livingstone's

famous quote "There are no races, there are only clines" is shorthand for the impossibility of successfully dividing human populations into 'races' based on groups of traits (Livingstone, 1962). Montagu questioned the validity of racial classifications and was one of the first authors to critically examine the use of race in scientific publications (Montagu, 1942a;Montagu, 1942b). Montagu was also instrumental in the drafting of the first United Nations Educational, Scientific, and Cultural Organization (UNESCO) statement on race in 1950; a strongly worded rejection of biological race declaring that "likenesses among men are far greater than their differences" (Brattain, 2007;Graves, 2001;UNESCO, 1961). The UNESCO statement was eventually revised a year later in 1951 as there was not yet complete agreement about the invalidity of the race concept (Barkan, 1992:341;Barkan, 1996;Graves, 2001:151).

Around the 1960s, however, most anthropologists and anthropology textbooks began to reject a biological notion of race (Lieberman et al., 1989;Littlefield et al., 1982). One notable exception is Carleton Coon (1904-1981), who adhered to the view that races evolved into *Homo sapiens* independently, a throwback to the polygeny of the 19th century (Jackson, 2001). His book, *The Origins of Races* (Coon, 1962), was severely criticized, as by that time, most of anthropology had moved away from the concept of biological races and separate species (Washburn, 1963).

Anthropology and the natural science disciplines have demonstrated that bones, blood, and genes do not adhere to any kind of racial classification strategy (Brown & Armelagos, 2001;Templeton, 2002). Human variation of most physical and biological attributes is continuous and distinctions between 'types' are largely arbitrary the often the result of political or other cultural boundaries (Goodman & Armelagos, 1996;Weiss,

1998). Variation of skeletal morphology and genetic traits are best described by clines, which show gradual changes in frequencies of a trait over large geographic regions (Lewontin, 1972;Livingstone, 1962). Biological traits in human populations vary independently, which means that there is no group of genes or group of skeletal characteristics that consistently occur together to define any racial group (Goodman, 2000;Keita & Kittles, 1997). Currently, the American Anthropological Association (AAA, 1998) statement on race and the American Association of Physical Anthropology statement on race (AAPA, 1996) views race as a social construction that has no basis in genes or biology.

Thus we come to an important and interesting moment in skeletal biology. The "skull doctors" both ushered in the idea of biological race with cranial morphology and measurements and also ushered it out with the work of Boas and others. Osteology and skeletal morphology have ceased to be the marker and measure of innate biological difference as it was throughout the 19th century. However, today a different kind of bone measurement is often being interpreted as evidence for biological racial variation. Technological advances in anthropological and clinical science have enabled bone mineral density (BMD) measurements, which measure the amount of bone mineral present in a given area of bone scanned, to become easily available for both clinical and research purposes. Systematic differences in BMD measurements exist in human populations, much like any other skeletal trait, however, differences in BMD measurements are commonly described in terms of 'racial' variation and interpreted as evidence for biological or inherent differences between racial groups. This practice reflects the routine use of racial and ethnic categories in medical, clinical, and public

health research, and the failure of anthropological race theory to permeate much of clinical literature. Interpretations of biological racial difference based on BMD are partially fueled by the unrestricted and sweeping manner in which racial categories are included in clinical science. The proliferation of racial categories in clinical science and the disconnection between these practices and anthropological critique is a major contributor to how bone mineral density, like the bone measurements studied by the "skull doctors" of the past, has become associated with inherent racial differences between human groups.

Chapter 2

The Problem of Race in Bone Density Research

While skeletal biology and anthropology may have demonstrated that racial groups are not biologically different, the use of racial categories has remained a common practice in scientific research. The use of racial categories is quite standard in a variety of disciplines and has been noted with increasing frequency in medical, clinical, epidemiological, and public health research (Comstock et al., 2004;Drevdahl et al., 2001;Jones et al., 1991). The use of racial categories in medical science shares its historical roots with the anatomists and physicians of the 18th and 19th centuries who also helped shaped the future of skeletal biology and physical anthropology. Notions of race in the medical literature, however, did not undergo the same transformation that in skeletal biology led to the rejection of biological racial groups. Instead, racial categories are quite commonly used to delineate populations and classify observed biological, inherited, or ancestral shared characteristics. This chapter explores the origins of the use of racial categories in medical, clinical, and epidemiological research and examines their pervasive presence in bone density and osteoporosis studies.

Racial Health Disparities in 19th Century America

Physicians, anatomists, and the "Skull Doctors" of the 18th and 19th century were the leading scientific authorities on the debate concerning the origins, humanity, and

equality of black African slaves brought to the American Colonies and United States. As discussed in Chapter 1, the "Skull Doctors" were interested in skeletal differences between racial groups to examine ways in which whites and blacks were biologically distinct, or perhaps even different species. In the same way, many physicians had noticed differences in health and susceptibility to disease between whites and blacks, and began to examine these differences for similar purposes (Duster, 2006;Krieger, 1987).

The prevailing ideology in the early decades of the 1800s concerning the poor health of black slaves compared to whites attributed these differences to innate black inferiority. Many physicians during this time argued that racial health disparities were evidence of fixed biological differences, and not the unequal social landscape of slavery. The illnesses and medical needs of black slaves were believed to be substantially different from that of whites and this was perceived as evidence that blacks were separate biological species or entities (Hodl, 2002;Savitt, 1978:10). A number of Southern U.S. physicians wrote articles outlining the diseases to which blacks were especially susceptible, and suggested alternatives from the typical 'white' remedies to treat black slaves (as cited in: (Krieger, 1987;Savitt, 1978:8). Samuel Cartwright (1793-1863) was a prominent pro-slavery advocate who suggested runaway slaves suffered from "drapetomania," explicitly arguing that the natural condition of blacks was subservience since running away was a 'sickness' (Cartwright, 1851).

The presumption that innate inequality underlies racial health disparities remained entrenched in clinical studies until the 1860s when several social, political, and scientific upheavals dramatically altered medical inquiry towards more direct examination of social and environmental conditions for disease. The aftermath of the Civil War, Darwin's *On*

the Origins of Species, and the first black doctors in the U.S. all contributed to this paradigm shift (Foner, 1983;Savitt, 2007). Dr. James McCune Smith (1813-1885), who received his medical degree from the University of Glasgow in Scotland was the first university educated black doctor in the U.S. (Foner, 1983:268). He argued that characteristics perceived as innate may actually be the consequence of social inequality and the living conditions of poverty (Smith, 1968). Smith speculated that "bandy legs," or rickets was a disease exacerbated by a poor diet, and not evidence that black slaves were sub-human, since many of the poor classes in Ireland suffered the same condition: "And if this peculiar bend should be sufficient to rule the blacks out from the circle of man-dom, it would, if rigidly applied, rule out many who have a white complexion" (Smith, 1968:230).

Smith's work helped usher in a shift towards examining environment, diet, and other behavioral and external contributions to disease in the late 19th and early 20th century (Krieger, 1987;Savitt, 2007). The blatant racism and unfounded hypotheses of Samuel Cartwright and those like him were marginalized from major medical and public health journals with the changing political and scientific climate of the 20th century (Krieger, 1987). However, presumptions of inherent biological differences between races cling to how racial concepts are used in clinical, medical, and epidemiological research. This is the exact opposite of what happened within the discipline of anthropology in the mid 20th century where the concept of race was re-defined as a social-cultural construct with no biological legitimacy (see Chapter 1).

Medical science never underwent the same kind of focused reflection on the concept of race that occurred in anthropology, and therefore biological ideas of race

currently linger in the definition and interpretation of racial categories. For instance, current definitions of "race" in the medical sciences include some aspect of ancestry or heredity. Taber's Medical Dictionary (Thomas, 1997) defines race as: "a distinct ethnic group characterized by traits that are transmitted through the offspring." Merriam-Webster's online medical dictionary entry for race includes: "a category of humankind that shares certain distinctive physical traits" and "breed" (Medline Plus, 2003). The current use of racial categories in much of medical, clinical, and public health literature reflects the idea that race is at least partially biological and contributes to the assumption of biological differences between racial groups.

While the use of racial categories remains a lasting legacy in medical science, it has recently become the subject of intense scrutiny and debate. Critical reflection about the use of racial categories in medical and clinical research has been developed by several authors (Aspinall, 1998;Bhopal & Donaldson, 1998;Cooper, 1984;Dressler et al., 2005;Hahn & Stroup, 1994;Kaufman & Cooper, 1995;Kaufman & Cooper, 2001;LaVeist, 1994;Stolley, 1999). The current discussion over the use of these categories often makes the claim that they are crude proxies for a number of undefined biological, environmental, cultural, and economic characteristics that contribute to health and disease incidence.

In addition, reviews have demonstrated that racial categories are rampant in medical literature and question their efficacy when genetic and DNA technology can quantify biological relationships much more effectively (Comstock et al., 2004;Drevdahl et al., 2001;Jones et al., 1991;Ma et al., 2007). To date, some similar arguments and critique about the use of race has entered the bone density and osteoporosis literature, but

the debate has not yet developed a sustained dialog about these issues (see for example: Leslie & Lentle, 2006;Nelson & Barondess, 1997;Villa, 1994). Despite the controversy in medical, clinical, and epidemiological literature over their appropriateness in scientific contexts, racial and ethnic categories are ubiquitous features in bone density and osteoporosis literature.

This practice is partially fueled by bone density's clinical importance to osteoporosis and technological advancements that made measurements widely available. Concurrent with bone density's increasing relevance to osteoporosis, federal guidelines for reporting race were adopted by the National Institutes of Health in the 1990s, further fostering the association between racial categories and bone density variation. The next section introduces bone density and its importance to osteoporosis and examines how racial categories are commonly used in the bone density and osteoporosis literature.

Bone, Bone Density, and Osteoporosis

In order to examine how bone density research routinely uses racial and ethnic categories, it is necessary to first discuss what bone density is, its relationship to skeletal health, and its importance to clinicians. Bone is a composite material that has evolved to reflect its many functions: support and protection of soft tissues; mechanical leverage for movement; production of red blood cells (haematopoiesis); and maintenance of calcium homeostasis (Lee & Einhorn, 2001). Bone is composed of approximately 70% mineral and inorganic matter, 5% water, and 25% organic matter, which is mostly collagen. This combination enables bone to be both flexible and strong.

There are two major types of bony tissue that contribute to its strength. Cortical

bone makes up the hard, solid, outer surfaces of nearly all bones and is extremely rigid and strong. It is able to withstand high compressive loads, but only in the direction of typical loading. For instance, the femur is able to withstand high compressive forces from running and jumping without fracturing, but those same loads applied from a transverse direction would likely result in fracture (Cullinane & Einhorn, 2002). Trabecular bone is porous and found primarily inside the long bones and vertebrae and it helps facilitate the production of red marrow and red blood cells. It is made up of thousands of bony struts arranged to withstand additional compressive loading (Martin et al., 1998).

Bone strength is a key and necessary attribute for healthy bones to withstand the demands of support, movement, and protection without fracturing. Osteoporosis is a skeletal disorder defined by compromised bone strength, predisposing individuals to an increased risk of fracture (NIH, 2001c). There are two main contributors to bone strength, bone quality and bone quantity (NIH, 2001c). Bone quality refers to the micro-architecture of trabecular bone such as the thickness of the bony struts and their orientation (Martin et al., 1998). The mechanical loading properties of long bones are partially dependent on trabecular bony architecture and several studies have noted its importance to determining fracture risk (Crabtree et al., 2000;Nelson et al., 2000;Nelson et al., 2004;Nelson & Villa, 2003).

Bone quantity, or bone mineral density (BMD) is a measure of the amount of bone mineral (hydroxyapatite) in a given area, or volume, of bone. A bone density measurement at any given skeletal site is bone mineral content (or bone mass) divided by the area scanned. Measures of BMD average bone mineral amounts in scanned areas,

which are most commonly the hip, wrist, or lumbar spine. BMD accounts for about 70% of bone strength, and is the most widely used measurement to assess fracture risk worldwide. Progress in bone density measurement technology in the last 50 years has been matched with equal advancement in the diagnosis, prevention, and treatment of osteoporosis (Marcus, 2002;Rodan et al., 2002). In fact, the ease and availability of BMD measurements has fueled medical research in nearly all aspects of osteoporosis.

This brings us to an important point with regards to bone density as an object of study. Currently, it is the key variable for a great deal of health related research on osteoporosis and of interest to physicians primarily due to its usefulness in predicting fracture risk (Kanis et al., 1994;NIH, 2001c;WHO, 2003). Its value as a clinical variable has influenced the way that bone density variation has been characterized, and has shaped what is viewed as important factors for osteoporosis risk. These characterizations and attributes of BMD have largely occurred within the clinical and medical orienting frame of race. Documenting racial group differences, examining external factors that may cause racial differences, and investigating racial effects on BMD represent a considerable portion of BMD research. This particular focus is evident as bone densitometry technology became widely available, facilitating its importance to the diagnosis of osteoporosis. In addition, federal requirements for the inclusion of women and minorities in funded biomedical studies were implemented in 1993 (NIH, 2001b). The combination of these events has encouraged the routine use of racial and ethnic categories in bone density and osteoporosis research.

Bone Densitometry and Clinical BMD Studies

Precise bone densitometry technology that was widely available did not become a reality until the decade of the 1960s. Prior to this, studies of osteoporosis were limited to the use of x-rays which lacked precision for determining bone loss over time (Albright et al., 1941;Kesson et al., 1947). Technology designed to specifically measure bone density was first developed in the late 1940s at Texas Women's University by Pauline Beery Mack (Mack et al., 1949). Radiographic photodensitometry used x-ray images and BMD was quantified using a scanning photodensitometer, producing more accurate measures of BMD than relying on human assessment. This technology, however, was not readily available to most researchers.

Without accessible, precise, methodologies to determine BMD on living subjects, its clinical utility and usefulness to osteoporosis research was rather limited. Instead, studies employing bone ash weights, and dry bone volume/weight ratios from skeletal collections were common avenues for BMD research during this time (Broman et al., 1958;Merz et al., 1956;Trotter et al., 1959). Before BMD became important in clinical contexts, studies were examining BMD variation within racial contexts using skeletal material. This research on bone density highlighted differences between 'whites' and 'negroes', concluding that 'negroes' had denser bones than 'whites' (Baker & Angel, 1965;Trotter et al., 1960). These studies represent some of the first research specifically examining racial variation of this skeletal trait, and are still cited frequently as evidence of racial differences in BMD.

The invention of single-photon absorptiometry (SPA) machines in the 1960s ushered in the modern era of precise and convenient densitometry technology. SPA

machines use a single beam of radioactive isotope to measure tissue resistance (Cameron & Sorenson, 1963). Denser tissues attenuate, or absorb, photon energy and differences in the attenuation of the photon beam are quantified to assess bone density. SPA machines were followed by dual-photon absorptiometry (DPA) in the 1980s and were used throughout the early 1990s (Dunn et al., 1980). Dual energy x-ray absorptiometry (DXA) machines, invented in the late 1980s, use two x-ray beams instead of photons. X-rays do not degrade like the photon source of SPA and DPA machines, making DXA machines more stable and accurate over time. DXA technology provides precise BMD measurements and faster scans for several commonly measured skeletal sites such as the wrist, lumbar spine, and hip. DXA machines are currently the diagnostic 'gold standard' for osteoporosis (Kanis et al., 1994;NIH, 2001c;WHO, 2003).

All three of these machines, SPA, DPA, and DXA produce a measure of areal bone density as grams of mineral per area scanned, or g/cm^2 which describes the amount of bone mineral normalized for the total area in the scanned field. The widespread availability of DPA and DXA machines especially has corresponded to growth in bone density and osteoporosis research since the 1960s. This is evident in the results of a Medline search for keywords 'bone density' and 'osteoporosis' which shows a staggering increase in publications in the 1980s through the 1990s and is a general indicator of the expansion of bone density measurements as it relates to osteoporosis (see Table 1). Table 1 depicts the results of three related Medline searches: one for "bone density" alone; one for "osteoporosis" alone; and one combining the terms with the "and" operator.

Table 1. Medline search for keywords 'Bone Density;' 'Osteoporosis;' and 'Bone Density'/'Osteoporosis' combined. (Search performed January 6, 2009)

Years	'Bone Density'	'Osteoporosis'	'Bone Density' AND 'Osteoporosis'
	Number of articles returned in search		
1960-1970	22	523	9
1971-1980	105	1,384	25
1981-1990	907	3,151	405
1991-2000	9,182	9,374	4,281
2001-2008	13,998	15,190	7,162

Each search term dramatically increases in both the 1980s and 1990s, a time period that corresponds to the availability of DPA and DXA densitometry methods. Simultaneous with this striking expansion were the implementation of federal requirements for reporting race and ethnicity in funded biomedical research (NIH, 2001a;NIH, 2001b). The National Institutes of Health (NIH) Revitalization Act of 1993 renewed federal funding for the NIH, and also included legislation requiring the inclusion of women and minorities in funded biomedical research (Epstein, 2004;Hunt & Megyesi, 2008a). In implementing these requirements for the reporting of race and ethnicity, the NIH has followed the guidelines used by the Office of Management and Budget (OMB) Race and Ethnic Standards for Federal Statistics and Administrative Reporting, which are the same categories that are used in the US census (OMB, 2008). The categories that are required for adherence to federal guidelines are comprised of these 5: American Indian or Alaska Native, Asian, Black or African American, Native Hawaiian or Other Pacific Islander, and White (Census Bureau, 2008b).

These contemporaneous events produced abundant data reporting BMD variation using racial categories and documenting the observation that BMD is higher in Black

Americans than White Americans. Many large-scale public health research projects investigating BMD report differences in BMD according to racial categories in compliance with federal guidelines. These studies include: The National Health and Nutrition Examination Survey (NHANES)(Looker et al., 1995;Looker et al., 1997;Looker et al., 1998); Study for Osteoporotic Fractures (SOF)(Cauley et al., 2005b;Cauley et al., 2005c); Study of Women's Health Across the Nation (SWAN)(Finkelstein et al., 2002;Greendale et al., 2006;Sowers et al., 2003); and the Health Aging and Body Composition Study (Health ABC)(Schwartz et al., 2005;Taaffe et al., 2003a).

Quite commonly, there is a focus on comparisons between black and white subjects to determine what factors may be causing black racial groups to have higher BMD than white groups (Barondess et al., 1997;Bell et al., 1995;Cohn et al., 1977;Daniels et al., 1997;Nelson et al., 1991;Nelson et al., 2000). Often studies will routinely examine the effect of 'race' on BMD (Aloia et al., 1999;Cauley et al., 1994;DeSimone et al., 1989;Ettinger et al., 1997;Liel et al., 1988;Luckey et al., 1989). Racial differences in BMD continue to be a focus for many current studies. There is a large body of recent research specifically setting out to identify racial differences in BMD and osteoporosis (see for example: Dvornyk et al., 2003;Evans et al., 2005;Jen et al., 2005;Sun et al., 2003;Wallace et al., 2005;Wampler et al., 2005;Wang et al., 2005).

Bone density research routinely explores racial differences in skeletal health and has observed numerous examples of BMD differences between white and black groups in the U.S. The use of racial categories and racial variables are conventional procedures in a great deal of bone density and osteoporosis research. This practice reflects an historical

precedent with regard to the use of race in medical contexts and mirrors most other clinical, medical, and epidemiological literature in this regard. Only recently has the inclusion of racial variables and categories been subjected to critical examination and review by a number of authors (Braun et al., 2007;Winker, 2006). Central to the debate is the appropriateness of racial categories to scientific research and their use as an imperfect proxy for a myriad of biological, cultural, and social attributes.

Despite this controversy, the use of these categories in bone density and osteoporosis research remains largely unexamined. The customary association between racial groups and BMD variation is widely practiced in this literature, but has rarely been the subject of a critical investigation. Since the use of these categories is the subject of much controversy in other areas of clinical medicine, it seems appropriate to examine their use in light of this debate. How are conclusions about racial differences in BMD reached? The next chapter examines the use of these categories in more detail and explores how the conceptualization, application, and interpretation of racial and ethnic variables may contribute to flawed conclusions about BMD variation.

Chapter 3

The Use of Racial and Ethnic Categories in Bone Density and Osteoporosis Research: A Literature Review

For decades, anthropologists and other scientists have been arguing that race and/or ethnicity is a social construct and not a biological category. Some of this debate has focused on the words 'race' and 'ethnicity,' where 'race' is often defined as biological ancestry or lineage, and 'ethnicity' as an individuals socio-cultural group affiliation (Bhopal & Donaldson, 1998;Caspari, 2003;Wolf, 1994). Race and ethnicity, however, are often used interchangeably and since both concepts are pertinent to the points made in this dissertation, they will hereafter be referred to as one construct "race/ethnicity."

In the midst of the controversy over racial/ethnic concepts, efforts to document and understand how racial/ethnic groups differ in disease susceptibility and treatment response proceed unabated in much clinical, public health, and genetics research. Recently, contention over the use of racial/ethnic categories in clinical, epidemiological, medical, and public health research has intensified as several reviews have noted the increasing use of these categories in scientific contexts (Comstock et al., 2004;Drevdahl et al., 2001;Jones et al., 1991;Ma et al., 2007). The controversy over the inclusion of these categories as a research variable is central in the ongoing discussion about the

meaning of race/ethnicity that spans several disciplines (Armelagos & Goodman, 1998;Braun, 2006;Collins, 2004;Gravlee & Sweet, 2008;Kittles & Weiss, 2003;Rotimi, 2004).

Racial/ethnic categories are routinely used to define comparison groups in studies of an impressive array of diseases. For example, disease incidence of common chronic illnesses such as diabetes, cardiovascular disease, and osteoporosis is quite commonly studied in this way. Conventionally, beginning with the assumption that racial/ethnic group members share some important characteristics relevant to these diseases, researchers set out to document and examine these differences. The central variable in such studies, racial/ethnic identity, is most often taken to be self-evident, obvious, and easily identified. Racial/ethnic identities, however, to the contrary are quite complex, and are highly interrelated with a wide variety of other attributes that can impact health.

There is a long history of dialog about these issues in medical, clinical, and epidemiological literature (see: Cooper, 1984;Hahn & Stroup, 1994;Kaufman & Cooper, 1995;LaVeist, 1994;Stolley, 1999), yet these categories continue to be used in a great deal of medical and health research as if they are not problematic. Critical discussion of conclusions about racial differences and causes of racial variation has as yet been largely neglected in bone density and osteoporosis research (however see: (Fausto-Sterling, 2008) for a review regarding some of these issues in bone density research). Several authors have raised questions regarding the usefulness of racial/ethnic categories for determining fracture risk based on bone densitometry measures (see: Chen et al., 1998;Leslie & Lentle, 2006;Nelson et al., 2008;Nelson & Barondess, 1997), but an in-depth analysis of the procedures and practices related to how conclusions about racial

differences in BMD are reached has not been undertaken for bone density and osteoporosis research. This chapter presents a review of these ubiquitous concepts and practices in current research on osteoporosis and bone density. It is argued that focusing on racial/ethnic variables most often obscures rather than clarifies the underlying causes of variation in skeletal health.

The Complexity of Race/Ethnicity and Biological Reductionism

Social scientists widely agree that race and ethnicity are indeed real. They are social categories that have important effects on the lives of real people. However, they are not inherent, intrinsic, or biological categories, but instead are socially generated designations rooted in shifting political, economic, and social contexts, which can and do change across time (Keita et al., 2004;Lewontin, 1972;McCann-Mortimer et al., 2004;Stocking, 1994). Racial/ethnic identities are contextual and are intimately intertwined with other variables important to health and disease such as income, access to health care, class, and discrimination. Because it is so highly interrelated with a host of important variables, race/ethnicity has very little explanatory power when treated as a singular variable, rather than a complex, multi-faceted variable (Krieger, 2005;Shields et al., 2005). When disease risk or disease incidence is observed to vary by race/ethnicity, there are surely several related factors that contribute to at least some of the disparity (Dressler et al., 2005;Goodman, 2000). To presume that racial/ethnic variation in disease risk reflects biological differences between groups would seem an ambitious presumption indeed, in the absence of examination of at least some of these related factors.

However, the complexity of race/ethnicity is often neglected in health research and instead it is treated as a discrete characteristic, much like any other easily measured variable such as age, sex, or weight. Reviews of the use of racial/ethnic categories in medical and epidemiological research have noted that these categories frequently serve as convenient indicators of biological relatedness (Comstock et al., 2004;Ma et al., 2007;Sankar et al., 2007;Shanawani et al., 2006). Researchers regularly treat racial/ethnic classifications as loose indicators of continental ancestry, and thus of important biological differences presumed to exist between continental populations. Several authors have argued that one consequence of routinely presuming biological differences between racial groups is that socioeconomic, environmental, and class contributions to racial health disparities are vastly underestimated (Braun, 2004;Dressler et al., 2005;Goodman, 2000).

Racial/Ethnic Variables in Bone Density and Osteoporosis

Consistent with a common trend in health and medical research across disciplines, the use of racial/ethnic categories in studies of bone density and osteoporosis has increased in the last 35 years. This is strikingly depicted in Table 2, which shows the number of articles indexed on Medline for "bone density" or "osteoporosis" in the first column, and of those, articles that include indexes for "race" or "ethnicity" in the second column. This table illustrates two phenomena: the marked escalation of bone density and osteoporosis research over time, and the related growth in the number of those articles that include race/ethnicity.

Table 2. Results of Medline search for keywords "bone density" or "osteoporosis" and keywords "race" or "ethnicity," 1960-2008

Years	"Bone Density" *OR* "Osteoporosis"	AND "Race" *OR* "Ethnicity"
1960-1970	564	16
1971-1980	1,530	47
1981-1990	4,027	83
1991-2000	17,332	477
2001-2008	26,106	892

The importance of race/ethnicity to bone density and osteoporosis is apparent in the screening and diagnostic interpretation of bone density scans. As is true for current diagnostic practices for a variety of conditions, the reference populations for the "norm" against which osteoporosis test results are interpreted are racial/ethnic group specific and therefore, result in what might be called a "sliding scale" for diagnosing the condition based on racial/ethnic identity (Chen et al., 1998;Leslie et al., 2005;Leslie & Lentle, 2006;Levasseur et al., 2003). The diagnostic criteria for osteoporosis are based on T-scores and Z-scores of femoral neck bone density, where osteoporosis is defined by a T-score of –2.5 or less (WHO, 2003). These normative values are calculated separately for different racial/ethnic groups, so that in practice, the "cut-off" point for diagnosing osteoporosis will vary depending on which group is chosen (Davey, 2001;Leslie et al., 2006a).

Racial/ethnic categories are therefore both ubiquitous in the BMD literature and figure prominently the diagnosis and screening of osteoporosis. Unlike other areas in epidemiological and clinical science, the use of racial/ethnic variables has not been subject to careful scrutiny within BMD and osteoporosis literature. In the previous chapter, I explained how these categories are commonly treated as shortcuts for

biological or genetic attributes in a great deal of medical and epidemiological literature. Since these categories have assumed such importance in bone density and osteoporosis research, it is important to ask: how are the conclusions about racial/ethnic differences that form the basis of their conspicuous insertion in these practices reached? As yet, the implications of this focus on racial/ethnic categories in the diagnosis and study of osteoporosis and BMD have remained largely unexamined.

This chapter presents a review of the recent literature linking race/ethnicity to variation in BMD and osteoporosis, and will argue that conclusions based on racial/ethnic differences in BMD are largely the result of 3 flaws: 1) Concepts of race/ethnicity are varied and unclear; 2) Assignment and application of racial/ethnic variables to the analysis are undefined and often unsuited to the complexity of these variables; and 3) Interpretations often tautologically presume biological racial/ethnic differences without independent evidence. These flaws in the use of racial/ethnic variables contribute to widely held erroneous assumptions about inherent racial/ethnic difference in BMD and obscures rather than clarify causes of BMD variation.

Systematic Literature Review

For this systematic review, I searched Medline for articles indexed for "bone density" or "osteoporosis" AND "race" or "ethnicity." The search was limited to articles published in 2002 to 2006 and restricted to publications in English on human subjects. Reviews, letters, editorials, comments, and clinical conferences were excluded. Articles were included which used racial/ethnic categories and bone density as key variables. This resulted in a sample of 55 articles, of which nearly half (26/55) are from 4

prominent osteoporosis and bone density journals (Osteoporosis International, Journal of Bone and Mineral Research, Journal of Clinical Densitometry, and Bone). The remaining articles are from a variety of epidemiological, medical, and clinically oriented journals. Table 3 lists the citations for all of the articles used for this review.

Each article was carefully reviewed to determine how racial/ethnic variables were conceptualized, how they were assigned and applied in the analysis, and how they were interpreted in the discussion in order to explore how conclusions about racial/ethnic differences in BMD were reached. Descriptive analysis of these data was performed using SPSS software, version 16.0 (SPSS, 2007).

Table 3. Citations for Articles Included in Review (N=55)

Alekel, Peterson, Werner et al. (2002)	Lauderdale and Rathouz (2003)
Alver, Meyer, Falch et al. (2005)	Lenchik, Hsu, Register, et al. (2004)
Andersen, Boeskov, and Laurberg (2005)	Leslie, Metge, Weiler et al. (2006b)
Barondess, Singh, Hendrix et al. (2002)	Li, Wagner, Holm et al. (2004)
Barrett-Connor, Siris, Wehren et al. (2005)	McCabe, Martin, McCabe et al. (2004)
Bass, Ford, Brown et al. (2006)	Melton, Marquez, Achenbach et al. (2002)
Bonilla, Shriver, Parra et al. (2004)	Morris, Jacques, Selhub (2005)
Carbone, Bush, Barrow et al., (2003a)	Morton, Barrett-Connor, Kritz-Silverstein et al. (2003)
Carbone, Tylavsky, Cauley et al. (2003b)	Nelson, Pettifor, Barondess et al. (2004)
Castro, Joseph, Shin et al. (2005)	Opotowsky and Bilezikian (2003)
Cauley, Fullman, Stone et al. (2005a)	Pescatello, Murphy, Anderson et al. (2002)
Cauley, Lui, Stone et al. (2005c)	Quandt, Spangler, Case et al. (2005)
Cauley, Lui, Ensrud et al. (2005b)	Robbins, Hirsch, and Cauley (2004)
Cobb, Kelsey, Sidney et al. (2002)	Roy, Swarbrick, King et al. (2005)
Corwin, Hartman, Maczuga, et al. (2006)	Ryder, Shorr, Bush et al. (2005)
Deng, Lei, Li et al. (2006)	Schwartz, Sellmeyer, Strotmeyer et al. (2005)
Duan, Wang, Evans et al. (2005)	Shepherd, Meta, Landau et al. (2005)
Dvornyk, Liu, Long et al. (2005)	Sowers, Finkelstein, Ettinger et al. (2003)
Evans, Ross, Heinrichs et al. (2005)	Strotmeyer, Cauley, Schwartz, et al. (2004)
Fielding, Backrach, Hudes et al. (2002)	Sun, Heshka, Heymsfield et al. (2003)
Finkelstein, Lee, Sowers et al. (2002)	Taaffe, Lang, Fuerst et al. (2003a)
George, Tracy, Meyer et al. (2003)	Taaffe, Simonsick, Visser et al. (2003b)
Goh, Low, and DasDe (2004)	Tracy, Meyer, Flores et al. (2005)
Greendale, Chu, Ferrell et al. (2006)	Wallace, Ballard, Holiday et al. (2005)
Hamson, Goh, Sheldon et al. (2003)	Wampler, Chen, Jacobsen et al. (2005)
Holm, Dan, Wilbur et al. (2002)	Wang and Dixon (2006)
Jen, Buison, Darga et al. (2005)	Wang, Duan, Beck et al. (2005)
	Yarbrough, Williams, and Allen (2004)

Conceptualization of Race/Ethnicity

All of the articles targeted in this review used terminology to describe both the *concept* of race/ethnicity, and specific *labels,* which referred to a particular group or groups participating in the study. First, I identified the specific terminology used in each article to describe the *concept* of race/ethnicity. Unique phrases used to describe racial/ethnic concepts were cataloged wherever they occurred in the article. Language used for concepts of race/ethnicity varied considerably among the articles reviewed.

Table 4 is a list of all the different terms used to describe racial/ethnic concepts for all articles reviewed. These were recorded exactly as they were published, therefore some expressions may seem redundant such as "racial/ethnic" and "ethnic/racial," which insures that all unique instances of style are preserved. Twelve exclusive designations were identified indicating a wide variety of nomenclature describing these concepts is apparent in BMD research. However, it is noteworthy that none of the articles offered a definition of these concepts or a rationale for choosing a particular signifier out what is apparently a diverse field of acceptable phrasings. The failure to include definitions of racial/ethnic concepts leaves room for a great deal of ambiguity in determining the designations for the specific group *labels*. All unique labels used to describe specific groups were recorded for each article and are summarized in Table 5.

Table 4. Diversity of terminology used to refer to the concepts of race and ethnicity in N= 55 reviewed articles

Ancestry	Ethnicity	Race
Ethnic Group	Heritage	Racial/Ethnic
Ethnic Origin	Nativity Group	Race/Ethnicity
Ethnic/Racial	Nativity/Ethnicity	Racial and Ethnic

The variety of racial/ethnic labels used to classify study participants are strikingly diverse, exhibiting a wide range of classificatory criteria. This is illustrated in Table 5, which lists each unique label and also includes the basis of division they appear to represent. These classifications are meant to be descriptive and correspond to the definition of the labels (also see: (Hunt & Megyesi, 2008b). The underlying principles of the group labels are derived from combining a wide variety of attributes, such as skin color, place of birth, country of origin, and geographic region. Presented in this format, it appears as a strikingly arbitrary and haphazard manner in which to combine study subjects for the purpose of bone density research.

An additional problem is that the same label is used across studies to refer to fundamentally different groups of people. For example, the label 'Caucasian' is used to refer to people living in Greenland, the United States, Australia, and England without explaining how people in these countries are selected to be so labeled (Andersen et al., 2005;Carbone et al., 2003a;Roy et al., 2005;Wang et al., 2005). The label 'Chinese' is used to refer to subjects from Shanghai in one study, and indicates people with Chinese ancestry living in Australia in another study (Deng et al., 2006;Duan et al., 2005). The labels are apparently meant to function as indicators that these groups share characteristics, presumably important to bone density. However, these characteristics are never explicitly stated. It is unclear from the label what attributes these groups may share that the authors believe to be important to bone density.

Table 5. Basis of classification for labels used in N= 55 articles reviewed

Basis of Classification	**Labels**
Skin Color	Black, Caucasian, White, Non-White
Skin Color + Language	Non-Hispanic Black, Non-Hispanic White, Black/Hispanic
Skin Color + Political Unit	US Black, South African Black, South African White, US White
Skin Color + Region	European-Caucasian
Skin Color + Ancestral Group	Multi-Racial
Political Unit	Chinese, Japanese, Somali, Puerto Rican, American, Norwegian, Indian, Malay, Pakistani
Political Unit + Religion	Gujarati Hindu, Pakistani Muslim
Political Unit + Ancestral Group + Place of Birth	Canadian Aboriginal, Mexican-American, Alaska Native, Native Hawaiian/Pacific Islander
Political Unit + Ancestral Group + Continent	American Indian, Native American, European-American, African-American, Asian-American
Political Unit + Place of Birth + Skin Color	US Born Black, Mexican Born Mexican-American, US Born Mexican American, US Born White
Region	Asian, South Asian, West African, European,
Two Political Units	Indian/Pakistani
Language	Hispanic, Latina
Language + Political Unit	Filipina, Gujarati, Chinese Han
Language + Region	Inuit

The next section examines how these labels are assigned in the analysis of bone density. The method of classification used to assign racial/ethnic labels is investigated along with an examination of how these variables are used in the study.

The Assignment of Racial/Ethnic Variables

The methodology used to assign racial/ethnic labels to subjects was often not provided, applied inconsistently, or ambiguous. Nearly half of the articles (24/55) did not supply any information about how race/ethnicity were ascribed to subjects. In these

cases, the reasoning behind grouping study subjects under a particular label is entirely unknown. The shared characteristics that formed the basis of classification, which are presumably relevant to bone density and osteoporosis, are not revealed in these studies. While in a handful of studies the approach for assigning labels was more explicit, uniformly these practices were unsystematic and vague.

For example, several studies described procedures for classifying some subjects, but not others. Such articles consider criteria for only the non-white subjects including location (2 articles), place of birth (4 articles), surname analysis (2 articles), and a tribal registry system (1 article). It seems telling that no article mentioned how a "white" racial/ethnic designation was reached. A few articles included subjects from the Gujarati state of India, who were classified as "Gujarati" based on a surname analysis, but there was no discussion of criteria for classifying the comparison group subjects as "white." Other articles described how place of birth was used to designate "Chinese" or "Asian" subjects, but no methodology was described for assigning groups with a "white" racial/ethnic label.

By far the most common practice (22/55) for assigning racial/ethnic labels was based on "self-identification." Although quite commonly used across disciplines, self-identification is a very abstract methodology and is highly problematic (Cho, 2006;Kaufman & Cooper, 2001;Winker, 2004). The basis for an individual choosing any particular category is unknown. Identities are by definition, idiosyncratic, based on a wide and shifting array of influences such as personal history, experiences, and family relationships. These criteria assuredly will vary dramatically from person to person, and there is therefore no way to know on what basis any particular category may have been

chosen. In addition, the articles reviewed provide very little clarification about how self-identification was determined. Did subjects pick from a list, or volunteer their own term?

The Application of Racial/Ethnic Variables

After racial/ethnic labels are assigned to study subjects, these labels are used as variables in bone density and osteoporosis research. How are these variables applied in the study? I reviewed the methods and results sections of each article to investigate the way in which racial/ethnic variables were used in the analysis. Three major unique purposes for racial/ethnic variables were identified. These included their use to compare groups, as control variables, and as covariates. This is a wide range of functions that may not be appropriate for complex variables that are not clearly defined.

Over half of the articles (32/55) made racial/ethnic comparisons between or within groups. This focus seems rooted in the assumption that meaningful attributes related to BMD are shared within a group, and that these attributes are different between groups. However, none of the articles offered any indication of what important characteristics these groups are thought to share, nor their importance for the purposes of bone density and osteoporosis research. Basing comparisons on vague or undefined variables simply recycles these categories while providing no reasons why race/ethnicity should be considered significant delineations in this research.

A few articles (8/55) treated race/ethnicity as confounding variables and adjusted or controlled for them. For these studies, the main variable of interest was something other than racial/ethnic identity. For instance, the effect of fat intake, aspirin use, or magnesium intake on BMD was being considered and race/ethnicity was a control

variable in multivariate models. Some authors have criticized adjusting for racial/ethnic variables because this practice does not elucidate the role or meaning of race/ethnicity in the analysis (Kaufman & Cooper, 2001;LaVeist, 1994). Any important factors affecting BMD or osteoporosis contained within the racial/ethnic variable would remain unknown and undiscovered. This practice does not add any information about what, if anything, racial/ethnic variables contribute to bone density and osteoporosis research.

The remaining articles (15/55) used racial/ethnic variables to determine a racial/ethnic effect on BMD. Race/ethnicity were used as covariates in ANOVA or multi-linear regression analysis. For these studies, the racial/ethnic label was entered into the statistical analysis in the same manner as more easily classified and measured environmental or behavioral variables. The value of this practice seems highly questionable. Racial/ethnic variables are complex proxies for a wide variety of potential factors that can influence BMD and osteoporosis. They have diverse meanings and are based on many attributes that can be significant to skeletal health. As such, they are not suited to analyses in which they are treated as a singular easily defined variable. When race/ethnicity is found to have a significant effect on BMD in these analyses, the substantive causal factors are unexplained.

Interpretation of Race/Ethnicity

Now I consider how racial/ethnic variables were interpreted in the discussion and conclusion section of the articles reviewed. First, I surveyed each article for results that were reported with racial/ethnic labels. Then I examined the different ways in which racial/ethnic findings were discussed and interpreted.

The articles reviewed can be split into those that included discussion of findings related to racial/ethnic results and those that did not. About one third of the articles (18/55) did not frame discussions around racial/ethnic variables. This is surprising given that all of the articles reviewed used racial/ethnic categorizations in ways central to their analyses. Eight out of these 18 were the articles that adjusted or controlled for race, where other effects on BMD were the primary research question. One might wonder why racial/ethnic categories were even included these studies, when it would seem that they serve little purpose for these articles.

However, for the remaining 10 articles that did not discuss their racial/ethnic results, racial/ethnic variables were prominent in the research design and results. The interpretation of racial/ethnic differences in these studies is never clarified and is left up to the reader. It seems failure to discuss the implications of racial/ethnic differences presumes that their meaning to the analysis is obvious and self-evident, when in fact quite the opposite is true.

The remaining articles (37/55) did address race/ethnicity in considering the implications of their results. Table 6 lists the factors proposed as reasons for the observed racial/ethnic differences they reported. The table is organized according to the type of cause: genetic/biological, physiological, environmental, and social/behavioral. Presented in this manner, the widely varying ideas about the influence of racial/ethnic differences in BMD are impressive. The potential interpretations regarding racial/ethnic differences are so varied that one wonders what are racial/ethnic labels adding to our understanding of BMD variation? Do they add anything concrete to our understanding of why BMD may vary between groups?

Table 6. Reasons for racial/ethnic variation proposed in 37 articles in which causal interpretations were discussed

Genetic / Biological	**Physiological**	**Environmental**	**Social/Behavioral**
Genetic or 'inherent' (15)*	Bone/Body size (8)	Environment (2)	Lifestyle (4)
	Weight (7)	Region (1)	Socioeconomic factors (3)
	Hormones (5)		Sociopolitical factors (1)
	Bone remodeling rates (4)		Physical activity (1)
	Calcium homeostasis (4)		Diet (1)
	Bone metabolism (3)		
	Biochemical factors (3)		
	Growth (2)		
	Body composition (2)		
	Microarchitecture (2)		

*Numbers in ( ) indicates the number of articles mentioning a particular reason, and since many articles specified numerous factors, totals will be greater than 37.

Further inspection of Table 6 reveals that genetic causes were provided as explanations for racial/ethnic differences more times than any other single factor. Attributing racial/ethnic differences to genetic or inherent effects included the following examples: "Genetic diversity may play a role in explaining the demonstrated racial differences" (Opotowsky & Bilezikian, 2003). "This suggests there may be bigger inherent differences in the races than previously thought, after controlling for other important modifiable covariates" (Wallace et al., 2005). "The importance of race/ethnicity for BMD likely reflects the important role of genes in determining BMD" (Cauley et al., 2005a).

It is noteworthy that while 15 different articles proclaim that racial/ethnic differences in BMD could be attributed to genetic causes, only 3 of these articles actually

included genetic analysis (Bonilla et al., 2004;Dvornyk et al., 2005;Greendale et al., 2006). Thus, it was common for articles to make claims about genetic racial/ethnic differences without the benefit of actual genetic data. This is a disturbing fact, both in its occurrence and the lack of criticism it draws. Could it reflect as Stanfield has argued (Stanfield, 1993), that racial/ethnic variables are not subject to rigorous scientific standards and spurious notions about innate differences between groups are allowed to drive interpretations?

Presuming genetic explanations for racial/ethnic differences in BMD can also preclude social and environmental explanations. This is also evident in Table 6, where environmental, socioeconomic, and behavioral causes are put forth much less often than genetic factors. If one accepts that since race/ethnicity is not a biological designation, the fundamental causes of BMD group differences would necessarily be rooted in social, economic, and political contexts. However, these causal factors are only rarely examined in the articles reviewed.

Discussion/Conclusions

This literature review was undertaken in order to examine how conclusions about racial/ethnic differences in BMD and osteoporosis were reached. Investigation of racial/ethnic variables reveals several flaws in their conceptualization, assignment, application, and interpretation. The relevant attributes of racial/ethnic variables to the analysis are never clearly stated, leading to dubious conclusions about inherent racial/ethnic differences in bone density and osteoporosis.

This review revealed that racial/ethnic concepts were undefined and that highly varied categories are in play resulting in ambiguous ideas of what race/ethnicity meant to the analysis. The vocabulary used to describe racial/ethnic labels for study subjects was inconsistent and based on a wide array of arbitrary and unrelated attributes. Unclear notions of race/ethnicity lead to additional problems with applying and interpreting these variables in many of the articles reviewed.

Vague racial/ethnic concepts contributed to flaws with assigning and applying race/ethnicity to the analysis. The methods and practices in which racial/ethnic labels are assigned are most often not described, not applied consistently, or lack clarity. Insufficient attention to methodological procedures in assigning racial/ethnic labels reflects a common assumption that these labels are obvious and self-evident. This imprecision leaves the door open to lend apparent support to the fallacious notion that these labels are natural biological divisions and legitimate categories for bone research.

Examination of the function that racial/ethnic variables play in analyses exposed a number of additional problems stemming from lack of clear definition. These problems included: assuming shared characteristics related to BMD and osteoporosis; failure to identify what racial/ethnic variables are contributing to the analysis; and treating complex racial/ethnic variables as singular qualities. In each of these applications, the pertinent attributes of these variables and how they correspond to BMD and osteoporosis are never clearly stated. Instead, there is an implied presumption that race/ethnicity is important to the analysis, due to presumed innate racial/ethnic differences in BMD. Together, these flaws in the conceptualization and application of racial/ethnic variables contribute to

overall weaknesses in the interpretation of racial/ethnic differences in BMD, fostering misleading interpretations of inherent racial/ethnic differences in BMD and osteoporosis.

For articles that did provide explicit interpretations of racial/ethnic differences, the scope of suggested causes was so varied that it further calls into question the value of these variables to understanding BMD variation. Racial/ethnic variables were used as if their meaning and implications to this research were obvious and clearly defined, when to the contrary, the significant features of these variables remained quite hidden. Consequently, genetic causes were often invoked for interpretations of racial/ethnic differences.

Genetic causes for racial/ethnic differences in BMD and osteoporosis were frequently proclaimed without evidence. Thus, interpretations about racial/ethnic differences in some studies did not follow from the data analysis, but instead seemed to simply reiterate a starting assumption of inherent differences between groups. A priori assumptions of genetic and biological causes of racial/ethnic differences in BMD and osteoporosis research inhibits seeking explanations in other areas such as social, cultural, political, or economic factors.

The flaws presented in this review of bone density research indicate that our current understanding of racial/ethnic differences in BMD and osteoporosis may not be very well reasoned. The weaknesses evident in the use of racial/ethnic variables suggest that reliance on these poorly defined variables obscure, rather than clarify the forces and factors that may be causing variation in BMD. A more productive undertaking would include examining social and economic causes more directly, without assuming racial/ethnic variables add significantly to the analysis. In order to introduce a research

design that incorporates this approach, it is necessary to first review what is already known about the causes and contributors to BMD variation. The next chapter reviews the factors that are currently understood to be the most influential to bone density. A research design that considers these factors and the critique developed here will be introduced.

Chapter 4

Bone Physiology and Factors Affecting Bone Mass and Bone Density in Adults

As discussed in Chapter 3, racial/ethnic biological variation is an unsound causal explanation for variation in bone density and race/ethnicity are flawed variables on which to presume shared traits influential to bone density. The review presented in Chapter 3 of the practices associated with the use of racial/ethnic categories in bone density and osteoporosis research has revealed weak arguments that rely on biological interpretations of racial/ethnic difference. These flaws often exclude consideration of social and environmental contributions to bone density variation. If we reject that racial/ethnic differences in bone mass are rooted in biological differences, what should we examine to explain systematic bone mass variation? To answer this question, we need to know what factors are most influential to bone mass.

This chapter briefly outlines the major contributors to bone density and bone mass in adults. Several of the primary demographic, behavioral, and lifestyle factors that affect bone remodeling and bone mass in adult-hood are discussed. The elements contributing to bone density in adults are varied and include the amount of bone mass acquired during growth in addition to many factors that can affect bone remodeling such as biochemical and genetic regulators, hormones, physical activity, nutrition, body composition, age, sex, and weight. It is increasingly recognized that socioeconomic conditions may affect bone

mass, albeit through indirect action on lifestyle, diet, activity, and environment (Krieger et al., 2003;Krieger et al., 2005). The effect of socioeconomic status on bone mass and bone density will also be reviewed.

Peak Bone Mass

Peak bone mass is an important consideration in the study of adult bone density. Bone increases in length, size, strength, and bone mass during growth primarily through intramembranous and endochondral ossification and bone modeling. Bone modeling and bone remodeling are two separate activities with different purposes and bone remodeling will be discussed later. Bone modeling is the process that shapes bones by adding bone in some places and removing it in others so that cortical bone thickness and overall morphology maintain their correct proportions as bones increase in length. Endochondral ossification is the main process by which the cartilaginous model of the skeleton, formed during fetal development, is replaced by bone. This process adds bone mineral to the skeleton and dramatically increases bone mass, especially during adolescence and puberty when skeletal growth rate increases (Bonjour & Rizzoli, 2001;Gilsanz et al., 1991;Li et al., 1989). Ossification of the skeleton begins in utero and continues until growth stops, sometime around age 20. Total skeletal mass peaks a few years after the end of bone growth (Heaney et al., 2000).

Peak bone mass is the point between active bone mass acquisition that marks skeletal growth, and bone loss, which occurs over time with advancing age. Peak bone mass usually occurs between the ages of 20 and 30 years. Adult bone density at any age is a function of both peak bone mass, and subsequent bone loss. In addition, peak bone

mass is used in the calculation of T-scores to diagnose osteoporosis. Young adult mean BMD is used to represent peak bone mass. T-scores are calculated by the formula:

$$\frac{\text{Patient BMD - Young adult mean BMD}}{\text{1 standard deviation of young adult mean}}$$

The young adult mean BMD and standard deviation is calculated from the NHANES III database for bone density measures on individuals ages 20-30 (Bonnick, 2004;Looker et al., 1998). The current definition of osteoporosis is a T-score at the femoral neck of –2.5 or less (Kanis et al., 1994;WHO, 2003).

Bone growth and the acquisition of peak bone mass are events that are restricted in time and both endochondral ossification and bone modeling cease in early adult-hood. Bone remodeling, in contrast, is a process by which bone is removed and replaced throughout life to repair bone and adjust to mechanical stress. Bone density in older adults is regulated and maintained by bone remodeling. Variation in adult BMD is largely dependent on the hormonal, behavioral, dietary, and physical influence on the bone remodeling system. Bone physiology related to the bone remodeling process is complex and consideration of these functions will identify several causal factors that influence bone density in adults.

Bone Physiology and Bone Remodeling

Bone is composed of 4 different cell types that originate in bone marrow. Osteoblasts, osteocytes, and bone lining cells are formed from mesenchymal progenitors and osteoclasts are derived from hematopoietic progenitors. Osteoblasts form new bone by creating the proteins that form the organic matrix of bone and regulate the mineralization of this matrix into bone (Lee & Einhorn, 2001). Deposition of bone

matrix and subsequent mineralization will surround the osteoblast, essentially trapping it in a space within the bone, the lacunae, and causing the cell to mature into an osteocyte. Osteocytes have long extensions on each cell that permit communication between neighboring osteocytes, bone-lining cells, and blood vessels present throughout the bone. Osteocytes are responsible for bone maintenance, and while the exact mechanisms are unknown, they can activate bone lining cells and osteoblasts to direct bone remodeling to accommodate mechanical strain and repair microfractures (Marks & Odgren, 2002).

Bone lining cells are former osteoblasts that are flat, inactive cells and they line the surfaces of bone. Their function is still under study, but they have receptors for hormones that initiate bone remodeling (Martin et al., 1998). Osteoclasts are large, multinuclenated cells with a distinctive ruffled border along which they resorb bone by demineralizing it with acid and dissolving the collagen with enzymes.

The purpose of remodeling is to repair and replace old bone in order to maintain structural integrity and calcium homeostasis (Martin & Rodan, 2001). Remodeling is directed through the action of osteoclasts and osteoblasts in a Basic Multicellular Unit (BMU) (Frost, 1973;Frost, 1994). A BMU consists of approximately 10 osteoclasts, several hundred osteoblasts, and is supported by blood vessels, nerves, and loose connective tissue (Martin et al., 1998). In photomicrographs, a BMU resembles a comet, with several osteoclasts clustered at the leading head and trailing osteoblasts (Martin et al., 1998).

The operation of BMU's are usually described in 3 stages: activation, resorption, and formation (Parfitt, 2008). Activation of a BMU is not entirely understood, but occurs when a chemical or mechanical signal causes osteoclasts to form and begin to remove

bone on the skeleton (Martin et al., 1998). Resorption of bone proceeds in a tunnel (in cortical bone) or a ditch (along individual trabeculae) that is approximately 200 microns wide, moving forward about 40 microns a day. The resorption stage lasts about 3 weeks in humans (Martin et al., 1998). After bone has been removed, osteoblasts form new bone to fill in the area. Bone formation is slower than resorption and takes approximately 3 months.

In cortical bone, remodeling creates a new packet of bone that is laid down in several concentric, circular layers called lamellae. This new bone is referred to as a secondary osteon, or a Haversian system, and features a blood vessel at the center to provide oxygen and nutrients to the surrounding osteocytes arranged in the lamellar bone. Haversian systems are usually not present in trabecular bone because individual trabeculae are usually too narrow to support them. Remodeling of trabecular bone replaces, removes, and reorganizes the microarchitecture of the bony struts. Remodeling rates vary during life, and within the skeleton. Approximately 5% of cortical bone each year is replaced by remodeling in adult humans (Martin et al., 1998). By contrast, trabecular bone has a much higher remodeling rate where approximately 25% is replaced each year, but this rate varies widely throughout the skeleton (Martin et al., 1998).

Bone remodeling in adults, like any other physiological process, is influenced by numerous variables. The causes of bone density variation can be thought of as the sum of the factors that regulate osteoclastic bone resorption, ostoeblastic bone formation, and the link between them. What are some attributes that might affect bone remodeling and influence bone density in adults? The next section reviews some demographic, lifestyle,

biological, and socioeconomic contributing factors to explaining how and why BMD varies in adults.

Factors Affecting Bone Density in Adults

It is widely accepted that environmental, behavioral, and lifestyle factors contribute to differences in bone remodeling, osteoporosis risk, and bone density in adults. This section reviews how age, sex, estrogen and menopausal status, weight, body size, diet, and socioeconomic conditions can affect bone density in adults. What is known about how these attributes affect BMD? Since racial/ethnic variables are unlikely to have a clear causal factor, investigations of lifestyle and behavioral characteristics may contribute to interpretations of BMD variation.

Age

Bone mass declines over time. This phenomenon is universal and age was noted as a key consideration in the etiology of osteoporosis in studies over 50 years ago (Albright et al., 1941;Kesson et al., 1947). More recent studies have shown that age is directly associated with less bone mass for males and females in the NHANES III study (Looker et al., 1997). The primary reason for the loss of bone mass with age is due to an imbalance in the remodeling cycle. The supply of osteoblasts available to create new bone reduces with age in proportion to the demand for them (Manolagas & Jilka, 1995). Replacement bone created by osteoblasts decreases with age and does not quite equal the bone removed by osteoclasts. This deficit in osteoblasts and replacement bone magnifies over time with the cumulative effect of each remodeling cycle (Marcus, 2002). One

consequence of this imbalance is that any factor that increases the remodeling rate will tend to accelerate bone loss.

Sex

Men have greater skeletal bone density than women, due in part because they tend to have a larger skeletal volume and larger skeletal size than women (Nieves, 2008). DXA bone density measurements only partially control for body size, and tend to overestimate BMD of larger bones. Quantified computer tomography (QCT) is a method of densitometry adapted from computer tomography techniques in the late 1970s. QCT produces a volumetric bone density measurement in g/cm^3 and controls for bone size in 3 dimensions in contrast to areal DXA measurements that control for bone size in 2 dimensions (Cann & Genant, 1980).

Volumetric measurements of BMD with QCT tend to find less difference between the sexes than areal BMD measurements. This indicates that sex differences in BMD may be exaggerated by DXA measurements when body size is not carefully accounted for. Riggs et al. (2004) found that young women have slightly higher vBMD in the lumbar spine and femoral neck than young men. This difference disappeared over time as females tended to lose more mass than males as they age. This finding emphasizes the importance in accounting for body size with DXA BMD measurements.

Sex differences in body size, however, only partially explain the differences in bone mass. Boys accrue bone mass during growth and development for a longer period of time than do girls, and accumulate greater peak skeletal bone mass (Orwoll & Klein, 2008). Evidence suggests that men have greater cortical bone thickness compared with women of similar body size (Nieves et al., 2005). Thicker cortices confer greater bone

strength and bone density. Men also lose bone mass at a lesser rate than women as they age (Duan & Seeman, 2002). Greater bone density in men is attributable to other core differences in physiology between men and women that relate to bone remodeling. These pertinent differences in hormones, weight, and body composition as they relate to bone density will be incorporated into the sections that follow.

Estrogen, local regulators, and menopausal status

The loss of bone mass over time increases significantly in post-menopausal women (Pacifici, 2008). Menopause marks the cessation of hormone production by the ovaries, either surgically or naturally (Marcus, 2002). Estrogen is the primary female sex hormone produced by the ovaries and it has diverse effects on bone remodeling and bone metabolism. Estrogen, in general, has a positive effect on bone mass (Pacifici, 2008). Estrogens' effect on bone mass has been well known for over 50 years and continues to be a subject of intense interest in osteoporosis research (Albright et al., 1941;Komm & Bodine, 2001).

The mechanism by which estrogens regulate bone remodeling is complex and currently being studied by a number of researchers (Komm & Bodine, 2001;Manolagas & Jilka, 1995;Rodan et al., 2002). Bone cells produce a number of molecules that act on other bone cells and their precursors in bone marrow to control cell differentiation, activation, and proliferation (Raisz, 2008). These molecules are commonly called 'local regulators' because of their control over a variety of bone cell functions and they are typically divided into two types: growth factors, and cytokines (Raisz, 2008). Both growth factors and cytokines are regulated by the effect of systemic hormones.

Estrogen blocks the production of cytokines that stimulate bone resorption and osteoclasts (Pacifici, 1996). Decreased estrogen levels associated with menopause increases the production of cytokines that produce more osteoclasts, elongate their life spans, and increase the activity of mature osteoclasts (Manolagas & Jilka, 1995;Raisz, 2008). Postmenopausal women can experience a daily calcium loss of approximately 60mg daily due to lack of estrogen and resulting changes in bone remodeling rates (Marcus, 2002).

In women, estrogen is a primary influencing factor on the bone remodeling process and the maintenance of bone mass. Estrogen is important to the growth and development of the skeleton for both sexes, but its action in remodeling and maintaining bone mass in men is less clear (Orwoll & Klein, 2008). Systemic hormones may be less important to the maintenance of bone mass in men than other factors, such as mechanical loading, and body composition (Cauley et al., 2005a).

Body weight, body composition, and body size

There is a positive correlation between body weight and BMD in both males and females (Nieves, 2008). Several studies have shown that adults with higher body weight have higher bone mass (Cifuentes et al., 2003;Edelstein & Barrett-Connor, 1993). Higher body weight in obese and overweight individuals provides a protective effect against bone loss over time when compared to normal weight individuals (Castro et al., 2005;Kirchengast et al., 2002). The reason for this association between body weight and bone mass is related to both an increase in mechanical loading on the skeleton and greater fat mass that provides a source of estrogen (Edelstein & Barrett-Connor, 1993;Reid et al., 1992a).

Sustained mechanical loading of the skeleton will increase bone strength and bone mass via remodeling of skeletal tissues. Research is ongoing to discover the mechanism by which mechanical loads are translated through bone to create a remodeling response that affects bone strength. Most evidence suggests that mechanical loads applied to the skeleton direct bone remodeling through the cellular network of osteocytes embedded in bone tissues (Cullinane & Einhorn, 2002;Lanyon, 1990). Body weight affects the mechanical loads the skeleton must support and increased weight is associated with a corresponding increase in skeletal bone mass and strength.

Measures of lean mass are associated with bone density due to the importance of mechanical loading via the action of muscles on maintaining bone strength. Studies indicate that mechanical loading and muscle strength exert the greatest influence on the male skeleton (Orwoll & Klein, 2008). Higher lean mass corresponds to greater bone density in men of all ages (Barondess et al., 1997;Orwoll & Klein, 2008). Lean mass is also important to bone density in females, but some research suggests that the strongest correlation to bone density occurs in pre-menopausal women (Li et al., 2004;Mizuma et al., 2006).

Obese and overweight individuals often have higher percentages of body fat (Kucamarski, 1992;Williamson et al., 1991). Fat mass is associated with greater bone density in postmenopausal women largely due to its function as a reserve for estrogen, which protects against bone loss (Reid et al., 1992a;Reid et al., 1992b). The androgen androstenedione is converted into estrogen primarily in fat tissue (Cauley et al., 1994;Nelson et al., 1991). Several studies demonstrate a positive association between bone density and fat mass in post-menopausal women (Chen et al., 1997;Mizuma et al.,

2006;Nelson et al., 1991). Fat mass has not been associated with bone density in men (Reid et al., 1992b).

Body size is also an important consideration in bone density research, especially since weight is strongly correlated with bone density. Comparing body size between individuals or populations is more complicated than comparing weights due to differences in body proportions. Body mass index (BMI) is a ratio calculated by the formula: weight (kg)/height (m^2) that provides a height-free measure of weight. Ideally, BMI adjusts body weight for height so that body size is somewhat standardized and comparable.

Many studies find that weight is more strongly correlated with bone density than body mass index (Edelstein & Barrett-Connor, 1993;Finkelstein et al., 2002). This is somewhat surprising because BMI should be a better measure of overall body size than weight alone. It is likely that the standard BMI index exaggerates differences in body size between tall and short individuals (Kleerekoper et al., 1994;Robbins et al., 2006). Population specific BMI indexes have been suggested by a few authors to correct the limitations of applying a standard formula for all body types (Kleerekoper et al., 1994;Robbins et al., 2006). Kleerekoper et al. (1994) found that using population specific BMI indexes eliminated body size differences between groups, so that despite differences in body proportions, body size could be compared across groups.

Physical activity

Bone functions optimally when subjected to vigorous mechanical loading through the action of muscle contractions on bone origin and insertion sites (Cullinane & Einhorn, 2002;Frost, 1994). This is evident in the dramatic loss of bone mass that occurs after

immobilization seen in spinal cord injury or wheelchair-bound patients (Pluskiewicz et al., 2006). All physiological and biochemical processes influencing the skeleton function optimally when bone is subject to loading forces during locomotion and other movements. In most cases these forces are substantially magnified by the action of muscles and the skeleton is designed to withstand temporary high impact forces several times the force of body weight (Uusi-Rasi et al., 2008).

Physical activity, therefore, has positive effects on bone mass. Bone structure, including bone mass and trabecular architecture adapts to mechanical demands so that bone strength is adequate for long-term function. New bone is laid down via remodeling on skeletal regions subject to strain that exceed the customary loading range, and bone is removed from regions that experience strains below customary loading ranges. Bone response to physical activity is site specific as evidenced by side-by-side comparisons of bone density in the arms of racket sports players where significantly higher bone density is found in the playing arm (Daly et al., 2004).

The capacity for bone remodeling to adapt to physical activity differs substantially between growing children and adults (Uusi-Rasi et al., 2008). Intense physical activity in childhood can affect bone modeling and increase the size and shape of epiphyses (Uusi-Rasi et al., 2008). In adults, physical activity has less effect on bone size, but can modify cortical bone thickness and trabecular architecture. There are many studies that demonstrate physically active adults have more bone mass and lose less bone mass over time than non-active counterparts.

Meta-analyses in pre and postmenopausal women find that exercise training programs prevented or reversed bone loss in the lumbar spine and femoral neck over non-

exercising controls (Wolff et al., 1999). Other studies have demonstrated that physical activity has a significant effect on bone density at the lumbar spine in postmenopausal women (Berard et al., 1997). There are fewer exercise intervention trials carried out with men than with women, but there is strong evidence that exercise and physical activity positively impacts bone density in men (Beck & Marcus, 1999). Several studies have demonstrated that jogging, resistance training, and general activity levels in men have a positive correlation with bone density (Cauley et al., 2005a;Uusi-Rasi et al., 2008).

Diet, nutrition, and calcium

According to Robert Heaney, after holding weight constant, the 3 most important factors affecting bone mass are physical activity, gonadal hormones, and nutrition (Heaney, 2008). Nutrient intake affects bone in two major ways: first, bone cellular processes are dependent on nutrients to operate, and second, the skeleton functions as a large nutrient reserve for two minerals, calcium and phosphorous. Nutritional effects on bone mass and bone density are significant, but they are difficult to measure. Studies of various nutritional effects on bone are incredibly numerous and complicated by the fact that nutrition does not impact bone in isolation, but influences all bodily processes (Heaney, 2000). Nutrients in food and supplements work in concert with each other and interactions can dramatically affect absorption, retention, and availability of any particular vitamin or mineral. Furthermore, bone is relatively isolated from immediate nutritional consequences. Only currently forming bone would be affected by extant nutritional circumstances, leaving the bulk of bone mass and structure unaffected. These facts make studying the effect of nutritional intake on bone challenging, but despite these

problems, several aspects concerning the link between nutrition and bone mass have been established.

Two nutrients have unequivocal importance to bone health: calcium and vitamin D. These nutrients are unique from other vitamins and minerals in several ways. Vitamin D is not actually a vitamin, but a hormone that is synthesized when skin is exposed to sunlight. It is a major regulator of calcium homeostasis, and crucial for normal bone mineralization (Feldman et al., 2008). Vitamin D enhances calcium and phosphate absorptions from the small intestine so that these minerals are available to form new bone both during growth and remodeling. The importance of vitamin D to bone mineralization is evident in cases where insufficient vitamin D causes rickets, a disease characterized by deformed weight bearing bones and other skeletal changes. Inadequate mineralization, especially during growth, causes the femora and tibias to bend and bow outward (Feldman et al., 2008).

The importance of calcium nutrition to bone mass and bone density probably cannot be overstated. It is also essential for a variety of cellular and extra-cellular processes. The human skeleton at birth contains approximately 25 grams of calcium as bone mineral and increases to 1000 to 1200 grams in the adult, all by the acquisition of calcium through food sources (Heaney, 2008). Calcium physiology is complicated by the fact that skeletal bone mass is in many ways, a secondary function of calcium in the body. Calcium is the only nutrient in which the reserves possess a secondary and important function, in this case providing bone strength and bone mass.

The skeleton operates as both a source and reservoir of calcium so that serum calcium levels remain tightly regulated, while bone mass may be added or resorbed

depending on supply and demand (Heaney, 2004). There is no biochemical assay that reflects calcium nutritional status. Insufficient calcium nutrition will inexorably lead to depletion of skeletal calcium reserves and subsequent loss of bone mass and strength. However, bone mass is only an indirect measure of calcium nutrition at best. Bone mass develops over time and is dependent on a number of other variables, regardless of calcium intake.

Higher calcium intake is clearly associated with higher bone mass (Heaney, 2008). Nearly all studies published relating calcium intake to bone density show that calcium intake above recommended daily values are associated with a bone mass benefit (see reviews: Heaney, 2000;Heaney, 2008;Prentice, 2004). Several studies provide evidence that calcium supplements are positively associated with higher bone mass in men and women (Aloia et al., 1994;Dawson-Hughes et al., 1990;Dawson-Hughes et al., 1997;Recker et al., 1996;Reid et al., 1994).

The pertinent questions regarding calcium and bone mass for researchers is determining *how much* calcium is needed during every life stage so that calcium is not the limiting factor for bone mass acquisition or an aggravating factor of bone loss. Given the complexity of nutritional effects on bone, studies continue to examine what the optimum calcium intakes should be to insure maximum bone mass and the least bone loss at every life stage (NIH, 1994). This model threshold for calcium intake is difficult to pinpoint given the variety of factors that influence bone density. Methods for determining recommended calcium intakes are limited to what can be measured by metabolic calcium balance and bone mass changes as measured by absorptiometry (IOM,

1997;NIH, 1994). Recommended daily intakes of calcium vary by sex and age and current recommendations for adults range from 1000 to 1200 mg per day (USDA, 2008).

Socioeconomic conditions

The factors discussed thus far have direct effects on bone mass physiology. Socioeconomic status (SES) is likely to be influential to many of the external conditions that act on bone, such as physical activity and nutrition. Measures of socioeconomic status gauge income, education, and occupation to describe where an individual or family falls in relation to others. Studies examining the relationship between bone density and SES are rare, however a few recent studies have focused on this topic.

Higher education and higher incomes correspond to greater bone density measures in studies from the United States (Wang & Dixon, 2006); Britain (Pearson et al., 2004); Spain (Barquero et al., 1992); Italy (Varenna et al., 1999); Australia (Brennan et al., 2009); and China (Ho et al., 2005). In Iran, a recent study showed that absolute poverty, a measure of the minimum set of resources for survival, was associated with low bone mass and osteoporosis (Amiri et al., 2008). These studies indicate that higher socioeconomic status provides a beneficial effect to bone mass.

A few studies have found an opposite relationship between socioeconomic status and bone mass. Elliot et al. (1996), for example, found higher BMD at the femoral neck and lumbar spine in men of lower socioeconomic status in New Zealand. The authors caution that many of the men in the low SES group were manual laborers. Given the importance of physical activity and muscle mass to bone density in males, this may account for the contradictory findings of this study compared with most others.

However, these few studies fail to clarify what is likely to be a complex relationship between socioeconomic status and BMD.

It is widely accepted that health disparities between groups in the U.S. fall along socioeconomic lines, however measures of socioeconomic status are rarely investigated in bone density research. As evidenced in the previous chapter, social, economic, or environmental interpretations of racial/ethnic differences in BMD are seldom offered in studies. The focus on racial/ethnic categories in many bone density studies often ignores social and economic factors as causes of differences in bone density. Further investigation of the relationship between SES and bone density would add to current knowledge about how social conditions can affect skeletal health.

Summary and Introduction of Research

The beginning of this chapter asked: If we reject that racial/ethnic differences in bone mass are rooted in biology, what should we examine to explain systematic bone mass variation? This chapter has briefly outlined several demographic, lifestyle, behavioral, and socioeconomic effects on bone remodeling and bone mass. These factors make up the investigative thrust of this dissertation examining how demographic, lifestyle, and socioeconomic variables affect bone mass. Much is known about the importance of age, sex, body size, physical activity, and diet on bone mass. Few bone density studies consider the effect of socioeconomic conditions, despite many research claims that health status and economic conditions are closely linked (Dressler, 1993;Krieger et al., 2003;Krieger et al., 2005). Appraisal of these issues leads us to the primary research question of this dissertation: Can what is often automatically assumed to

be inherent racial/ethnic variation in bone density be attributed to social, economic, behavioral, and lifestyle variables?

This broad question is the guiding focus of this dissertation research. However, we have seen from the literature review presented in Chapter 3 that a great deal of bone density research investigates similar questions, but relies on racial/ethnic groups to form the basis of these comparisons. The use of racial/ethnic categories in bone density research has often lead to erroneous assumptions of biological racial/ethnic differences and flawed conclusions about variation in bone density. How can we study systematic variation in bone mass and bone density while avoiding default assumptions of inherent racial/ethnic differences associated with their use that contribute to the flaws described in Chapter 3?

This question has been considered in a broad sense by several epidemiological and health disparities researchers. Several authors suggest one way to overcome spurious conclusions about biological racial/ethnic differences is to limit investigations to one racial/ethnic group (Bhopal, 2006;Comstock et al., 2004;Kaufman & Cooper, 1995;LaVeist, 1994). Documenting variation (of bone mass or any trait) within one racial/ethnic group shifts racial/ethnic identity to a descriptive function, and away from being treated as a causal or attributive mechanisms of difference. This practice should reduce erroneous interpretations of biological racial/ethnic differences based on tautological presumptions of inherent group differences.

In light of such discussions within the public health, epidemiological, and medical literature, a small but growing body of literature has begun to address this issue in bone density and osteoporosis research (Fausto-Sterling, 2008;Leslie & Lentle, 2006). These

authors suggest that there is significant value to studies that analyze within group variation in BMD. Race/ethnicity is likely to be confounded with lifestyle, diet, activity, and socioeconomic status. Limiting studies of BMD variation to one racial/ethnic group can help clarify what may be causing the systematic racial/ethnic differences seen in BMD in the U.S. This tactic would limit interpretations that offer inherent racial/ethnic differences as valid causal mechanisms and instead investigate social and environmental factors that are more likely the underlying cause.

These initiatives in bone density and osteoporosis research have lead to additional recommendations to examine within-group variation in racial/ethnic groups where bone density and bone mass is less often studied (Melton et al., 2002;Melton & Marquez, 2008;Morton et al., 2003;Robbins et al., 2004). A great deal of bone density and osteoporosis research conducted in the U.S. focuses on white post-menopausal women (Barrett-Connor et al., 2005;Cauley et al., 2005b;Finkelstein et al., 2002). Studies focusing exclusively on bone mass of non-white groups in the U.S. occur less frequently, but this trend may be reversing (see: Melton et al., 2002;Robbins et al., 2004;Unson et al., 2005;Yarbrough et al., 2004).

Therefore, we see that by limiting investigations of bone mass to one, traditionally understudied group, several pitfalls associated with racial/ethnic categories are avoided and advantages are gained. Investigation of social, economic, demographic, and behavioral characteristics important to bone mass can be examined directly without resorting to ambiguous notions of racial/ethnic difference. In addition, gaps in current knowledge about bone mass distributions across the U.S. are addressed.

Careful consideration of these issues has lead the way to the creation of this dissertation project. Heeding several of these authors' recommendations, this research explores intra-group bone mass variation in a sample of African-American participants from Detroit, Michigan. This dissertation is designed to examine how much variance in bone mass can be explained by demographic (age, sex, and body size), lifestyle (diet, physical activity and smoking), and socioeconomic status (income, education, occupation and other social features). This primary focus is considered through the following questions:

- What is the association between socioeconomic status and bone mass?
- How much variance in bone mass can be explained by different measures of body size?
- How much variance in bone mass can be explained by lifestyle and socioeconomic variables when age, sex, and body size are held constant?
- How much variance in bone mass can be attributed to individual lifestyle and socioeconomic variables when age, sex, and body size are held constant?

These questions form the analytical framework of this dissertation. The analysis will test the association between variables that are known to influence bone mass (such as age, sex, and body size) in addition to variables in which their significance to bone mass is less well understood (such as socioeconomic status). Results of this study will contribute to models about the effects of environment and social class on bone mass in addition to providing information about variation in bone mass within a traditionally understudied population. The next chapter describes the subjects and variables used for this project in more detail.

Chapter 5

Materials and Methods

This dissertation is a secondary analysis of data collected for an existing and ongoing research program at the Wayne State University Center for Urban and African-American Health (CUAAH), in Detroit, Michigan. The CUAAH project is one of 8 centers launched nationally in 2003, as part of the National Institutes of Health Centers for Population Health and Health Disparities (CPHHDs) (NIH, 2008). The primary goal of this initiative is to explore the determinants of health disparities by conducting multilevel, transdisciplinary research among a variety of minority populations employing social, behavioral, biological, and clinical theory and methods (Warnecke et al., 2008). Collaboration among the 8 CPHHD centers will help identify and explore how policies, relationships, social conditions, institutions, individual behaviors, and biological responses and pathways affect a variety of diseases such as cancer, cardiovascular disease, and diabetes. A total of 21 different research projects across the 8 centers are currently being conducted through the CPHHD (Holmes et al., 2008;Paskett et al., 2008).

There are 3 CUAAH projects at Wayne State University and their primary focus is exploring how breast cancer and cardiovascular health outcomes are influenced and mediated by social support, neighborhood characteristics, and biological pathways (Artinian et al., 2007;Paskett et al., 2008). These projects are: 1) Obesity, Nitric Oxide, Oxidative Stress and Salt Sensitivity (ONOSS), which examines the effects of obesity,

nitric oxide, oxidative stress, and salt sensitivity on cardiovascular disease risk; 2) Women's Healthy Lifestyle Study (WHLS), which explores the effects of various experiences and lifestyle attributes on cardiovascular disease risk; and 3) Exploring Changes in Experiences and Lifestyles (EXCEL), which examines the effects of individual relationships and social support factors on breast cancer outcomes (Artinian et al., 2007;Paskett et al., 2008).

The CUAAH dataset for all three of these studies included 828 participants. The sex ratio of the entire CUAAH dataset is 76% female and 24% males. Baseline measures included demographic, lifestyle, socioeconomic attributes, and DXA bone density measurements. CUAAH clinical staff collected baseline measures used in this analysis this through questionnaires and participant clinical visits in an identical fashion for all three studies. Only 438 (53%) of the CUAAH participants have baseline DXA bone density data available. Bone mass measurements were not a primary objective for any of the CUAAH projects; therefore many participants opted out of this procedure. There is no evidence of systematic bias in which participants opted out, and which decided to participate, however, reasons were not recorded. My analysis will only focus on the 438 participants with DXA bone density measurements. The Michigan State University Office of Regulatory Affairs (IRB # X07-182) and the Wayne State University Human Investigation Committee (HIC # 072007B3X) approved the use of CUAAH data for this dissertation.

Participants

Each CUAAH project was a randomized controlled trial that included only African-American subjects. All CUAAH participants were identified as African-American based on self-declaration from the following categories: Black or African-American, American Indian or Alaska Native, Asian, Native Hawaiian or Pacific Islander, White, and "Other." ONOSS included males and females over age 35, WHLS included females aged at least 18 years, and EXCEL included males and females at least 18 years of age. All participants were recruited from Detroit, MI and the surrounding metro area between 2003-2007. Primary referral sites included clinics and health care practices associated with Wayne State University and the Detroit Medical Center and include the Karmanos Cancer Institute and Harper University Hospital (Paskett et al., 2008).

Measurement of Bone Density, Bone Mass, T-scores, and Z-scores

Bone measurements used in this research were obtained using Dual-Energy X-ray Absorptiometry (DXA) using a fan-beam instrument (QDR 4500, Software version 11.2, Hologic, Inc. Bedford, MA). Measurements included Bone Mineral Density (BMD) and Bone Mineral Content (BMC). Participants included in this analysis had DXA measurements of the proximal femur and whole body.

As discussed previously in Chapter 2, DXA is the 'gold standard' method for measuring bone density in the clinical setting (Kanis et al., 1994;NIH, 2001c;WHO, 2003). DXA technology utilizes two photon beams emitted from an X-ray source that pass through the region of interest (e.g. proximal femur). Bone and soft tissue have

different absorption properties and these differences are detected and quantified to estimate the amount of bone and soft tissue in the region of interest. Absorptiometric data as measured by DXA are expressed as areal densities that are obtained by dividing BMC by the bone area scanned. The relationship between BMC and BMD can be expressed by this equation:

$$\text{BMD } (g/cm^2) = \frac{\text{BMC (g)}}{\text{Area } (cm^2)}$$

This size correction is an estimate of the ratio between BMC and bone area and it standardizes differences in bone size between individuals. The exact relationship between BMC and area for any individual is dependent on the skeletal site, body size, and scanning conditions (Prentice et al., 1994). Note that BMD is not a true densitometric measurement because it is not expressed as mass per unit volume, but as mass per area. Consequently, BMD as measured by DXA does not always adequately correct for bone and body size differences between individuals or populations. Part of the variation within a population, or between them, will be due to bone size differences between individuals. Thus, a number of different techniques have been used to adjust the BMD obtained by DXA for differences in bone and body size.

One such technique is the estimation of volumetric bone density called Bone Mineral Apparent Density (BMAD). BMAD is one method of adjusting BMD for body size that is frequently used in bone density studies (Katzman et al., 1991). BMAD can be calculated for any region of interest, and for my analysis, I used it to approximate volumetric total body BMD. I calculated total body BMAD by the formula:

$$\text{BMAD} = \frac{\text{WBBMC}}{\text{Area}^2 \div \text{Height (cm)}}$$

Whole body bone mineral content (WBBMC) is a measure of the amount of bone mineral in the total body. WBBMC is preferred instead of whole body bone density for total body measurements. Whole body skeletal geometry is difficult to model, and dividing by the area scanned makes little sense when other measures of total body size such as height or weight are better approximations. All of the DXA bone mass and bone density variables used in this analysis are summarized in Table 7.

Femoral neck (FN) and total hip (TH) are regions of the proximal femur illustrated in Figure 1. DXA software uses a coordinate system to define the regions of interest at the proximal femur. A point of minimal width, depicted by the * in Figure 1 is calculated and this point serves as a point of origin for delineation of subsequent regions. The region scanned for femoral neck BMD corresponds to the rectangular area labeled in Figure 1, and includes the soft tissue on either side of the bony femoral neck. A line drawn from the origin of the coordinate system to the base of the trochanter defines the lower edge of the trochanteric region. The intertrochanteric region extends distally from the femoral neck and trochanteric region to the inferior border of the total area scanned, and includes part of the femoral shaft. Ward's area defines a region in the femoral neck of low density formed at the intersection of 3 trabecular bundles, and is the small square area labeled in Figure 1. Total hip includes the femoral neck, Ward's area, trochanter, and intertrochanteric regions (Bonnick, 2004;Looker et al., 1995).

Table 7. DXA Bone Density and Bone Mass Measures

Bone Density Measurement	**Abbreviation**	**Description**
Whole Body Bone Mineral Content	WBBMC	Bone mass measurement for entire body
Bone Mineral Apparent Density	BMAD	Approximates volumetric bone density for whole body
Femoral Neck Bone Density	FN	Bone density in the femoral neck
Total Hip Bone Density	TH	Bone density in the total hip

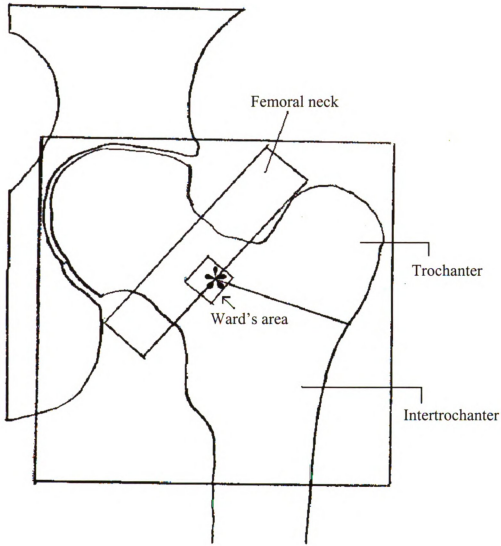

Figure 1. Schematic representation of femoral neck, trochanter, intertrochanter, and Ward's area as measured by Hologic DXA instrument. Total hip is the combination of these 4 areas. Based on Looker et al. (1995).

In addition to these 4 BMD and BMC measures, I calculated T-scores and Z-scores for the femoral neck and total hip. These standardized scores are based on Standard Deviation (SD) units so that values created from different scales can be compared. In general statistics, the average value for a set of raw data is arbitrarily assigned a Z-score of zero, and a T-score of 50. For each SD unit increase above or below the mean, the Z-score increase by a value of 1 and a T-score by a value of 10. T-scores and Z-scores tell you how many SDs above or below the mean a value lies.

T-scores and Z-scores are used extensively in bone densitometry; however, their use and interpretation are distinct from general statistics. In bone densitometry, T-scores always use a mean value that is the peak, or young adult sex-matched BMD mean. T-scores measure bone density changes from young adult BMD. By contrast, Z-scores always use an average value of mean BMD for age and sex-matched peers. These conventions in bone densitometry have been adopted by most major manufacturers of DXA instruments and enable one to immediately understand what reference average is used.

T-scores are calculated with the formula:

$$\frac{\text{Subject BMD - Young Adult Mean}}{\text{1 Standard Deviation of Population Mean}}$$

and Z-scores are:

$$\frac{\text{Subject BMD - Age Matched Mean}}{\text{1 Standard Deviation of Population Mean}}$$

T-scores are primarily used to diagnose osteoporosis in postmenopausal women and men over age 50 years (ISCD, 2007). Osteoporosis is defined by a femoral neck T-

score of –2.5 (Kanis et al., 1994;WHO, 2003). This cut-off point identifies individuals with femoral neck BMD 2.5 standard deviations below the mean young adult BMD. T-scores are less applicable for younger women and men because they are designed to measure bone loss from a young adult mean.

Z-scores are the preferred measure for premenopausal women or men younger than age 50 (ISCD, 2007). They provide a standardized score in which to measure bone loss or gain compared to age and sex-matched means. This sample includes both pre and postmenopausal women and men of various ages; therefore age-matched Z-scores are an appropriate measure of relative bone loss or gain for this analysis. Z-scores will be used as dependent variables in the statistical analysis in this study along with WBBMC, BMAD, FN, and TH.

Software installed with Hologic DXA machines includes reference bone mass data from the NHANES III study which is used to compute the young adult mean, age matched mean, and standard deviations (Looker et al., 1997;Looker et al., 1998). T-scores and Z-scores are standard clinical measures in densitometry and by convention, are calculated with racial/ethnic matched reference data. The assumptions of racial/ethnic homogeneity behind such practices are challenged in this research; however, I used conventional reference samples to calculate T-scores and Z-scores. They are appropriate as standardized measures of bone loss or gain relative to age-matched peers. Using different reference standards would not be particularly useful for the purposes of this study (also see: (Chen et al., 1998;Leslie et al., 2005;Leslie et al., 2006a;Levasseur et al., 2003).

The exact formulas I used to calculate T-scores for males and females in the femoral neck and total hip are listed in Table 8. Tables 9 and 10 list the mean and standard deviations for femoral neck and total hip BMD I used to calculate Z-scores.

Table 8. Hologic DXA African-American reference data used to calculate Femoral Neck and Total Hip T-scores. From Bonnick (2004:389-391)

	Femoral Neck	**Total Hip**
Females	$\frac{\text{subject BMD} - 0.951}{0.142}$	$\frac{\text{subject BMD} - 1.031}{0.156}$
Males	$\frac{\text{subject BMD} - 1.073}{0.156}$	$\frac{\text{subject BMD} - 1.177}{0.172}$

Table 9. Hologic DXA African-American female reference data used to calculate Femoral Neck and Total Hip Z-scores. From Bonnick (2004:389)

Subject Age Range	**BMD Mean Femoral Neck**	**Standard Deviation Femoral Neck**	**BMD Mean Total Hip**	**Standard Deviation Total Hip**
18-30	0.951	0.142	1.030	0.156
31-40	0.913	0.142	1.004	0.156
41-50	0.925	0.142	1.031	0.156
51-60	0.839	0.142	0.945	0.156
61-70	0.769	0.142	0.879	0.156
71-80	0.728	0.142	0.832	0.156
81-85	0.670	0.142	0.755	0.156

Table 10. Hologic DXA African-American male reference data used to calculate Femoral Neck and Total Hip Z-scores. From Bonnick (2004:390-391)

Subject Age range	BMD Mean Femoral Neck	Standard Deviation Femoral Neck	BMD Mean Total Hip	Standard Deviation Total Hip
18-30	1.073	0.156	1.177	0.172
31-40	1.008	0.156	1.125	0.172
41-50	0.918	0.156	1.062	0.172
51-60	0.903	0.156	1.053	0.172
61-70	0.870	0.156	1.025	0.172
71-80	0.811	0.156	0.973	0.172
81-83	0.756	0.156	0.900	0.172

A single DXA machine located in the Harper Professional Building, Detroit, MI was used for all densitometry. Standardized procedures for participant positioning and scan analysis were performed for all scans. Short- and long-term accuracy of the densitometer was verified by scanning a manufacturer's spine phantom of a known density. All DXA scans were performed by a certified densitometry technologist and analyzed by investigators trained in scan analysis.

Demographics, Lifestyle, and Socioeconomic Status

The CUAAH clinical research staff collected a great deal of demographic, behavioral, medical, psychological, dietary, social, and economic details from participants via interviews and questionnaires for all three studies. Baseline variables were collected in an identical fashion for each study and included demographics, lifestyle, and socioeconomic status. For this analysis, I selected specific independent variables from these baseline CUAAH variables. Selection for inclusion in this analysis was dependent upon a number of criteria. Variables had to be present in a majority of subjects to insure sufficient sample sizes in multiple linear regressions. In addition,

related variables could not be closely correlated with each other. This insures that each separate independent variable brings unique information to the multiple linear regression models used for the majority of the statistical tests in this project.

Table 11 lists the independent variables used in this analysis. Most of these variables were measured and collected by CUAAH staff, however, I calculated a few of these variables specifically for this analysis from other CUAAH data. The next section reviews each of these variables in detail, explaining how either I, or CUAAH staff, measured/calculated these variables.

Table 11. Independent variables used in this analysis

Demographic	**Lifestyle**	**Socioeconomic**
Age	Smoking (current/ex smoker, and never smoked)	Yearly Income
Sex	Physical Functioning	Number of People to Support on Income
Weight kg	Percentage of Average Daily Calories from Carbohydrates	Income Per Person
Height cm	Percentage of Average Daily Calories from Protein	Highest Level of Schooling
Body Mass Index (a) $\frac{\text{Weight kg}}{\text{Height m}^2}$	Percentage of Average Daily Calories from Sweets/Desserts	Neighborhood Satisfaction
Body Mass Index (b) $\frac{\text{Weight kg}}{\text{Height m}^{1.3}}$	Supplemental Calcium (average daily intake in mg)	Occupation
Hip Circumference cm	Dietary Calcium (average daily intake in mg)	Status Composite Score
	Dietary Fat (average daily intake in grams)	

Demographics

As discussed previously in Chapter 4, bone mass is significantly influenced by the effects of age, sex, and body size. For any study of BMD, it is important to collect this

information. CUAAH clinical staff collected Age, Sex, Weight, Height, and Hip Circumference data during participant clinical visits to the Harper Professional Building, Detroit, MI. CUAAH staff recorded the Age and Sex of participants at the time of the DXA bone scans. Weight was measured with a digital scale that was calibrated at regular intervals by clinical staff. Height was measured with a wall mounted Harpenden stadiometer with the head placed in the Frankfort horizontal plane. Subjects were asked to remove shoes for both measures. Maximum Hip Circumference was measured by finding the widest part of the hip between the waist and top of the thigh. This was determined by circling the area with the tape measure and moving it up and down to the find the maximum circumference.

I calculated Body Mass Index (BMI) using the weight and height measures taken by CUAAH staff. BMI is a ratio of weight over height that is an additional method of determining body size in many public health and bone mass studies. A BMI index reduces the influence of height on weight so that body size is standardized and differences in body proportions between individuals are minimized. The conventional BMI formula $\frac{\text{Weight kg}}{\text{Height m}^2}$ used by the CDC squares height in the denominator and is an approximation of the best index to reduce the effect of height on weight. The Centers for Disease Control and Prevention uses BMI as a measure of overall fatness and to define 'normal,' 'overweight,' and 'obese' individuals (CDC, 2009).

Several authors have criticized this "one-size-fits-all-approach" and suggest that this index does not minimize the effect of height for all populations (Lee et al., 1981;Robbins et al., 2006). Kleerekoper et al. (1994) created a population-specific index based on weight and height data from a body composition study of African-Americans

from Detroit. MI. Every power function for height was tested until an index that was maximally correlated with weight and minimally correlated with height for this sample was created. Kleerekoper et al's (1994)BMI is calculated by the formula: $\frac{\text{Weight kg}}{\text{Height m}^{1.3}}$. I calculated both the CDC's formula: BMI (a) and Kleerekoper et al's (1994) formula: BMI (b) in order to determine which method of BMI accounts for more variation in bone mass and bone density in this sample. This analysis will be one of the first studies to test Kleerekoper et al's (1994) BMI (b) for its effectiveness at accounting for body size in a sample from the population in which it was created.

Lifestyle

The lifestyle variables included in this analysis focus on diet, smoking, and physical activity since these variables are known to have a significant influence on bone mass (see Chapter 4). Dietary information about typical eating habits for CUAAH participants was obtained using the Block Food Frequency Questionnaire (Block & Subar, 1992). This questionnaire was administered by CUAAH staff, or in some cases, self-administered and completed at home by CUAAH participants. The Block Food Frequency Questionnaire is designed to estimate typical food intakes using a 110-item food list and questions about portion size. Computerized software from the Nutrition Coordination Center based at the University of Minnesota analyzed the CUAAH dietary questionnaires and produced a daily nutrient intake dataset. The daily nutrient intake dataset for CUAAH participants was available for my analysis.

For this analysis, I was particularly interested in calcium and fat intakes, and overall diet. Specific variables from the daily nutrient intake dataset used in this analysis are listed in Table 11. These dietary variables were selected because they are not closely

correlated with each other and represent relatively unique dietary contributions of calcium, fat, protein, carbohydrates, and sweets that can be examined in multiple linear regressions.

The CUAAH dataset included information on tobacco cigarette smoking history and current smoking habits for study participants. I used this information to create two groups: current/former smokers, and never smokers for comparisons. All CUAAH participants completed a questionnaire about overall health known as the 36 Item Short Form health survey (SF-36) (McHorney et al., 1993;Ware & Sherbourne, 1992). For this analysis, I used the Physical Functioning component of the SF-36 as a measure of general physical activity. This segment of the SF-36 is composed of 10 questions about typical daily activities such as walking, climbing stairs, and strenuous exercise with 3 standardized responses to measure how much these activities are limited by health ('a lot', 'a little', and 'not at all'). Responses are added together for a composite score that ranges between 10 and 30. Low scores designate very limited activities and high scores indicate that activities are not limited by health.

Socioeconomic status

One of the primary questions of this dissertation is to determine what variation in bone mass may be attributed to socioeconomic (SES) variables. Socio-economic variables included Income, Number of People to Support on Income, Highest Level of Education, Neighborhood Satisfaction, and Occupation. Income was recorded in the CUAAH database as ordinal data where participants were grouped according to yearly income amounts within specific ranges. Participants were also asked how many people they supported with their income. I calculated Income Per Person by dividing the rank of

the yearly income ranges with the Number of People to Support on Income. This variable is an ordinal measure of Income Per Person that provides an additional dimension of overall socioeconomic status.

Highest Level of Education was coded by CUAAH into the following categories: 8th grade, Some High School, High School Diploma/GED, Some College, Associates Degree, Bachelors Degree, Beyond Bachelors but no Masters Degree, Masters degree, Doctorate, and Other. Neighborhood Satisfaction ratings represent a mean measure of satisfaction with 9 neighborhood features: safety, grocery stores, physical appearance, recreational facilities, streets, lighting, sidewalks, parks, and restaurants. Participants were asked to rate each one on a 4-point scale (4 = very satisfied, 3= satisfied, 2= dissatisfied, and 1= very dissatisfied). This variable is part of the Social Provisions Scale designed for CUAAH projects (Holmes et al., 2008).

Past and current occupations were coded by CUAAH according to the U.S. Bureau of Labor Statistics Major Occupational Group codes (BLS, 2002). Nine major categories of occupations are described: Professional and technical; Executive, administrative, and managerial; Sales; Administrative support including clerical; Precision, production, craft, and repair; Machine operators, assemblers and inspectors; Transportation and material moving; Handlers, equipment cleaners, helpers and laborers; Service, except private household. I combined past and current occupation codes into one variable, 'Occupation,' for the purposes of this analysis. When there was a rare discrepancy between past and current job, the current job was used.

For this analysis, I wanted to create a composite measure of socioeconomic status that combined different measures of economic strain. The purpose of this composite

measure is to combine separate aspects social and economic strain into one variable denoting overall status and explore its association with bone mass. I called this composite variable the Status Composite Score, which I created by combining yearly Income, Number of People to Support on Income, and Education. I assume a low Income, high Number of People to Support, and low Educational Attainment corresponds to increased economic and social stress (low status). I assume a high Income, low Numbers of People to Support, and high Educational Attainment is associated with lower social and economic stress (high status).

I recoded each separate variable making up the Composite Status Score into a 4-point scale. Table 12 is a chart explaining how each separate element of the Composite Status Score was classified into 4 ranked numerical variables. The separate status scores were then added together to create a Composite Status Score. For example, an individual with a yearly income of $20,000, an Associates degree, and with only themselves to support on their income (one person) would receive a Composite Status Score of 9. The Composite Status Score has a minimum of 3 and maximum of 12, where 3 corresponds to 'low' SES and high stress, and 12 corresponds to 'high' SES and low stress.

Table 12. How separate elements of the Composite Status Score were recoded into a 4-point scale

Status Score	Yearly Income	Education	Number of People to Support on Income
1	0-$19,999	8th grade, some high school	5 or more people supported on income
2	$20,000-$34,999	High school diploma, some college	3 or 4 people supported on income
3	$35,000-$49,999	Associates degree, bachelors degree, or beyond bachelors	2 people supported on income
4	$50,000 and above	Masters, Doctorate, or other	1 person supported on income

Statistical Analysis

The CUAAH participants used in this research represents 3 somewhat independently recruited groups of participants for projects not related to bone mass. It is possible that there are systematic differences in bone mass between these study groups related to recruitment, health conditions, or other bias. Before the 3 CUAAH study groups were combined for this analysis, I performed an ANOVA test to determine if the study groups (ONOSS, WHLS, or EXCEL) were significantly different with respect to Age, Weight, Body Mass Index (a) and (b), and Whole Body Bone Mineral Content, Bone Mineral Apparent Density, Femoral Neck, and Total Hip listed in Table 7 (WBBMC, BMAD, FN, and TH).

Weight, BMI (a) and (b), and the BMD and BMC measures did not differ significantly between any of the study groups for males or females. Age was significantly different between study groups; both males and females are significantly younger in the ONOSS study group. However, age is controlled for in the statistical

methods used in this analysis, therefore, the 3 study groups are combined for this analysis.

Descriptive statistics and frequencies were calculated for all variables used in this analysis. The end of Chapter 4 identified several questions that form the investigative basis of this dissertation. Using the materials and CUAAH participants described here, this research tests some specific relationships between demographic, lifestyle, and socioeconomic factors and BMD/BMC. The goal of this dissertation is to examine how much variation in bone density/bone mass in this sample can be attributed to differences in demographics, lifestyle, and socioeconomic status.

The statistics of this study are designed to answer 3 main questions about variation in bone density and bone mass in this sample. The first primary research question is: What is the association between socioeconomic status and bone density? There are few studies directly testing the relationship between economic strain, socioeconomic status, and BMC/BMD. Are indicators of higher SES associated with greater BMC/BMD in this population as demonstrated in a few previous studies? Which social and economic measures are significantly associated with BMC/BMD and is this influence negative or positive? Bivariate correlation and ANOVA will test associations of separate and composite measures of SES and BMC/BMD measures. These statistical tests will clarify the relationship between SES and BMC/BMD for this population.

The second primary research question is: What is the contribution of demographic, lifestyle, and socioeconomic factors to variation in BMC/BMD and what individual factors are significant? Multiple linear regression models using demographic, lifestyle, and socioeconomic variables will test for their contribution to variation in

BMC/BMD. There is abundant evidence that the variables used in the demographic model (Age, Sex, Weight, and other body size measures) have significant effects on BMC/BMD. Will the demographic regression model account for most of the variation in BMC/BMD, or will the inclusion of lifestyle and SES attributes improve these regression models? Which variables have significant effects on BMC/BMD in these models? Multiple linear regression analyzes the separate contribution of each variable to the overall model. With this statistical test, the effect of lifestyle and socioeconomic variables to BMC/BMD can be determined when age, sex, and body size are held constant.

The third and final primary research question is: Which BMI formula accounts for more variation in bone mass and bone density in this sample? Kleerekoper et al's (1994) BMI formula has not been tested on a sample from the population in which it was derived. Body size is a crucial consideration to BMD studies and this test may provide evidence that population-specific indexes for BMI should be considered in BMD studies. In addition, systematic differences in body size between racial/ethnic groups in the U.S. may be contributing to differences seen in BMD and BMC between racial/ethnic groups. Multiple linear regression will test the amount of variation in BMC/BMD that could be explained by different formulas of Body Mass Index.

For all statistical tests, the dependent bone mass and bone density measures are: Whole Body Bone Mineral Content (WBBMC), Bone Mineral Apparent Density (BMAD), Femoral Neck BMD (FN), Total Hip BMD (TH), and Z-scores of the Femoral Neck (ZFN), and Total Hip (ZTH). All data analyses were conducted using SPSS version 16.0 (SPSS, 2007).

Chapter 6

Results

Descriptive Statistics

The sample used in this analysis is made up of 438 participants from the CUAAH database with DXA bone density measurements. Table 13 summarizes the frequencies of a variety of attributes of these participants. The majority of the participants in this analysis are female (77%), which reflects the percentage of females in the entire CUAAH dataset (see Chapter 5). The majority of subjects were between 41 and 60 years of age with a range of 18-84 years and a mean age 53.4 years. Participants have lower incomes than would be expected based on Michigan median yearly income, indicating that this sample is more representative of lower income groups (Census Bureau, 2008a).

Table 14 lists the means and standard deviations of the remaining variables used in this analysis. The mean weights of male and female participants in this analysis are similar to the mean weights reported in other studies investigating bone mass of African-American participants from large U.S. cities (Finkelstein et al., 2002;George et al., 2003). Females weighed less on average than male subjects, yet Hip Circumference and mean Body Mass Index (a) and (b) was higher in females than males.

The mean BMI (a) for this sample indicated that most participants were either obese or overweight. According to the Centers for Disease Control and Prevention, overweight individuals are defined by a BMI of 25-29.9 and obesity is defined by a BMI

of over 30 (CDC, 2009). The mean BMI (a) for females was 32.19 and for males 28.56 (Table 14). This may reflect the increasing incidence of obesity in the U.S. as a whole (CDC, 2008;Kucamarski, 1992;Williamson et al., 1991). One question that will be further investigated in this analysis is how well various measures of body size account for BMD and BMC variation

Nutritional intakes of CUAAH participants with DXA measurements are similar to those reported for African-American participants of the National Health and Nutrition Examination Survey (NHANES III) (Corwin et al., 2006). However, average daily dietary fat intake is higher for CUAAH women (79.22) than reported for African-American women in NHANES III (65.3) (see Table 14).

Table 13. Descriptive characteristics of CUAAH participants with DXA bone density measurements used in this analysis

Variable	N	%
Sex (N=438)		
Female	337	77%
Male	101	23%
Age (N=424)		
18-30	13	3%
31-40	34	8%
41-50	118	28%
51-60	158	37%
61-70	73	18%
71-80	22	5%
80+	6	1%
Education (N=405)		
1-12 years	87	21%
Some college, but no degree	140	35%
Associates or Bachelors degree	122	30%
Graduate degree	56	14%
Occupation (N=324)		
Professional and technical operations	69	21%
Executive, administrative, and managerial occupations	42	13%
Sales	28	9%
Administrative support including clerical	66	20%
Precision production, craft, and repair occupations	26	8%
Machine operators, assemblers and inspectors	18	5%
Transporting and material moving	16	5%
Handlers, equipment cleaners, helpers, and laborers	7	2%
Service occupations, except private households	52	17%
Income (N=421)		
0-$19,999	100	24%
$20,000-$34,999	67	16%
$35,000-$49,999	72	17%
$50,000 and above	182	43%

Table 13 (con't)

Variable	N	%
Number of people to support on income (N=419)		
One	106	25%
Two	166	40%
Three	70	17%
Four	45	11%
Five	17	4%
Six or more	15	3%
Smoking (N=417)		
Current or former smoker	232	56%
Never smoked	185	44%

Table 14. Descriptive statistics for body size, DXA measurements, dietary intakes, and other CUAAH variables used in this analysis

Variable	Females			Males		
	Mean	Standard Deviation	N	Mean	Standard Deviation	N
Body Size Variables						
Weight kg	86.04	15.27	337	89.87	16.21	101
BMI (a)[1]	32.19	5.41	312	28.56	4.81	98
BMI (b)[2]	45.44	7.51	312	42.61	7.18	98
Height cm	163.84	6.38	312	177.22	7.66	98
Hip Circumference cm	116.71	10.72	282	107.24	9.72	90
DXA Bone Mineral Density and Bone Mineral Content Measures						
WBBMC[3]	2281.99	347.30	337	2922.21	518.00	101
BMAD[4]	0.10	0.01	312	0.09	0.008	98
FN[5]	0.89	0.15	320	0.94	0.14	97
TH[6]	0.99	0.15	321	1.01	0.16	97
Average daily intakes of selected nutrients						
% Carbs	49.52	9.66	249	49.91	9.23	66
% Protein	14.34	3.33	249	14.12	2.65	66
% Sweets	15.07	9.33	249	15.61	8.75	66
Supplemental CA mg	295.70	418.72	249	134.09	284.68	66
Dietary CA mg	600.90	379.24	249	597.80	465.40	66
Dietary Fat g	79.22	55.71	249	87.65	76.85	66
Other CUAAH variables used in this analysis						
Physical Functioning score	24.18	5.68	326	24.66	4.89	101
Neighborhood Satisfaction score	2.74	0.59	321	2.71	0.60	100

[1] (a) Body Mass Index = weight (kg) / height m^2
[2] (b) Body Mass Index = weight (kg) / height $m^{1.3}$
[3] Whole Body Bone Mineral Content
[4] Bone Mineral Apparent Density
[5] Femoral Neck Bone Density
[6] Total Hip Bone Density

T-scores and Z-scores

Distributions for T-scores and Z-scores for the femoral neck and total hip are illustrated in Figures 2 through 5. T-scores are a standardized measure of bone loss from peak or young adult bone density and Z-scores are a standardized measure of relative bone density compared to age-matched peers. Both T-scores and Z-scores at the femoral neck and total hip are normally distributed in this sample. Osteoporosis is defined by a T-score of less than –2.5 at the femoral neck. In Figure 2, the small portion of the sample with osteoporosis are the 9 individuals that make up the area of the bar chart to the left of the –2.5 on the far left hand side of the graph. As stated previously in Chapter 5, the age-matched Z-scores will be used as dependent bone density variables along with Whole Body Bone Mineral Content (WBBMC), Bone Mineral Apparent Density (BMAD), Femoral Neck BMD (FN), and Total Hip (TH) (see Table 7). By using BMC and BMD at various skeletal sites, this analysis can compare the effect of the independent variables between skeletal sites.

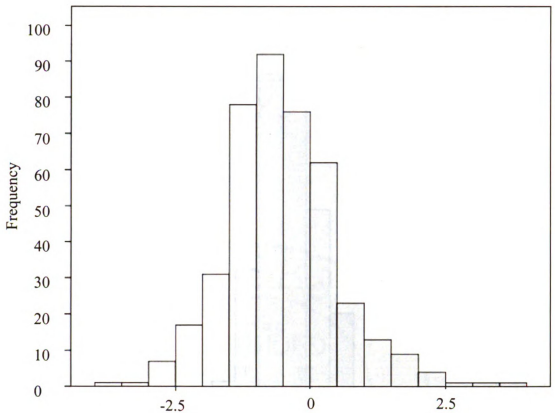

Figure 2. Distribution of T-scores for femoral neck (N= 417)

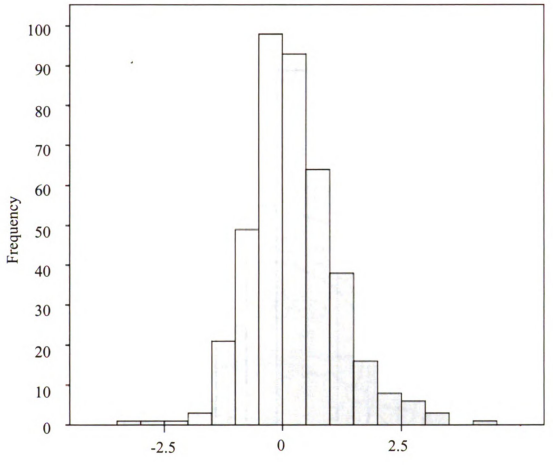

Figure 3. Distribution of Z-scores for femoral neck (N= 403)

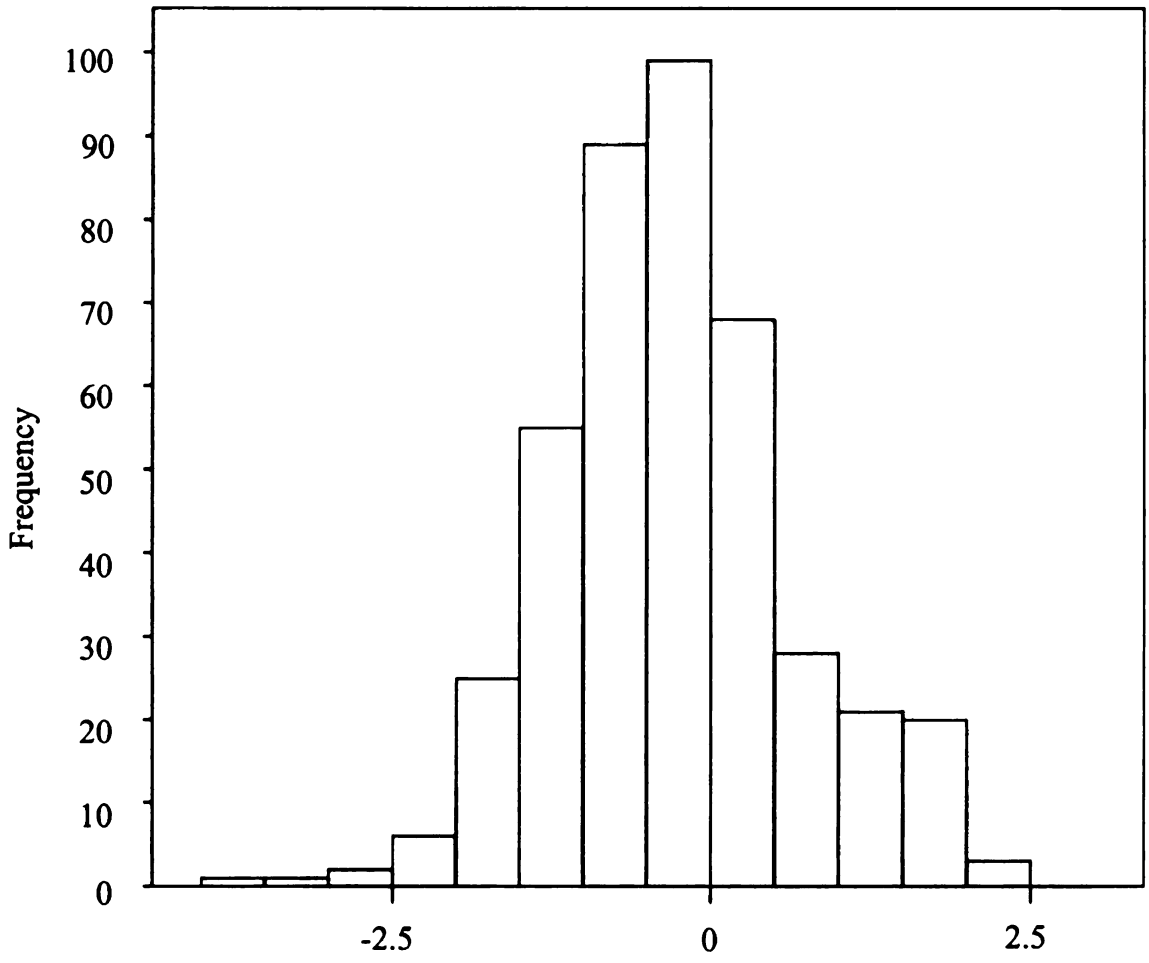

Figure 4. Distribution of T-scores for the total hip (N= 418)

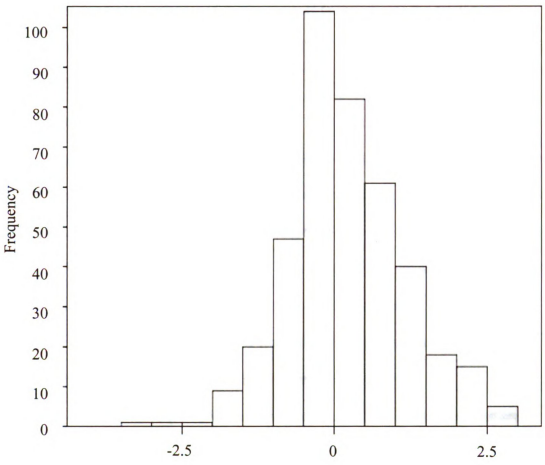

Figure 5. Distribution of Z-scores for the total hip (N= 404)

Hypothesis Testing

One of the major purposes of this analysis is to investigate the relationship between socioeconomic status and BMC/BMD. The first tests I conducted were bivariate tests of correlation between the Composite Status Score and Income Per Person, and BMC/BMD measures. Bivariate correlation tests the hypothesis that differences in Composite Status and Income Per Person would be associated with differences in bone mass and bone density. Linear non-parametric correlations (Spearman's rho) showed no significant correlation between the Composite Status Score or Income Per Person and WBBMC, BMAD, FN, TH, ZFN, and ZTH. This indicates that these composite socioeconomic measures are not strongly associated with bone mass. Examining the effect of specific socioeconomic variables via ANOVA may reveal some further associations between social and economic elements and BMC/BMC.

Analysis of variance (ANOVA) is a way to test the hypothesis that there are significant differences in the means between two or more groups. I tested the hypothesis that different Income groups, Number of People to Support on Income, and levels of Education would have significantly different mean bone mass. Income, Education, and Number of People to Support were each divided into 4 groups as explained in Chapter 5, Table 12. Tukey's test of honest significant difference identifies the groups whose means are statistically different from each other. The mean BMC/BMD for each group is listed in Tables 16, 17, and 18. Significant mean differences are flagged with an *.

Table 15. Mean BMC/BMD for Yearly Income groups

	Yearly Income			
	0-$19,999	$20,000-$34,999	$35,000-$49,999	$50,000 and above
WBBMC	2403.41	2308.18*	2527.94*	2454.21
BMAD	.09	.09	.09	.09
FN	.89	.88	.91	.90
TH	.99	.97*	1.04*	1.02
ZFN	.09	.24	.33	.24
ZTH	.08	.12	.39	.25

*p<.05
**p<.01

Table 16. Mean BMC/BMD for Number of People to Support on Income groups

	Number of People to Support on Income			
	5 or more	3 or 4	2	1
WBBMC	2583.97	2460.79	2426.93	2358.48
BMAD	.09	.09	.09	.09
FN	.97*	.91	.89*	.87*
TH	1.05	1.03	1.02	.98
ZFN	.39	.19	.27	.13
ZTH	.23	.16	.31	.12

*p<.05
**p<.01

Table 17. Mean BMC/BMD for Education groups

	Highest Level of Education			
	8th grade, some high school	High school diploma, some college	Associates degree, bachelors degree, or beyond bachelors	Masters, Doctorate, or other
WBBMC	2284.99	2424.74	2461.21	2440.48
BMAD	.09	.09	.09	.09
FN	.83*	.90	.92*	.89
TH	.95	1.02	1.03	1.00
ZFN	-.08	.21	.36	.17
ZTH	-.06	.24	.30	.17

*p<.05
**p<.01

Measures of social and economic conditions do correspond to differences in mean bone mass and bone density. WBBMC and TH means are significantly greater in the group whose yearly income is $35,000 – $49,999 than the group whose yearly income is $20,000 – $34,999 (Table 15). The difference in income between these 2 groups is not extreme, yet greater bone density is associated with the higher income group. Femoral Neck bone density is significantly greater in groups with an Associates or Bachelors degree than those with only 8th grade or some high school (Table 17). Higher Incomes and Educational attainment are associated with greater bone density, which may indicate that higher socioeconomic status and lessened economic stress are beneficial to skeletal health.

There is a general agreement within the bone density literature that groups with higher SES have greater bone mass (Barquero et al., 1992;Unson et al., 2005). Several studies demonstrate positive associations between income, education, and bone mass (Ho et al., 2005;Lauderdale & Rathouz, 2003;Varenna et al., 1999;Wang & Dixon, 2006). These authors claim that the underlying causal mechanism behind this association is related to the greater access and knowledge about food choices, nutrition, and bone health in higher educated and higher income groups. A similar situation may be the cause underlying the results of this study.

Femoral Neck bone density is significantly greater in groups that have 5 or more People to Support on Income, than in groups that only support one or two people (Table 16). In this instance, it seems that a characteristic of greater economic stress (more people to support) is associated with greater bone density. This result is somewhat

contradictory to the association between less economic stress (higher income and educational attainment) with greater BMC/BMD.

A few bone density/osteoporosis studies examine overcrowding in the household, which is similar to what is being measured here, as an additional component to socioeconomic status. Farahmand (2000) found household crowding associated with an increase risk of hip fracture, but did not examine bone density directly. Pearson (2004) found low socioeconomic status, which included a variable for number of people in the household, associated with low heel bone density. The results of my analysis, which examines this variable in isolation, suggest that the number of people to support is influencing BMC/BMD differently than other measures of socioeconomic status. The directionality of BMC/BMD changes associated with number of people to support is opposite of its relationship to economic strain. In this instance an attribute that usually identifies households and individuals as lower SES (and in many cases, *lower* bone density), is associated with *greater* bone density.

The results of the bivariate correlation and ANOVA analyses indicate that some measures of social and economic strain are correlated with BMC/BMD, but only when measured in isolation. The Composite Status Score and Income Per Person were not significantly correlated with any measure of BMC/BMD. The next section explores the multiple linear regression models.

Multiple Linear Regressions

The multiple linear regression analysis is designed to answer the following 2 related questions: 1) How much total variation in WBBMC, BMAD, FN, TH, ZFN, and

ZTH can be explained by demographic, lifestyle and socioeconomic variables? And 2) What independent variables are significantly contributing to variation in WBBMC, BMAD, FN, TH, ZFN, and ZTH in these models?

The first question is addressed by calculating the multiple coefficient of determination (R^2) value for 3 different regression models for demographic, lifestyle, and socioeconomic variables. The R^2 values indicate the amount of variation in the dependent variable accounted for by the model. Comparison across R^2 values can identify which model (demographic, lifestyle, or socioeconomic) accounts for more variation in BMC/BMD.

The second question can be answered by examining the significance of each independent variable in the model. Multiple linear regression calculates the partial slope coefficient of each independent variable when all other variables in the model are held constant. This statistical control over the other variables in the model enables multiple regression to measure the separate effect that each variable has on the dependent variable. The partial slope coefficient is a measure of how much the dependent variable changes with each one unit increase in the independent variable (one unit refers to whatever unit of measurement is used for the independent variable).

In the following tables (Tables 19 through 24) the columns for "unstandardized coefficients" are the partial slope coefficients for each variable. For example, in Table 18, a one-year increase in Age is associated with a 3.59 loss of WBBMC and a one-kilogram increase in Weight is associated with a WBBMC gain of 9.68. Sex was scored as 1= male and 2= female, therefore a one unit 'increase' in the Sex variable corresponds to the difference to BMC/BMD in females. Smoking was scored as 0= never smokers

and 1= current or former smoker, therefore a one unit increase in 'Smoking' corresponds to the difference to BMC/BMD in current/former smokers.

The variables included in the demographic model varied depending on the bone mass or bone density measurement. WBBMC, FN, and TH demographic variables included Age, Sex, Weight, Height, and Hip Circumference. Only Age and Sex were tested with BMAD, since BMAD factors body size into its measurement. Since Z-scores are age and sex matched, only Weight, Height, and Hip Circumference were included. Subsequent regression models for lifestyle and socioeconomic status for all BMC/BMD measures included significant demographic variables[7]. Inclusion of significant demographics (such as Age, Sex, and Weight) holds these variables constant so that the effect of lifestyle and socioeconomic variables on BMC/BMD can be measured in these models.

[7] There is one exception to this rule. Table 19 shows that 'sex' was not significant to BMAD in the demographic model, but 'sex' was included in subsequent models because this is a key demographic variable.

Table 18. Difference in Whole Body Bone Mineral Content (WBBMC) per one unit of change in independent variable (unstandardized coefficients) and multiple coefficient of determination (R^2) for demographic, lifestyle, and socioeconomic variables

Independent Variables	**I Demographic**	**II Demographic + Lifestyle**	**III Demographic + Socioeconomic Status**
	Unstandardized Coefficients		
Constant	-788.51	-788.88	-1126.63
Age	-3.59**	-3.97*	-2.46
Sex	-321.47**	-303.64**	-311.76**
Weight kg	9.68**	8.34**	8.95**
Height cm	19.45**	20.25**	20.51**
Hip Circumference cm	-.928		
Smoking		-41.15*	
Physical Functioning		.42	
% Carbohydrates		-1.64	
% Protein		-.23	
% Sweets		-3.44	
Dietary CA mg		.04	
Dietary Fat g		-.27	
Supplemental CA mg		-.03	
Income			-.31
Number of People to Support on Income			10.07
Neighborhood Satisfaction			-52.51*
Highest Level of Education			22.22*
Occupation			6.24
R^2 values	**.56**	**.55**	**.57**

* $p<.05$
** $p<.01$

Table 19. Difference in Bone Mineral Apparent Density (BMAD) per one unit of change in independent variable (unstandardized coefficients) and multiple coefficient of determination (R^2) for demographic, lifestyle, and socioeconomic variables

Independent Variables	**I Demographic**	**II Demographic + Lifestyle**	**III Demographic + Socioeconomic Status**
	Unstandardized Coefficients		
Constant	.10	.11	.10
Age	-1.28×10^{-4}**	-1.51×10^{-4}**	-1.15×10^{-4}**
Sex	3.99×10^{-4}	.001	3.2×10^{-4}
Weight kg			
Height cm			
Hip Circumference cm			
Smoking		-.001*	
Physical Functioning		-2.05×10^{-4}	
% Carbohydrates		-6.8×10^{-5}	
% Protein		6×10^{-6}	
% Sweets		-1.1×10^{-3}	
Dietary CA mg		2×10^{-6}	
Dietary Fat g		-1.4×10^{-5}	
Supplemental CA mg		-2×10^{-6}	
Income			-8.3×10^{-5}
Number of People to Support on Income			4.7×10^{-5}
Neighborhood Satisfaction			-1.01×10^{-4}
Highest Level of Education			2.52×10^{-4}
Occupation			1.0×10^{-5}
R^2 values	**.024**	**.048**	**.005** (model not significant)

* $p<.05$
** $p<.01$

Table 20. Difference in Femoral Neck BMD (FN) per one unit of change in independent variable (unstandardized coefficients) and multiple coefficient of determination (R^2) for demographic, lifestyle, and socioeconomic variables

Independent Variables	**I Demographic**	**II Demographic + Lifestyle**	**III Demographic + Socioeconomic Status**
	Unstandardized Coefficients		
Constant	.98	.91	.81
Age	-.004**	-.003**	-.004**
Sex	-.05*	-.03	-.042**
Weight kg	.004**	.004**	.004**
Height cm	-8.29×10^{-4}		
Hip Circumference cm	-1.79×10^{-4}		
Smoking		-.03**	
Physical Functioning		1.31×10^{-4}	
% Carbohydrates		-.002	
% Protein		-7.55×10^{-4}	
% Sweets		3.5×10^{-5}	
Dietary CA mg		3.7×10^{-5}	
Dietary Fat g		-1.57×10^{-4}	
Supplemental CA mg		-1.5×10^{-5}	
Income			.001
Number of People to Support on Income			.001
Neighborhood Satisfaction			-.007
Highest Level of Education			.005
Occupation			.001
R^2 values	**.28**	**.28**	**.28**

* $p<.05$
** $p<.01$

Table 21. Difference in Total Hip BMD (TH) per one unit of change in independent variable (unstandardized coefficients) and multiple coefficient of determination (R^2) for demographic, lifestyle, and socioeconomic variables

Independent Variables	**I Demographic**	**II Demographic + Lifestyle**	**III Demographic + Socioeconomic Status**
	Unstandardized Coefficients		
Constant	1.19	.97	.88
Age	-.002**	-.002**	-.002**
Sex	-.12**	-.08**	-.096**
Weight kg	.005**	.004**	.005**
Height cm	-.002		
Hip Circumference cm	-3.9×10^{-5}		
Smoking		-.02*	
Physical Functioning		-5.27×10^{-4}	
% Carbohydrates		-.001	
% Protein		.003	
% Sweets		2.24×10^{-4}	
Dietary CA mg		2×10^{-5}	
Dietary Fat g		-1.22×10^{-4}	
Supplemental CA mg		-4.1×10^{-5}*	
Income			.003
Number of People to Support on Income			-.002
Neighborhood Satisfaction			-.01
Highest Level of Education			.002
Occupation			2.17×10^{-4}
R^2 values	**.27**	**.27**	**.32**

* $p<.05$
** $p<.01$

Table 22. Difference in Z-score of Femoral Neck (ZFN) per one unit of change in independent variable (unstandardized coefficients) and multiple coefficient of determination (R^2) for demographic, lifestyle, and socioeconomic variables

Independent Variables	**I Demographic**	**II Demographic + Lifestyle**	**III Demographic + Socioeconomic Status**
	Unstandardized Coefficients		
Constant	-.95	-1.07	-1.93
Weight kg	.03**	.02**	.03**
Height cm	-.008		
Hip Circumference cm	.001		
Smoking		-.16*	
Physical Functioning		-.006	
% Carbohydrates		-.01	
% Protein		-.003	
% Sweets		-1.83×10^{-4}	
Dietary CA mg		3.54×10^{-4}	
Dietary Fat g		-.002	
Supplemental CA mg		-9.1×10^{-5}	
Income			.008
Number of People to Support on Income			-.021
Neighborhood Satisfaction			-.08
Highest Level of Education			.02
Occupation			.001
R^2 values	**.17**	**.15**	**.18**

* $p<.05$
** $p<.01$

Table 23. Difference in Z-score of Total Hip (ZTH) per one unit of change in independent variable (unstandardized coefficients) and multiple coefficient of determination (R^2) for demographic, lifestyle, and socioeconomic variables

Independent Variables	**I Demographic**	**II Demographic + Lifestyle**	**III Demographic + Socioeconomic Status**
	Unstandardized Coefficients		
Constant	-.49	-1.47	-1.86
Weight kg	.03**	.023**	.03**
Height cm	-.009		
Hip Circumference cm	-.001		
Smoking		-.111	
Physical Functioning		-.012	
% Carbohydrates		-.004	
% Protein		.03	
% Sweets		.001	
Dietary CA mg		2.12×10^{-4}	
Dietary Fat g		-.001	
Supplemental CA mg		-2.59×10^{-4}*	
Income			.021
Number of People to Support on Income			-.05
Neighborhood Satisfaction			-.10
Highest Level of Education			-.004
Occupation			-.001
R^2 values	**.17**	**.16**	**.19**

* $p<.05$
** $p<.01$

Comparison of R^2 Values

The inclusion of socioeconomic variables improves multiple linear regression models and accounts for more variation in BMC/BMD than models that do not include these variables. The amount of variance in BMC/BMD explained by the socioeconomic regression model was greater than the demographic or lifestyle model for all sites except BMAD and FN. All regression models explained the same amount of variance in FN, and the socioeconomic regression model was not significant for BMAD.

The socioeconomic regression model explained 57% of the variance in WBBMC, 28% in FN, and 32% in TH. Other published studies that do not include socioeconomic variables in regression models find their models only account for approximately 20% of the variation in the femoral neck and 29% in the total hip (Morton et al., 2003). Cauley et al's (2005a) study on factors affecting bone density in men show that multivariate models which included lifestyle, medical history, and demographic information only explained 19% of the variance in the femoral neck. This suggests that including socioeconomic variables improves prediction models of bone density in the femoral neck and total hip.

Z-scores were less influenced by the variables included in the regression models than WBBMC, FN, or TH as evidenced by their lower R^2 values (Tables 23 and 24). This is expected because Z-scores are already age and sex-adjusted, which eliminates some variation in the scores. The socioeconomic regression model for ZFN and ZTH shows that 18% and 19% of variation respectively can be attributed to variation in weight and SES variables.

Comparison of the R^2 values between these multiple linear regression models indicates that including socioeconomic variables improves the prediction model for BMC and BMC variables. The R^2 values are a gauge of the models as whole. We now turn our attention to some of the significant independent variables identified in these models.

Significant Independent Variables

Many of the unstandardized coefficients in Tables 19 through 24 are very small numbers (especially for BMAD, FN, TH, ZFN, and ZTH). These small numbers are a function of both the small measurement units of the independent variables (grams and milligrams for some of the dietary variables) and the small numbers of measurement for most of the bone density variables (see the means for BMC and BMD measures in Table 14). Although the unstandardized coefficients often denote small numerical changes in BMC/BMD, many of these changes are statistically significant.

Demographic variables

The importance of age, sex, and body size to BMC/BMD is apparent in this study. Age was a significant negative covariate for BMC/BMD for all sites and all regression models, except for WBBMC in the socioeconomic model. In this case, Age is negatively associated with WBBMC, but is not a significant covariate. This suggests that age may be less important to WBBMC when socioeconomic covariates are included (Robbins et al., 2004).

Female sex was a significant negative covariate for nearly all sites and all regression models with a few exceptions. Sex was not significant for FN in the lifestyle regression model. This was unexpected, but may indicate that including smoking as a

covariate diminishes the amount of variation explained by sex. Sex was not significant for any of the regression models for BMAD. This result, in addition to the small overall R^2 values for BMAD and the fact that the socioeconomic model was not significant for BMAD warrants some discussion.

BMAD is a measure of total body bone mineral content that approximates a volumetric measurement. This measure accounts for body size by incorporating height and the area scanned by the DXA machine (see Chapter 5 for how BMAD is calculated). The results of the regression models for BMAD indicate quite clearly that after accounting for body size, very little variation in bone mass can be explained by other factors. The importance of body size on all bone mass and bone density sites is evident when we examine the results of the other multiple regression models.

Weight is one the most effective means of controlling for body size. Weight was a significant contributor to increased bone mass and bone density at all sites where it was included in the regression model. The substantial contribution of weight to explain variation in bone mass and bone density is commonly reported (Nieves, 2008). Other measures of body size were less effective at predicting variation in BMC/BMD than weight alone. Height was only significant for WBBMC and Hip Circumference was not significant at any site. Hip Circumference does not seem to add anything significant to BMC/BMD variation over and above what is captured by Weight.

Lifestyle variables

Being a current or former smoker significantly decreased BMC/BMD for all regression models except ZTH. This analysis supports many previous claims about the influence of smoking on BMC/BMD in this sample. Current or former smokers had

significantly less BMC/BMD for all sites except ZTH. This effect was independent of Sex, Age, Weight, or other lifestyle variables included in the regression model. Gerdhem & Obrant (2002) found bone density in the femoral neck and total body was significantly less for current smokers compared to never smokers. Other studies have found that BMC/BMD is significantly less in former or current smokers when compared to never smokers (Cauley et al., 2005a;George et al., 2003;Nieves, 2008).

Physical Functioning was not significant in any of the regressions. There are several reasons that may explain this result. This analysis used a physical activity assessment that was available for nearly all participants, but was limited in scope. The physical activity component of the 36 item short form health survey (SF-36) may not capture the information relevant to differences in bone density (Holm et al., 2002;Ware & Sherbourne, 1992). Other studies have found that physical activity assessments via similar questionnaires were not statistically significant (Morton et al., 2003;Robbins et al., 2004). More quantitative measures of physical activity may be warranted in bone density studies. Measurements of physical activity for many bone density studies are based on exercise tests, muscle strength, or studies comparing athletes and non-athletes (Berard et al., 1997;Daly et al., 2004;Wolff et al., 1999). The results from this analysis indicate that physical activity as assessed by the SF-36 may be too limited to be of use for bone density studies.

This analysis found few dietary variables that were significant in the lifestyle regression model. Dietary Calcium and Dietary Fat were not significant covariates at any site; however, their effect on BMC/BMD supported the results of previous research. Dietary Calcium was positively associated with BMC/BMD at all sites, which supports

many previous studies that find a similar relationship (Heaney, 2000). Studies on both animals and humans indicate dietary fat intakes are negatively associated with bone density (Corwin et al., 2006;Wohl et al., 1998). The results of this study also found that Dietary Fat had a negative influence on BMC/BMD at all sites. Dietary Fat and Percentage of Calories from Carbohydrates had a negative effect on BMC/BMD at all sites, but neither were significant covariates. The Percentage of Calories from Protein and the Percentage of Calories from Sweets had both negative and positive effects on BMC/BMD depending on the site.

The only dietary variable that was significant in the lifestyle regression model was Supplemental Calcium. Supplemental Calcium decreased bone density for TH and ZTH. Calcium supplements had a significant negative effect on bone density in the TH and ZTH. This contradicts numerous studies that demonstrate calcium supplements increase bone density (Aloia et al., 1994;Dawson-Hughes et al., 1990;Dawson-Hughes et al., 1997;Recker et al., 1996). However, Cauley et al. (2005a) in a study on the effects of several factors on bone density in men found a similar negative association between calcium supplements and bone density. One reasonable explanation for this incongruous finding is an indication bias where individuals with low bone mass were advised to take calcium supplements.

Socioeconomic variables

Education and Neighborhood Satisfaction were significant independent covariates in the socioeconomic model for WBBMC. Each ranked increase in educational attainment was associated with a 22.22 g increase in WBBMC. This result provides additional evidence that educational attainment is positively associated with bone mass.

Neighborhood Satisfaction was negatively associated with WBBMC, where increasing Neighborhood Satisfaction corresponded to a decrease in WBBMC. It is unclear at this time why this may be the case. It is possible that opinions of the neighborhood may vary systematically with age. Very few bone density studies measure neighborhood satisfaction or a similar attribute, therefore comparisons with previous research are not applicable.

It is clear from the results of the multiple regression models that Weight, Age, and Sex are the variables that have the largest influence on bone mass and bone density. Smoking was the only lifestyle variable to be significant for nearly all the BMC/BMD measures. Education and Neighborhood Satisfaction were only significant for WBBMC. The next part of this analysis looks more carefully at Body Mass Index and examines how predictive this measure of body size is for BMC/BMD.

Body Mass Index

I compared two different formulas for calculating BMI for their contribution to variation in BMC/BMD. Which formula explains more variation in bone mass and bone density in this sample? Comparison of R^2 values from multiple linear regressions testing BMI formula (a) and BMI formula (b) (see page 81 for description of these variables) will determine which formula is most predictive of BMC/BMD when age and sex are held constant. Two different regression tests are run with WBBMC, FN, TH, ZFN, and ZTH as the dependent variables. BMAD was not included as a dependent variable because it incorporates body size in its calculation. Age and Sex were not included as independent variables for ZFN and ZTH because these bone density measures are already

adjusted for age and sex. First, Age, Sex, and BMI (a) are run together in a multiple regression, then Age, Sex, and BMI (b) are run. Table 24 lists the unstandardized coefficients and R^2 values for these multiple linear regression tests.

Table 24. Unstandardized coefficients and multiple coefficient of determination (R^2) for Age, Sex, and BMI variables on WBBMC, FN, TH, ZFN, and ZTH

	Constant	Age	Sex	BMI (a)	BMI (b)	R^2
	Unstandardized Coefficients					
WBBMC	3263.72	-4.07*	-702.24**	19.99**		**.39**
	3027.67	-3.81*	-680.23**		18.09**	**.43**
FN	.94	-.004**	-.09**	.01**		**.25**
	.88	-.004**	-.08**		.008**	**.27**
TH	1.01	-.002**	-.15**	.01**		**.27**
	.955	-.002**	-.13**		.009**	**.28**
ZFN	-1.74			.06**		**.13**
	-1.98				.05**	**.15**
ZTH	-1.81			.07**		**.14**
	-2.03				.05**	**.16**

* $p<.05$
** $p<.01$

BMI (b) has higher R^2 values compared to BMI (a) for all of the BMC/BMD measures. This analysis is one of the first to directly compare the BMI (b) formula from Kleerekoper et al. (1994) with the conventional BMI (a) formula for their effectiveness at accounting for body size. The results demonstrate that BMI (b) contributes to more variation in bone mass/density than BMI (a). This finding supports claims that tailor-made indexes based on weight and height data from the population of interest accounts for body size more effectively than the conventional BMI used by the CDC (CDC, 2009;Kleerekoper et al., 1994;Lee et al., 1981).

However, despite the higher R^2 values for BMI (b) compared to BMI (a) in this study, weight and height alone may be the best method to account for body size in bone

mass studies. Weight and Height used as separate variables account for more variation in WBBMC than using BMI. Compare the R^2 values for BMI (b) (Table 24) to the R^2 values for the demographic regression model for WBBMC (Table 18). Age, Sex, Weight, Height, and Hip Circumference account for 56% of the variation in WBBMC, while Age, Sex, and BMI (b) account for only 43%. For studies using WBBMC, it seems that weight and height are better controls for body size than using BMI (b).

Other studies have found that weight alone or weight and height account for more variance in BMC/BMD than BMI (Edelstein & Barrett-Connor, 1993;Finkelstein et al., 2002). The results of this study support that claim, but only for WBBMC. Differences between the demographic regression model and the BMI (b) regression are not as great for FN, TH, ZFN, and ZTH (only 1-2% difference). The demographic regression R^2 values are slightly larger for FN, ZFN, and ZTH and slightly smaller for TH. For other bone density sites, it appears that either BMI (b) or weight alone would be equally effective at controlling for body size in similar populations.

Results Summary

The results of the statistical investigation of these data collected for this project has examined the association between several demographic, lifestyle, and socioeconomic variables and BMC/BMD measures. This study found that regression models that include socioeconomic variables have slightly higher R^2 values for most bone mass sites when compared to models that include lifestyle factors and compared to models that only include demographics. Over half (57%) of the variation WBBMC is explained by Age, Sex, Weight, Height, and SES variables. No difference was found between any of the

regression models for FN. The socioeconomic regression model was not significant for BMAD, and the other regression models only accounted for a very limited amount of variation as evidenced by their small R^2 values.

Several variables were identified in these models as significant contributors to BMC/BMD. Smoking is significantly associated with a decrease in bone mass at all sites. Education and Neighborhood Satisfaction were significant only for WBBMC, and Supplemental Calcium intakes were significant for only TH.

Tests of association investigated the relationship between SES and BMC/BMD. These tests found significant differences between mean BMC/BMD and Income levels, Education attainment, and Number of People to Support on Income. This study also examined the difference between two BMI formulas in predicting BMC/BMD. Comparison of R^2 values indicates that BMI (b) contributes to more variation in bone mass at all sites than BMI (a). The next chapter discusses the meaning and implications of the results presented here.

Chapter 7

Discussion and Conclusions

The purpose of this investigation was to examine factors that may contribute to what is frequently and erroneously presumed to be inherent racial/ethnic differences in Bone Mineral Content (BMC) and Bone Mineral Density (BMD). This analysis examined intra-group variation of BMC/BMD associated with several demographic, lifestyle, and socioeconomic attributes. Several previously unrecognized correlates that may contribute to systematic racial/ethnic differences in BMC and BMD are revealed in this analysis. The findings presented here indicate that racial/ethnic differences in BMC/BMD are not inherent or innate, but instead an artifact of systematic racial/ethnic variation in individual and non-biological traits.

Body Size

Systematic racial/ethnic differences in weight and body size may be a primary contributor to the racial/ethnic differences BMC/BMD documented in the U.S. This analysis indicated that weight and body size were the most important contributors to variation in BMC/BMD in this sample. As discussed in Chapter 4, there is a strong positive correlation between weight and BMC/BMD (Cifuentes et al., 2003;Edelstein & Barrett-Connor, 1993). Weight varies systematically between African-Americans and White Americans in U.S. (Kucamarski, 1992;Williamson et al., 1991). According to

recent national data, African-American adults are heavier than White Americans and have a higher prevalence of obesity (Ogden et al., 2006). The sample used in this analysis was predominantly overweight, and over half of the female subjects had a BMI of greater than 30, which is categorized as 'obese' according to CDC standards (CDC, 2008;CDC, 2009).

Finkelstein et al., (2002) demonstrate that most racial/ethnic differences in BMD can be attributed to differences in weight. However, Finkelstein et al., (2002) and others occasionally find that racial/ethnic differences in BMD persist after accounting for weight (Barondess et al., 1997). This has often been interpreted as indicating that genetic or inherent racial/ethnic differences are responsible for the remaining difference (see literature review in Chapter 3). I suggest that instead of resorting to such assumptions, that we may need more careful consideration of accounting for body size in Dual Energy X-ray Absorptiometry (DXA) studies.

As discussed in Chapter 5, BMD as measured by DXA does not always adequately correct for bone and body size differences between individuals or populations (Prentice et al., 1994). To compensate for this, various measures of body size are often used to adjust BMD measurements or used in multiple linear regressions to control for body size. My analysis used both of these techniques. Bone Mineral Apparent Density (BMAD) was calculated as an estimation of volumetric whole body BMD, and 2 different formulas for Body Mass Index (BMI) were used to examine the variation in bone density in multiple linear regressions.

The results of my analysis using BMAD provides further evidence that differences in body size are associated with most of the variation in bone mass. The

small R^2 values from the multiple linear regressions (Table 19) indicate that very little variation in BMAD is accounted for by any of the demographic, lifestyle, or socioeconomic variables included in the regression models. For this sample, after accounting for skeletal size, there is very little difference in whole body BMC between individuals.

BMI is often used in bone density studies to account for body size differences between individuals. Height is strongly correlated to overall skeletal size; taller individuals tend to have larger skeletons. DXA measurements also tend to overestimate the BMD of taller individuals due to their larger skeletal size. BMI measures minimize differences in height to produce a height free measure of weight. The standard ratio for calculating BMI divides weight by height squared, but this ratio is an estimate of the contribution of height to weight and the actual ratio varies depending on the individual or population being studied (CDC, 2008).

Similar to the systematic racial/ethnic differences in weight documented in the U.S., there are also systematic differences in body size and body proportions between populations. Several authors recommend the use of population specific BMI ratios to account for these differences in weight and height ratios and better control for body size in BMD studies (Kleerekoper et al., 1994;Lee et al., 1981;Robbins et al., 2006). My analysis demonstrates that the calculation of BMI for the specific population under study should be seriously considered in BMD research. More variation in BMD was accounted for in my analysis when the BMI ratio specifically formulated for the body proportions of African-Americans was used in contrast to the 'standard' BMI ratio.

Studies that don't carefully consider weight, height, and BMI for DXA

measurements may find racial/ethnic differences in BMD that are originating in issues stemming from body size estimations. Insufficient attention to systematic racial/ethnic body size variation may partially contribute to the racial/ethnic differences in BMD documented in the U.S. Since DXA BMD measurements are sensitive to skeletal and body size, regard for these details is important so that erroneous conclusions about the magnitude and source of racial/ethnic difference in BMD are minimized.

It is well known that body size is an important consideration for DXA BMD studies, and this analysis demonstrates that a great deal of intra-group BMC/BMD variation can be accounted for by body size. Weight, height, BMI, and the estimation of volumetric BMD (such as whole body BMAD) should be addressed carefully so that any racial/ethnic body size variation is accounted for in BMD studies. This analysis also investigated several lifestyle variables for their association with BMC/BMD.

Lifestyle

The lifestyle variables included in this analysis, were, for the most part, not statistically significant covariates in the multiple linear regression models at any of the BMC/BMD skeletal sites. This may be partially due to the superficial dietary and physical activity variables used in this analysis. As discussed in Chapter 4, there is abundant evidence that physical activity and diet influence bone mass, but this analysis may not have included sufficiently detailed variables to detect an effect. The lifestyle variables selected from the CUAAH database for this analysis were intended to provide general information about health and diet. More often, research reporting statistically significant effects of diet and physical activity on bone mass are longitudinal studies and

incorporate more detailed information about nutrition and physical activity (Daly et al., 2004;Heaney, 2000;Uusi-Rasi et al., 2008).

Smoking was one exception; it was a statistically significant covariate at all skeletal sites except for Z-score of the Total Hip (ZTH). As discussed previously in Chapter 6, this analysis is in agreement with several other studies that also find a negative association between smoking and bone mass (Gerdhem & Obrant, 2002;Nieves, 2008). The mechanism for how smoking may affect bone mass is unclear, and its covariance with BMC/BMD in this analysis may be indirect. Smoking may be an indicator of health, diet, or activity in this sample. My analysis could be picking up variation associated with other characteristics smokers and non-smokers may posses that influence bone mass.

Few studies have examined additional dietary factors that were included in this study such as percentage of calories from protein, carbohydrates, or sweets. Their lack of significance in the lifestyle regression model for all sites suggests that their influence on BMC/BMD is minimal. However, as stated previously in Chapter 4, the influence of dietary factors on BMC/BMD is difficult to measure due to the fact that current nutritional intake only affects the small portion of actively remodeling bone. This analysis lacks longitudinal data that may demonstrate a nutritional effect on BMC/BMD over time.

Physical activity as measured by the physical functioning component of the SF-36 may not be specific or sensitive enough for BMD studies. This component is only composed of 10 questions, and most research on the effect of physical activity on BMC/BMD implements a more extensive survey or monitored physical efforts such as

walking speed or grip strength (Berard et al., 1997;Wolff et al., 1999). It is likely that the lack of inclusion of dietary and physical activity details for this analysis contributed to results in which these factors were not statistically significant covariates.

Socioeconomic Status

It is evident from this analysis that some BMC/BMD measures are sensitive to social and economic situations, but these conditions only influence bone mass and bone density indirectly. The relationship between bone mass and socioeconomic conditions is likely to be complex because the effect would be mediated through physiological processes that have a more direct effect on bone remodeling. This analysis considered several socioeconomic variables not commonly studied in bone mass research and revealed previously unrecognized correlations between measures of socioeconomic status and bone mass. Nuanced interpretations of the relationship between socioeconomic variables and bone mass are rare in studies that frequently examine socioeconomic status superficially. What is often attributed to inherent racial/ethnic differences may be due to perfunctory treatment of socioeconomic variables and more careful consideration of socioeconomic conditions is warranted in future studies.

The relationship between socioeconomic variables and BMC/BMD is complex and inconsistent. The variables used to measure economic stress in this analysis; Income, Number of People to Support, and Education, do not have a uniform association with BMC/BMD. Economic stress *per se* is not likely to be the factor of importance with regards to BMC/BMD, but rather how economic circumstances are borne out in ways that have more direct influence on BMC/BMD. Therefore, we find what appear to be

contradictory results concerning the relationship between socioeconomic measures and BMC/BMD.

This analysis demonstrates that one measure of economic strain; more people to support, is associated with *greater* BMC/BMD at all skeletal sites. Other measures of economic strain, such as lower incomes, and educational attainment were associated with *lower* BMC/BMD. The number of people to support is not exerting the same influence on BMC/BMD as the other socioeconomic variables.

The reason for this association may be due to the fact that subjects with 5 or more people to support are younger with children living at home, or that they are more active and less sedentary than subjects with only 2 or 3 people to support. A post hoc test of this assumption finds that subjects with 5 or more people to support are significantly ($p<0.05$) younger than those with only 1 or 2 people to support. While this is a likely causal factor underlying this relationship, more evidence is needed to make a definitive statement about the reasons for the association between number of people to support and BMD.

Combining measures of social and economic strain may be counter productive to elucidating complex relationships between these variables and BMC/BMC. Measures of economic stress as measured in this analysis have opposite influence on BMC/BMD. This is probably why, when Income, Education, and Number of People to Support were combined in the Composite Status Score, the correlations were not significant. When ranked according to relative economic strain, the association between these three variables and BMC/BMD are actually at cross-purposes to each other. Combining them into one composite score nullifies their separate effects in the correlation.

This socioeconomic discordance between Income and Number of People to Support may also be the reason Income Per Person was not significant in correlations with BMC/BMD. Income and Number of People to Support have opposite effects on BMC/BMD. Dividing Income by Number of People to Support reduces the ratio for every increase in the Number of People to Support. This is contradictory to the relationship between Number of People to Support and BMC/BMD; where with every increase in the Number of People to Support, there is an increase in BMC/BMD. Therefore, this ratio effectively cancels out the separate, but opposite effects of Income and Number of People to Support.

Researchers should consider the possibility that measures of socioeconomic status may not have a uniform influence on bone mass. Careful consideration of socioeconomic variables such as examining attributes separately for isolated effects on bone mass may aid in such endeavors. This analysis revealed several previously unrecognized socioeconomic correlates with bone mass and included attributes not commonly examined in bone mass research. Number of People to Support, Neighborhood Satisfaction, and Occupation helped distinguish the influence of socioeconomic effects on bone mass in this analysis. The results of the socioeconomic regression models indicate that including socioeconomic variables improves predictions of BMC/BMD and accounts for more variation than regression models that do not include them.

Socioeconomic variables are often not considered carefully in bone mass research and treated rather superficially. As discussed in the literature review in Chapter 3, socioeconomic variables are rarely included at all bone mass studies, or when they are included, they are used as control variables. Socioeconomic attributes are only

occasionally examined directly as agents of variation in a great deal of bone mass research. In addition, interpretations of racial/ethnic differences in bone mass are seldom attributed to social or economic explanations. The customary and usual treatment of socioeconomic variables in bone mass research is not sufficiently nuanced to illuminate the complex and indirect relationship between bone mass and socioeconomic status.

What is commonly, but erroneously, put forth in many bone mass studies is that after ostensibly controlling for socioeconomic status, the remaining variation is attributed to inherent racial/ethnic differences. Racial/ethnic differences in bone mass are likely to be partially rooted in long-standing socioeconomic inequality between racial/ethnic groups in the U.S. Cursory analysis of socioeconomic attributes is unlikely to reveal much about its relationship to bone mass and skeletal health.

This analysis examined the association between socioeconomic status and bone mass more directly. I tested the correlation between several different measures of social and economic strain for their significance to BMC and BMD. In addition, I included socioeconomic variables in multiple linear regression models in order to estimate their contribution to variance in BMC and BMD. Very few bone density studies investigate socioeconomic status this thoroughly. The results of this analysis demonstrate that careful consideration of these variables is warranted in future studies.

Limitations

This research was limited in its analysis and subsequent interpretations by a few conditions related to the CUAAH dataset. The participants of the CUAAH studies were recruited for cardiovascular and breast cancer research projects. Since bone mass was not

the primary focus of these studies, some information that is typically included in many bone mass studies is lacking. For instance, I do not have information on previous fracture history or if participants are taking medications that affect bone mass, such as hormone replacement therapy or osteoporosis medications. The inclusion of these variables may have accounted for more variation in the regression models, or enabled an analysis of the interaction between socioeconomic status and osteoporosis medications.

There is some evidence that this sample does not represent a large range of incomes and other socioeconomic variables. Nearly 25% of the participants recorded yearly incomes of $20,000 or less. Approximately 56% of the participants have yearly incomes less than the Michigan median yearly income of $49,699 (Census Bureau, 2008a). This indicates a higher proportion of participants have lower incomes than would be expected, and that this sample as a whole is more representative of lower income groups. Despite the relative homogeneity of incomes, BMC/BMD differences were found between income groups. This suggests that perhaps income is even more influential to BMC/BMD than reported here.

Analysis of the relationship between income and BMC/BMD was somewhat limited by the fact that yearly income was not recorded as a continuous variable, but as an ordinal one. The income ranges provided in the CUAAH database put some constraints on the income groups I constructed for this analysis. Incomes between $50,000 and $99,999 were lumped into one category in the CUAAH database and could not be meaningfully separated or counted; therefore all incomes greater than $50,000 are grouped together. Finer distinctions between incomes would have been possible with

continuous data and may have revealed further differences in BMC/BMD between income groups.

I was not able to calculate BMAD for the femoral neck. In order to calculate BMAD, one needs BMD and BMC from the DXA bone scan and I only had access to the DXA BMD measurement for the femoral neck. BMAD approximates volumetric BMD by modeling the skeleton geometrically. Femoral neck BMAD is easily modeled because it resembles a cylinder. It is possible that this additional BMD measurement could have added information to this analysis. Femoral neck BMAD will likely be included for subsequent studies using this data so that the investigation is thorough.

Future Studies

This dissertation serves as a model for investigating social, economic, demographic, and lifestyle variables more directly in bone mass research. The methods applied in this analysis such as limiting investigations to one racial/ethnic group and carefully considering socioeconomic and body size variables can be applied to future bone mass studies. In fact, a few authors have already recommended similar practices for bone mass research (Fausto-Sterling, 2008;Leslie & Lentle, 2006).

This analysis revealed several new and significant relationships between BMC/BMD variation and income, education, number of people to support, neighborhood satisfaction, BMI, weight, and smoking. Inclusion of these variables is recommended in future studies employing a similar approach so that patterns of bone density variation may be recognized within a social and environmental landscape. Many anthropologists and public health experts advocate moving away from presumptions of racial/ethnic

biological difference and towards explanations based in social and environmental causes. This research represents one facet of that initiative which considers bone mass and bone density.

The conclusions of this research can be applied to future studies examining BMC/BMD. The complex and discordant relationship between economic strain and BMC/BMD warrants further investigation. Subsequent research examining BMC/BMD in participants of diverse social and economic conditions may reveal further associations. A few authors have recently focused on regional bone mass variation (Kaptoge et al., 2008;Langsetmo et al., 2008;McCloskey et al., 2004). Geographically based bone mass studies are an important component of research seeking to identify environmental, economic, and social patterns of bone mass variation.

This analysis has demonstrated that population or sample-specific BMI ratios should be considered for bone mass research. The importance of body size to DXA studies is well-known and has received much attention (Edelstein & Barrett-Connor, 1993;Katzman et al., 1991;Riggs et al., 2004;Robbins et al., 2006). Addressing systematic body size differences in studies that make racial/ethnic BMD comparisons should be a priority.

Summary

A great deal of bone density and osteoporosis research relies on poorly defined racial/ethnic variables that often obscure, rather than clarify the forces and factors that may be causing variation in bone mass. This research design applies a different approach in which inherent racial/ethnic differences in bone mass are not assumed *a priori*. Unlike

many studies examining causes of variation in BMC/BMD, this analysis began with the assumption that racial/ethnic differences in bone mass are not inherent or primarily based in biology. Instead of accounting for variation by an ambiguous 'race' variable, which often leads to interpretations of inherent racial/ethnic differences, I examined measures often confounded with race directly. I directly examined several attributes that vary systematically by race/ethnicity in the U.S., and are also influential to BMC/BMD.

The use of poorly defined racial/ethnic categories in bone density and osteoporosis research frequently contributes to biological interpretations of racial/ethnic bone mass differences. Careful consideration of integral problems in the conceptualization, application, and interpretation of racial categories in bone density research led to the construction of this analysis. Participants in this analysis only include subjects who self-identify as African-American so that intra-group variation in bone mass can be investigated. This practice offers several advantages over more common inter-racial/ethnic comparisons: Investigation of social, economic, demographic, and lifestyle characteristics important to bone mass can be examined directly without assuming racial/ethnic variables add significantly to the analysis; and gaps in current knowledge about bone mass distributions across the U.S. are addressed.

This approach has identified several new and significant findings for the causes of BMC/BMD variation. This research uncovered previously unrecognized correlates with bone mass such as number of people to support and neighborhood satisfaction. These results reveal that the relationship between SES and BMC/BMD is not uniform in its directionality, and there is likely to be discordance between variables associated with economic strain and BMC/BMD. Some socioeconomic attributes have a positive

association with BMC/BMD and some are negative. Research designs that include a conglomerate socioeconomic status score may not be effective at discovering conflicting associations between BMC/BMD and social and economic conditions.

Future studies employing research designs in which intra-groups variation is examined may be able to uncover previously unrecognized relationships with social and economic variables. This approach is a reasonable and recommended research design for bone mass studies.

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