DEMOGRAPHIC AND LIFESTYLE VARIABLES AFFECT BONE MINERAL DENSITY IN PREMENOPAUSAL WOMEN

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Bachelor of Science

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1998

Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
MASTER OF SCIENCE
December, 2000

DEMOGRAPHIC AND LIFESTYLE VARIABLES

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PREMENOPAUSAL WOMEN

Thesis Approved:

Thesis Advisor

Dean of the Graduate College

ACKNOWLEDGEMENTS

I would like to thank several people who have contributed to my success in completing this thesis. First, I would like to express my gratitude to my major advisor, Dr. Kathryn S. Keim, and my committee members, Dr. Gail E. Gates, and Dr. Christine A. Johnson for their infinite wisdom, invaluable encouragement, and immense assistance throughout the research and writing process. In addition to my committee members, I would like to thank the Nutritional Sciences Department for support during my research, especially Dr. Barbara J. Stoecker for her encouragement to come back for my Master's degree.

I would also like to recognize my family for all of the support they have given—emotionally and physically. Here's to you guys—Mom and Dad; Dena, Joshua, and Elizabeth; Esther, Robert, Andrew, and Samantha; Debbie, Doug, and the new baby (Ashley?); Sarah, David, Lexie, and Grant; Bryce and Zachary. And, of course, special thanks to those who helped me proofread everything—Aunt Orma (who said I had to get my Master's degree!), Esther, Dena, and Bryce.

Lastly, I would like to recognize that I could not and would not have been capable of doing this without the knowledge that God is in control and He is guiding my life.

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CHAPTER I

INTRODUCTION

Osteoporosis is a skeletal disorder distinguished by reduced bone mass and a change in the microstructure of bone resulting in an increased risk of bone fracture due to bone weakness (National Osteoporosis Foundation, 1999). Osteoporosis can strike people of all ages and ethnic backgrounds. Currently, 10 million people in the U.S. have osteoporosis. Eighty percent of those 10 million are women. Another 18 million people are at risk for osteoporosis due to low bone density. The end product of osteoporosis is osteoporotic bone fractures, which occur over 1.5 million times per year. Often people do not realize they have osteoporosis until they experience a fracture.

Osteoporosis has negative impacts on society, according to the National Institute of Health's Consensus Statement on Osteoporosis (2000). Financial expenses for treatment of fractures related to osteoporosis are approximately 10 to 15 billion dollars per year. This does not include the indirect costs, such as lost wages and reduced productivity. Negative physical impact of osteoporosis occurs on other body systems, such as respiratory, gastrointestinal, and genitourinary systems. Psychosocial impacts include inability to perform activities of daily living and increased symptoms of depression.

There are many risk factors related to osteoporosis. Being older, female, or postmenopausal, having low body weight, or having a family history of osteoporosis all increase the risk for osteoporosis (National Osteoporosis

Foundation, 1999; World Health Organization, 1999). Other risk factors include inadequate dietary intake of calcium, a sedentary lifestyle, smoking, excessive alcohol use, and use of certain medications (National Osteoporosis Foundation, 1999; World Health Organization, 1999).

The World Health Organization (WHO) (1994) defined osteoporosis as a bone mineral density (BMD) of at least 2.5 standard deviations below the reference value for the mean BMD of young adults. Looker et al. (1997) used non-Hispanic whites between 20 and 29 years of age for the young adult reference group, although the WHO did not indicate the race, sex, or age of the reference group. Osteoporotic fractures are best predicted by low BMD measurements, and the existing standard for assessing proximal femur and lumbar spine BMD is the dual energy x-ray absorptiometry (DEXA) (Sahota and Masud, 1999). Kanis et al. (1994) reiterates the idea that the risk factor for osteoporosis is low BMD and the clinical expression is fractures.

The Centers for Disease Control and Prevention (CDC) and the National Institutes of Health (NIH) collaborated on the "Arthritis, Osteoporosis, and Chronic Back Conditions" focus area of Healthy People 2010 (CDC and NIH, 2000). Healthy People 2010 objectives include reducing the overall number of cases of osteoporosis to 8 percent of adults aged 50 years and older as measured by low total femur BMD (CDC and NIH, 2000). Another Healthy People 2010 objective is to reduce the proportion of adults who are hospitalized for vertebral fractures associated with osteoporosis to 11.6 hospitalization per 10,000 population aged 65 years and older. Additional objectives include

reducing hip fractures among adults 65 years and older to 491.0 per 100,000 for women and to 450.5 per 100,000 for men.

The objective of this study was to determine the relation between BMD, demographic variables, and health behaviors in premenopausal women. If health professionals know which factors may be related to BMD, they can determine which clients are at greater risk for developing osteoporosis and target those individuals for intervention. By improving those health behaviors that may be related to BMD, individuals may prevent osteoporosis when they are older. Individuals may be unaware of their need to improve or prevent further loss of BMD because osteoporosis takes years to develop.

CHAPTER II

REVIEW OF THE LITERATURE

Reviewing the literature will help clarify why this study is needed. First, a definition of osteoporosis will be given and a brief overview of bone metabolism will be covered. Next, the prevalence of osteoporosis and bone fractures will be reviewed. Finally, variables, including nutrient intake, which may influence BMD will be discussed.

Osteoporosis and Bone Metabolism

Osteoporosis is a skeletal disorder distinguished by reduced bone mass and a change in the microstructure of the bone resulting in an increased risk of bone fracture due to bone weakness (National Osteoporosis Foundation, 1999). In order to understand osteoporosis, it is important to briefly review the basics of bone metabolism. There are two types of bone structure, cortical and trabecular (Arnaud and Sanchez, 1996). Cortical bone is the densely packed outer bone, whereas trabecular is the spongy inner bone.

The human body has very tight metabolic controls to maintain calcium homeostasis in the blood. The bone provides a storage area for calcium, so when the blood levels of calcium are outside of the tight range for homeostasis, the bone either releases or accepts calcium (Arnaud and Sanchez, 1996). This process of releasing or accepting calcium is called bone remodeling.

The three types of cells involved in bone remodeling are osteoclasts, osteoblasts, and osteocytes (Arnaud and Sanchez, 1996). Osteoclasts are the cells that resorb bone, or release calcium and other minerals from the bone to go into circulation. Osteoblasts are the cells that lay down bone matrix proteins at the site of resorption by the osteoclasts. After the proteins are laid down, the collagen matrix then becomes mineralized. The osteoblasts become trapped in the bone matrix that they just made. Once the osteoblasts become encased in the collagen and mineral matrix, they are then called osteocytes, or literally "bone cells".

Not only are there several different cells involved in bone remodeling, there are many hormones involved in bone remodeling (Arnaud and Sanchez, 1996). These include parathyroid hormone (PTH), calcitonin, and 1,25-dihydroxycholecalciferol (calcitriol). Calcitriol is considered the most active form of vitamin D. However, there are other forms of vitamin D that are metabolically active, but not as active as calcitriol.

In order to maintain tight metabolic control of calcium levels in the blood, the body immediately tries to compensate for either low blood calcium levels or high blood calcium levels (Arnaud and Sanchez, 1996). In response to low calcium concentrations, PTH secretion increases and calcitonin secretion decreases. PTH and calcitonin act in three main areas of the body: bone, intestine, and kidney. PTH stimulates bone resorption, so there will be more calcium in the blood. Also, PTH stimulates formation of active vitamin D (calcitriol) in the kidney, which, in turn, enhances calcium absorption in the

intestine, so more calcium will be absorbed from the food consumed. PTH also reduces renal excretion of calcium, so less calcium is lost via the urine.

Just the opposite happens in response to high concentrations of calcium in the blood, calcitonin secretion increases and PTH secretion decreases (Arnaud and Sanchez, 1996). Calcitonin inhibits bone resorption, so calcium is not released from the bone. Calcitonin also decreases calcium absorption in the intestine by inhibiting the activation of vitamin D in the kidney. In addition to the other compensatory mechanisms to lower blood calcium levels, calcitonin also increases renal excretion of calcium.

Not only are hormones important in bone formation, but various minerals are also important. Two of the most important minerals involved in the formation of bone are calcium and phosphorus (Arnaud and Sanchez, 1996). Calcium and phosphorus are both a part of hydroxyapatite, the mineral crystals that form on the collagen matrix. Calcium and phosphorus are closely interconnected through the action of PTH and calcitonin and their regulatory mechanisms. Both calcium and phosphorus must be present at the proper time and in the proper amounts to be part of the hydroxyapatite that will be laid down on the collagen matrix.

Other important minerals involved in bone formation are copper, zinc, and magnesium. Copper is a part of lysyl oxidase, an enzyme important for collagen cross-linking (Linder, 1996). The collagen cross-linking is vital so there can be a protein matrix on which the minerals can be "stuck". Zinc is also necessary for collagen formation (Cousins, 1996). Magnesium is important for bone formation, but the role it plays is not clear (Shils, 1996). Magnesium may be a part of the

hydroxyapatite. It is known that magnesium deficiency leads to low magnesium, low calcium, and low potassium levels in the blood. It also may decrease the levels of circulating PTH.

Bone Mineral Density and Osteoporosis

Measurement of bone mineral density

Bone mineral density (BMD) is the measurement of mass per area of bone to determine the density, or strength of the bone. The unit of measurement is grams per centimeter squared (g/cm^2). Sites within the proximal femur measured using dual-energy x-ray absorptiometry (DEXA) in NHANES III are the femoral neck, trochanter, intertrochanter, Ward's Triangle, and total femoral region (National Center for Health Statistics, 1994) (Figure 1).

The most commonly used methods of measuring bone mineral density are single photon absorptiometry (SPA), dual photon absorptiometry (DPA), and dual-energy x-ray absorptiometry (DEXA). SPA measures bone mineral content instead of BMD, and requires a uniform thickness of tissue around the bone being measured (Wahner, 1996). SPA is generally used in appendicular bones, such as the radius, that have uniform distribution of tissue around the bone. DPA measurements can be performed on areas of the body with irregular thickness of tissue, such as lumbar spine or proximal femur (Wahner, 1996). However, DPA scans are time consuming and require complicated procedures. DEXA is useful in determining the BMD in areas of the body that do not have a constant thickness, such as the lumbar spine, proximal femur, radius, and whole body

(Shore and Poznanski, 1999). This is a major advantage of DEXA over SPA. DEXA is useful because it measures both cortical and trabecular bone, which are useful in evaluating overall bone mineral status (Shore and Poznanski, 1999). DEXA is more accurate and faster than DPA (Wahner, 1996).

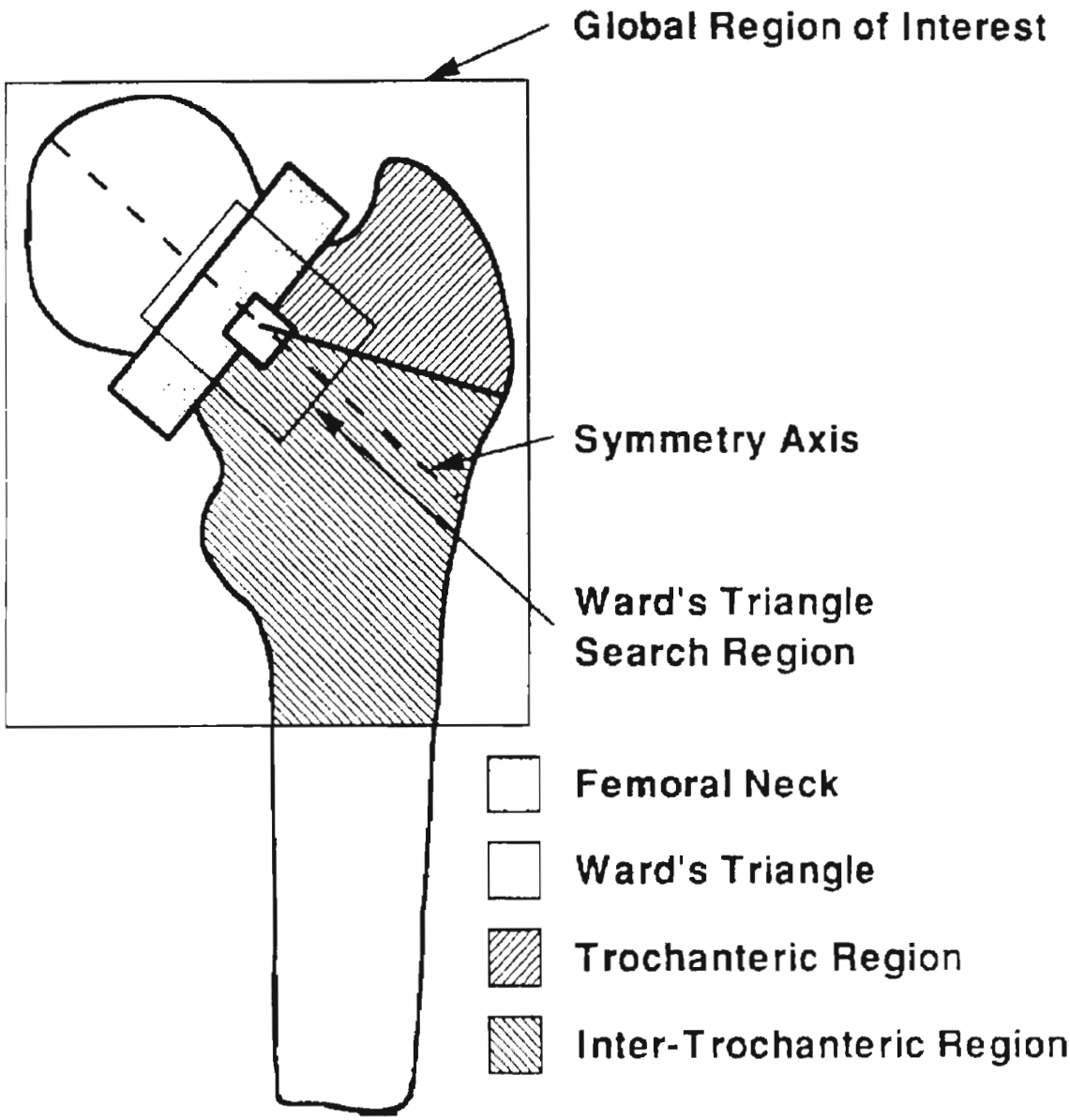

Figure 1. Bone sites of proximal femur (Hologic, 1998).

Trabecular bone has a much greater amount of surface area and therefore has a higher metabolic turnover than cortical bone, which is four times more dense than trabecular bone (Einhorn, 1996). Einhorn (1996) determined that various areas of the skeleton have different amounts of trabecular bone. The amount of trabecular bone in various areas is as follows: vertebrae, 66-90%; intertrochanter, 50%; femoral neck, 25%; distal radius, 25%; mid-radius, 1%; and femoral shaft, 5% (Einhorn, 1996). Einhorn (1996) also states that osteoporosis will produce a loss of trabecular bone and result in fractures in areas of the skeleton that are composed of a large percentage of trabecular bone, such as the vertebra and the intertrochanter. Although the femoral neck is composed primarily of cortical bone, which usually has a protective periosteum layer of bone, the femoral neck is not protected by a layer of periosteum (Einhorn, 1996).

Relation between bone mineral density and osteoporosis

Bone mineral density measurements have been used to predict osteoporotic fractures (Black et al., 1992; Melton et al., 1993; Faulkner et al., 1993). Lower BMD was associated with higher risk of osteoporotic fractures. If an individual has a greater risk of osteoporotic fractures, he or she can work with health care providers to try to reduce that risk.

Black et al. (1992) prospectively studied 8134 women aged 65 years and older from the Study of Osteoporotic Fractures. Researchers wanted to see if appendicular BMD in the radius (forearm) and calcaneus (heel), as measured by SPA predicted fractures as well as axial BMD in the spine and proximal femur, as

measured by DEXA. In addition, researchers wanted to see if proximal femur and spine BMD predicted fractures. A decreased BMD in the spine and proximal femur was strongly associated with about a 1.4 times higher age-adjusted relative risk of all fractures ($p=0.05$). Relative risk of 1.4 means that a female with a BMD value 1 standard deviation (SD) below the mean has a 1.4 times greater risk of fracture than a female with a BMD value at the reference mean. No measure of BMD at the proximal femur, spine, heel, or forearm was significantly better at predicting fractures than any other measure of BMD at those sites.

Melton et al. (1993) followed 304 Minnesota women aged 30-94 years for a median of 8.3 years to determine if BMD at the spine and femur, as measured by DPA, and BMD at the radius, as measured by SPA, could predict fractures. The age-adjusted risk of fracture at all fracture sites was 1.4 for lumbar spine BMD and 1.3 for femoral neck BMD. That means if a women has a BMD 1 SD lower than the reference mean at the femoral neck, she would have a 1.3 times greater risk than a woman who has a BMD at the reference mean. Of all the fracture types the researcher studied, fractures from mild to moderate trauma are most likely related to osteoporosis.

Faulkner et al. (1993) studied hip DEXA scans of 8074 white females aged 67 years and older from the Study of Osteoporotic Fractures. These researchers specifically studied the geometry of the proximal femur (hip) in relation to predicting hip fractures. The controls and cases had significantly different femoral neck BMD (0.652 ± 0.094 g/cm^2 and 0.556 ± 0.118 g/cm^2,

respectively) ($p<0.05$). The age-adjusted relative risk for hip fracture is 2.7 for femoral neck BMD ($p=0.05$).

Prevalence of Osteoporosis and Bone Fracture

The Centers for Disease Control and Prevention (CDC) and the National Institutes of Health (NIH) collaborated on the Healthy People 2010 objectives (CDC and NIH, 2000). Osteoporosis was one of the focus areas of Healthy People 2010. Along with objectives to reduce the prevalence of osteoporosis and fractures, baseline prevalence data was given. The prevalence data for number of cases of osteoporosis was taken from the Third National Health and Nutrition Examination (NHANES III). The prevalence data for hospitalizations relating to osteoporosis was taken from to the National Hospital Discharge Survey (NHDS).

Prevalence by sex

According to CDC and NIH (2000), approximately 16% of women and 3% of men over the age of 50 have osteoporosis as measured by low BMD. Although prevalence rates by sex differ widely, the number of hospitalizations for vertebral fractures, per 10,000 people aged 65 years and older were very similar; 15.2 for women and 13.0 for men. The number of hip fractures, per 100,000 people aged 65 years and older, were 1120.9 for women and 563.1 for men.

Prevalence by race

According to CDC and NIH (2000), the prevalence of osteoporosis, as measured by low BMD, for adults aged 50 years and older by race is as follows: 7% for blacks or African Americans, 10% for whites, and 10% for Mexican Americans. The number of hospitalizations for vertebral fractures, per 10,000 people aged 65 years and older, according to NHDS in the Healthy People 2010 (CDC and NIH, 2000), was 6.5 for blacks or African American, 11.1 for whites, but the data were statistically unreliable for Mexican Americans.

The number of hip fractures, per 100,000 people aged 65 years and older, according to NHDS (CDC and NIH, 2000), were 492.0 for black or African American women, 932.1 for white women, and 469.4 for white men. The data for black or African American men were statistically unreliable.

Prevalence by age

As age increases, the hospitalization rates for vertebral fractures also increases. According to the NHDS in Healthy People 2010 (CDC and NIH, 2000), hospitalization rates were 5.5 per 10,000 people for male and female adults aged 65-74 years; 16.5 per 10,000 for those aged 75-84 years; and 47.6 per 10,000 for those aged 85 years and older.

Demographic and Lifestyle Factors that Influence Bone Mineral Density

Family history and bone mineral density

Genetics may influence up to 70% of BMD (Sambrook et al., 1996). A family history of osteoporosis can affect calcium deposition, calcium resorption, and BMD. O'Brien et al. (1998) studied the relation between family histories of osteoporosis and calcium intake in girls and women. Females without family histories of osteoporosis had less bone calcium resorption, but only small positive changes in bone calcium deposition with high calcium intakes. On the other hand, females with family histories of osteoporosis had more bone calcium deposition and resorption with high calcium intakes, so they did not have positive changes in bone calcium deposition. In addition, O'Brien et al. (1998) found the total body BMD in the mothers with a family history of osteoporosis was significantly lower than in the mothers without a family history of osteoporosis ($p<0.05$). Other researchers determined that a family history of osteoporosis was negatively correlated with BMD ($p<0.05$) (Rubin et al., 1999).

Sex and bone mineral density

Much of the osteoporosis research was conducted in females (Anai et al., 1996; Black et al., 1992; Cadogan et al., 1997; Clark and Sowers, 1996; Cumming and Nevitt, 1997; Etherington et al., 1996; Faulkner et al., 1993; Franceschi et al., 1996). Fewer studies included males, in addition to females (Dennison et al., 1998; Egger et al., 1996; Felson et al., 1995). This may be due

to the fact that a 3 times more females had osteoporosis than did males (CDC and NIH, 2000).

Race and bone mineral density

Much of osteoporosis research was conducted with non-Hispanic whites (Cadogan et al., 1997; Clark and Sowers, 1996; Laitinen et al., 1993; Lloyd et al., 1993; Mazess and Barden, 1991; O'Brien et al., 1998; Rubin et al., 1999). However, preliminary information from the National Osteoporosis Risk Assessment (NORA) suggests that minority women (Hispanics, Asians, Native Americans and blacks) are at higher risk for osteoporosis than previously thought (Anonymous, 1999). Over 50% of each of these groups of minority women have a BMD below 1 standard deviation of the reference mean, which places them at risk for osteoporosis.

In a longitudinal study, Dennison et al. (1998) investigated elderly men and women in Britain and Japan. At baseline, British women had significantly higher BMD at the trochanter (0.65 g/cm^2) and lumbar spine (0.92 g/cm^2), as measured by DEXA, than did Japanese women (0.50 g/cm^2 and 0.78 g/cm^2, respectively). However, British women had significantly greater bone loss rates in the trochanter and femoral neck than did Japanese women.

Age and bone mineral density

BMD changes differently with age at various bone sites. If an individual has attained peak bone mass as an adult, then eventually BMD will only

decrease from that point forward and any factor that inhibits that bone loss is beneficial. A decrease in BMD eventually occurred in all individuals as a result of age, so any factor that inhibits optimum BMD will also increase the chance of osteoporosis as the individual ages (Masi and Bilezikian, 1997). Mazess and Barden (1991) found that femoral neck BMD, as measured by DPA, negatively correlated with age when they studied 200-300 women aged 20-39 years ($p<0.05$). However, the differences in femoral neck BMD were not significant among the four age groups (20-24, 25-29, 30-34, 35-39) (Mazess and Barden, 1991).

Once again, BMD changes occur differently depending on the bone site and age. Age positively correlated with lumbar spine BMD, but age negatively correlated with femoral neck BMD in females aged 18-35 ($p<0.05$) (Rubin et al., 1999). Guthrie et al. (1996) studied 167 women, 46 to 57 years of age, to determine the association of various factors and BMD, as measured by DEXA. These women were either premenopausal, perimenopausal, or postmenopausal. Researchers used multivariate analyses to determine that femoral neck BMD was negatively correlated with age for all women, but lumbar spine BMD was negatively correlated with menopausal status for all women and with age for those women in perimenopause.

Body mass index and bone mineral density

There is an overwhelming positive correlation between BMI and BMD. Orozco and Nolla (1997) studied 72 premenopausal women aged 42 years to

see if body morphology was related to BMD as measured by DEXA. Weight and BMI was positively correlated to BMD at the intertrochanter and the total femur ($p<0.05$) (Orozco and Nolla, 1997). Weight was also positively associated with femoral neck BMD ($p<0.05$) (Orozco and Nolla, 1997). According to Franceschi et al. (1996), BMI strongly determined lumbar spine BMD, as measured by DPA, in the 733 premenopausal women they studied. Additional research involving Caucasian women 18-35 years old indicated that height and weight were positively associated with BMD in the femoral neck and lumbar spine ($p<0.05$) (Rubin et al., 1999). Slemenda et al. (1990) noted that in 124 perimenopausal women with a mean age of 50.8 years, weight was positively related to BMD in the femoral neck, lumbar spine, and the radius, as measured by SPA and DPA ($p<0.05$).

BMI is positively associated with BMD in women of all ages. Michaelsson et al. (1996) chose a cross-sectional random sample of 175 women aged 28-74 years to study the correlation of body measurements to BMD. There was a positive correlation between weight and BMD, height and BMD, and BMI and BMD at the lumbar spine, femoral neck, and total body ($p<0.05$). In the Dubbo Osteoporosis Epidemiology Study (DOES), Nguyen et al. (2000) related the body mass index of 1075 women with a mean age of 69.4 years to their BMD measurements. There was a positive linear relation between BMI and femoral neck and lumbar spine BMD in these women (Nguyen et al., 2000).

Even in women who have confounding factors, such as being smokers or being postpartum, BMI is positively correlated with BMD. Premenopausal

smokers with a BMI of less than 25 kg/m^2 had a lower BMD than did smokers with a higher BMI ($p<0.05$) (Jones and Scott, 1999). Anai et al. (1996) found that both higher weight and higher BMI in 126 postpartum Japanese women correlated significantly with lumbar spine BMD ($p<0.05$), as measured by DEXA.

Income and bone mineral density

With all the osteoporosis research being performed, there is very little, if any, that included information about income. Within this literature search, there was no article that directly related income to BMD. However, the researchers in the current study speculated that income has intervening variables between it and BMD because income might have affected nutrient intake, physical activity, and BMI (Kurinij et al., 1986; Diez-Roux et al., 1999; Pomerleau et al., 1997; Kahn et al., 1991).

Physical activity and bone mineral density

Few researchers found that physical activity has no effect on BMD. Mazess and Barden (1991) studied 200-300 women aged 20-39 years and found no significant relation between physical activity, as measured by accelerometer or pedometer, and BMD of the femur, spine, or radius, as measured by SPA and DPA. However, many researchers found that either overall or weight bearing physical activity positively influences BMD (Etherington et al., 1996; Rubin et al., 1999; Uusi-Rasi et al., 1998; Lohman et al., 1995).

Overall physical activity positively influenced BMD. Research involving Caucasian women 18-35 years old indicated that overall physical activity level was positively associated with BMD in the femoral neck and lumbar spine ($p<0.05$) (Rubin et al., 1999). In a cross-sectional study of 422 women aged 25 to 65 years of age, a higher level of exercise was positively associated with femoral neck BMD, as measured by DEXA ($p<0.05$) (Uusi-Rasi et al., 1998). In the Dubbo Osteoporosis Epidemiology Study (DOES), Nguyen et al. (2000) related the overall physical activity of 1075 women with a mean age of 69.4 years to their BMD, as measured by DEXA. Researchers found that the women's physical activity index was positively associated with lumbar spine and femoral neck BMD ($p<0.05$), even when adjusting for age.

Physical activity, as manifested as competitive sports participation, also positively affected BMD. When researchers studied smoking, sports participation, and BMD, they found that smokers who played competitive sports had greater BMD ($p<0.05$) than did smokers who did not play sports (Jones and Scott, 1999). Nonsmokers who played competitive sports had a greater BMD than did nonsmokers who did not play competitive sports, but the difference was not significant.

Not only can physical activity increase BMD, but it can also help prevent the risk of osteoporotic fractures because of the higher BMD. Turner et al. (1998) analyzed data from Phase 1 of NHANES III to determine the importance of physical activity in prevention of osteoporosis. All activity levels with a frequency of 2 or more times per week were considered "physical activity".

Among women over 50 years of age, physical inactivity had an odds ratio of 1.838 ($p<0.05$). An odds ratio of 1.838 means that a women who is not physically active at least two times per week has over an 80% higher risk for osteoporotic fracture than does a women who is physically active at least two times per week.

Several meta-analyses have related physical activity and osteoporosis. A meta-analysis performed on randomized trials by Berard et al. (1997) showed that for postmenopausal women, aged 50 years and older, physical activity without additional drug or supplement therapy helped prevent BMD loss at the lumbar spine, but not at the forearm or proximal femur. Wolff et al. (1999) performed a meta-analysis on both randomized and non-randomized trials for both pre- and post-menopausal women using not only exercise treatments, but also calcium supplementation or hormone replacement therapy (HRT). Despite the limitations of having the effect of the exercise treatment confounded by also including calcium supplementation or HRT, the researchers determined that exercise programs might slightly inhibit or reverse loss of BMD at both the lumbar spine and the femoral neck in both pre- and post-menopausal women.

In addition to overall physical activity, weight bearing physical activity is also associated with increased BMD. Etherington et al. (1996) determined that long-term weight bearing exercise is important for BMD. Researchers retrospectively studied the BMD of the lumbar spine, femoral neck, and forearm, as measured by DEXA, along with activity levels of 83 ex-elite female athletes and 585 female controls, who were not ex-elite athletes, matched for age. The

controls were separated by level of activity: active, moderately active, and inactive. The athletes had significantly higher femoral neck BMD than did the controls, the active controls had significantly higher femoral neck BMD than did the inactive controls, and the moderately active controls had a significantly higher femoral neck BMD than did the inactive controls.

Other researchers focused their attention on "non-athletes" to see the association between weight bearing exercise and BMD. Lohman et al. (1995) studied the effects of resistance training on BMD in women aged 28-39 years. In addition to the randomized 18-month strength-training program, all subjects consumed a supplement containing 500 mg calcium per day. The exercise group performed 12 weight lifting exercises with 3 sets of 8-12 repetitions approximately 3 days per week. BMD (g/cm^2) for the exercise group significantly increased from baseline in the lumbar spine at 5 and 12 months and the femur trochanter at 5, 12, and 18 months ($p<0.05$).

Smoking and bone mineral density

There is a general consensus that smoking is negatively correlated with BMD. Premenopausal female smokers and nonsmokers were studied to determine the associations between BMD and other variables (Jones and Scott, 1999). Nonsmokers had a significantly greater femoral neck, lumbar spine, and total body BMD than did smokers ($p<0.05$). Franceschi et al. (1996) studied 733 premenopausal women to determine the relation between smoking and other factors and BMD. The outcome variable was lumbar spine BMD as measured by

dual photon absorptiometry (DPA). In this study, smokers who smoked more than 15 cigarettes per day had significantly lower BMD than did women who had never smoked (Franceschi et al., 1996). Mazess and Barden (1991) studied 200-300 women aged 20-39 years. Smokers in the Mazess and Barden (1991) study had significantly lower spine BMD, as measured by DPA, than did nonsmokers ($p<0.05$). BMD as measured by SPA and DPA, at the femur, humerus, and radius was not significantly lower for smokers than for nonsmokers.

Not only is smoking detrimental to BMD in premenopausal women, but also in postmenopausal women. Egger et al. (1996) studied 186 women between the ages of 63 and 73 years to see if there was an association between smoking and lumbar spine and femoral neck BMD, as measured by DEXA. There was a non-significant negative correlation between smoking status and BMD at both measured sites. Kiel et al. (1996) used data from the Framingham Study to evaluate the effect of smoking on BMD, as measured by SPA and DPA, in subjects 68-98 years of age. After adjusting for age, weight, caffeine intake, alcohol consumption, and years of estrogen use, current smokers who had used estrogen had significantly lower BMD at the trochanter and radius than did women who had used estrogen and never smoked ($p<0.05$).

The mechanism by which smoking is related to lowered BMD may be through calcium absorption. Smoking decreased calcium absorption, which, in turn, affected BMD (Krall and Dawson-Hughes, 1999). Elderly smokers aged 65 and over had lower calcium absorption than did nonsmokers, after adjusting for

age, sex, dietary calcium and vitamin D intakes, and supplementation. A significant reduction in femoral neck and total body BMD ($p<0.05$) was hastened by inefficient calcium absorption related to smoking.

While many studies show a negative correlation between smoking and BMD, others show no effect of smoking on BMD. Law and Hackshaw (1997) performed a meta-analysis of 29 cross-sectional studies, 13 of which included premenopausal women. In these studies, BMD was measured at the femoral neck, radius, and calcaneus. In the studies including primarily premenopausal women, there is no significant difference in BMD between smokers and nonsmokers. Kiel et al. (1996) used data from the Framingham Study to evaluate the effect of smoking on BMD, as measured by SPA and DPA, in subjects 68-98 years of age. In current smokers who had not used estrogen, smoking had no significant effect on BMD at any measured site.

Nutrient Intake Influences on Bone Mineral Density

Calcium, from food and supplements, and bone mineral density

In order for peak bone mass to be reached, calcium intake must be adequate during the formative years of bone growth (Lloyd et al., 1993). In a double blind, randomized, 18-month calcium supplement study of 94 girls with a mean age of 11.8 years, Lloyd et al. (1993) found calcium intake was positively associated with total body BMD and lumbar spine BMD. In this study, the supplement group consumed approximately 350 mg of calcium per day more than did the control group. In addition, the supplement group gained significantly

more bone, in both the lumbar spine and total body, than did the control group ($p<0.05$).

Cadogan et al. (1997) studied 82 white girls with a mean age of 12.2 years to determine if milk supplementation affected total body bone mineral gain. The group that received on average an additional half-pint of milk per day had a calcium intake of 1125 mg/day versus the group that consumed a normal diet whom had a calcium intake of 703 mg/day. The 44 girls in the milk group gained significantly more total body BMD than did the 38 girls in the control group ($p<0.05$). In addition, the girls in the milk group had significantly greater increases in BMD in the pelvis and leg regions ($p<0.05$).

Although it is well known that calcium is needed during the formative years of bone growth, in adult women the importance of calcium intake in relation to BMD is controversial. Some studies indicated that in adult women calcium intake does not correlate with BMD (Kardinaal et al., 1999; Uusi-Rasi et al., 1998). Kardinaal et al. (1999) studied 375 European women aged 20-23 years stratified into quartiles of calcium intake. Quartile 1 was 78-637 mg/day; quartile 2 was 638-875 mg/day; quartile 3 was 876-1238 mg/day; and quartile 4 was 1239-3384 mg/day. Kardinaal et al. (1999) determined that calcium intake was not significantly related to radius BMD. In a cross-sectional study of 422 women aged 25 to 65 years of age, a higher level of calcium intake was not associated with BMD in the femoral neck (Uusi-Rasi et al., 1998). Uusi-Rasi et al. (1998) separated the subjects into high calcium intake, with a mean of 1475 mg/day, and low calcium intake, with a mean of 638 mg/day.

Other researchers (Ulrich et al., 1996; Teegarden et al., 1999) determined lifelong milk consumption did not correlate with BMD. Ulrich et al. (1996) studied 25 mother (mean age 72 years)-daughter (mean age 41 years) pairs to determine the effect of lifelong milk consumption and calcium intake from supplements on axial and peripheral BMD, as measured by DEXA. Total calcium intake was not reported, but calcium intake from supplements over the past two years was 640 mg/day for the mothers and 214 mg/day for the daughters. Ulrich et al. (1996) found no significant relation between milk consumption and BMD or calcium intake from supplements and BMD. Teegarden et al. (1999) also found no significant relation between milk consumption during childhood and adolescence and BMD. Teegarden et al. (1999) asked 224 women aged 18-31 years to place their milk consumption during childhood and adolescence into three categories: infrequent or never, sometimes, and at every or almost every meal. Total body, lumbar spine, femoral neck, and radius BMD were measured. Previous milk consumption correlated significantly with total body and radius BMD ($p<0.05$), but not lumbar spine or femoral neck BMD (Teegarden et al., 1999).

Other studies, however, indicated that calcium intake in adult women positively correlated with BMD (Rubin et al., 1999; Nguyen et al., 2000). Rubin et al. (1999) suggested that increased calcium intake (separated into quartiles of ≤250, 251-500, 501-750, and >750 mg/day) was associated (using Chi-square analysis) with a higher femoral neck BMD ($p<0.05$). Higher calcium intake was associated with higher lumbar spine BMD, but the relation was not significant

($p=0.08$). In the Dubbo Osteoporosis Epidemiology Study (DOES), Nguyen et al. (2000) related the dietary calcium intake of 1075 women with a mean age of 69.4 years to their BMD measurements. The women had a mean calcium intake of 642 mg/day. The subjects' dietary calcium intake was positively correlated with femoral neck BMD ($p<0.05$), but was not correlated significantly with the lumbar spine BMD (Nguyen et al., 2000).

In a study of 200-300 women aged 20-39 years, there was a significant difference in calcium intake among each of four age groups (Mazess and Barden, 1991). The calcium intake for the 20-24 year old group was 1030 mg; for the 25-29 year old group, 925 mg; for the 30-34 year old group, 857 mg; and for the 35-39 year old group, 848 mg. Mazess and Barden (1991) found no significant relation between calcium intake and BMD at the femur, spine, humerus, or radius as measured by DPA and SPA. However, when researchers grouped calcium intake by quartiles, there was a non-significant trend for higher BMD at all sites for the grouped quartiles 2-4 (mean calcium intake of 1047 mg) as compared to quartile 1 alone (mean calcium intake of 510 mg).

Other researchers determined that lifelong milk consumption positively correlated with BMD. Murphy et al. (1994) studied 284 females aged 44-74 years to determine the effect of lifelong milk consumption on BMD. The outcome variables were BMD at the lumbar spine, total femur, femoral neck, trochanter, intertrochanter, and Ward's triangle. Murphy et al. (1994) asked subjects to place their previous milk consumption before age 25 into three categories: less than one glass per week, more than one glass per week but less than one glass

per day, or at least one glass per day. Murphy et al. (1994) determined that milk consumption up to age 25 accounted for 1.5-2% of the explained variance in the BMD for all hip sites (total femur, femoral neck, trochanter, intertrochanter, and Ward's triangle) ($p<0.05$).

Other researchers determined that calcium is important in decreasing hip fracture risk. Cumming and Nevitt (1997) performed a meta-analysis of epidemiological studies of dietary calcium intake and hip fracture outcomes in postmenopausal women. The researchers calculated an odds ratio of 0.96 per 300 mg/day increase in dietary calcium, which corresponds to about a 4% decrease in hip fracture risk per 300 mg/day increase in dietary calcium.

Calcium Dietary Reference Intakes

According to the Dietary Reference Intakes (DRI), the recommended calcium intake for 19-50 year old women is 1000 mg per day (Food and Nutrition Board, 1997). However, the 1994 NIH Consensus Panel recommended for women over 25 years of age, depending on their menopausal status and hormone replacement therapy status, to consume 1000-1500 mg of calcium per day. However, it is doubtful that calcium intake alone can prevent age-related bone loss (Masi and Bilezikian, 1997).

Alcohol and bone mineral density

There is some debate if alcohol is correlated with BMD. Some researchers found no difference in BMD between alcohol dependent women and

those who were not alcohol dependent. Clark and Sowers (1996) studied 50 white women in their late twenties: 25 alcohol dependent women and 25 controls matched on age, height, and weight. Femoral neck and lumbar spine BMD were significantly lower for the alcohol dependent women with a paired mean difference of 0.089 g/cm^2 and 0.103 g/cm^2, respectively ($p=0.05$). However, most of the alcohol dependent women also smoked, so the researchers controlled for smoking (in the 10 pairs of women who matched for smoking). When researchers controlled for smoking in the pairs of women, there were no significant differences in BMD at either measured site. Laitinen et al. (1993) studied 18 alcohol dependent non-cirrhotic women with a mean age of 38 years and 157 controls who were not alcohol dependent with a mean age of 37 years to determine if alcohol abuse was associated with BMD, as measured by DEXA. The alcohol dependent women consumed an average of 186 g of alcohol per day during their most recent drinking episode. Researchers found no statistically significant difference in the lumbar spine, femoral neck, or trochanter BMD between the alcohol dependent and women who were not alcohol dependent.

Other researchers found information that suggests that a moderate intake of alcohol is associated with higher BMD. Felson et al. (1995) used information from the Framingham Study to see if there was a correlation between alcohol use and BMD in men and women aged 68 to 96 years. Alcohol use was measured in ounces per week, averaged over the years 1967 to 1989, and put in four categories for women (<1 oz/week, 1-2 oz/week, 3-6 oz/week, ≥7 oz/week). Women who consumed more than seven ounces of alcohol per week had

significantly higher trochanter and lumbar spine BMD, after adjusting for age, weight, height, smoking, age at menopause, and estrogen use, than did women who consumed less than one ounce of alcohol per week ($p<0.05$). Holbrook and Barrett-Connor (1993) prospectively studied 267 women with a mean age of 59.8 years. Researchers divided alcohol intake into quartiles per week: none; low, 0.1-48.6 g; medium, 48.7-120.4 g; and high, ≥120.5 g. An average alcoholic beverage (12 oz beer, 4 oz wine, 1 oz liquor) contains approximately 10 g of alcohol (USDA, 1999). Holbrook and Barrett-Connor (1993) determined that increasing alcohol intake significantly increased lumbar spine BMD ($p<0.05$).

Other researchers related alcohol intake to the risk of fractures. Tuppurainen et al. (1995) prospectively studied 3140 women to determine the relation between possible risk factors and the incidence of fractures during a 2.4-year follow-up period. The women who reported fractures during this time had a significantly higher alcohol intake (49.9 g/week) than did women who reported no fractures (32.2 g/week) ($p<0.05$).

Summary of Literature Review

Osteoporosis is a major health concern in the U.S because prevalence of osteoporosis is high and may increase due to the aging of the baby boomer population, or those persons born 1946 to 1964 (Poulous and Smith Nightengale, 2000). Because BMD can help predict osteoporotic fractures, it is important to determine factors that influence BMD. Some factors that may influence BMD are family history of osteoporosis, race, sex, age, income, physical activity level, BMI,

smoking, calcium intake, and alcohol consumption. If health professionals know that these factors may be related to BMD, they can determine which clients or patients are at greater risk for developing osteoporosis and target those individuals for intervention.

CHAPTER III

METHODOLOGY

The purpose of this study was to determine the relation between bone mineral density, demographic variables, and health behaviors in premenopausal women. This study analyzed data from the Third National Health and Nutrition Examination Survey, 1988-94 (NHANES III). The unit of analysis for this study was an individual, and the time dimension was cross-sectional over all six years.

Sampling and Subjects

NHANES III target population was the U.S. population. However, the target population for this study was premenopausal women from 20 to 45 years of age. The sampling unit for the NHANES III study was the United States civilian non-institutionalized population aged 2 months and older. Young children (younger than 5 years old), older persons (60 years old and over), blacks and Mexican-Americans were over-sampled to contribute toward research for these populations. This survey can be generalized to the entire U.S. civilian non-institutionalized population aged 2 months and over. For this study only premenopausal women from 20 to 45 years of age were used for analysis.

Participants were asked questions about their dietary intake, smoking habits, and alcohol consumption that they may find sensitive. To resolve these ethical concerns, interviewers assured the participants that all of their responses

were held in strict confidence. Participants signed an informed consent form before participating in NHANES III.

The sampling frame for the NHANES III was the 1980 census and the 1990 census, and the sampling procedure was a stratified random sampling. NHANES III used a stratified multistage probability design. A nationally representative sample was drawn from the entire civilian, non-institutionalized population aged 2 months and older in the United States. First, 81 primary sampling units (PSU's), primarily individual counties or adjacent counties, were randomly selected and 89 locations within the PSU's were designated. The 89 survey locations were randomly assigned into 2 phases of equal sample size. From these PSU's, 2138 area segments, or housing blocks, were randomly chosen. From all the area segments selected, all households and eligible group quarters (such as dormitories) were listed and a random subsample of 106,000 households/dwelling units were interviewed to identify potential sample individuals living in the household. Within approximately 20,000 dwelling units/households that were chosen from the screening, a random subsample of 40,600 people was selected based on age, sex, race or ethnicity from all eligible individuals in the household. Of these 40,600 people who were selected, about 35,000 were actually interviewed and 30,100 were physically examined. For the present study, one woman per household was randomly selected if more than one woman per household met the selection criteria.

Exclusion criteria

Persons were excluded from this analysis that took drugs known to increase calcium loss. The drugs included phenytoin, phenobarbital, thyroid hormone, corticosteroids, methotrexate, cyclosporin, lithium, tetracycline, aluminum-containing antacids, heparin, and phenothiazine derivatives (Anderson, 2000). Other exclusion criteria for this study included males, pregnant or lactating females, presence of chronic diseases such as diabetes mellitus, and anyone taking estrogen replacement therapy, or thyroid replacement therapy (Hanna et al., 1998). Females over 45 years of age were excluded because they may have been entering menopause (Reid, 1999). Additional women were excluded if there were more than one female per household who met the inclusion criteria, so there would not be the confounding factor of related females. If a sample individual could not participate in the exam at the MEC, a home examination was scheduled. For this study, however, these individuals were excluded because they did not have bone densitometry measurements performed.

Data Collection

Westat, Incorporated collected the NHANES III data for the National Center for Health Statistics (NCHS). Approximately 10 days before the screening interviews began in the households in the PSU, a letter was mailed to each sample household to inform them that an interviewer would be contacting them soon. Screening interviews were administered to identify household

members, their sex, birthdate, age, ethnicity, race, and relationship to the head of household. Once sample individuals were selected based on the screening interviews, the interviewer administered the Household Adult Questionnaire to any sample adult living in the household in either English or Spanish. The Household Adult Questionnaire consisted of items relating to various health conditions, diet, exercise, tobacco use, vitamin/mineral supplementation, and medication use. The final part of the interview was the administration of the Family Questionnaire to an adult household member. Included in the Family Questionnaire were items relating to characteristics of the entire family such as educational levels, occupations, health insurance coverage, ethnicity, and family income.

After administering the various questionnaires, the interviewer explained the various components of the health examination, which would be conducted at the mobile examination center (MEC) on a future date. Once each sample individual had read the Sample Person Brochure and signed the consent form for the health examination, the interviewer scheduled an appointment for each sample individual to come to the MEC. A reminder notice was sent to the sample person several days before the scheduled health examination. In addition, on the day prior to the examination, a reminder telephone call was made to the sample person. If a sample individual could not participate in the exam at the MEC, a home examination was scheduled.

Once the sample individual arrived at the MEC, four types of data collection methods were used. A professional expert physically examined the

sample individual during the dental and physician's exams. Health technicians and ultrasonographers performed other tests and measurements, such as bone densitometry and body measurements. Interviewers collected nutrition-related information, data on sensitive issues, and tests of cognitive achievement. Phlebotomists and medical technicians performed the final method of data collection, blood and urine collection.

The nutrition-related information was important for this study because of the need for accurate estimates of nutrient intake. Interviewers collected 24-hour dietary recalls during face-to-face interviews using a computer based interviewing system, the NHANES III Dietary Data Collection (DDC) system. Sample individuals provided information about what foods were eaten, the time and place those foods were consumed, the type of meal or snack in which the foods were eaten, and how much of each food was consumed. The interviewer probed for exact names, brands, preparation methods, amounts of foods, and foods/beverages that are frequently forgotten.

Variable List

Bone mineral density (BMD) is the measurement of mass per area of bone to determine the density, or strength, of the bone. The unit of measurement for BMD was grams per centimeter squared (g/cm^2). BMD at four bone sites, femoral neck, total femur, intertrochanter, and trochanter, were used as outcome variables. Because of variations in measurement between the two phases of NHANES III, Ward's Triangle was not used for analysis in this study. Physical

activity level was calculated from the self-reported activities of the subject. Lifelong milk consumption, calcium supplementation, and alcohol use was determined from the subject's self-reported behaviors. Other nutrients besides calcium were not analyzed in relation to BMD because other researchers (Mazess and Barden, 1991) have determined that the intakes of 14 nutrients (phosphorus, protein, iron, magnesium, zinc, and energy, among others) did not significantly predict BMD at the lumbar spine, femoral neck, or radius.

Data processing procedures

BMI was calculated by dividing weight in kilograms by standing height in meters squared (BMI=kg/m^2) (National Center for Health Statistics, 1994).

Poverty income ratio (PIR), or poverty index, was a value computed by NHANES (National Center for Health Statistics, 1994). PIR was calculated by dividing the midpoint of the family income category by the poverty threshold the year in which the interview occurred and the family reference person's age. The family reference person was the person who responded to the family questionnaire. The poverty threshold values came from the Census Bureau and are adjusted annually for inflation. Using the PIR instead of annual income allows for comparisons across all six years of the NHANES. PIR data has a high potential for bias because of nonresponse.

A separate score was created for non-weight bearing exercise and weight bearing exercise. The non-weight bearing exercise score included activities such as walking, biking, swimming, gardening, and other activities deemed to be non-

weight bearing. The weight bearing exercise score included activities such as jogging, running, aerobics, dancing, calisthenics, lifting weights, and other activities deemed to be weight bearing. There were about 50 activities that were not in the general categories (e.g., walk, jog/run, bike, swim, aerobics, dance, calisthenics, garden, lift weights) specified in the questionnaire. Researchers based some decisions about the category (i.e. non-weight bearing or weight bearing) in which to place those 50 activities on the Etherington et al. (1996) article. After determining the category in which to place the extra activities, the number of times a respondent reported performing an activity in the past month was multiplied by the MET score. The MET score is a measure of how many mL of oxygen is consumed per kilogram body weight per minute of performing a specific exercise. The resulting number for each activity was added together in either the total weight bearing or the total non-weight bearing score. Each of the exercise scores were not an exact MET measure because subjects did not report the length of time that each activity was performed. However, the exercise scores still indicated the intensity of the activities.

Subjects were separated into nonsmoking versus smoking categories using their serum cotinine levels. Cotinine is a sensitive indicator of nicotine in the blood (Wallach, 2000). According to Wallach (2000), the level of cotinine for smokers is greater than 8 mcg/L. NHANES measured cotinine in ng/mL which converts directly to mcg/L (ng/mL=mcg/L). Since there was an abnormal distribution of cotinine levels that could not be corrected by transformations (such as log transformations, etc), the researchers in the current study recoded all

values less than and equal to 8 ng/mL as "nonsmoker", and all values more than 8 ng/mL as "smoker".

Dietary supplements containing calcium were only used in analysis when they had a certainty index of "1" or "2". A certainty index of "1" indicated the NCHS staff could exactly match the product to a specific supplement. A certainty index of "2" indicated the NCHS staff could match the product to a specific supplement with a reasonable degree of certainty. For each calcium-containing supplement, the frequency with which the respondent consumed the supplement over a one-month time period was multiplied by the quantity consumed each time the respondent took the supplement. The number calculated was an estimate of the total calcium from each supplement consumed per month. For respondents who took more than one calcium-containing supplement, the total calcium per month for each supplement was added together to obtain the total calcium per month for all supplements. Then, the resulting number was divided by 30 to get the calcium intake from supplements per day.

Each antacid consumed was studied to see if it contained calcium and if so, how much calcium it contained. Various sources were used to determine the amount of calcium contained in each antacid, including *Drug Facts and Comparisons 2000* (54th ed.), web sites (Tums), and personal correspondence with antacid manufacturers (Mylanta-Johnson & Johnson Merck). If the source only listed the calcium content as calcium carbonate, the amount of calcium carbonate was multiplied by 0.40 to determine the amount of elemental calcium (calcium carbonate is 40% elemental calcium). If the dose was not clearly

specified, the assumed dose was 2 teaspoons for a liquid dose, and 1 tablet/pill/piece for a solid dose. If a respondent consumed a "calcium antacid-type not specified", it was assumed to contain 500 mg of calcium carbonate. Assumptions of how much calcium each product contained were made for different types (i.e. extra strength, regular) of the same brand of antacid. For example, regular Rolaids contained 550 mg of calcium carbonate, so the assumption was made that Extra Strength Rolaids and Rolaids-type not specified also contained 550 mg of calcium carbonate. For those stating they consumed a certain brand but did not specify the type (i.e. liquid or solid), the unit of measure (i.e. teaspoon, tablet, etc.) was important in determining how much calcium the respondent consumed. For example, one respondent reported taking Mylanta-type not specified and reported the unit of measure as teaspoons. The assumption was made that a liquid product is usually measured in teaspoons and the liquid Mylanta did not contain any calcium, so the amount of calcium in that product for that respondent was changed to zero.

Once the amount of calcium was determined for each antacid consumed, the daily amount of calcium from antacids was calculated. This was done by multiplying the amount of elemental calcium in each antacid by how often that antacid was consumed in the past month, by how much antacid was taken each time, then dividing the resulting number by 30. Then the daily amount of calcium from supplements and the daily amount of calcium from antacids were added together to obtain a total amount of daily calcium from all non-food sources. For

convenience, the total amount of daily calcium from antacids and supplements was referred to as "calcium from supplements".

A lifelong milk consumption score was calculated using the responses from the four questions "How often did you drink any type of milk when you were a child, teenager, young adult, and middle-aged adult?" First, the categorical responses were recoded to place the categories in ascending amounts (0=never, 1=less than once per week, 2=once per week, 3=less than once per day but more than once per week, 4=once per day, and 5=more than once per day). Then each categorical response (0-5) was multiplied by the number of years in each stage of life. For example, if a woman was 37 years old, her childhood (ages 5-12) milk response was multiplied by eight and her teenage (ages 13-17) milk response was multiplied by five. Then her young adult (ages 18-35) milk response was multiplied by 18, and her middle-aged adult (ages 36-65) milk response was multiplied by two because she had only been in the middle-aged adult stage of life for two years. Then the products of each multiplication were added together and divided by the respondent's age minus four (because there was not a question about the stage of life during the first four years of life). This final lifelong milk consumption score was related back to the original categories, so if there was a milk consumption of 3.87, that would mean that the respondent drank milk almost once per day (category 4) on average over a lifetime.

Alcohol consumption was manipulated to report it as drinks per year. This number was calculated by multiplying the number of days in the past 12 months that the respondent consumed alcoholic beverages times the average number of

drinks consumed on those days. A drink was considered either 12 ounces of beer, 4 ounces of wine, or 1 ounce of liquor.

The completed data set for this study included data from the household adult questionnaire data file, the examination data file, and the total nutrient data file. Demographic variables, such as age, sex, race, ethnicity, and poverty income ratio, were included from the household adult questionnaire data file. Other questions pertaining to this study included in this data file relate to disease conditions, vitamin and mineral supplementation, prescription medicine use, exercise behaviors, lifelong milk consumption, and tobacco use. Body measurements, specifically body mass index, weight, and height; and bone densitometry values were obtained from the examination data file. Information on specific intakes for nutrients such as calcium, vitamin D, and phosphorus was selected from the total nutrient data file. Cotinine levels were included from the laboratory data file. Then exclusion criteria were applied to all subjects, which resulted in a final sample for analysis.

Statistical Analysis

The data were analyzed using the Statistical Package for the Social Sciences (SPSS, Inc., 1998, Chicago, IL), version 9.0. NHANES III data were weighted to adjust for the unequal probability of selection because of over-sampling of certain groups and for non-response rates. Preliminary analyses were performed without the weights; however, all final analyses incorporated the weights using WesVar (Westat, 1997, Rockville, MD), version 2.12, and weights

using SPSS. Descriptives, such as frequencies, characterized the demographics of the sample. Analysis of variance (ANOVA) determined significance using Scheffe's post hoc test for the differences in BMD by race/ethnicity. Correlation analysis determined the relation of demographic variables and lifestyle behaviors to bone mineral density. Researchers checked for multicollinearity among the independent variables and excluded any variables where Pearson's r was greater than 0.4. However, no variables had a Pearson's r greater than 0.4, so none were excluded from analysis. Preliminary forward selection stepwise multiple regression analysis was performed using weights in SPSS. Then in WesVar, backward elimination stepwise multiple regression analysis was used to determine which variables best predict optimum bone mineral density at each of the four bone sites: femoral neck, intertrochanter, total femur, and trochanter. Backward elimination was done by performing the multiple regression analysis with all the variables of interest, then selecting the least significant variable to eliminate. This was done several times until the only variables remaining were significant. All results were presented as means ± standard error of the mean (SEM).

Research Questions and Hypotheses

Note: BMD is the dependent variable, all others are independent or mediating variables.

1. What is the relation between race/ethnicity and BMD?

    1HO: There will be no relation between race/ethnicity and BMD.

1HA: Blacks will have higher BMD than other ethnic groups.

2. What is the relation between age and BMD?

   2HO: There will be no relation between age and BMD.

   2HA: As age increases, BMD will decrease.

3. What is the relation between body mass index (BMI) and BMD?

   3HO: There will be no relation between BMI and BMD.

   3HA: As BMI increases, BMD will increase.

4. What is the relation between poverty income ratio (PIR) and BMD?

   4HO: There will be no relation between PIR and BMD.

   4HA: As PIR increases, BMD will increase.

5. What is the relation between non-weight bearing exercise status and BMD?

   5HO: There will be no relation between non-weight bearing exercise status and BMD.

   5HA: As non-weight bearing exercise status increases, BMD will increase.

6. What is the relation between weight bearing exercise status and BMD?

   6HO: There will be no relation between weight bearing exercise status and BMD.

   6HA: As weight bearing exercise status increases, BMD will increase.

7. What is the relation between smoking status and BMD?

   7HO: There will be no relation between smoking status and BMD.

7HA: Smokers will have lower BMD than nonsmokers.

8. What is the relation between calcium intake from food and BMD?

   8HO: There will be no relation between calcium intake from food and BMD.

   8HA: As calcium intake from food increases, BMD will increase.

9. What is the relation between calcium intake from supplements and BMD?

   9HO: There will be no relation between calcium intake from supplements and BMD.

   9HA: As calcium intake from supplements increases, BMD will increase.

10. What is the relation between lifelong milk consumption and BMD?

   10HO: There will be no relation between lifelong milk consumption and BMD.

   10HA: As the amount of milk consumed over a lifetime increases, BMD will increase.

11. What is the relation between alcohol intake and BMD?

   11HO: There will be no relation between alcohol intake and BMD.

   11HA: As alcohol intake increases, BMD will increase.

12. What are the predictors of BMD in premenopausal women?

   12HO: None of the variables (race/ethnicity, age, BMI, PIR, non-weight bearing exercise, weight bearing exercise, smoking, calcium intake from food, calcium intake from supplements,

lifelong milk consumption, or alcohol intake) will predict BMD.

12HA: Any of the variables above will predict BMD.

CHAPTER IV

RESULTS

Table 1 summarizes the demographic/lifestyle characteristics of the sample. The women in this study were about 34 years old, with a body mass index (BMI) of approximately 26. The reported income using PIR was relatively high, about 2 ½ times the poverty index. The sample had non-weight bearing and weight bearing exercise scores of approximately 50 and 40, respectively. Descriptions of these exercise scores are located in Chapter III "Methodology". The mean cotinine level for the women was about 86 ng/mL. The women had a low calcium intake from food and supplements, almost 700 mg from food and 40 mg from supplements. The lifelong milk consumption score for the sample was 3.47 ± 0.04, which corresponds to the midpoint between the category of "I drank milk less than once per day but more than once per week" and the category of "I drank milk once per day" in a lifetime. The women consumed approximately 1860 kilocalories. The mean alcohol consumption was low at about 83 drinks per year.

The women in this study had a mean bone mineral density of 0.842 ± 0.004 g/cm^2 at the femoral neck and 1.103 ± 0.005 g/cm^2 at the intertrochanter (Table 2). Mean BMD was 0.940 ± 0.004 g/cm^2 at the total femur and 0.700 ± 0.003 g/cm^2 at the trochanter. Table 2 summarizes the BMD ranges for the sample in comparison to reference ranges (Looker et al., 1997). Some subjects could be defined as having either osteopenia (11.8-15.5% of the sample,

depending on the bone site) or osteoporosis (0.1-0.5% of the sample, depending on the bone site), in that a value below the lowest BMD cutoff for osteopenia is considered a diagnosis for osteoporosis (World Health Organization, 1994).

Using population weights changed the race/ethnicity characteristics of the study and is summarized in Table 3. After using the population-based weights, the final demographic percents were similar to the U.S. population (US Census Bureau, 2000).

Table 4 summarizes selected characteristics of the sample by race/ethnicity. The data in Table 4 were not statistically analyzed among the race/ethnic groups because the characteristics were not the focus of this study. Average age was similar across all groups. BMI for all race/ethnic groups was between 25-28. Income varied among the groups, with a low of 147% of PIR for Mexican Americans and a high of 275% of PIR for whites. Non-weight bearing exercise scores ranged from near 30 for blacks and Mexican Americans to about 60 for whites. However, weight bearing exercise scores were highest for blacks at approximately 47 and lowest for Mexican Americans at near 30. Average cotinine levels were lowest for Mexican Americans at 17 and highest for whites and blacks at about 97. The "other" group and blacks tended to have the lowest calcium intake from food; while Mexican Americans and whites tended to have higher calcium intakes from food. Calcium intake from supplements was between 20-50 mg for all race/ethnic groups. Lifelong milk consumption scores were lowest for the "other" group at about 2.5, while whites, blacks, and Mexican Americans had scores around 3.5. Average alcoholic drinks per year varied from

the lowest for "others" of 30 drinks per year to the highest for whites and blacks of 90 and 88 drinks per year, respectively.

As women became older, a smaller percentage reported drinking milk more often than once per day and a larger percentage reported never drinking milk (Table 5). Approximately 50% reported they drank milk more than once per day during childhood (5-12 years), but about 10% reported they drank milk at that frequency during middle-aged adult years (36-45 years). About 8% reported they drank milk once per week or less during childhood (5-12 years) compared to approximately 39% during middle-aged adult years (36-45 years).

The majority of women reported doing some type of exercise (Table 6). About 50% reported doing some weight bearing exercise, whereas almost 75% reported performing some non-weight bearing exercise.

Women who exercised were significantly different from women who did not exercise for all demographic/lifestyle characteristics ($p<0.05$) (Table 7). Women who exercised were older, had a lower BMI, and had a higher income than women who did not exercise. The cotinine level for women who performed no exercise was higher than for women who performed some exercise. Women who exercised had a higher calcium intake from food and from supplements than women who did not exercise. In addition, women who exercised had a higher lifelong milk consumption score than women who did not exercise. The number of alcoholic drinks per year for women who performed no exercise was lower than for women who performed some exercise.

Women who performed non-weight bearing exercise were significantly different from women who did not perform non-weight bearing exercise for all demographic/lifestyle characteristics ($p<0.05$) (Table 8). Women who performed some non-weight bearing exercise were older, had a lower BMI, and had a higher income than women who did not perform non-weight bearing exercise. Women who performed some non-weight bearing exercise had a significantly higher weight bearing exercise score than women who did not perform non-weight bearing exercise. The cotinine level for women who did not perform non-weight bearing exercise was higher than for women who performed some non-weight bearing exercise. Women who performed some non-weight bearing exercise had a higher calcium intake from food, calcium intake from supplements, and lifelong milk consumption score than women who did not perform non-weight bearing exercise. The number of alcoholic drinks per year for women who did not perform non-weight bearing exercise was lower than for women who performed some non-weight bearing exercise.

Women who performed weight bearing exercise were significantly different from women who did not perform weight bearing exercise for all demographic/lifestyle characteristics ($p<0.05$) (Table 9). Women who performed weight bearing exercise were younger, had a lower BMI, and had a higher income than women who did not perform weight bearing exercise. Women who performed some weight bearing exercise had a significantly higher non-weight bearing exercise score than women who did not perform weight bearing exercise. The cotinine level was higher for women who performed no weight bearing

exercise was higher than for women who performed some weight bearing exercise. Women who performed some weight bearing exercise had a significantly higher calcium intake from food and from supplements than women who performed no weight bearing exercise. In addition, women who performed some weight bearing exercise had a higher lifelong milk consumption score than women who did not perform weight bearing exercise. The number of alcoholic drinks per year for women who performed no weight bearing exercise was lower than for women who performed some weight bearing exercise.

Table 10 summarizes selected characteristics of the sample according to smoking status as defined by blood cotinine levels. Smokers were significantly different from nonsmokers for all demographic/lifestyle characteristics ($p<0.05$). Nonsmokers were older, had a higher BMI, and had a higher income than smokers. Nonsmokers had significantly higher non-weight bearing and weight bearing exercise scores than did smokers. Smokers had higher cotinine levels than did nonsmokers. Nonsmokers consumed more calcium from food and from supplements than did smokers. Smokers had a higher lifelong milk consumption score than did nonsmokers. Smokers consumed more alcoholic drinks per year than did nonsmokers.

Table 11 displays the mean bone mineral density (BMD) for each race/ethnic group. BMD at the femoral neck, the intertrochanter, the total femur, and the trochanter varied according to race/ethnicity. At all four bone sites, blacks had a significantly higher BMD than all other race/ethnic groups ($p<0.05$). Mexican Americans also had a significantly higher BMD at the femoral neck than

did whites and others ($p<0.05$). The intertrochanter BMD of whites did not differ significantly from Mexican Americans or the "other" group, but the intertrochanter BMD of Mexican Americans was significantly higher than that of the "other" group ($p<0.05$). The total femur BMD of Mexican Americans and whites was not significantly different from each other, but they both were significantly higher than the total femur BMD of the "other" group ($p<0.05$). The trochanter BMD of Mexican Americans and whites was not significantly different from each other, but they both were significantly higher than the trochanter BMD of the "other" group ($p<0.05$).

Table 12 summarizes the BMD range for each race/ethnic group. For BMD at all sites, if the minimum value of the range was used, Mexican Americans had the lowest value, followed by whites, then blacks, and finally "others".

Tables 13 - 15 show that the mean BMD for each bone site was significantly different due to exercise status, non-weight bearing exercise status, and weight bearing exercise status ($p<0.05$). The mean BMD at the femoral neck was higher for those who did not exercise than for those who did exercise (Table 13) and for those who did not do non-weight bearing exercise than for those who did non-weight bearing exercise (Table 14) ($p<0.025$). However, at all other bone sites, the reverse was true for the exercise and non-weight bearing exercise dichotomies. The mean BMD at all bone sites was significantly higher for the weight bearing exercise category than the no weight bearing exercise category (Table 15) ($p<0.025$).

Table 16 displays the differences in BMD by smoking status as defined by blood cotinine levels. At the femoral neck, intertrochanter, total femur, and trochanter, nonsmokers' BMD was significantly higher than smokers' BMD ($p<0.025$).

Table 17 shows correlations between demographic/lifestyle characteristics and BMD at four bone sites. Several demographic and lifestyle characteristics correlated significantly with BMD ($p<0.05$). Age correlated negatively with femoral neck BMD ($p<0.05$), however BMI correlated positively with BMD at all bone sites ($p<0.05$). Income correlated negatively with BMD at the total femur, the femoral neck, and the intertrochanter ($p<0.05$). Both non-weight bearing and weight bearing exercise scores positively correlated with intertrochanter, total femur, and trochanter BMD ($p<0.05$). Calcium intake from food or supplements did not significantly correlate with any bone sites, but lifelong milk consumption positively correlated with BMD at all bone sites ($p<0.05$). Alcohol intake did not significantly correlate with any bone site.

After controlling for all other variables in a stepwise multiple regression analysis, several variables were significantly associated with higher BMD at each of the bone sites (Table 18). The final model varied by bone site. Higher BMI was associated with higher BMD at all bone sites. The predictor of race was significantly associated with BMD at all bone sites. The partial regression coefficients for race were positive as the race "black" was coded "1" and all other races coded "0". Higher lifelong milk consumption scores were significantly associated with higher BMD at all bone sites, but the partial regression

coefficients were smaller than were those for BMI. Higher weight bearing exercise scores were significantly associated with higher BMD at all bone sites, but the partial regression coefficients were once again smaller than were those for BMI. Lower income was associated with higher BMD only at the femoral neck. Higher non-weight bearing exercise scores were associated with higher BMD at the total femur, trochanter, and the intertrochanter, but not at the femoral neck. Younger age was associated with higher BMD at the total femur, femoral neck, and intertrochanter, but not at the trochanter.

Table 1. Demographic/lifestyle characteristics of the sample[1].

Demographic/lifestyle characteristics	N	Mean	SEM
Age (y)	1892	33.6	0.2
BMI (kg/m^2)	1889	25.6	0.2
Poverty Income Ratio (%)	1740	248	5
Non-weight bearing exercise score	1892	49.5	2.8
Weight bearing exercise score	1892	40.1	2.7
Cotinine (ng/mL)	1809	86.42	4.97
Calcium from food (mg)	1859	692	15
Calcium from supplements (mg)	1892	40	4
Lifelong milk consumption score	1890	3.47	0.04
Calories (kcal)	1859	1859	26
Phosphorus (mg)	1859	1080	18
Vitamin D (mcg)	1859	3.9	0.2
Alcoholic drinks per year	1892	82.9	6.0

[1]Sample sizes refer to the number of unweighted observations in the data set.

Table 2. Bone mineral density ranges of the sample.

BMD (g/cm^2)	Mean ± SEM	Current Study	Reference Range[1]	BMD cutoff-Osteopenia[2]
Femoral Neck	0.842 ± 0.004	0.467-1.650	0.560-1.283	0.560-0.740
Intertrochanter	1.103 ± 0.005	0.696-1.921	0.717-1.588	0.740-0.950
Total Femur	0.940 ± 0.004	0.594-1.707	0.635-1.379	0.640-0.820
Trochanter	0.700 ± 0.003	0.422-1.441	0.480-1.051	0.460-0.610

[1]Mean BMD of 20-29 year-old non-Hispanic white women, NHANES III 1988-1994. From Table 1 in Looker et al., 1997.
[2]From Table 1 in Looker et al., 1997. BMD values less than the lowest value in the range were defined as osteoporosis.

Table 3. Sample size and percents[1] of race/ethnicity.

Race/Ethnicity	N	Percent
Non-Hispanic white	530	68.6
Non-Hispanic black	660	15.3
Mexican-American	614	6.8
Other (includes non-Mexican Hispanics)	88	9.3

[1]Sample sizes refer to the number of unweighted observations in the data set; percents reflect sample weights.

Table 4. Demographic/lifestyle characteristics[1] of the sample by race/ethnicity.

	White			Black		
Demographic/lifestyle characteristic	N	Mean	SEM[2]	N	Mean	SEM
Age (y)	530	33.9	0.2	660	32.4	0.2
BMI (kg/m^2)	530	24.9	0.3	660	27.9	0.3
Poverty Income Ratio (%)	510	275	6	609	173	7
Non-weight bearing exercise score	530	57.5	4.0	660	31.4	3.0
Weight bearing exercise score	530	39.2	3.7	660	47.0	4.1
Cotinine (ng/mL)	511	96.41	7.02	626	97.88	7.38
Calcium from food (mg)	522	738	21	643	546	17
Calcium from supplements (mg)	530	46	5	660	28	5
Lifelong milk consumption score	529	3.64	0.05	660	3.36	0.05
Alcoholic drinks per year	530	90.4	8.7	660	88.5	12.9

	Mexican American			Other		
Demographic/lifestyle characteristic	N	Mean	SEM	N	Mean	SEM
Age (y)	614	32.2	0.5	88	34.4	1.0
BMI (kg/m^2)	611	27.5	0.2	88	25.9	0.7
Poverty Income Ratio (%)	545	147	6	76	228	26
Non-weight bearing exercise score	614	31.2	3.0	88	33.8	8.3
Weight bearing exercise score	614	30.4	3.4	88	43.0	12.3
Cotinine (ng/mL)	587	17.19	3.36	85	44.72	13.37
Calcium from food (mg)	609	742	29	85	542	40
Calcium from supplements (mg)	614	24	6	88	21	6
Lifelong milk consumption score	613	3.37	0.05	88	2.54	0.16
Alcoholic drinks per year	614	65.9	14.1	88	30.8	10.4

[1]Sample sizes refer to the number of unweighted observations in the data set.
[2]Standard error of the mean with weights.

Table 5. Percent reporting[1] how often drank milk at different age groups, NHANES III, 1988-1994.

	Percent Reporting					
Age Groups	0[2]	1	2	3	4	5
5-12 y	3.9	2.4	1.6	9.4	30.7	51.9
13-17 y	7.2	4.2	4.5	17.2	32.5	34.4
18-35 y	8.6	10.2	7.3	26.9	27.9	19.0
36-45 y	13.5	13.5	11.7	24.2	26.5	10.6

[1]"Percent reporting" reflects sample weights.
[2]0=never, 1=less than once per week, 2=once per week, 3=less than once per day but more than once per week, 4=once per day, 5= more than once per day.

Table 6. Sample sizes and percents[1] of population that exercised.

	Yes		No	
	N	Percent	N	Percent
Any Exercise[2]	1452	84.3	440	15.7
Weight Bearing Exercise	888	50.8	1004	49.2
Non-Weight Bearing Exercise	1241	74.2	651	25.8

[1]Sample sizes refer to the number of unweighted observations in the data set; percents reflect sample weights.
[2]Any exercise refers to either weight bearing or non-weight bearing or both.

Table 7. Demographic/lifestyle characteristics[1] of the sample by exercise status.

Demographic/ lifestyle characteristic	Did Not Do Exercise- Any Kind			Did Some Exercise- Any Kind		
	N	**Mean**	**SEM**	**N**	**Mean**	**SEM**
Age (y)	440	32.6[2]	0.6	1452	33.8	0.2
BMI (kg/m^2)	440	26.0	0.5	1449	25.6	0.2
Poverty Income Ratio (%)	391	187	9	1349	258	5
Non-weight bearing exercise score	NA[3]	NA	NA	1452	58.7	3.0
Weight bearing exercise score	NA	NA	NA	1452	47.6	3.1
Cotinine (ng/mL)	420	100.56	13.04	1389	83.81	5.29
Calcium from food (mg)	435	654	33	1424	699	17
Calcium from supplements (mg)	440	23	6	1452	43	5
Lifelong milk consumption score	440	3.29	0.12	1450	3.51	0.05
Alcoholic drinks per year	440	64.8	14.0	1452	86.2	6.2

[1]Sample sizes refer to the number of unweighted observations in the data set.
[2]Means across rows were significantly different at $p<0.05$ using independent t-test and sample weights.
[3]NA=not applicable.

Table 8. Demographic/lifestyle characteristics[1] of the sample by non-weight bearing exercise status.

Demographic/ lifestyle characteristic	Did Not Do Non-Weight Bearing Exercise			Did Some Non-Weight Bearing Exercise		
	N	**Mean**	**SEM**	**N**	**Mean**	**SEM**
Age (y)	651	32.1[2]	0.4	1241	34.2	0.2
BMI (kg/m^2)	649	26.2	0.4	1240	25.4	0.2
Poverty Income Ratio (%)	591	191	8	1149	267	5
Non-weight bearing exercise score	651	NA[3]	NA	1241	66.7	3.1
Weight bearing exercise score	651	23.0	4.0	1241	46.1	3.4
Cotinine (ng/mL)	620	101.14	10.04	1189	81.36	5.53
Calcium from food (mg)	642	650	28	1217	706	18
Calcium from supplements (mg)	651	35	7	1241	41	6
Lifelong milk consumption score	651	3.34	0.09	1239	3.52	0.05
Alcoholic drinks per year	651	82.1	12.4	1241	83.2	7.1

[1]Sample sizes refer to the number of unweighted observations in the data set.
[2]Means across rows were significantly different at $p<0.05$ using independent t-test and sample weights.
[3]NA=not applicable.

Table 9. Demographic/lifestyle characteristics[1] of the sample by weight bearing exercise status.

Demographic/ lifestyle characteristic	Did Not Do Weight Bearing Exercise			Did Some Weight Bearing Exercise		
	N	**Mean**	**SEM**	**N**	**Mean**	**SEM**
Age (y)	1004	34.0[2]	0.3	888	33.3	0.3
BMI (kg/m^2)	1003	26.1	0.3	886	25.1	0.3
Poverty Income Ratio (%)	913	230	8	827	264	6
Non-weight bearing exercise score	1004	43.2	3.0	888	55.6	3.8
Weight bearing exercise score	1004	NA[3]	NA	888	78.9	4.7
Cotinine (ng/mL)	964	96.65	6.84	845	76.44	6.08
Calcium from food (mg)	985	672	22	874	710	21
Calcium from supplements (mg)	1004	27	4	888	52	7
Lifelong milk consumption score	1003	3.40	0.07	887	3.55	0.05
Alcoholic drinks per year	1004	80.9	9.4	888	84.8	6.8

[1]Sample sizes refer to the number of unweighted observations in the data set.
[2]Means across rows were significantly different at $p<0.05$ using independent t-test and sample weights.
[3]NA=not applicable.

Table 10. Demographic/lifestyle characteristics[1] of the sample by smoking status as defined by blood cotinine levels.

Demographic/ lifestyle characteristic	Nonsmokers			Smokers		
	N	Mean	SEM	N	Mean	SEM
Age (y)	1274	34.4[2]	0.2	535	32.3	0.3
BMI (kg/m^2)	1271	26.1	0.3	535	24.8	0.3
Poverty Income Ratio (%)	1157	269	7	509	209	9
Non-weight bearing exercise score	1274	49.9	2.8	535	48.7	5.0
Weight bearing exercise score	1274	45.9	3.7	535	28.7	3.6
Cotinine (ng/mL)	1274	0.46	0.04	535	246.84	6.24
Calcium from food (mg)	1253	713	18	524	657	35
Calcium from supplements (mg)	1274	42	6	535	36	5
Lifelong milk consumption score	1272	3.39	0.06	535	3.62	0.07
Alcoholic drinks per year	1274	60.0	5.2	535	128.4	12.9

[1]Sample sizes refer to the number of unweighted observations in the data set.
[2]Means across rows were significantly different at $p<0.05$ using independent t-test and sample weights.

Table 11. Bone mineral density means for each race/ethnic group.

	Femoral Neck BMD (g/cm^2)		Intertrochanter BMD (g/cm^2)		Total Femur BMD (g/cm^2)		Trochanter BMD (g/cm^2)	
	Mean	**SEM**	**Mean**	**SEM**	**Mean**	**SEM**	**Mean**	**SEM**
White	$0.824^{a,1}$	0.005	$1.086^{a,b}$	0.007	0.927^{a}	0.006	0.695^{a}	0.004
Black	0.932^{b}	0.007	1.198^{c}	0.009	1.018^{b}	0.007	0.747^{b}	0.006
Mexican American	0.872^{c}	0.007	1.126^{a}	0.008	0.958^{a}	0.007	0.702^{a}	0.006
Other	0.806^{a}	0.013	1.052^{b}	0.015	0.894^{c}	0.012	0.659^{c}	0.010

[1]Different letter superscripts indicate significant differences in the mean BMD at each site between race/ethnic groups at the $p<0.05$ level using weighted ANOVA and Scheffe's post hoc test.

Table 12. Bone mineral density ranges for each race/ethnic group.

	Femoral Neck BMD Range (g/cm^2)	Intertrochanter BMD Range (g/cm^2)	Total Femur BMD Range (g/cm^2)	Trochanter BMD Range (g/cm^2)
White	0.563-1.284	0.712-1.724	0.607-1.420	0.422-1.057
Black	0.600-1.650	0.735-1.921	0.640-1.707	0.481-1.441
Mexican American	0.467-1.214	0.696-1.755	0.594-1.419	0.422-1.030
Other	0.626-1.260	0.763-1.488	0.676-1.245	0.485-0.966

Table 13. Differences in BMD by exercise status.

	No Exercise (n=440)		Some Exercise (n=1452)	
BMD (g/cm^2)	**Mean**	**SEM**	**Mean**	**SEM**
Femoral Neck	0.845[1]	0.009	0.842	0.005
Intertrochanter	1.092	0.012	1.105	0.006
Total Femur	0.930	0.010	0.942	0.005
Trochanter	0.684	0.007	0.703	0.003

[1]Means across rows were significantly different at $p<0.025$ using independent t-test and sample weights.

Table 14. Differences in BMD by non-weight bearing exercise status.

	No Non-Weight Bearing Exercise (n=651)		Some Non-Weight Bearing Exercise (n=1241)	
BMD (g/cm^2)	**Mean**	**SEM**	**Mean**	**SEM**
Femoral Neck	0.853[1]	0.009	0.838	0.005
Intertrochanter	1.101	0.010	1.103	0.006
Total Femur	0.938	0.009	0.941	0.005
Trochanter	0.691	0.007	0.703	0.004

[1]Means across rows were significantly different at $p<0.025$ using independent t-test and sample weights.

Table 15. Differences in BMD by weight bearing exercise status.

	No Weight Bearing Exercise (n=1004)		Some Weight Bearing Exercise (n=888)	
BMD (g/cm^2)	**Mean**	**SEM**	**Mean**	**SEM**
Femoral Neck	0.835[1]	0.006	0.849	0.006
Intertrochanter	1.092	0.008	1.114	0.009
Total Femur	0.930	0.006	0.949	0.007
Trochanter	0.692	0.005	0.707	0.005

[1]Means across rows were significantly different at $p<0.025$ using independent t-test and sample weights.

Table 16. Differences in BMD by smoking status as defined by blood cotinine.

	Nonsmoker (n=1357)		Smoker (n=535)	
BMD (g/cm^2)	**Mean**	**SEM**	**Mean**	**SEM**
Femoral Neck	0.845[1]	0.005	0.836	0.007
Intertrochanter	1.113	0.006	1.084	0.009
Total Femur	0.947	0.005	0.927	0.007
Trochanter	0.703	0.004	0.693	0.005

[1]Nonsmoker and smoker means were significantly different at $p<0.025$ using independent t-test and sample weights.

Table 17. Correlations between demographic/lifestyle characteristics and BMD at various sites.

Demographic/lifestyle characteristic (N[1])	Femoral Neck BMD		Intertrochanter BMD		Total Femur BMD		Trochanter BMD	
	r^2	p	r	p	r	p	r	p
Age (1892)	-0.1378	**0.0000**	-0.0000	0.4189	-0.0361	0.0944	-0.0173	0.2744
BMI (1889)	0.4042	**0.0000**	0.5112	**0.0000**	0.4837	**0.0000**	0.3501	**0.0000**
Poverty Income Ratio (1740)	-0.1905	**0.0000**	-0.0985	**0.0034**	-0.1072	**0.0017**	-0.0548	0.0688
Non-weight bearing exercise score (1892)	-0.0100	0.3763	0.0600	**0.0392**	0.0656	**0.0289**	0.1091	**0.0015**
Weight bearing exercise score (1892)	0.0374	0.1442	0.0728	**0.0423**	0.0707	**0.0472**	0.0843	**0.0187**
Calcium from food (1859)	0.0245	0.2477	0.0436	0.1041	0.0566	0.0640	0.0557	0.0699
Calcium from supplements (1892)	0.0000	0.4773	0.0100	0.4069	0.0141	0.3514	0.0316	0.1697
Lifelong milk consumption score (1890)	0.1034	**0.0004**	0.0707	**0.0104**	0.1015	**0.0004**	0.1288	**0.0001**
Alcoholic drinks per year (1892)	-0.0100	0.4145	-0.0265	0.1922	-0.0141	0.3435	0.0100	0.3899

[1]Sample sizes refer to the number of unweighted observations in the data set.
[2]Pearson's r.

Table 18. Weighted stepwise multiple regressions for various bone sites.

Femoral Neck BMD[1]	β	SEE[2]	p
Y-intercept	0.1551	0.0611	0.0142
BMI	0.5279	0.0458	0.0000
Race/Ethnicity	0.0726	0.0072	0.0000
Poverty Income Ratio	-0.0178	0.0073	0.0185
Lifelong milk consumption score	0.0124	0.0031	0.0002
Weight bearing exercise	0.0109	0.0043	0.0141
Age	-0.0026	0.0006	0.0001

[1]R^2=0.2621, Prob>F=0.0000
[2]SEE=standard error of estimate.

Intertrochanter BMD[1]	β	SEE[2]	p
Y-intercept	-0.1430	0.0672	0.0381
BMI	0.8598	0.0509	0.0000
Race/Ethnicity	0.0799	0.0089	0.0000
Weight bearing exercise	0.0185	0.0060	0.0033
Non-weight bearing exercise	0.0167	0.0047	0.0009
Lifelong milk consumption score	0.0112	0.0039	0.0063
Age	-0.0012	0.0006	0.0373

[1]R^2=0.3237, Prob>F=0.0000
[2]SEE=standard error of estimate.

Total Femur BMD[1]	β	SEE[2]	p
Y-intercept	-0.0396	0.0593	0.5073
BMI	0.6796	0.0448	0.0000
Race/Ethnicity	0.0668	0.0074	0.0000
Non-weight bearing exercise	0.0152	0.0041	0.0006
Weight bearing exercise	0.0141	0.0050	0.0066
Lifelong milk consumption score	0.0121	0.0032	0.0004
Age	-0.0015	0.0005	0.0040

[1]R^2=0.3059, Prob>F=0.0000
[2]SEE=standard error of estimate.

Trochanter BMD[1]	β	SEE[2]	p
Y-intercept	0.0873	0.0545	0.1154
BMI	0.3848	0.0391	0.0000
Race/Ethnicity	0.0455	0.0070	0.0000
Non-weight bearing exercise	0.0150	0.0039	0.0004
Lifelong milk consumption score	0.0120	0.0027	0.0000
Weight bearing exercise	0.0103	0.0039	0.0100

[1]R^2=0.1886, Prob>F=0.0000
[2]SEE=standard error of estimate.

Table 19. Null hypotheses reject or fail to reject summary.

Null Hypotheses There will be no relation between variables below (1-11) and BMD.	**Reject HO**	**Fail to Reject HO**
1[1]: race/ethnicity	[2]FN, IN, TO, TR	No bone sites
2[3]: age	FN	IN, TO, TR
3: BMI	FN, IN, TO, TR	No bone sites
4: PIR	FN, IN, TO	TR
5[4]: non-weight bearing exercise status	IN, TO, TR	FN
6: weight bearing exercise status	FN, IN, TO, TR	No sites
7: smoking status	FN, IN, TO, TR	No sites
8: calcium intake from food	No bone sites	FN, IN, TO, TR
9: calcium intake from supplements	No bone sites	FN, IN, TO, TR
10: lifelong milk consumption	FN, IN, TO, TR	No bone sites
11: alcohol intake	No bone sites	FN, IN, TO, TR
12[5]: None of the above variables will predict BMD.	FN, IN, TO, TR	No bone sites

[1]1HO was determined from weighted ANOVA and Scheffe's post hoc tests on Table 11.
[2]FN=femoral neck, IN=intertrochanter, TO=total femur, TR=trochanter.
[3]2HO-4HO & 8HO-11HO were determined from weighted correlations on Table 17.
[4]5HO-7HO was determined from independent t-tests and sample weights on Tables 14-16.
[5]12HO was determined from multiple regression analyses on Table 18.

CHAPTER V

DISCUSSION

The objective of this study was to determine the relation between BMD, demographic variables, and health behaviors in premenopausal women. Demographic variables are not modifiable, but health behaviors are modifiable. Health care professionals need to understand what health behaviors are related to BMD and to know which individuals are at highest risk for osteoporosis. By improving modifiable health behaviors that are related to BMD, individuals may prevent the loss of bone that leads to osteoporosis when they are older.

Descriptives

Comparisons in physical activity levels between the current study and others are difficult to make because of the differences in measuring physical activity (Table 1). For example, Mazess and Barden (1991) measured activity using a pedometer and an accelerometer. Rubin et al. (1999) calculated a physical activity score based on level of activity at work, distance walked per week, amount of recreational exercise the subjects did, and if the subjects worked up a sweat during any regular activity. Uusi-Rasi et al. (1998) had a more complex method of determining physical activity level that will not be explained here. Nguyen et al. (2000) used a physical activity index (PAI) somewhat similar to the exercise score used in the current study, but the PAI was not directly comparable to the exercise score of the present study.

Etherington et al. (1996) described physical activity by units of time; however, the current study does not include units of time.

Comparisons in smoking status between the current study and others are difficult to make because of the differences in measuring smoking status (Table 1). For the current study blood cotinine levels were used to determine smoking status because cotinine is a more accurate indicator than is self-reported smoking status. Other researchers determined smoking status differently. Jones and Scott (1999) asked if the subject currently smoked and if the subject replied "yes", asked the number of cigarettes smoked per day. Michaelsson et al. (1996) asked about current or former smoking and about number of cigarettes smoked per day. Mazess and Barden (1991) defined smokers as subjects who smoked more than two cigarettes per day for at least one year.

Women in this study consumed approximately 69% (692 mg) of the DRI for calcium from food (Table 1). Subjects consumed between 15 and 4969 mg of calcium from food in the current study. However, about 17% of the sample in the present study consumed supplemental calcium (mean of 186 mg for those who consumed supplements and 40 mg for the entire sample; 19% and 4% of the DRI, respectively) to bring them closer to the recommended intake. Kardinaal et al. (1999) determined calcium intake in European women aged 20-23 years ranged from 139-2985 mg/day with a mean of about 900 mg/day, which is higher than the mean dietary calcium intake in the present study. Kardinaal et al. (1999) reported calcium intake from food, but it is unclear if Kardinaal et al. (1999) also included calcium intake from supplements. Uusi-Rasi et al. (1998) selected

women with high dietary calcium intake (around 1400 mg/day) and women with low dietary calcium intake (around 600 mg/day) for their research. The dietary calcium intake values in the current study are close to the low dietary calcium intake values from the Uusi-Rasi et al. (1998) study. Rubin et al. (1999) separated the 18-35 year old women into quartiles by calcium intake. Most of the women (60%) had dietary and supplemental calcium intakes in the middle range of 251-750 mg (Rubin et al., 1999). The current study has dietary and supplemental calcium intakes higher than the intakes that Rubin et al. (1999) reported. Mazess and Barden (1991) determined that the mean dietary and supplemental calcium intake for women aged 20-39 years was 909 mg/day, which is higher than the dietary and supplemental calcium intake of the women in the current study. Calcium intake from supplements alone was 214 mg/day for the seven daughters who took calcium supplements in a mother-daughter pair study (Ulrich et al., 1996). The calcium intake from supplements in the Ulrich et al. (1996) study was higher than the calcium intake from the current study, but Ulrich et al. (1996) had a much smaller sample size than did the current study.

This study did not analyze the relation among nutrients other than calcium and BMD because other researchers (Mazess and Barden, 1991) have found that 14 other nutrients (phosphorus, protein, iron, magnesium, zinc, and energy, among others) did not significantly predict BMD at the lumbar spine, femoral neck, or radius. However, the mean intake for phosphorus and vitamin D in the current study was 154% (1080 mg) of the DRI for phosphorus, and 78% (3.9 mcg) of the DRI for vitamin D (Table 1).

The amount of alcohol consumed by subjects in this study compared with the amount of alcohol consumed by subjects who were not alcohol dependent in other studies (Table 1). The current study reports alcohol consumption as drinks per year and other research studies generally report alcohol consumption as drinks per week, so direct comparisons were made by dividing the number of drinks per year by 52 weeks per year for the current study. Clark and Sowers (1996) reported the alcohol consumption for women who were not dependent on alcohol and for women who were dependent on alcohol as one to two drinks and 46 drinks per week during the past year, respectively. Laitinen et al. (1993) reported the alcohol consumption for the women who were not dependent on alcohol as 28 g per week, which corresponds to approximately three drinks per week. For women who were dependent on alcohol, Laitinen et al. (1993) reported the alcohol consumption as 186 g per week, which corresponds to about 18 to 19 drinks per week. The amounts reported by Clark and Sowers (1996) and Laitinen et al. (1993) for the women who were not dependent on alcohol were close to the alcohol consumption for subjects in the current study of less than two drinks per week (Table 1; 82.9 drinks per year divided by 52 weeks per year). Felson et al. (1995) studied data from the Framingham Study and reported that approximately 75% of the female subjects consumed fewer than three drinks per week, which is slightly more than the amount of alcohol consumption in the current study.

Bone Mineral Density Compared to Reference Values

Table 2 summarizes the variability of BMD for the sample and compares those values to reference values from Looker et al. (1997). The BMD minimum values in this study were higher than the reference range from Looker et al. (1997), because researchers for the present study excluded women known to have had chronic diseases that affect BMD or women who were taking prescription medications known to affect bone metabolism. At all bone sites, the sample for the present study had values that fell below the lowest value in the range for osteopenia, which indicates that some women could be diagnosed as having osteoporosis because any value lower than the lowest value in the range for osteopenia was considered osteoporosis.

Hypothesis One

Blacks will have higher BMD than other groups.

Blacks had a significantly higher BMD than other race/ethnic groups, as hypothesized (Table 11). This agrees with the CDC and NIH (2000) statement that blacks have lower prevalence of osteoporosis as measured by low BMD. The race/ethnicity variable significantly predicted part (β=0.0455 - β=0.0799) of the explained variability in BMD at each bone site in the multiple regression analyses (Table 18). Researchers of the current study speculate that a possible reason for blacks having higher BMD is that they have higher BMI and lower income (which is associated with higher BMI and therefore higher BMD) than

most other race/ethnic groups, except Mexican Americans (Table 4). However, by performing multiple regression analysis BMI and income were statistically controlled for, so this speculation may not be correct.

Hypothesis Two

As age increases, BMD will decrease.

Age did not significantly correlate with BMD in the present study at any site except the femoral neck, where age negatively correlated with BMD using Pearson's correlation ($p<0.05$) (Table 17). Despite the lack of a significant correlation at the other bone sites, younger age was a significant independent predictor of higher BMD at the total femur, femoral neck, and intertrochanter when using stepwise multiple regression analysis (Table 18). However, older age explained only part (β= -0.0026, -0.0012, -0.0015,) of the decrease in BMD at the femoral neck, intertrochanter, and total femur, respectively. Other researchers (Mazess and Barden, 1991; Rubin et al., 1999; Guthrie et al., 1996) have found inconsistent correlations between age and BMD. This may be due to the fact that the researchers studied different bone sites of women at different ages. Mazess and Barden (1991) studied the BMD of women aged 20-39 years. They found that age was positively associated with lumbar spine and radius shaft BMD, but negatively associated with femoral neck, Ward's triangle, trochanter, distal radius, and humerus neck BMD (Mazess and Barden, 1991). Rubin et al. (1999) studied 18-35 year-old women and found that age is positively correlated

with lumbar spine BMD, but negatively correlated with femoral neck BMD. Guthrie et al. (1996) studied women aged 46-57 years and found that age was negatively correlated with lumbar spine BMD in perimenopausal women, but not pre- or postmenopausal women. They also found that age was negatively correlated with femoral neck BMD in all three groups of women, but the results were only statistically significant for the perimenopausal group (Guthrie et al, 1996).

Hypothesis Three

As BMI increases, BMD will increase.

Body mass index (BMI) and BMD strongly correlated at all bone sites using Pearson's correlation in the present study ($p<0.05$) (Table 17). In the final regression model for each bone site, BMI accounted for part ($\beta=0.3848$ - $\beta=0.8598$) of the explained variability (Table 18).

Other researchers (Orozco and Nolla, 1997; Franceschi et al., 1996; Rubin et al., 1999; Michaelsson et al., 1996) also found a strong positive correlation between BMI, or height and weight, and BMD. Higher weight correlated significantly with BMD at the femoral neck, intertrochanter, and total femur, whereas higher BMI correlated significantly with BMD at only the intertrochanter and total femur (Orozco and Nolla, 1997). Depending on the multiple regression model used, weight accounted for 29% (at the femoral neck) to 34% (at the intertrochanter) of the explained variability in BMD; and BMI accounted for 26% (at the total femur) to 31% (at the intertrochanter) of the

explained variability in BMD (Orozco and Nolla, 1997). Franceschi et al. (1996) determined that in premenopausal women each 1 unit increase in BMI accounted for 0.5% of the explained variability in BMD in the regression model. Weight and height accounted for 0.3% and 33.5% of the explained variability in BMD in the multiple regression model for the femoral neck, whereas weight accounted for 0.3% of the explained variability in BMD in the model for the lumbar spine (Rubin et al., 1999). Michaelsson et al. (1996) determined BMI accounted for 26%, 18%, and 12% of the explained variability in BMD using multiple regression analysis at the total body, lumbar spine, and femoral neck, respectively.

Although researchers (Franceschi et al., 1996; Michaelsson et al., 1996; Orozco and Nolla, 1997) agree that there is not a clear answer as to why higher BMI is strongly correlated to higher BMD, they suggest a few explanations. One explanation is that being overweight increases the amount of sex hormones, such as estrogen, that are associated with higher BMD (Franceschi et al., 1996; Orozco and Nollas, 1997). Another explanation is that being overweight exerts additional mechanical forces on the bone and therefore leads to higher BMD (Michaelsson et al., 1996; Orozco and Nolla, 1997).

Hypothesis Four

<u>As PIR increases, BMD will increase.</u>

Income, as measured by PIR, negatively correlated with BMD at the femoral neck, intertrochanter, and total femur using Pearson's correlation ($p<0.05$) (Table 17). However, in the stepwise multivariate regression analysis,

PIR was negatively associated with BMD (β= -0.0178) only at the femoral neck (Table 18). At all other bone sites, PIR was not part of the model to predict BMD. No other study in the search of the literature related income to BMD. The researchers of the current study speculate that this is because income was statistically controlled for in each of the other studies that used multiple regression analysis. Income may affect nutrient intake, physical activity and BMI (Kurinij et al., 1986; Diez-Roux et al., 1999; Pomerleau et al., 1997; Kahn et al., 1991), but in this study all variables in the multiple regression model were controlled for statistically.

Hypothesis Five

<u>As non-weight bearing exercise level increases, BMD will increase.</u>

When compared with women who did not perform non-weight bearing exercise (Table 14), women who performed non-weight bearing exercise had significantly higher BMD at all sites except the femoral neck ($p<0.025$). At the femoral neck, women who did not perform non-weight bearing exercise had a significantly higher BMD than did women who did perform non-weight bearing exercise ($p<0.05$). The different finding at the femoral neck may be due to the low percentage (25%) of trabecular bone in the femoral neck (Einhorn, 1996). Using Pearson's correlation, the intertrochanter, total femur, and trochanter were the sites at which higher non-weight bearing exercise scores correlated significantly with BMD ($p<0.05$) (Table 17). The non-weight bearing exercise score accounted for part (β=0.0167, 0.0152, 0.0150) of the explained variability in

the multiple regression models at the intertrochanter, total femur, and trochanter, respectively, in the present study (Table 18).

In the present study the non-weight bearing exercise score included activities such as walking, biking, swimming, gardening, and other activities deemed to be non-weight bearing (Etherington, 1996). The number of times a respondent reported performing an activity in the past month was multiplied by the MET score. The resulting number for each activity was added together to create the total non-weight bearing score. The exercise score was not an exact MET measure because subjects did not report the length of time that each activity was performed. However, the exercise score still indicated the intensity of the activities.

Many other researchers did not separate total physical activity into non-weight bearing and weight bearing categories, so comparisons between those studies and the present study are difficult to make. In addition, other researchers had different methods to assess physical activity levels. However, when looking at total physical activity, many researchers (Rubin et al., 1999; Uusi-Rasi et al., 1998; Nguyen et al., 2000) found that overall physical activity positively influenced BMD in a wide age-range of women. Only one group of researchers (Mazess and Barden, 1991) found no significant correlation between overall physical activity and BMD.

Hypothesis Six

As weight bearing exercise level increases, BMD will increase.

When compared with women who did not perform weight bearing exercise (Table 15), women who performed weight bearing exercise had a significantly higher BMD at all sites ($p<0.025$). Using Pearson's correlation, the intertrochanter, total femur, and trochanter were the sites at which higher weight bearing exercise scores correlated significantly with BMD ($p<0.05$) (Table 17). The weight bearing exercise score explained part ($\beta=0.0103$ to $\beta=0.0185$) of the accounted variability in the multiple regression models at all sites (Table 18).

In the present study the weight bearing exercise score included activities such as jogging, running, aerobics, dancing, calisthenics, lifting weights, and other activities deemed to be weight bearing. The number of times a respondent reported performing an activity in the past month was multiplied by the MET score. The resulting number for each activity was added together in the total weight bearing score. The exercise score was not an exact MET measure because subjects did not report the length of time that each activity was performed; however the exercise score still indicated the intensity of the activities.

Several researchers have found similar positive associations between weight bearing exercise and BMD (Etherington et al., 1996; Lohman et al., 1995), although neither of these studies placed weight bearing exercise alone (instead of total exercise) in a regression model. Direct comparisons in physical activity

levels between the current study and others are difficult to make because of the differences in measuring physical activity. Etherington et al. (1996) described the weight bearing physical activity scores of the 40-65 year old women by units of time; however, the current study does not include units of time. Etherington et al. (1996) also mentioned that there is not a "gold standard" for assessing physical activity levels for groups of people. However, using their method of assessing exercise levels, Etherington et al. (1996) found that more active individuals have a significantly higher lumbar spine and femoral neck BMD ($p<0.05$). In the Etherington et al. (1996) study, the ex-elite athletes had significantly higher BMD than the active, low activity, and inactive controls; the active controls had significantly higher BMD than the inactive controls; and the low activity controls had significantly higher BMD than the inactive controls. Lohman et al. (1995) prospectively observed 28-39 year old women participating in an 18-month strength training study along with taking 500 mg calcium supplements. The exercise group performed 12 different weight lifting exercises (three sets of 8-12 repetitions) using free weights three times per week. The outcome measures were total body, lumbar spine, femoral neck, Ward's triangle, and trochanter BMD. The exercise group significantly increased their BMD from baseline at 5 and 12 months for lumbar spine and at 5, 12, and 18 months for trochanter ($p<0.05$), while the there was no significant change in the control group.

Hypothesis Seven

Smokers will have lower BMD than nonsmokers.

When compared with smokers (Table 16), nonsmokers had significantly higher BMD at all sites ($p<0.025$). However, there was no significant relationship between smoking status and BMD in the stepwise multivariate analysis (Table 18). This may be due to the fact that in the present study the method of determining smoking status is different from other studies. In the present study subjects were separated into nonsmoking versus smoking categories based on their serum cotinine levels. Cotinine was chosen as an indicator of smoking status because it is a sensitive indicator of nicotine in the blood (Wallach, 2000). Since there was an abnormal distribution of cotinine levels that could not be corrected by transformations (such as a log transformation), the researchers in the current study recoded all values 8 ng/mL and less as "nonsmoker" and all values more than 8 ng/mL as "smoker". The present study is not directly comparable to other studies relating smoking to BMD, because of using cotinine as an indicator of smoking status. The other studies in this discussion used a question to determine smoking status, "Do you smoke?" or "Have you smoked in the past?"

Other researchers (Jones and Scott, 1999; Franceschi et al, 1996; Mazess and Barden, 1991), found that nonsmokers had significantly greater BMD at various sites than did smokers, as in the present study. Jones and Scott (1999) studied women with a mean age of 33 and used unpaired t-tests to

determine that smokers have significantly lower femoral neck, lumbar spine, and total body BMD than did nonsmokers ($p<0.05$). Franceschi et al. (1996) used multiple regression analysis to determine that premenopausal women who smoked more than 15 cigarettes per day had a greater risk of low lumbar spine BMD. As in the present study, Mazess and Barden (1991) found that while smokers had significantly lower lumbar spine BMD than did nonsmokers in a study of 20-39 year old women, there was not a significant relation between smoking status and BMD in a multiple regression analysis.

In contrast, others found no significant relation between smoking and BMD (Law and Hackshaw, 1997; Kiel et al., 1996). One potential reason for differing results could be different methods of measuring smoking status. Law and Hackshaw (1997) performed a meta-analysis of 13 cross-sectional studies that included premenopausal women and determined that there was no significant difference between smokers and nonsmokers BMD at the femoral neck, radius, and calcaneus. Kiel et al. (1996) used data from the Framingham Study to determine that in women aged 68-98, there was no significant difference in radius, femoral neck, trochanter, Ward's triangle, or lumbar spine BMD among subjects who currently smoke, who formerly smoked, and who never smoked.

Hypothesis Eight

<u>As calcium intake from food increases, BMD will increase.</u>

There was no significant correlation between current calcium intake from food and BMD at any site using Pearson's correlation (Table 17) or multivariate

regression analysis (Table 18) in the present study. The results agree with some researchers (Kardinaal et al., 1999; Uusi-Rasi et al., 1998), but not with others (Cadogan et al., 1997; Rubin et al., 1999). This apparent inconsistency in results may be due to the various methods used in the studies (either reporting only dietary calcium intake or both dietary and supplemental calcium intake), the ages of the females in the studies, and the amount of calcium from food that was being consumed. In the current study, there was a wide range of calcium intakes (from 15 to 4969 mg/day), so there should have been enough variability to see an effect of calcium intake on BMD if there was an effect. Perhaps the greatest effect of calcium intake on BMD is in the adolescent years (Cadogan et al., 1997).

Some researchers found similar results as the current study. Kardinaal et al. (1999) determined dietary calcium intake in European women aged 20-23 years was about 900 mg/day, but it is unclear if Kardinaal et al. (1999) also included calcium intake from supplements. Kardinaal et al. (1999) found no significant relation between calcium intake and BMD at the radius. Uusi-Rasi et al. (1998) compared women with high dietary calcium intake (~1475 mg/day) to women with low dietary calcium intake (~650 mg/day) to determine that calcium intake was not significantly related to either femoral neck or radius BMD.

Other researchers found different results than the current study. Cadogan et al. (1997) studied girls with a mean age of 12 years to determine that girls in the milk group had significantly greater increases in total body BMD than did girls in the control group ($p<0.05$). Girls in the milk group consumed approximately

1125 mg calcium per day compared to the girls in the control group who consumed about 700 mg calcium per day. Rubin et al. (1999) separated 18-35 year old women into quartiles by calcium intake from both food and supplements. Using Chi-square analysis, Rubin et al. (1999) found that women consuming higher amounts of calcium had significantly higher lumbar spine and femoral neck BMD ($p<0.05$). However, there was not a significant relation between calcium intake and femoral neck or lumbar spine BMD using multiple regression analysis in the Rubin et al. study (1999).

Hypothesis Nine

As calcium intake from supplements increases, BMD will increase.

There was no significant correlation between calcium from supplements and BMD at any site in Pearson's correlation (Table 17) or multivariate regression analysis (Table 18) in the present study. Lloyd et al. (1993) randomized 12 year-old girls into either a calcium supplement group (mean total calcium=1370 mg/day) or a control group (mean total calcium=935 mg/day) for a study lasting 18 months. Lloyd et al. (1993) found that greater total calcium intake from supplements and food correlated significantly with greater increases in total body and lumbar spine BMD ($p<0.05$). Cadogan et al. (1997) also studied 12 year-old girls to determine if milk supplementation (not from a pill) affected bone acquisition. Girls in the milk group (1125 mg calcium/day) had significantly greater increases in total body BMD than did girls in the control group (700 mg calcium/day) ($p<0.05$). The discrepancies between the current study and either

the Lloyd et al. (1993) or the Cadogan et al. (1997) study may be because the subjects in the current study were older (20-45 years of age). Uusi-Rasi et al. (1998) results agree with the present study in that they found no relation between calcium intake from supplements and food and BMD in women aged 25-65. One group of researchers (Ulrich et al., 1996) reported the relation between calcium intake only from supplements and BMD. Ulrich et al. (1996) results also agree with the present study in that they did not find any significant correlation between calcium intake from supplements and BMD in women with a mean age of 41 years.

Hypothesis Ten

As the amount of milk consumed over a lifetime increases, BMD will increase.

Higher lifelong milk consumption scores correlated significantly with BMD at all sites using Pearson's correlation in the present study (Table 17). The lifelong milk consumption score explained part (β=0.112 - β=0.0124) of the variability in the multiple regression models at all bone sites (Table 18). Perhaps the lifelong milk consumption score did not account for more of the explained variability because of the method in which the score was calculated or because the subjects' recall of previous milk consumption was inaccurate.

A lifelong milk consumption score was calculated using the responses from the four questions "How often did you drink any type of milk when you were a child, teenager, young adult, and middle-aged adult?" Calculations were performed on each categorical response (0=never to 5= more than once per day)

to obtain a lifelong milk consumption score. This lifelong milk consumption score was related back to the original categories, so if there was a milk consumption score of 3.87, that would mean that the respondent drank milk almost once per day (category 4) on average over a lifetime.

The current study agreed with Murphy et al. (1994) who studied 284 females aged 44-74 years to determine the effect of lifelong milk consumption on BMD. Murphy et al. (1994) asked subjects to place their previous milk consumption before age 25 into three categories: less than one glass per week, more than one glass per week but less than one glass per day, or at least one glass per day. Murphy et al. (1994) determined that milk consumption up to age 25 accounted for 1.5-2% of the explained variance in the BMD for all hip sites (total femur, femoral neck, trochanter, intertrochanter, and Ward's triangle) ($p<0.05$).

The current study conflicted with Ulrich et al. (1996) who found no significant relation between milk consumption and BMD. Ulrich et al. (1996) studied 25 mother (mean age 72 years)-daughter (mean age 41 years) pairs to determine the effect of lifelong milk consumption and calcium intake from supplements on axial and peripheral BMD, as measured by DEXA. Milk consumption was estimated for each of the following age time periods: childhood (0-12 years), teens (13-19 years), early adulthood (20-39 years), midadulthood (40-59 years), and late adulthood (60+ years). Ulrich et al. (1996) asked the subjects how often they drank the equivalent of one cup of milk. Then Ulrich et al. (1996) calculated an average milk consumption score over each of the age

periods and weighted the childhood and teen periods by half. The mean milk consumption scores for the subjects were around 3, which corresponded to drinking milk at least once per day and was about the same as the present study. The present study differs from Ulrich et al. (1996) in that the present study had more choices on how often milk was consumed, it weighted each age period by the exact number of years that it contained, and that it included younger women (20-45 years).

The current study also conflicted with Teegarden et al. (1999), who found no significant relation between milk consumption during childhood and adolescence and BMD. Teegarden et al. (1999) asked 224 women aged 18-31 years to place their milk consumption during childhood and adolescence into three categories: infrequent or never, sometimes, and at every or almost every meal. Previous milk consumption correlated significantly with total body and radius BMD ($p<0.05$), but not lumbar spine or femoral neck BMD. Teegarden et al. (1999) did not provide as many categories in which to place previous milk consumption as did the present study. In addition, Teegarden et al. (1999) did not present a milk consumption score, so that could not be directly compared to the current study.

Lifelong milk consumption was a part of the stepwise multiple regression models for all bone sites (Table 18). Calcium intake from food and supplements was not included in any of the regression models in the present study. Perhaps there are other nutrients found in milk, such as phosphorus, vitamin D, or protein,

which affect BMD. Or perhaps individuals who drink more milk over their lifetime may have some other characteristics of health behaviors that affect BMD.

Hypothesis Eleven

As alcohol intake increases, BMD will increase.

Alcohol intake, as determined by drinks per year, did not correlate significantly with BMD at any site in either Pearson's correlation (Table 17) or multivariate analysis (Table 18). These results agree with the findings of some researchers (Clark and Sowers, 1996; Laitinen et al., 1993) who determined that the BMD of alcohol dependent women versus women who were not alcohol dependent were not significantly different. However, the present study was not designed to compare women who were alcohol dependent to women who were not alcohol dependent. Other researchers (Felson et al., 1995; Holbrook and Barrett-Connor, 1993), however, found that a moderate intake of alcohol was associated with higher BMD.

When Clark and Sowers (1996) controlled for smoking, they determined there were no significant differences in BMD between women who were dependent on alcohol (n=25) and women who were not (n=25). Clark and Sowers (1996) reported the alcohol consumption for women who were not dependent on alcohol and for women who were dependent on alcohol as one to two drinks and 46 drinks per week during the past year, respectively. Laitinen et al. (1993) found no significant differences in lumbar spine, femoral neck, trochanter, Ward's triangle, or forearm BMD between 24-48 year old women who

were alcohol dependent (n=19) and controls of the same age who were not alcohol dependent (n=157). Laitinen et al. (1993) reported the alcohol consumption for the women who were not dependent on alcohol as 28 g per week, which corresponds to approximately three drinks per week. For women who were dependent on alcohol, Laitinen et al. (1993) reported the alcohol consumption as 186 g per week, which corresponds to about 18 to 19 drinks per week.

Felson et al. (1995) studied data from the Framingham Study to determine that after adjusting for other variables, such as age, moderate alcohol consumption (7+ drinks/week) was significantly associated with higher lumbar spine, trochanter, and radius BMD ($p<0.05$). Felson et al. (1995) separated 68-96 year old women into quartiles of alcohol consumption (<1 drink/week, 1 to <3 drinks/week, 3 to <7 drinks/week, and 7+ drinks per week). Holbrook and Barrett-Connor (1993) separated 267 women aged 45 years and older into quartiles of alcohol consumption per week (none; 0.1-48.6 g or ~0-5 drinks; 48.7-120.4 g or ~5-12 drinks; and more than 120.5 g or more than 12 drinks). Increased alcohol intake was significantly associated with increased lumbar spine BMD ($p<0.05$).

Limitations

The current study had several limitations. One limitation is that several variables, including alcohol consumption, had a high non-response rate. In the case of alcohol consumption, researchers in the current study made assumptions

that if there was no response to the question, then that subject consumed no alcohol. However, this may not be true. In addition, variables were not always in the best form for what the current study's researchers were studying, such as exercise or alcohol variables, so researchers in the present study made adjustments. However, this study did use a nationally representative sample (NHANES III data set), and selected each variable for the analysis based on previous studies.

CHAPTER VI

CONCLUSIONS AND IMPLICATIONS

The objective of this study was to determine the relation between bone mineral density (BMD), demographic variables, and health behaviors in premenopausal women. The present study revealed that demographic variables and health behaviors explained 19-32% of the variability in BMD at four measured sites (total femur, femoral neck, trochanter, and intertrochanter). Researchers in the current study determined that increased body mass index (BMI) was the strongest predictor for increased BMD, however this does not mean that women with a BMI in the normal or healthy range should gain weight to protect them against low BMD. However, health care providers should encourage women with very low BMI, such as women with anorexia nervosa, to gain appropriate amounts of weight to help prevent osteoporosis. Race/ethnic group was the next strongest predictor of BMD in the current study. In addition, in the present study, increasing age and increasing income predicted a small percentage of the decrease in BMD. Health care providers can use race/ethnic group, age and income as a part of a screening tool to identify women at risk for osteoporosis. Increasing non-weight bearing exercise, weight bearing exercise and lifelong milk consumption scores also predicted a small portion of increasing BMD in the current study. Health professionals can encourage women to increase their physical activity and milk consumption to help prevent osteoporosis. Smoking was correlated with lower BMD in the current study, so

health care providers can advocate smoking cessation programs for their clients to help prevent low BMD. In the present study, alcohol consumption did not significantly affect BMD, so health professionals can maintain their current alcohol consumption recommendations to their clients.

Only premenopausal women aged 20-45 years were studied in the current research project. If the researchers in the current study had also included postmenopausal women, the results might have been different. In fact, future research could be performed on the NHANES III data set to compare premenopausal women to postmenopausal women, in regards to demographic variables, lifestyle behaviors, and BMD. In addition, it would be interesting to perform a longitudinal study on the current subjects using NHANES III data as baseline data. In this longitudinal study, bone loss rates could be observed.

There are many additional areas for future research about osteoporosis and BMD. Using the same basic methods of the current study, other nutrients, such as vitamin D, phosphorus, copper, zinc, magnesium, or protein, could be examined for their relation to BMD. Perhaps instead of focusing on nutrients, research could be performed focusing on foods containing calcium, using the food frequency questionnaire (FFQ) from NHANES III, and their relation to BMD. Instead of just observing the calcium intake from the 24-hour food recall, using the FFQ would allow for observing calcium intake for a longer time period.

The present study and others informed health professionals of the factors that may be related to BMD. Those health professionals can then determine who is at greatest risk for developing osteoporosis and develop interventions for those

individuals. By improving those health behaviors that may be related to BMD, individuals may prevent osteoporosis when they are older. Because osteoporosis takes years to develop, individuals may be unaware of their need to improve or prevent further loss of BMD; but health care professionals can help raise their clients' awareness of their bone health.

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APPENDICES

Appendix A. Multiple regression analysis equations

Femoral Neck BMD Regression Equation. (n=1736)

y'=(0.1551) + (0.5279) (BMI) + (0.0726) (Race/Ethnicity) - (0.0178) (Poverty Income Ratio) + (0.0124) (Lifelong milk consumption score) + (0.0109) (Weight bearing exercise score) – (0.0026) (Age)

Intertrochanter BMD Regression Equation. (n=1887)

y'=(-0.1430) + (0.8598) (BMI) + (0.0799) (Race/Ethnicity) + (0.0185) (Weight bearing exercise score) + (0.0167) (Non-weight bearing exercise score) + (0.0112) (Lifelong milk consumption score) – (0.0012) (Age)

Total Femur BMD Regression Equation. (n=1887)

y'=(-0.0396) + (0.6796) (BMI) + (0.0668) (Race/Ethnicity) + (0.0152) (Non-weight bearing exercise score) + (0.0141) (Weight bearing exercise score) + (0.0121) (Lifelong milk consumption score) – (0.0015) (Age)

Trochanter BMD Regression Equation. (n=1887)

y'=(0.0873) + (0.3848) (BMI) + (0.0455) (Race/Ethnicity) + (0.0150) (Non-weight bearing exercise score) + (0.0120) (Lifelong milk consumption score) + (0.0103) (Weight bearing exercise score)

VITA

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