EFFECTS AND PREDICTORS OF BONE MINERAL DENSITY IN COLLEGE-AGED FEMALE ATHLETES

by

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Dedication

This body of work is dedicated to three of the most amazing women that I have known, my cousin Dawnene Patricia Barnett, my grandmother Elaine Mahoney Anderson, and my friend Kristina Leigh Jonathan. To my cousin, who took my hand as a child and gave me the wings to become all that I am, and to my grandmother, who modeled strength and integrity. Dawnie and Grandma, thanks for blazing a trail for me to accomplish this overwhelming task, you both are forever in my heart. To Kristina, your spirit has given me the courage to reach for mountain tops. Finally, this dissertation is dedicated to all who believe that they can reach higher than circumstances might dictate.

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Abstract

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The primary purpose of this study was to examine the effect of sport-type on bone mineral density (BMD) (predominately lower body or predominately upper/lower body) among university Division I-A, female athletes (ages 18-25 yrs). A secondary purpose was to determine to what extent evidence of an eating disorder, body mass index (BMI), percent body fat (PBF), and episodes of amenorrhea predict the bone mineral status of the same sample of athletes. There were 50 athletes from various lower and upper/lower body sports included in this sample. A MANOVA determined the effect of sport-type on BMD, while four separate regression analyses examined the predictors of BMD (dominant and non-dominant forearm and femoral neck). The results showed a significant relationship between type of sport and BMD; although, the sample was too limited to determine which sport-type produced the most significant relationship. The regression analyses revealed that BMI was the only significant predictor of BMD for both the upper and lower BMD measurements, while PBF was significant for the upper body measurements. These data suggest that sport-type does have an effect on the BMD that athletes attain. Additionally, BMI can be utilized to predict BMD issue among athletes.

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Chapter One

Introduction

Women's collegiate athletics have undergone tremendous change over the past 30 years. No longer are women denied the opportunity to excel in athletics at the collegiate level. This process, which has been gradual, began when Title XI of the Civil Rights Act of 1965, which prohibited discrimination of person by race, color, or nationality, become law. The Civil Rights Act of 1965 served as a forerunner to Title IX, opening the doors for certain individual groups. Title IX of the Education Amendments of 1972 was modeled after the Civil Rights Act, although, Title IX shed light on a different side of discrimination. Title IX emphasized equality for all females in federally funded programs. From that period on, institutions were required to provide similar educational and athletic opportunities to both sexes (United States Department of Education, 1997).

Title IX offered women's athletics the advantage of funding for collegiate programs at a similar level to male athletics. Consequently, scholarships became available for promising female athletes. Women's athletics began a metamorphosis that persists today. The number and influence of female athletes, and the level of competition continues to grow. Contemporary female athletes compete and train at levels far greater than prior to Title IX (Golden, 2002).

Coupled with the success of females in athletic competition is the cultural pressure that women must have the "ideal" body. Body image among females is a

growing concern in modern society, particularly among female athletes. Athletes receive constant pressure from their peers and coaches to train intensely and build extremely lean body mass. The result is often prolonged physical training, a restrictive diet, and a premature reduction in bone mineral density (BMD) through disruption of the hypothalamic-pituitary axis (Turk, Prentice, Chappell, & Fields, 1999).

Osteoporosis is formally defined as the incidence of low trauma fractures, and a BMD score of 2.5 standard deviations below the mean (Parsons, 2005; World Health Organization [WHO], 1994). Osteoporosis can be the result of compromised BMD. BMD is evaluated by a variety of bone density scans. The most accurate of which is dual x-ray absorpitometry (DEXA). A DEXA scan produces a value, represented as a T-Score. The T-Score is referenced to the benchmark (e.g., the average value for a woman who is the same height, weight, race, etc.).

Women develop their peak bone mass by age 30; with 60% to 70% formed by age 20 (Nattiv & Armsey, 1997; Teitz, 1997; Zeni, Street, Dempsey, & Staton, 2000). After this age, women will loose BMD at a rate of 0.5% per year. After a woman reaches menopause, the rate of bone loss can increase as much as five percent per year (Leslie & St. Pierre, 1999; Melton et al., 2004; Steinwig, 2002). Female athletes, in particular, can lose 25% of their BMD if their osteopenic condition is ignored throughout their competitive years (Yurth, 1995). Osteopenia, or low bone mass, is the precursor to osteoporosis, and is defined by BMD measurements between 1 and 2.5 standard deviations below the mean (Joy et al. 1997; WHO, 1994). Compromised BMD can place the athlete at risk for stress fractures and eventually lead to osteoporosis. Moreover, the skeleton of a 20-year old osteopenic female athlete can be equated with that of a

postmenopausal woman. Early detection and treatment of osteoporosis is imperative to spare the deterioration of the athlete's BMD. The increased level of competition that has become accessible to female athletes creates the opportunity for osteoporosis to become a mounting concern for the extended team surrounding the female athlete (e.g., coaches, trainers, etc.).

Females are no longer relegated to intramural competition. In contemporary sport, female athletes now have the opportunity to pursue their athletic goals and to use their skills to the ultimate degree. Women athletes may now compete at all levels of competition. For example, there has been an increase in female athletic participation in high school from just fewer than 300,000 during the 1971-1972 school year to 2,570,333 during the 1997-1998. In addition, there are more opportunities to participate in the Olympics; in 1900, 24 women competed in the entire Olympic field, in the year 2000, 38,000 women competed in the Olympics in Sydney, Australia (Golden, 2002). Women also have a growing opportunity to compete at the collegiate level. Being an intercollegiate female athlete also has disadvantages.

The most noteworthy disadvantage can be over training. Over training can expose the athlete to a greater likelihood of injury, as well as, disruption of the hypothalamic-pituitary axis. Another disadvantage is meeting the expectations of the coach, peers, and even the athlete herself in terms of optimal body size. Picard (1999) found that athletes that compete at elite levels displayed increased symptoms of pathological eating, and thus, were at an elevated risk for developing eating disorders.

Statement of the Problem

Osteoporosis is one of the three most devastating diseases that can affect women. Only cardiovascular disease and cancer rival the impact that osteoporosis has on the health of U.S. women (Ford, Bass, Turner, Mauromoustakos, & Graves, 2004). Nearly, eight million new cases of osteoporosis are registered annually, many of which are Caucasian women (National Osteoporosis Foundation [NOF], 2003a; Slovik, 2000). Caucasian women have been reported to have up to a 50% chance of experiencing an osteoporotic fracture in their lifetime (NOF, 2003a). Consequently, prevention and early detection are imperative.

Female competitive athletes' are at particular risk for osteoporosis. Previous research in athletes has shown that weight-bearing exercise contributes to bone health. Female athletes have ample opportunity to increase their BMD in the course of training for their sport and/or while competing. Unfortunately, these same athletes are put at an amplified risk of encountering problems with their BMD due to the disruption of their hypothalamic-pituitary axis, also known as the reproductive axis. The hypothalamic-pituitary axis is a negative feedback system that stimulates and monitors female's menstrual and reproductive functions (Rickenlund et al., 2003; Warren & Shantha,2000).

Purpose Statement

The purposes of this investigation are: (1) to examine the extent to which (BMD) will be impacted by the type of sport (predominately lower body and/or predominately upper/lower body) among university Division I-A, female athletes (ages 18-25 yrs) and (2) to determine to what extent evidence of an eating disorder, BMI, percent body fat

(PBF), and episodes of amenorrhea contribute to the bone mineral status of university Division I-A female athletes.

Research Questions

Research Question 1

What is the effect of type of sport (predominately lower body and predominately upper/lower body) on BMD when controlling for episodes of amenorrhea, incidence of stress fractures, evidence of an eating disorder, the athlete's PBF and BMI.

Research Question 2

When controlling for limb preference and type of sport, the evidence of an eating disorder, PBF, BMI, and amenorrhea will predict BMD of the forearm (dominant and non-dominant) and femoral neck (dominant and non-dominant)?

Hypotheses

Hypothesis 1

The type of sport will effect the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, when controlling for episodes of amenorrhea, evidence of an eating disorder, PBF, and BMI.

Hypothesis 2

When controlling for BMI, past episodes of amenorrhea, PBF, limb preference and type of sport, evidence of an eating disorder will be positively related to the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes.

Hypothesis 3

When controlling for BMI, evidence of an eating disorder, past episodes of amenorrhea, limb preference and type of sport, PBF will be positively related to the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes.

Hypothesis 4

When controlling for the evidence of an eating disorder, PBF, past episodes of amenorrhea, limb preference and type of sport, BMI will be positively related to the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes.

Hypothesis 5

When controlling for the evidence of an eating disorder, PBF, limb preference and type of sport, and BMI past episodes of amenorrhea will be positively related to the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes.

Operational Definitions

Amenorrhea (Primary) is delayed menarche (no menstrual bleeding by age 16) and delayed pubertal changes (Teitz, 1997)

Amenorrhea (Secondary) is the loss of menses for at least six months in women that have had a least one mense (Teitz, 1997).

Anorexia Nervosa is the absence of the menstrual cycle for three consecutive months, and the loss of at least 15% of an individual's body weight through depriving the body of adequate nutrients to maintain homeostasis (Teitz, 1997).

Athletic Amenorrhea is the absence of menses for at least three or four menstrual cycles (Beals, Brey, & Gonyou, 1999).

Bone Mineral Density is the end result of dividing the bone mineral content by the surface area of the bone that is scanned (Mehler, 2003)

Bone Modeling is the process that determines the overall shape of the bone in reaction to demands, either physiological or mechanical (Ericksen, Axelrod, & Fleming, 1994).

Bone Remodeling is the cycles of bone formation and resorption (Zeni et al., 2000).

Bulimia Nervosa is when the individual may experience loss of control around food, binge eating two or more times a week for a period of a minimum of three months, and an abnormal body image (Teitz, 1997).

Cortical Bone is approximately 80% of the total bone in the human body. It is highly compact and organized, and it is located in the shell of cuboid-like bones (e.g., vertebral bodies, tarsal, and carpal) and in the diaphysis (shaft) of long bones (the appendicular skeleton) (Eriksen et al., 1994; Zeni et al., 2000).

Diaphysis is the long shaft of appendicular skeletal bones (Zeni et al., 2000).

Disordered Eating/Sub-clinical/Anorexia Athletica is excessive exercising with eating habits that range from insufficient caloric intake or unhealthy eating habits to binging and purging (Nattiv & Lynch, 1994; Stone, 1999).

Dual X-ray Absorpitometry (DEXA) is the low dose x-ray machine that measures site-specific, regional, and total bone mineral density.

Endosteal is a type of cortical bone which is responsible for the bulk of remodeling activity (Mundy, Chen, & Oyajobi, 2003).

Female Athlete Triad is three separate, yet, interrelated disorders (disordered eating, amenorrhea, and osteoporosis) that are grouped together to form the Triad (Nattiv & Lynch, 1994).

Follicle Stimulating Hormone (FSH) refers to the FSH acting on granulose cells to promote the transformation of androgens to estradiol (Bonen & Keizer, 1984).

Gonadotrophic Releasing Hormone (GnRH) is secreted from the hypothalamus to stimulate the release of lutenizing hormone (LH) and FSH from the anterior pituitary gland (Bonen & Keizer, 1984).

Haversian Canals connects cortical osteons. Each canal contains capillaries and nerve cells (Brukner, Bradshaw, Kahn, White, & Crossley, 1996).

Haversian System is the end result of the remodeling process in cortical bone, also known as a cortical osteon (Eriksen et al., 1994). The Lamellae and the haversian canals form this system (Zeni et al., 2000).

Hypothalamic Amenorrhea is the cessation of menses due to diminished levels of GnRH (Trash & Anderson, 2000).

Hypothalamic-pituitary Axis is the release of GnRH from the hypothalamus to stimulate LH and FSH from the anterior pituitary to release the ovum (Bonen & Keizer, 1984).

Lamellae are thin sheets of bone matrix (Brukner, et al., 1996).

Lutenizing Hormone (LH) is a hormone that participates in the hypothalamic-pituitary axis by acting upon the theca cells to produce androgens in conjunction with FSH (Bonen & Keizer, 1984).

Metaphysis is the transitional area of a long bone where the shaft merges with the epiphysis (Eriksen et al., 1994; Mundy et al., 2003).

Oligomenorrhea is the gradual onset of amenorrhea with lengthened intervals between menses of 35 days or more (Teitz, 1997).

Osteoblasts are cells found in the periosteum that actively produce bone matrix (mineral salts and collagen) (Guyton, 1991).

Osteoclasts are the cells that are responsible for resorbing the existing bone by secreting a substance that dissipates the mineral salt crystals of the matrix (Wingard, 1994).

Osteocyte is a single bone cell (Brukner et al., 1996).

Osteon is a basic structural unit, the end product of the remodeling cycle in cortical and cancellous bone (Eriksen et al., 1994).

Osteopenia is when BMD values lie between 1 and 2.5 standard deviations below the mean (Joy et al., 1997; WHO, 1994).

Osteoporosis is when BMD values are greater than 2.5 standard deviations below the mean (Joy et al., 1997; WHO, 1994).

Periosteal is a type of cortical bone which regulates the volume and fracture repair of the bone (Mundy et al., 2003).

Resorption is the removal of established bone in order to form new bone (Eriksen et al., 1994).

Stress Fracture is a minute or traumatic fracture that results in the athlete's inability to withstand sub-maximal stress that is applied repeatedly (Trash & Anderson, 2000).

Trabecular Bone/Cancellous comprises approximately 15% of the bone in the body (Mundy et al., 2003). It is located in the pelvis, long bones at the epiphysis and metaphysic, the rounded ends of long bones, and in cuboid-like bones (the axial skeleton) (Eriksen et al., 1994; Mundy et al., 2003).

Assumptions

1) Participants completed all study materials, including the Eating Attitudes Test (EAT-26), honestly and accurately.
2) The female athlete's included in this study are a representative sample of all university Division I-A, female athletics that participate in collegiate volleyball, soccer, golf, cheerleading, track and field, and softball.
3) There were a representative sample of athletes that participate in lower body and upper and lower body collegiate athletics.
4) Investigator bias was controlled via coding answer sheets on the EAT-26 and the demographic data. The investigator could encounter bias issues if she is exposed to the names of the subjects.

Limitations

1) Athletes who were in season or on vacation during the data collection period were not included in this study.
2) The athletes volunteered to participate in this study, therefore, an proportionate sample of athletes from all collegiate sports was not included.
3) Data on the EAT-26 tool were collected by self-report. Therefore, these data are subject to limitations associated with self-report.

Delimitations

1) The athletes represented different sports and varying training regimes at the university Division I-A level of competition.
2) This study was limited to a sample of collegiate Division I-A athletes at a university in the Southeast.
3) Due to the limited access to all sports at the university, the sport-to-sport effect on BMD was not generalized to all athletes that participate in a particular sport (e.g., soccer, volleyball, etc.)
4) Confidentiality is a major concern to most individuals who complete questionnaires. To alleviate this concern, the participants were assured that their answers will be kept confidential. The athlete's names were not used on the questionnaires. To ensure confidentiality, the researcher coded all data sheets. A master list of codes and names were seen only be the researcher.

Chapter II

Review of Related Literature

Introduction

The three most catastrophic diseases to women are osteoporosis, cardiovascular disease, and cancer (Ford et al., 2004). Osteoporosis is characterized by bone mineral density (BMD) measurements, as well as the occurrence of low-trauma fractures (Parsons, 2005). BMD is the product when dividing the bone mineral content (BMC) by the surface area of the bone scanned (Mehler, 2003). As many as 44 million Americans are at risk of developing osteoporosis, and approximately 34 million are affected by osteopenia (low BMD) (National Osteoporosis Foundation [NOF], 2004b). According to Slovik (2000), over 10 million new cases of osteoporosis are reported each year, contributing $13.8 billion in healthcare costs annually. Surprisingly, 80% or 8 million of the new cases that are reported are women. During the mid-1990's nearly two and one half million office visits, 432,000 hospitalizations, and 180,000 admissions were attributed to osteoporotic conditions (Parsons). In addition to the financial burden, osteoporotic fractures can be quite painful, bringing deformity, causing complications such as pneumonia or blood clots, and reducing the quality of life (Slovik).

This review of literature will consist of an overview of osteoporosis, the morbidity, the associated risk factors, and test for and diagnose osteoporosis. The focus of the review will then switch to the physiology of bone formation. From the physiology

of bone growth and development, the review will approach the stress injuries that can occur to bone and the risk factors for stress injury. Next, the review will narrow and discuss how female athletes may encounter injury to bone. This will bring the female athlete triad (FAT) to the forefront of the review. The FAT, which includes three interrelated conditions (eating disorders, amenorrhea, and osteoporosis) is defined and provides the framework for which the remainder of the review unfolds. Each component of the FAT is described. The term "eating disorder" is defined and dissected into various expressions of the malady (e.g., anorexia, bulimia, etc.) and how BMD in the female athlete can be affected by the presence of the disorder. Eventually, a similar description is produced for amenorrhea. Finally, the review will evolve into the focus of this investigation, how training affects the BMD of female athletes.

The Morbidity of Osteoporosis

Osteoporosis results from an imbalance between bone formation and resorption (Whyte & Marting, 2004). It is considered the most widespread metabolic disease in the U.S. and is distinguished by low BMD (Leslie & St. Pierre, 1999). BMD is a representation of the bone's mineral content (Slovik, 2000). BMD is determined by scanning an area of bone mass and estimating the amount of mineralized tissue. It is measured in g/cm^2 (Slovik). The result of the scan is expressed as a T-score. The T-score is the standardized value that is considered the reference for determining osteoporosis. A diagnosis of osteoporosis is determined by standard deviations from what is considered the norm for age, gender, and race (Slovik, 2000; The World Health Organization [WHO], 1994). The WHO (1994) has established diagnostic categories for Osteoporosis. For example, a T-score of greater than 2.5 *SD* below the mean constitutes

a diagnosis of osteoporosis, while a T-score between 1 and 2.5 identifies osteopenia.

Siris et al. (2004) used data from the National Osteoporosis Risk Assessment to explore the risk of fracture within one year of measurement on 149,524 Caucasian postmenopausal women (50 – 104 years.). The researchers revealed that 2,340 new osteoporotic fractures had occurred over the year. Of the women who received fractures, 80% had T-scores greater than -2.5. According to the NOF (2004b), a T-score less than -2.0 or a T-score less than 1.5 accompanied by clinical risk factors, should receive treatment. Merely 22.6% of the fracture recipients in this study would have been targeted for treatment (Siris et al.).

Osteoporotic Fractures

Over 1.5 million fractures of the wrist (Colles' fracture), hip, and vertebral fractures occur annually in the U.S. (Paier, 1996). Fractures of the wrist, hip, and vertebrae, particularly in the elderly, can lead to social isolation, loss of independence, and open the door for a host of other illnesses (e.g., pneumonia and deep vein thrombosis) (Brew, 2003). According to the National Institute of Health (National Institute of Health [NIH], 2001), only one third of people who have experienced an osteoporotic fracture regain their pre-fracture mobility. Further, one in three elderly individuals that sustain a hip fracture will end up in a nursing home; while one in five dies within a year of their injury. The major causes of death related to osteoporotic fracture tend to be atherosclerosis, stroke, and pulmonary diseases (e.g., pneumonia, chronic obstructive pulmonary disease [COPD], etc.) (Kado et al., 1999; McBean, Forgac, & Finn, 1994).

Hip fractures tend to have the most far reaching health implications to the elderly.

The risk for Americans to encounter a hip fracture during their lifetime is 17.5% for women and 6.0% for men, and it is anticipated that this rate will nearly double by the year 2040 (Siris et al., 2004). The risk of an osteoporotic hip fracture doubles for every one standard deviation from the mean, (Cummings et. al., 1993). Cooper, Campion, and Melton (1992) predicted that there will be a five-fold increase in hip fractures for persons 65 and older by the year 2050. Further, persons 65 years and older in the United States, represent more than 340,000 hospitalizations yearly from hip fractures (Cummings & Melton, 2002). In one prospective study, the overall incidence of hip fracture annually occurred at a rate of 104/100,000, in a population heavily represented by 167 women, and 35 men, *M* age = 76 to 84 years (Rizzoli & Bonjour, 1997). Fractures of the hip can be the most debilitating fracture to the elderly. Generally, long-term rehabilitation and nursing care is warranted (Slovik, 2000). Advanced age also brings additional health issues, such as decreased flexibility and muscle tone and problems with equilibrium which can amplify the propensity of injury.

Osteoporotic vertebral fractures are the most common fractures (Boonen et al., 2004). The prevalence of vertebral fractures is 5% to 10% in men and women aged 50 to 54 years. The incident increases from 45% to 55% in men and women aged 80 to 89 years (Kanis & McCloskey, 1992). In women over 65, vertebral fractures area related to a 23% age-adjusted mortality rate (Siris et al., 2004). Vertebral fractures are painful and may leave the individual with a kyphotic posture. Perhaps the most dramatic expression of an osteoporotic fracture is experienced in the proximal femur.

A fracture to the femur can be most devastating. A femur fracture is not only painful, but carries with it the sign of significant osteoporotic fragility. Due to the rising

incidence of osteoporosis, the number of proximal femur fractures has exponentially increased (Rizzoli & Bonjour, 1997).

Osteoporotic Gender Differences

Caucasian women have a greater likelihood of experiencing an osteoporosis-related fracture than men or women of other ethnicity in their lifetime. In fact, the incidence of osteoporosis may be as high as 50% in Caucasian women (NOF, 2003b). Further, for every four men, one woman will develop osteoporotic symptoms (NOF, 2004b; Taylor, & Sicard, 2003). Women also are at an increased risk of developing osteoporosis due to estrogen fluctuations just prior to and during menopause (Parsons, 2005).

Men are not subject to the same hormonal fluctuations as women, yet, they are not impervious to osteoporosis; more than two million men in the United States are diagnosed with osteoporosis (NOF, 2000). Three and one half million American men have the precursor to osteoporosis, osteopenia (*Harvard Men's Health Watch*, 1999). Additionally, 25% of hip fractures annually are experienced by men. Research defining risk factors, diagnosis and treatment of osteoporosis for men are often overlooked.

Osteoporosis is serious health condition affecting elderly men and women which is often masked until a fracture transpires. Postmenopausal women are at a particular risk for developing osteoporosis. Men and women can suffer debilitating fractures that can hamper mobility and limit quality of life. Vertebral fractures are the most common osteoporotic fractures, however, hip and wrist fractures occur frequently as well. The risk factors for developing osteoporosis need to be acknowledged in order to lessen the chance for the onset of the disease.

Risk Factors of Osteoporosis

This section will examine the risk factors associated with osteoporosis. These risk factors express themselves in various ways. There are risk factors that cannot be controlled, such as age, postmenopausal status, and genetics (Melton et al., 2004; Thomas, 1997). There are also nutritional and lifestyle factors that can contribute to the occurrence of osteoporosis that are controllable. Lastly, there are certain drugs and medical conditions also contribute to osteoporotic bone loss.

Uncontrollable Osteoporotic Risk Factors

There are several risk factors associated with osteoporosis that are unable to be managed (Melton et al., 2004; Thomas, 1997). For example, the result of aging on bone health causes insufficient bone matrix development due to decreased levels of growth hormone and deteriorating protein anabolic functions (Melton et al.). Gender also plays a role in BMD management. Men and women experience bone loss at .3% to .5% around age 40 years (Leslie & St. Pierre, 1999). Moreover, women are likely to experience an additional bone loss of 3% to 5% per year for up to seven years, as a result of diminished levels of estrogen that occurs during and after menopause (Leslie & St. Pierre, 1999; Melton et al., 2004;). Certain genetic characteristics such as race and body type can place individuals at greater risk for osteoporosis. For instance, persons of Asian or Caucasian decent have a slight body frame (ecktomorphs) and are more likely to encounter osteopenia (Scheiber & Torregrosa, 1998). Genetic factors can contribute 70% to 80% of the potential for peak bone mass (Pollitzer & Anderson, 1989). The remaining 20% to 30% of risk associated with osteoporosis seems to be attributed to nutritional and lifestyle factors that can be controlled.

Controllable Osteoporotic Risk Factors

There are various nutritional and lifestyle features that contribute to low BMD. These features include low calcium and vitamin D intake and milk intolerance, vegetarian diet, high protein diets, lack of physical activity, and excessive exercise (Leslie & St. Pierre, 1999; Thomas, 1997). Calcium and vitamin D intake are important components of bone formation, maintenance, and retention (Leslie & St. Pierre). Calcium, unlike vitamin D, is not manufactured in the body. Vitamin D can be fabricated in the human body via exposure to sunlight. Yet, this source may not be enough to supply the adequate amounts the body needs, 400 to 800 IUs of Vitamin D daily is recommended (Parsons, 2005). Food sources rich in vitamin D include vitamin D enriched dairy products, egg yolks, and liver. Vitamin D is important to include in a balanced diet due to its ability to assists with the absorption of calcium, and calcium is necessary for proper bone development and retention.

Calcium deficiency is a problem among women of all ages, but especially in ages 12 to 29, which is the crucial period for developing peak bone mass (Fleming & Heimbach, 1994; Greer, 2005). Merely 10% of young women during adolescence, ages 12-19 years, partake in the RDA for calcium (Greer, 2005). The mean calcium intake in the 20 to 29 age group is 650 milligrams per day, far less than the recommended daily allowance (RDA) of 100 milligrams per day (Thomas, 1997). The RDA for women aged 29 to 50 is 1000 milligrams per day as well, and for women over 50, 1,500 milligrams is suggested (Fleming & Heimbach; Thomas). Approximately 99% of the calcium ingested is directed toward building and maintaining the bones and teeth (Thomas).

Certain parts of the skeletal system seem to be more greatly effected by calcium.

Moreover, calcium supplementation could have a site-specific effect on BMD. Winters-Stone & Snow (2004) in their study conducted with female distance runners (aged 23.7 +/- 4.7 years.), discovered that there was a site-specific effect of calcium supplementation to the femoral mid-shaft, a bone that consists predominately of cortical bone. The tibia, a site that is common to bone injury, is composed primarily of cortical bone like the mid-shaft of the femur (Winters-Stone & Snow). They concluded that female runners who partake in at least 1000 mg/day of calcium, protect cortical bone but the routine has little to no effect on trabecular bone.

Most women do not eat a diet that is rich in calcium; thus, they fail to ingest the appropriate daily amount, and supplementation should be considered. This trend is not only seen in the general population but in female athletes as well. Most female athletes are unaware of the nutritional needs of their body. Often their diets will be void of calcium and other important nutrients that their exercising bodies require to perform at the optimal level.

Conversely, female athletes can go to extremes in their diets by including an abundance of nutrients that can be detrimental to the body when taken in excess. A common misperception is that a diet that includes excessive protein can produce heightened muscle mass. Actually, a diet dominated by excessive amounts of protein can cause the body to retain excessive acid. In an attempt to buffer the acid, dissolution of the bone may occur (Leslie & St. Pierre, 1999). Nutritional balance is an important element in maintaining peak bone mass, yet, a variety of lifestyle factors are also associated with the maintenance of BMD.

One lifestyle factor that is inherent in female athletics is ongoing physical,

activity, specifically weight-bearing activity. The absence of physical activity can result in compromised BMD. In fact, Lanou, Berkow, and Barnard (2005) and Lloyd, Petit, Lin, and Beck (2004) concur that physical activity is the most significant modifiable lifestyle component associated with bone growth and strength. Several research studies have documented the benefit of weight-bearing exercise on the development and maintenance of BMD (e.g., Bassey & Ramudale, 1994; Drinkwater, 1994; Heinonen et al., 1996). For instance, Heinonen et al. (1996) studied 98 sedentary women aged 35-45 years. The group was divided equally into a high-impact exercise group and control. The results showed that BMD improved significantly in the training group (M = 1.6% [CI 0.8-2.4]) in comparison to the control group (0.6% [-0.2 to 1.4, p = 0.60). In contrast, bone can be negatively influenced by inactivity and non-weight bearing activity (Feicht, 1990; Second International Conference on Osteoporosis, 1997). Heinonen et al. (1996) measured bones at non-weight bearing sites (e.g., distal radius) and found no significant differences between the training and control groups. The result of this investigation indicates that weight-bearing activity aids in the fortifying of BMD. Results of a study performed by the Centers for Disease Control (CDC) in 1995 revealed that only 30% of college women aged 18-24 engage in weight-bearing exercises. Thus, 70% of college women aged 18-24 are potentially not building peak bone mass during a crucial period of time; therefore, placing them at risk for issues related to low BMD later in life. While, college-aged athletes participate in weight-bearing that positively affects BMD during training, other lifestyle factors that may accompany training and college life can negatively impact the BMD of female athletes.

The use of substances that adversely affect BMD, such as steroids, cigarettes,

alcohol, and caffeine are additional lifestyle factors that place all persons at risk for developing osteoporosis (Leslie & St. Pierre, 1999; Thomas, 1997). For example, steroid use increases calcium excretion during urination, while decreasing calcium absorption resulting in increased levels of parathyroid hormones. Elevated levels of parathyroid hormone can vary gonadal hormone secretion and cease osteoblast function (Leslie & St. Pierre). Bone loss has been recorded with as little as 7.5 milligrams of steroid taken per day for only one month (Leslie & St. Pierre). Females do not generally use steroids to the same extent as their male counterparts; nevertheless, other lifestyle factors such as cigarette smoking and alcohol consumption may be similar for both genders.

Cigarette smoking not only exposes women to an augmented risk for cardiovascular disease, but also elevates the probability of osteoporosis due to decreased levels of serum estrogen (Thomas, 1997). Additionally, smokers tend to have low body weight, perhaps due to the oral gratification that replaces eating experienced by most smokers (Wardlaw & Weese, 1995). Low body weight can further expose an individual to issues with their BMD. While, enhanced body weight has been associated with positive BMD outcomes due to the increased mechanical stress on the weight-bearing bones (Thomas). Therefore, the combination between low body mass and fading serum estrogen levels leads to an elevated risk for osteoporosis.

Other factors that contribute to loss of BMD are alcohol and caffeine consumption. Alcohol multiplies the risk of osteoporosis by depressing osteoblastic activity and interfering with proper nutrition (Lappe, 1994; Thomas, 1997). Caffeine affects BMD because, when taken in excess, caffeine elevates calcium excretion in the urine and reduces the available calcium in the blood (Wardlaw, 1993; Wardlaw & Weese,

1995). The controllable risk factors associated with decreased BMD are not limited to extraneous substances (e.g., caffeine, alcohol, etc.) that the athlete may consume, but also to substances that the athlete may require to maintain her health such as prescribed drugs and medical treatments.

There are certain prescribed medications that can amplify the risk for developing osteoporosis. The various drugs that are contraindicated are anticonvulsants, chemotherapy, chronic antacid and tetracycline (an oral antibiotic) use, cyclosporine A (used to prevent rejection of kidney, heart, or liver after transplants), glucocorticoid therapy (used for symptoms of rheumatoid arthritis), gonadotropin-releasing hormone, lithium (antipsychotic agent), methotrexate (antirheumatic agent), thyroid hormone, and phenothiazine derivatives (antipsychotic agents) (Martin & Yates, 1998; NIH, n.d.; Scheiber & Torregrosa, 1998; Thomas, 1997).

Finally, there are definite medical conditions that exasperate the risk for osteoporosis. The medical conditions include anorexia nervosa, Cushing's syndrome, diabetes mellitus type I, gastrointestinal dysfunction, hepatobiliary dysfunction, hemolytic anemia, mastocytosis, osteogenesis imperfecta, parathyroid over activity, prolonged parenteral nutrition, prolactinoma, low testosterone production, steroid therapy, some neoplastic diseases, gastric surgeries, rheumatoid arthritis and transient osteoporosis (Baskins, 1996; Scheiber & Torregrosa, 1998). Athletes that might have a medical condition that further predisposes the propensity for bone injury, should be watched for signs and symptoms of osteoporosis.

There are various risk factors that predispose an individual to osteoporosis. The risk factors can be delineated into two categories, uncontrollable risk factors and

controllable risk factors. Uncontrollable risk factors include gender and genetics, while controllable factors incorporate a number of lifestyle influences. For example, vitamin and mineral intake, vegan diets, high protein diets, inadequate physical activity, and/or excessive exercise can impact BMD. Additional sources of concern involve the use of steroids, certain prescribed medications, cigarettes, alcohol, and caffeine to excess. Certain medical conditions can also exasperate the propensity of encountering osteoporosis. Clearly, early diagnosis and testing is warranted to combat the onset of this debilitating disease.

BMD and Diagnosis of Osteoporosis

WHO (1994) has classified osteoporosis as a disease that results in micro architectural deterioration of bone tissue that leads to low bone mineral density (BMD) and fragility. Yet, the signs and symptoms often appear long after extensive damage has already been done to the skeleton. The National Osteoporosis Foundation (NOF) advocates that certain at risk individuals (e.g., estrogen-deficient women, amenorrheic female athletes, women with osteopenia or vertebral, and women with multiple risk fractures) should receive screening in order to diagnose the likelihood of developing osteoporosis or osteopenia (Erickson & Sevier, 1997; Kanis et al., 1994). Additional conditions should be recognized as potential threats to bone health and be submitted to BMD screening (e.g., abnormalities revealed on radiographs, patients who will undergo glucocorticoid therapy, individuals with asymptomatic primary hyperparathyroidism, and those who have experienced fractures that could be symptomatic of osteoporosis). If osteoporosis is diagnosed, the patients should be evaluated to determine if there are any variables that can be reversed to stop BMD loss. The assessment should consist of a

chemistry panel, sedimentation rate, thyroid-stimulating hormone (TSH) test, estrogen levels, LH and FSH, prolactin levels and a urine pregnancy test (Erickson & Sevier). This section will address the most commonly used methods to diagnose osteoporosis.

Diagnosis of Osteoporosis

Diagnosis of osteoporosis is not predicated upon the presence of a fracture or clinical expressions of the disease, but on quantitative measures, expressed as a T-score, from which the risk of fracture can be predicted (Rizzoli & Bonjour, 1997). There are no universal standards for predicting osteoporosis. The methods used most often to calculate BMD are bone biopsy, quantitative computed tomography (QCT), dual-photon absorptiometry (DPA), ultrasound and dual x-ray absorptiometry (DEXA) (Erickson & Sevier, 1997; Slovik, 2000). QCT is the most commonly utilized method to measure the BMD of the spine, but it can also be employed to measure other sites. DPA measures the spine, hip, and total body, yet DPA is used infrequently (NOF, 2003a). There are additional methods of calculating BMC that focus on peripheral sites, such as the wrist, heel, or finger. Peripheral dual e-ray absorpitometry (pDXA), single energy x-ray absorptiometry (SXA), peripheral quantitative computed tomography (pQCT), radiographic absorptiometry (RA), and single photon aborptiometry (SPA) are all noninvasive methods of computing BMD (NOF, 2003a). Bone biopsy can bring irrefutable results, however; it is not normally recommended due to the invasive nature of the procedure (Erickson & Sevier, 1997). Expense is a further consideration in osteoporosis scanning. Therefore, the quantitative ultrasound device is commonly utilized for its cost effectiveness and convenience.

The ultrasound device was approved for use to measure BMD in 1998 by the

Food and Drug Administration (Slovik, 2000). The ultrasound provides a BMD estimate by sending high frequency sound waves though the heel, shin, or kneecap. A computer reads how quickly the sound waves pass through the bone that is being measured. The BMD measurement will be expressed in g/cm^2 (Slovik). The ultrasound is a versatile method to measure BMD; it is not complicated to operate, and it is compact and lightweight. The DEXA is the most frequently employed method of measuring BMD (Erickson & Sevier, 1997; Slovik).

The DEXA is validated for measurement of the entire body, lumbar spine and proximal femur, and at peripheral skeletal sites such as the forearm. The lumbar spine and proximal femur are at particular risk of developing osteoporotic fractures, thus, the importance of the DEXA cannot be overlooked (Johnston, Slemenda, & Melton, 1991). It is now considered the gold standard for measuring BMD. The DEXA has a margin of error of 1% to 2% and provides an extremely low dose of radiation, approximately 5 mrem per scan (Rome, 2003). A chest x-ray emits 20 to 50 mrem, while a full dental x-ray emits 300 mrem (Erickson & Sevier, 1997; Snow-Harter, 1994). The DEXA was chosen as the BMD device in this investigation due to the high validity and reliability that has been established for the machine. The DEXA does emit a low dose of radiation, and it is exceptionally safe. Up to 30% bone loss can not be determined by routine x-ray. DEXA will diagnose marginal bone loss. Therefore, is the diagnostic tool of choice when determining osteopenia and/or osteoporosis (Kessenich, 2001).

The DEXA provides an approximation of the true volumetric density, as well as estimate the size and thickness of the bone in question (Rizzoli & Bonjour, 1997). This is achieved by using two different types of energies that delineate bone from the adjacent

soft tissue (Rome, 2003). The American College of Sports Medicine (American College of Sports Medicine [ACSM], 1995) has reported that a two to three-fold plunge in BMD can be attributed to a one *SD* decrease on the T-score. A diagnosis of severe osteoporosis is considered when one or more fractures have occurred in conjunction with a T-score of 2.5 *SD* below the mean (WHO, 1994). Longitudinal studies have indicated that a decrease of only one standard deviation from the mean score can expose an individual to the risk for fractures (Barrett-Conner, 1995; Marshall, Johnell, & Wedel, 1996; Melton, Atkinson, O'Fallon, Wahner, & Riggs, 1993). Prevention and treatment should be considered when a diagnosis of osteopenia or osteoporosis is established.

Prevention and Treatment of Osteoporosis

Exercise, estrogen replacement, and calcium supplementation have proven to be successful therapies for the prevention and treatment of osteoporosis (Erickson & Sevier, 1997). Yet, an active lifestyle that includes the proper diet and supplementation alone may not prevent the silent progression of osteoporosis. Pharmacologic intervention could possibly be necessary. There are drugs that have recently emerged on the medical scene for the treatment of bone loss.

The role of drug therapy in the treatment of compromised BMD. Pharmacological intervention (i.e., drug therapy), in conjunction with diet, exercise, calcium, and vitamin D, supplementation has become increasingly popular for the management of BMD. The therapeutic effect for majority of drug therapies operates in one of two ways, by either limiting bone resorption or by increasing BMD. The frequently used drug therapies for treating the evidence of osteoporosis include the bisphosphonates (alendronate and risedronate), calcitonin (Miacalcin), and raloxifene (Evista) (Lehne, 2004; NOF, 2003a).

These drug therapies are approved by the FDA, and work by limiting bone resorption, with the exception of raloxifene (NOF, 2004a). Raloxifene is also approved by the FDA, but it simply maintains BMD and lowers plasma levels of cholesterol (Parsons, 2005).

The biophosphonates (Alenronate and Risendronate) are two of the most universally prescribed drugs to treat osteopenia and osteoporosis (Adami, Prizzi, & Colapietro, 2001; Ringe, Faber, & Dorst, 2001). Researchers have shown that persons who take this drug reduce the incidence of fractures of the spine and hip by as much as 50% (NOF, 2003a). Alendronate is ingested in pill form either daily (5 milligram) or weekly (35 milligram) to limit bone resorption. Risedronate has demonstrated a 30% to 40% reduction rate in the incidence of fractures of the hip and vertebrae (Parsons, 2005).

An additional oral drug that lessens the frequency of fractures of the vertebrae by up to 40% is Raloxifene (Parsons, 2005). Raloxifene is a selective estrogen receptor modulator (NOF, 2003a). Raloxifene is taken in pill form (60 milligram daily). Further medical interventions for the treatment of osteoporosis can be administered via a nasal spray or by injection.

The NOF acknowledges that calcitonin is a viable intervention for the treatment of osteoporosis (NOF, 2003a). Calcitonin is administered by means of a nasal spray (100 IU) (Parsons, 2005). It may moderate the risk of fracture in the vertebrae by up to 35%. It is a polypeptide hormone that limits the osteoclasts activity, therefore lessening the resorptive action in the bone (Parsons). Yet, the most efficient treatment for osteoporosis could be presented in an injectable form.

One of the latest drugs approved by the Food and Drug Administration (FDA) (2002) for the treatment and prevention of osteoporosis is teriparatide (Forteo) (LoBuona;

2003; NOF, 2004d; NOF, 2004e). Teriparatide is the initial medication in an innovative class of bone-forming agents. This new class of drugs amplifies the development of new bone by boosting the action of the osteoblasts (Parsons, 2005). Teriparatide is an injectable that is dispensed daily in the thigh or abdomen. Teriparatide consists of parathyroid hormone, a major regulator of phosphate and calcium metabolism. Teriparatide seems to be a progressive solution in the treatment of osteoporosis, yet, the long-term effects are not known. Therefore, this treatment is recommended for limited use, generally for two years in duration (NOF, 2003a).

The newest drug that has been approved for human use is ibandronate sodium or Boniva (FDA, 2003). Boniva works by inhibiting osteoclasts, and therefore, limiting bone resorption. In postmenopausal women, Boniva limits levels of biochemical markers of bone turnover to that of premenopausal women. It is taken orally once per month, although, an injectable that can be administered once every three months is being investigated (Anonymous, 2005). Approximately 1400 postmenopausal women were subjected to a three year double blind trial on Boniva. A daily dose of 2.5 mg of Boniva and a placebo were administered to the participants. The results indicated that significant improvements were made to BMD at the lumbar spine (6.4% vs. 1.4% in the control sample), total hip (3.1% vs. -.7 in the control sample), femoral neck (2.6% vs. -.7 in the control sample), and (5.3% vs. .2% in the control sample) (FDA, 2003). Chesnut et al., (2005) found similar results in another double blind study of a sample of 2947 postmenopausal women who received either daily or intermittent (greater than two months) doses of Boniva. The data from this study found that North American women had an efficacy of 60% with daily doses and 54% with intermittent doses. This finding

suggests that Boniva remains effective over time, therefore, patients may not have to take it as frequently, and greater adherence rates can be expected.

Pharmacological therapy is producing positive results in treating osteoporosis and osteopenia; unfortunately, drug therapy, unaccompanied by additional therapies, will not successfully treat osteopenia in the female athlete. Another important form of treatment for female athletes, and women in general, is exercise.

The role of exercise in the treatment of compromised BMD. The American College of Sports Medicine (1995) has developed a position statement concerning the role of exercise in the prevention and treatment of osteoporosis. The statement makes three distinct recommendations concerning exercise and bone health for all women: (1) that further bone loss may be averted if sedentary women increase their physical activity level, (2) that physical strength building activities are advantageous, especially to non-weight bearing bones, and (3) that weight-bearing exercise is necessary for building and preserving BMD. Exercise is the first line of defense in promoting and preventing the loss of BMD. A number of studies have shown that exercise positively impacts the bones of women (e.g., Grove, & Londeree, 1992; Michel et al., 1989; Nelson et al., 1994). Michel et al. (1989) determined that women over the age of 50 years, who ran two to three times per week, had a 9.2% to 35% higher BMD in the lumbar spine than women who were sedentary (Michel et al. 1989). Augestad, Schei, Forsmo, Langhammer, and Flanders (2004) in their longitudinal, population-based health study discovered that premenopausal women, who had high levels of recreational and occupational physical activity, possessed significantly greater BMD of the forearm than premenopausal women who led a more sedentary life. It appears then that physical stress is an important

component to building and maintaining BMD integrity.

Physical stress exerted upon bones increases the stimulus of bone remodeling and strengthening. The remodeling is specific to the area that is receiving the stress (Lanyon, 1992). Bone must receive a stress stimulus that is greater than it is accustomed to in order to adapt and generate enhanced BMD. Short, but intense patterns of weight-bearing activity provides the appropriate motivation to stimulate remodeling; although, varying rates of strain provide the maximum osteogenetic response (Whyte & Marting, 2004).

High-impact and high-intensity exercise is generally anaerobic in nature. Conversely, aerobic physical activity that is low-impact and low-intensity (e.g., swimming and bicycling) does not provide the proper impetus to stimulate BMD (Whyte & Marting, 2004). An exercise regime that includes anaerobic exercise to promote bone health and aerobic exercise to foster cardiovascular fitness, can be a balanced method to maintain optimal health and vitality. Yet, many female athletes exercise both aerobically and anaerobically beyond what is necessary to compete in their sport, which can place their health in jeopardy.

Exercise that exceeds beneficial limits has a negative effect on the female athlete. Excessive exercise can take two forms, either the extra training is implemented by the athlete to meet an internal standard or the over training is mandated by the coaching staff, which provides an external stimulus. Excessive exercise can place undo stress on the female endocrine system resulting in an imbalance in the hypothalamic-pituitary axis. If the hypothalamic-pituitary axis has a breach in homeostasis, the athlete may lapse into a hypo estrogenic state which can result in amenorrhea (Erickson & Sevier, 1997).

Anaerobic exercise that stimulates bone formation exclusively, cannot negate the effects of damage that can occur due to a hypo estrogenic state in young amenorrehic athletes. Therefore, hormone replacement therapy can be advocated to overcome the hormonal deficit associated with amenorrhea (Keen & Drinkwater, 1995).

The role of hormone replacement therapy in the treatment of compromised BMD. There is some controversy regarding the role of hormone replacement therapy (HRT), specifically estrogen replacement, in the reversal of BMD loss (Erickson & Sevier, 1997). HRT is the use of certain sex hormones (e.g., estrogen, progesterone) to bring homeostasis to the fragile environment of the female endocrine system. The results of one investigation indicated that estrogen replacement did not bolster BMD (Keen & Drinkwater, 1995). This suggests that bone loss cannot be reversed. Subsequently, a number of reputable studies have refuted these findings. For example, the investigations included women ranging in age from 17 to 40 years; and showed an improvement of 2% to 8% BMD of the lumbar spine (Cumming, 1996; Gulekli, Davies, & Jacobs, 1994; Hergenroeder, Klish, Smith, & Ellis, 1995). For women over 50 years, there is a four percent chance of developing breast cancer from participating in estrogen replacement therapy. There are increased cardiovascular benefits to partaking in estrogen replacement therapy.

The risk of cultivating breast cancer is a disadvantage to estrogen therapy, but cardiovascular protection may provide an advantage. Women who are on estrogen replacement therapy have only a 15% risk of cardiac death as opposed to 30% risk to women who do not take therapy (Erickson & Sevier, 1997). Estrogen therapy alone may not be sufficient to prevent the loss of bone mass in female, maintaining a proper amount

of calcium and vitamin D in the diet can further negate the loss of BMD.

The role of vitamin and mineral therapy in the treatment of compromised BMD. Calcium and vitamin D are important elements in preserving and building BMD in women of all ages. Both are recommended for the treatment of osteoporosis. Calcium and vitamin D work in conjunction to promote bone health. Vitamin D helps the body absorb calcium, and calcium works to build BMD. The NIH (1994) released a statement regarding the amount of dietary calcium required for females. Female's ages 11 to 24 years are recommended to consume 1,200 to 1,500 milligram of calcium per day. Likewise, the NIH recommended that female athletes that are experiencing menstrual irregularities or have disordered eating behaviors should consume 1,200 to 1,500 milligram of calcium per day, as well as, ingesting 400 to 800 IU of vitamin D daily (NIH). Additionally women ages 25 to 65 should consume 1,000 milligram or calcium per day, and postmenopausal women between 50 and 65 who are taking estrogen should consume 1,500 milligram of calcium per day (NIH, 2001).

BMD testing and diagnosis is paramount in treating osteoporosis. A diagnosis is general determined by performing a BMD scan. The preferred method for scanning BMD is the DEXA. Supplementary tests should be executed to form a profile for the patient (e.g., a chemistry panel, sedimentation rate, TSH, estrogen levels, LH an FSH, prolactin levels, and a urine pregnancy test). A number of influences contribute to the treatment and prevention of osteoporosis. For example, drug therapy plays a role halting bone loss and fortifying BMD. Regular exercise, especially during the years prior to and post adolescence helps to maximize BMD. Finally, insuring the proper intake of vitamin D and calcium will provide an individual an opportunity to optimize and protect BMD by

contributing to the physiology of bone formation.

The Dynamic Process of Bone Formation

Bone is a dynamic living tissue with structural and metabolic mechanisms, which work in unison and separately from one another to produce and maintain BMD and regulate mineral homeostasis throughout a lifetime (Trash & Anderson, 2000; Zeni et al., 2000). Maintaining the skeletal constitution is synchronized by the structure mechanism; likewise, mineral homeostasis is sustained through the metabolic system. Both components are involved in bone remodeling (Zeni et al.). Bone remodeling consists of cycles of bone formation and bone resorption (Nattiv & Armsey, 1997). In approximately the first three decades of life bone formation exceeds bone resorption. After this period, the bone resorption process dominates and BMD begins to decrease (Nattiv & Armsey). Peak bone mass generally occurs between the ages of 25 and 30; although, there is some controversy over the exact age (Leslie & St. Pierre, 1999; Nattiv & Armsey; Zeni et al.). Females acquire the bulk of their BMD in their adolescent growth spurt (ages 11-14), while males begin somewhat later in adolescents (ages 13-17) (Nattiv & Armsey). After a woman has completely ceased menstruating (approximately 90% of her circulating estrogen has been depleted from her system) the rate of bone loss may increase to 3% or more per year (Steinwig, 2002). The presence or absence of estrogen seems to have an overwhelming impact on BMD in females.

Types of Bone in the Human Body

There are two types of bone in the mature human body, cortical and trabecular or cancellous bone. Cortical and trabecular bone are classified as lamellar bone (Zeni et. al, 2000). Lamellar bone is highly structured tissue that replaces the bone tissue (woven

bone) present in embryos and children up to age four. It is also anisotropic (the direction of the force applied will impact the mechanical properties of the bone) due to the stress-oriented collagen that is in its composition (Montelone. 1995) Nearly, 80% to 85% of maturing skeletal bone consist of cortical or compact bone. The remaining 15% to 20% is composed of trabecular (cancellous) bone (Mundy et al., 2003). Cancellous bone represents a fraction of the total skeleton, but it requires 50% of the metabolic activity of bone (Eriksen, Axelrod, & Melsen, 1994). The total mass of cortical bone is four times larger than that of trabecular bone (Zeni et al.).

Cortical bone. Cortical bone is primarily composed of compacted osteons that are present along the length of the bone and are connected by channels known as haversian canals. Osteoblasts build new bone by depositing a mineralized and collagen substance to create thin sheets of matrix called lamellae (Guyton, 1991). The lamellae form circular boundaries around the haversian canals, together form the Haversian system, also called an osteon. Outside the lamellae are minute fissures, or lacunae (Brukner et al., 1996). Lucanae house a single bone cell, known as an osteocyte. Osteocytes have thin projections (canaliculi) that extend from the lacunae into the canals within the bone to offer nutrition and metabolic transport within the bone (Guyton, 1991). Bone formation transpires and resorption ceases along the outer perimeter of an osteon. The perimeter becomes a rigid boundary (Zeni et al., 2000). Cortical bone is, highly organized, and withstands compression forces far greater than tension (force created by muscle exertion on bone) (Brukner et al., 1996). Cortical bone can be found primarily in the shell of cuboid-like bones (e.g., vertebral bodies, tarsal, and carpal) and in the diaphysis (shaft) of long bones (the appendicular skeleton) (Eriksen et al., 1994; Zeni et

al.). The metabolic turnover of cortical bone is eight times slower than trabecular bone.

Trabecular or cancellous bone. The density of trabecular bone is substantially inferior to that of cortical bone due to its elevated sensitivity to hormonal fluctuations (Klossner, 2000). As a result of this sensitivity, trabecular bone undergoes a greater amount of remodeling (Zeni et al., 2000). The lamellae in trabecular bone are arranged into an irregular network of mesh-like burrows, which is not as dense as cortical bone. The asymmetrical configuration defines how the trabecular bone endures stress (Romani, Gieck, Perrin, Saliba, & Kahler, 2002). Trabecular bone is composed of trabeculae (thin plates of bone). The trabeculae are interconnected. Hematopoiesis or blood-cell formation occurs between trabeculae (Wingard, 1994). Cancellous or trabecular bone is located in the pelvis, long bones at the epiphysis and metaphysic (the transitional area of a long bone where the shaft merges with the epiphysis), the rounded ends of long bones, and in cuboid-like bones (axial skeleton) (Eriksen et al., 1994; Mundy et al., 2003).

The BMD of trabecular bone is somewhat elusive to measure. The measurement phenomenon is due to the reduced density, greater sensitivity to hormonal vacillations, and accelerated bone turnover rate characteristic of trabecular bones. Accordingly, BMD is generally measured in regions containing principally cancellous bone (sacrum, vertebral bodies, and femoral trochanter). Logically, this is where the maximum variation in BMD is expected to be observed (Zeni et al., 2000).

Bone Remodeling

Bone remodeling is a process that takes place throughout the life span of an individual. During this procedure, bone is refurbished incessantly in various pockets throughout the skeletal system by removing established bone (resorption) and replacing it

with freshly synthesized bone matrix (bone formation). Additionally, remodeling, along with the kidneys, stomach, and intestines, helps facilitate calcium homeostasis (Eriksen et al., 1994; Frost, 1964). This action involves dismantling of aged bone, therefore setting calcium free to fulfill the body's need for calcium for various metabolic activities (e.g., muscle contraction, nerve impulses, etc.) (Wingard, 1994). The cells involved in remodeling respond within a basic framework called a bone remodeling unit (BRU) (Frost, 1964).

Local control of BRU activity is determined by the type of bone, cancellous or cortical, that is present. BRU action creates a bone structural unit (BSU), which is likely locally controlled by mechanisms that are present in each microenvironment (Frost, 1964; Mundy et al., 2003). Remodeling cycles are initiated by osteoclasts which resorb the existing bone by secreting a substance that dissipates the mineral salt crystals of the matrix (Eriksen et al., 1994; Romani et al., 2002; Wingard, 1994). Once resorption ends, the targeted area (hole) is engulfed by preosteoblasts that evolve into osteoblasts to establish new mineralized bone matrix. This process has been described as an activation-resorption-formation sequence (Eriksen et al.). It may also be broken down into two phases, erosion and formation.

Even though remodeling takes place continually throughout the skeletal system, there are certain sites that are particularly susceptible to osteoporotic fracture. These sites are the vertebrae, hip region and forearm. The sites that are of particular concern to this investigation are the hip region and the forearm. The most frequent site for osteoporotic fractures is the vertebral column (Mundy et al., 2003). The lumbar spine is predominantly composed of cancellous bone (greater than 66% of the total bone in the

lumbar spine and up to 75% in the other vertebrae) (Mundy et al.). Another common area of fracture is the hip region. The hip region is particularly prevalent in the elderly. It can be divided into two sections, the femoral neck and the intertrochanteric area. The femoral neck consists of 75% cortical bone and 25% cancellous, while the intetrochanteric area is composed of 50% cortical and 50% cancellous. Finally, the forearm is another site common to fracture. The central section of the radius contains only 5% of cancellous bone, whereas the distal end of the radius contains 25%. Although, cancellous bone represents only 15% to 20% of the total bone in the skeletal system, the remodeling that takes places in this type of bone, particularly after the age of 30, will predominately determine the incidence of osteporotic fractures. Between males and females, age-related trabecular bone loss declines at a similar rate, this same trend is not the case for cortical bone. Cortical bone mass loss is greater in females (Winters-Stone & Snow, 2004).

Cortical bone remodeling. There are two types of cortical bone: (1) periosteal, which regulates the volume and fracture repair of the bone, and (2) endosteal, which is responsible for the bulk of remodeling activity (Eriksen et al., 1994; Mundy et al., 2003). Resorption of cortical bone takes place mainly during endosteal resorption (increasing bone diameter) and resorption that takes place within the Haversian systems (increasing porosity) (Eriksen et al.; Mundy et al.). Normal cortical porosity is roughly 5% due to remodeling or resting haversian canals (Eriksen et al.). Eventually cortical bone will begin to wane, at around 40 years of age, and this bone loss accelerates in women five to ten years after menopause. Though, this trend decelerates approximately 15years post menopause (Mundy et al.).

The complete phase of remodeling in cortical bone takes 100 days (Eriksen et al., 1994). The entire formation period of cortical bone lasts approximately 90 days, and results in new bone that has 40 to 60 mm wall thickness (Eriksen et al.). The erosion (resorption) phase tends to last about 30 days (a tunnel roughly 150 mm in diameter is burrowed out). Next, the preosteoblasts invade the area followed by the matrix formation and mineralization by the osteoblasts. The result is newly remodeled cortical bone.

Cancellous bone remodeling. Cancellous bone is approximately 50 mm to 400 mm thick consisting of layers of trabeculae. The trabeculae will make adaptations to mechanical stress, therefore, fortifying the bone in the area that has increased mechanical loading (Eriksen et al., 1994). The BRUs in cancelleous bone are in the region of 300 mm in width and about three times as long (Kragstrup & Melsen, 1983). Remodeling in cancellous bone is initiated by the activation of the osteoclasts at specific focal sites, although the mechanism that instigates this activity is yet unknown (Mundy et al., 2003). The erosion (resorption) period lasts approximately 43 days, during which time a 60 mm deep tunnel is produced. The BRUs resorp and form cancellous bone on the trabecular surface, resulting in a wall of bone, or the BSU (or trabecular osteon), which constitutes the final phase of the remodeling session. The entire formation period of remodeling cancellous bone takes nearly 145 days, with a final wall thickness of 40 to 60 mm (Eriksen et al., 1994).

Mature human bone is composed of two varieties of bone, cortical and trabecular. The structure of each is determined by how the bone is remodeled and the manner of stress that is applied to the bone. Cortical bone comprises the majority of skeletal structure; though, trabecular requires 50% of the metabolic activity. Trabecular bone is

subject to heightened remodeling and diminished BMD. Therefore, injury to the bone (e.g., stress fracture) is likely to occur in areas that house trabecular bone, such as the vertebrae and sacrum.

Stress Fractures

Stress fractures, also called "march fractures" are common overtraining injuries among both male and female athletes. Stress fractures occur when the training intensity (load) placed on the skeleton exceeds the bones ability to repair and reform itself (Callahan, 2000). In other words, a fracture will transpire when the external forces to the skeleton surpass the structural micro- and macro architectural integrity of the bone at a particular site (Nattiv & Armsey, 1997). The fracture is not a traumatic injury but a partial or incomplete fissure resulting from an accumulation of stress applied sub maximally in a rhythmically and repeatedly manner to a localized area of bone (Nattiv & Armsey; Romani et al., 2002). Stress fractures were first diagnosed in 1855 by a Prussian surgeon named Breithraupt (Breithraupt, 1855). Breithraupt classified the injuries of military recruits that were experiencing tender inflamed feet as "marchers' fractures" (Callahan, 2000; Nattiv & Armsey). Four decades later, advances in roenterography permitted scientists to actually visualize the metatarsal stress fractures that Breithraupt hypothesized (Nattiv & Armsey). Advances to technology and radiology have afforded researchers the opportunity to further understand stress fractures. Today, it is believed that stress injury to bone occurs on a dynamic continuum that stretches from normal bone remodeling and periostitis to a frank cortical fracture (Nattiv & Armsey; Romani et al.).

Stress injury to bone occurs due to excessive bone strain as a result of amassed microdamage in addition to the bones inability to maintain appropriate remodeling and

repair (Zeni et al., 2000). The rate of fatigue in conjunction with certain strain rates can result in the accrual of microdamage, and lead to stress fractures (Zeni et al.).

Stress fractures can be classified into two categories: (1) fatigue, and (2) insufficiency fractures. Fatigue fractures generally materialize when abnormal stress is applied to normally elastic bone (Umans & Pavlov, 1994). Conversely, insufficiency fractures develop in bone that has compromised mineral levels or are unnaturally inelastic (Umans & Pavlov). Both varieties of stress fractures are relevant to this investigation, yet, the insufficiency fracture is of particular interest due to its relationship to mineral levels in the bone.

Risk Factors for Stress Fractures

There are certain risk factors that can somewhat predispose an athlete to a stress fracture. These factors include gender, ethnicity, age, mechanical factors, hormonal issues, nutritional issues, and an increase or change in exercise intensity (Callahan, 2000; Nattiv & Armsey 1997; Romani et al., 2002; Zeni et al., 2000). Yet, many of these factors remain unproven. Most of the research has been performed on males and military recruits, with relatively little research available on female athletes (Zeni et al.).

Gender. Nonetheless, gender does seem to be a factor that differentiates the risk for stress fractures. Female military recruits experience a 1.2 to 10 time greater occurrence of stress injury than their male counterparts (Brudvig, Gudger, & Obermeyer, 1983; Reinker & Ozburne, 1979; Scully & Besterman, 1982). This could be due to hormonal influences, the smaller size of the tibia (there is a positive correlation between having a narrow tibia and the incidence of stress fractures), and the reduced amount of muscle mass seen in female athletes in comparison to their male counterparts (Callahan,

2000; Nattiv & Armsey; Zeni et al.). More research is warranted to discover the rate of stress fractures in female athletes in comparison to male athletes.

Ethnicity and age. Ethnicity and age are additional risk factors attributed to the development of a stress fracture. For example, military researchers have found that Caucasian and Asian military women have a significantly higher incidence of stress fractures than do African American women (Brudvig et al., 1983; Friedl & Nuovo, 1992; Nattiv & Armsey, 1997). Racial differences could be due to the degree of increase in the development of BMD during puberty in African Americans is substantially higher than in Caucasians. This phenomenon was especially apparent late in puberty. This might suggest that hormonal and other metabolic factors may cause the discrepancy in race (Gilsanz, Roe, Mora, Costin, & Goodman., 1991). Mechanical factors can also contribute to the incidence of stress injury in bone.

Mechanical risk factors. Mechanical risk factors can be broken down into two categories: (1) extrinsic mechanical factors (e.g., training schedule, footwear, fitness level, and running surface or sport terrain), and (2) intrinsic mechanical factors (e.g., tibial bone width, BMD, bone geometry, foot structure, and leg length). Both categories of mechanical factors can bring stress injury to the bone of exercising individuals (Callahan, 2000; Nattiv & Armsey, 1997; Zeni et al., 2000).

Extrinsic mechanical risk factors. Athletes and other active exercisers are susceptible to contracting a stress fracture as a result of mechanical issues (e.g., footwear, the fitness level of the exerciser, and the training intensity) in their training regimes (Goldberg & Pecora, 1994; Milgrom, Simkin, Eldad, Nyska & Finestone, 2000; Nattiv & Armsey, 1997). For example, a drastic change in a training routine is a major mechanical

risk factor for stress fractures. Goldberg and Pecora (1994) established that 67% of 58 stress fractures in intercollegiate college athletes were freshmen. This could be due to the change in training intensity experienced at the varsity level when entering the college arena. Similar results have been discovered in military populations (Protzman & Griffis, 1997). Likewise, Armstrong, Rue, Wilckens, and Frassica, (2004) discovered that in their population of male and female U.S. Naval Academy summer trainees, most of the stress fractures that occurred happened on or before the fourth week of training. The authors suggested that as mechanical forces compound, bone resorption exceeds remodeling and actual repair of the bone structure. Eventually, the loading area declines; yet, the stress does not decelerate leading to a fracture (Armstrong et al., 2004). The training regimen mandated by the level of competition or by authority figures is just one peripheral mechanical factor that can affect bone health.

Foot wear is another extrinsic risk factor for stress fractures. Price, shock-absorbing capability, etc., have been issues researched that have not proved significant. Although, the age of the shoe does appear to be noteworthy, individuals wearing new shoes were less likely to experience a stress fracture than individuals wearing older shoes (Nattiv & Armsey, 1997). Although, the fitness level of an individual has a much more significant effect on bone health.

Fitness level reflects the risk rate of stress fractures. In epidemiologic investigations with military recruits, for example, recruits who participated in ball sports regularly two years prior to induction in the military sustained 50% less stress fractures than individuals who did not participate in ball sports (Milgrom et al., 2000). Possible contributing factors to these results include substandard aerobic capacity, inadequate

flexibility, and inferior muscular strength (Zeni et al., 2000). However, the main determining factor of fitness effects on stress fractures is inferior muscular strength. According to Markey's (1987) fatigue theory, early muscular fatigue in which muscles disperse the load on the bone translates to greater forces diffused to the bone. The result can be a stress fracture. Armstrong et al. (2004) also theorized that muscular fatigue can result in a stress fracture. The nature of the surface on which the training is performed on can also be explained by Markey's fatigue theory. For example, running on hard, soft, or uneven surfaces can provide mechanical stress that compounds the risk for a stress fracture. Each of these conditions can cause unnecessary fatigue to the muscles (Nattiv & Armsey, 1997). Running on "hard" surfaces has been shown to cause additional mechanical loading to the bone, often resulting in a stress fracture (Nattiv & Armsey). There are mechanical factors that are intrinsic in nature as well.

Intrinsic mechanical risk factors. There are a few of intrinsic mechanical factors, such as the foot structure of the exerciser, the leg length of the exerciser and the tibial size, which can exposure the training individual to the possibility of attaining a stress fracture. For example, foot structure can have an impact on the likelihood of developing a stress fracture. The shape of the arch of the foot can impact the dispersion of force to the tibia and fibula. A foot with a high, rigid arch (i.e., pes cavus) is unable to absorb impact as well as a foot with a more flexible, low arch (i.e., pes planus). The result is that the pes cavus does not absorb the stress load and disperses greater force to the tibia and fibula than the pes planus (Zeni et al., 2000). How the foot accepts and disperses ground forces can determine the consequential affect on the anatomy of the lower leg and potential injuries.

The consistency in leg length and tibial dimension is an additional mechanical consideration that can increase the risk of a stress fracture (Friberg, 1982). In theory, females are at a greater risk of stress fractures of the tibia due to reduced size of the tibia (Nattiv & Armsey, 1997). The tibia sustains a tremendous amount of stress due to the biomechanical compression and tension forces accumulated when athletes run and/or jump continually (Nattiv & Armsey). The amount of strain that a bone can take is related to its cross-sectional area (Zeni et al., 2000). Bones that have a smaller cross-sectional area have a decreased ability to withstand repeated stress. A reduction in tibial width produces a sizeable decline in resistance to bending (Armstrong et al., 2004). This places the athlete with smaller bone size at a greater risk of stress fracture. Moreover, female athletes may have an elevated risk of stress fracture due to the BMD issues, hormonal and otherwise, that can transpire as an athlete enters into the FAT.

Female athletes are at further risk of attaining a stress fracture due to alterations in their hormonal profile. A number of hormonal anomalies may occur that can impact BMD. For example, the age when an athlete reaches menarche may affect the attainment of peak bone mass and may lead to future menstrual issues (Bennell et al., 1995). Menstrual disturbances cause female athletes to have a depressed basal estrogen level and a disruption of the hypothalamic-pituitary axis. It is possible that hormonal imbalance increases the set point for bone modeling and remodeling, therefore, impeding the cellular response that is needed to allow bone to adapt to extraneous loads (Loucks & Horvath, 1985).

Finally, the nutritional risk factors of low calcium and vitamin D intake, and disordered eating can also impede the maintenance and development of BMD.

Disordered eating patterns, overtraining, or insufficient energy intake can create a negative energy balance that can lead to weight loss. Armstrong et al. (2004) concluded that military recruits that have a continuous negative energy balance can result in weight loss which can be a risk factor for stress fractures. Disordered eating/eating disorders are generally the entry point to the female athlete triad, an inter-related disorder of eating disorders, amenorrhea and osteoporosis (ACSM, 1997).

Stress fractures consist of insufficiency fissures that disrupt the integrity of bone. These fissures are brought about generally by overuse, but there are additional factors that play a role in instigating a stress fracture. Mechanical influences have some bearing on the incidence of stress fractures. These mechanical risk factors can be divided into to groupings, intrinsic and extrinsic. Moreover, gender, ethnicity, age, nutritional status, and hormonal insufficiencies all are part of the milieu of issues that can predispose an individual for bone injury. Females, even more specifically, females that participate in high levels of physical activity can have an elevated risk of incurring a stress injury.

The Female Athlete Triad

The exponential increase in the females contributing to the athletic population in the US primarily materialized via the passage of Title IX of the Education Amendment Act in 1972. In its most basic form, Title IX produced a directive that mandated females a similar opportunity to participate in sports as their male counterparts had been afforded for generations (Golden, 2002). In addition to Title IX, society in American became more aware of the health benefits of exercise. Jazzercise and aerobics developed into a trend in American pop culture, and therefore, women began to exercise at a greater rate than ever before.

As increasing numbers of females began training for competition and health-related fitness results, a variety of related symptoms emerged. Eventually, in 1992, at the Task Force on Women's Issues of the ACSM, the inter-related symptoms and risk factors of what became known as the triad was discussed, and the term Female Athlete Triad was birthed (Yeager, Agostini, Nattiv, & Drinkwater, 1993).

The FAT consists of three interconnected maladies that can transpire alone or in unison in physically active women (Rust, 2002; Teitz, 1997; Trash & Anderson, 2000). The components for the FAT are eating disorders, amenorrhea, and osteoporosis. The presence of the triad can affect the athlete's ability to perform, as well as, placing the athlete's physical and mental health in jeopardy (Rust). This section will introduce the FAT and discuss the risk factors associated with entering into the triad.

Risk Factors of the Female Athlete Triad

There are certain risk factors that predispose a female athlete to enter into to the milieu of the female athlete triad. The general risk factors associated with the triad are: never-ending calorie restriction, altered self-esteem, low body fat levels and/or loss of fat stores in the femoral region (hips, buttocks and thighs), a family history of dysfunction, abuse (physical/sexual), improper body image, perfectionism, vegetarianism, and an inadequate understanding of nutrition (Benson, Engelbert-Fenton, & Eiseman, 1996; Otis, Drinkwater, Johnson, Loucks, & Wilmore, 1997; Sanborn, Horea, Siemers, & Dieringer, 2000). It has been hypothesized that there are specific areas that a woman must maintain fat in order to be viable for reproduction. When the body looses these fat stores, the metabolic activity of these areas is altered, resulting in the body's inability to sustain pregnancy and lactation (Benson et al.; Brownell, Steen, & Wilmore, 1987). There are

other sport-specific triggers which push the athlete toward entering into the inter-related cycle of the triad. These risk factors include: an unbalanced view of her body weight in relation to her performance, an unending pressure to be thin from influential members of the athletes support structure (coaches, peers, and parents), an obsessive compulsion to win at any cost, an identity as an athlete (no view of self outside of being an athlete), changes in training or abrupt alterations in training, training while injured, an imbalance between energy intake and energy expenditure, withstanding some sort of traumatic event (e.g., an injury, change of coaches, etc.), and when the athlete is in the midst of vulnerable episodes (e.g., entering college, depression, etc.) (Otis et al., 1997; Sanborn et al., 2000). Entry into the triad generally begins with episodes of disordered eating and/or the evidence of an eating disorder.

The FAT is a condition encountered by physically active women. The triad includes three interrelated components (eating disorders, amenorrhea, and osteoporosis) that work to compromise the performance, physical and mental health of female athletes. The next section will elaborate on and define eating disorders.

Definitions and Prevalence of Eating Disorders and Disordered Eating

Female athletes are under tremendous pressure to maintain a lean physique. Coaches push the athletes to achieve "perfect bodies", while athletes place themselves under tremendous pressure to attain low body fat percentages. The drive to stay thin is further fueled by Western society's continual shift toward an expectation of maintaining a thinner physique. This ideal is projected via the media (Tiggemann, Gardiner, & Slater, 2000). The pressure to be thin has lead female athletes to engage in dieting that is

detrimental to performance and eating behaviors that compromise their health and psychological well-being (Hausenblas & Mack, 1999).

According to Nattiv and Lynch (1994), athletes who are at particular risk to enter into the FAT tend to participate in sports that require excessively lean body mass (e.g., figure skaters, divers, endurance runners). In one Norwegian study of elite female athletes, ages 12-25 years., Sundgot-Borgen (1994) found a significantly higher prevalence of eating disorders and training intensity among athletes competing in aesthetic or endurance sports, such as gymnastics, diving, figure skating, distance running, cross-country, and skiing, as opposed to athletes in other varieties of sports. Rosen, McKeag, Hough, and Curley (1986) discovered that 47% of runners and 25% of athletes that participate in softball, track, tennis, and volleyball exhibit detrimental behaviors. There seems to be a link between the type of sport and the severity and nature of the eating disorder.

Eating Disorders Defined

Women are approximately ten times as likely to develop a disordered eating behavior as men (Clarks, 1994; Stone, 1999). There are three types of eating disorders: (1) anorexia nervosa, (2) bulimia nervosa, and (3) disordered eating. Disordered eating does not fit into the first two specific categories (National Collegiate Athletic Association [NCAA] Guidelines, 2001-2002). Additional authors categorize the third class, disordered eating, as a sub-clinical eating disorder (Cobb et al., 2003; Nattiv & Lynch, 1994; Stone; Otis et al., 1997).

Nearly two-thirds of female athletes practice disordered eating (Nattiv, Agostini, Drinkwater, & Yeager, 1994). Athletes engaging in a sub-clinical behavior or disordered

eating display certain characteristics of each of the above stated classes, but they do not present evidence of an illness. Instead, the athletes' exhibit behaviors that normally include excessive exercising, with eating habits that range from insufficient caloric intake or unhealthy eating habits to binging and purging (Nattiv & Lynch, 1994; Stone, 1999). This practice generally leaves the body at a chronic energy deficit (Cobb et al., 2003; Otis et al., 1997). According to Skolnick (1993), "Many in the field prefer the term disordered eating to eating disorders because it implies not a definitive end point but a spectrum of abnormal behavior, which at its extreme includes anorexia nervosa and bulimia nervosa" (p. 2). The American Psychiatric Association (APA) has defined disordered eating as "eating disorder not otherwise specified" (NOS). In fact, the most recent version of the Diagnostic and Statistical Manual fourth edition (DSM-IV) established a criterion for defining the behavior (APA, 1994). According to the new manual, the criterion for anorexia and bulimia are met except that the individual maintains menstruation (or sometimes not), their weight is within normal boundaries, and binge eating followed by purging (e.g., purging via forcing oneself to vomit, or by taking diuretics) happen less than two times per week for three months (APA; Rome 2003). In addition to these symptoms the athlete will practice repeated episodes of binge-eating without purging. Although, they may frequently chew food and then spit it out without swallowing (APA, 1994). Sundgot-Borgen (1994) categorizes the sub-clinical disorder as anorexia athletica. Other categories of eating disorders include anorexia and bulimia nervosa.

Anorexia nervosa. A commonly known eating disorder is anorexia nervosa. Anorexia generally begins in the adolescent years , and is experienced predominantly by

females (Mehler, 2003). Unfortunately, the adolescent does not realize the damage that she is creating for her developing skeletal structure. The BMD of adolescent anorectics will not increase within the normal growth range, even years after the anorexia has been cured (Mehler). One study revealed a three fold rise in the fracture incidence in participants with anorexia as compared to age matched controls (Lucas, Melton, Crowson, & Fallon, 1999). Greater than 50% of females that are diagnosed with anorexia nervosa eventually cultivate osteoporosis (Powers, 1999; Treasure & Serpell, 2001). Excessive exercise can further exasperate the BMD of anorectics. The result can range from a negative impact or at least no protective effect on the BMD of anorexic females (Mehler; Sungot-Borgen, Bahr, Falch, & Schneider, 1998). Conversely, Sungot-Borgen et al. (1998) reported that bulimics who exercised regularly in their study saw improvement in BMD in weight bearing sites as compared to anorectics who did not improve.

Anorexia is difficult to diagnose in its early stages. The criteria to diagnose its' presence is two-fold, (1) when there is the absence of the menstrual cycle for three consecutive months and (2) the loss of at least 15% of an individual's body weight through depriving the body of adequate nutrients to maintain internal homeostasis (APA, 1994). Anorexia has been classified into two varieties: (1) restricting anorexia nervosa, denoted by the lack of purging, and (2) bulimic anorexia nervosa, fasting is a regular practice, but is accompanied by purging (e.g., vomiting, laxatives, over exercising, etc.) (Rome, 2003). An unrealistic body image, refutation of the severity of low body weight, amenorrhea, and a desperate fear of being fat are additional traits of anorexia (APA;

Rome, 2003). Symptoms of anorexia nervosa should not be confused with indicators for bulimia nervosa.

Bulimia nervosa. Bulimia nervosa was first defined in 1976. One of the most predominant signs of bulimia is reoccurring periods of binge eating (APA, 1994; Rome, 2003). In the Greek, bulimia truly stands for, "appetite like a bull" (Rome). The percentage of college females who have bulimia could be as high as 19% (Rome). Binge eating behavior includes the actions behaviors: eating a large amount of food in a short period of time coupled with a feeling of being out of control in regards to their eating frenzy during these episodes (APA; Rome; Teitz 1997). Another telltale sign is some sort of compensatory action to insure that the effects of the binge episode (e.g., weight gain) will not transpire (APA). The compensatory action might include: fasting, excessive exercise, self-provoked vomiting, and the abuse of laxatives, enemas, diuretics or other medications. In order for a diagnosis of bulimia to be secured, the disordered behavior must take place two times per week for three months. The individual with bulimic behavior will also have an unrealistic body image (Rome; Teitz, 1997). Again, binge eating is often the most notable sign.

In a 1997 study of National Collegiate Athletic Association (NCAA) athletes, 10% of the female athletes participated in binge eating at least weekly (NCAA Guidelines, 2001-2002). Research on female athletes has revealed that 15% to 62% of female athletes show evidence of having pathogenic weight-control behaviors. Conversely, only one percent of the universal female population (predominately adolescents and young adults) meet the DSM-IV criteria for anorexia nervosa, while just another 1% to 3% meet the criteria for bulimia nervosa (Nattiv & Lynch, 1994). There

seems to be a physiological connection between the athletic personality type and the tendency of an athlete to develop disordered eating habits. The athlete's personality profile may be impacted by biochemical factors that are inherent to the athlete.

Some evidence suggests that there are biochemical factors within the athlete that predispose them to disordered eating. For example, the psychodynamics between the coach and the athlete can be affected by biochemical imbalances. How psychodynamics affects the brain is unknown, however certain anti-depressants have been used successfully to treat athletes with disorders (Thornton, 1990). Athletes may not only have a physiological bias to disorder eating, but unfortunately, the environment that the athlete trains and competes in, and how the athlete perceives herself in that environment can incline them toward eating disorders as well.

Body Image and Eating Disorders

The results of past investigations have indicated that Americans may be more predisposed to abnormal body image perceptions due to mounting cultural pressure. This can be confirmed by the increased rate of eating disorders over the past two decades. Eating disorders can have a subtle inception. The individual who is unsatisfied with their body weight may begin with temperate dieting but soon switch to an overwhelming fixation with diet and eating (Rome, 2003). Historical evidence from the Middle Ages further supports that certain cultures and populations are more likely to practice self-imposed disordered eating. Therefore, cultural pressure for the "ideal" body presents a predominate theme in addition to the demands of achieving athletic excellence for the female athlete (Turk et al., 1999). The American culture has bought into this idea. Cultural pressure is just one of the many triggers that lead the athlete to disordered eating.

Triggers of Eating Disorders

Work conducted by Pliner and Haddock (1996), who provide a profile of an athlete who is inclined to disordered eating. According to Pliner and Haddock's investigation, females with anorexic behaviors were more greatly affected by the words, opinions and feedback of others. This research suggests that athletes who are predisposed to partake in an eating disorder are more likely to seek the approval of significant figures, especially coaches. As stated by the *NCAA Guidelines Sportsmedicine Handbook* (2001-2002), eating disorders can be triggered by the coach's pressure regarding the athlete's body weight and/or body composition. Certain coaches have gone as far as threatening their athletes with loosing their scholarship unless the athlete conforms to the expected body fat percentage. This threat and other issues can affect the athlete's body image.

A myriad of performance issues, social issues (e.g., Westernized female body ideals), psychological factors, (e.g., altered self-image, insufficient coping skills, etc.) and personality traits (e.g., perfectionism and compulsiveness) can lead an athlete to practice disordered eating patterns (Johnson, 1994). Unfortunately, athletes that practice disordered eating behaviors may experience a decrease in performance and an elevated risk for injury. These phenomena happen due to the limited energy intake and electrolyte imbalance which in turn affect endurance, reaction time, speed, concentration, and strength (American Academy of Pediatrics [AAP], 2000).

Sundgot-Borgen (1994) administered the Eating Disorder Inventory (EDI) to elite female athletes in Norway ($N = 603$) to determine if this population was in greater danger of developing eating disorders. Sundgot-Borgen classified 117 athletes as at risk,

reporting that, 103 of the 117 "at risk" athletes were administered a thorough clinical interview for eating disorders. From this sample a list of risks and triggers for disordered eating was compiled. Out of the entire list, 15% of respondents gave no specific reason for their disorder. The most cited reason stated was the practice of prolonged periods of dieting and accompanying weight fluctuations (37%). The next most frequent response was the loss of a coach (30%), followed by the presence of an injury or illness (23%). Further risks and triggers include: casual comments (19%), leaving home or failure at school or work (10%), a problem in a relationship (10%), family problems (7%), illness or injury to family members (7%), death of a significant other (4%), and sexual abuse (4%). When the same athletes were asked why they dieted, the sample agreed that dieting was part of enhancing athletic performance. Most of the athletes stated that dieting was recommended by their coach (67%). Supplementary reasons ranged from recommendations from parents (15%) and friends (8%) to improving physical appearance (40%). The complicated factors that trigger an episode of disordered eating should be noted by coaches, individuals that work closely with athletes, and athlete's to better educate themselves to the origins of an eating disorder. This alone, will enable all involved to be prepared to predict and detect the signs and symptoms that will ensue.

Signs and Symptoms Eating Disorders

The symptoms of eating disorders are varied, but can be broken down into two distinct groups: (1) behavioral, and (2) physical. Beals et al. (1999) state that the physical symptoms include: chronic fatigue, anemia, frequent gastrointestinal problems, cold intolerance, lanugo (baby fine hair on the body), tooth erosion, irregular or absent menstrual cycles, frequent musculoskeletal injuries (e.g., stress fractures), and prolonged

wound healing. Other physical symptoms include swelling of the parotid glands (chipmunk cheeks), hypo tension, bradycardia and Russell's sign, nail changes of the first and second digits of the dominant hand (Stone, 1999).

Burney and Brehm (1998) offer a list of observable behavioral and psychological symptoms. The behavioral symptoms include the following: frequently eating alone, frequent trips to the bathroom (especially after meals), compulsive and excessive exercise, use of laxatives or diuretics, and use of diet pills. Psychological symptoms consist of: preoccupation with food and weight, anxiety about eating in public or in another's presence, concerns about being fat or feeling fat (even when weight is below normal), refusal to maintain a minimal or normal weight for their sport, low self-esteem, poor body image, and depression (Burney and Brehm). In addition to these symptoms athletes may experience a loss of aerobic and anaerobic conditioning, diminished coordination of body movements, and impaired visual judgment (NCAA Guidelines, 2001-2002). Clearly, the trained observer must spot the signs and symptoms of disordered eating before the habit moves into a full-blown eating disorder. Early detection is the key to avoid long-term physical consequences to the athlete.

Long-term Physical Consequences of Eating Disorders

The long-term ramifications of untreated disordered eating can lead to a number of serious health issues. Trash and Anderson (2000) contend that problems include a compromised immune system and chronic fatigue to morbidity. In addition, electrolyte and acid-base imbalances and other deficiencies (e.g., nutrient, micronutrient, and hormonal) can affect the body's homeostatic condition. Women can also suffer from reduced cognitive ability, seizures, cardiac arrhythmia, and myocardial infarction (Rome,

2003). Unfortunately, the physical consequences can go by undetected, yet, one outcome of disordered eating particularly relevant to female athletes is amenorrhea.

Contemporary female athletes are being persuaded to have a lean body mass, especially in endurance and aesthetic sports. This drive to stay thin has put female athletes at a greater risk of developing one of three eating disorders (anorexia nervosa, bulimia nervosa, binge eating and sub-clinical disorder). As many as 62% of all female athletes have some sort of pathogenic weight-control behavior. There is some evidence that there is a genetic link to this behavior. Environment can also be a factor by predisposing an athlete to an "ideal" body image. Early detection by recognizing the triggers and signs and symptoms can stop the athlete from developing long-term physical consequences. Amenorrhea may be a direct result of restrictive eating behaviors.

Definition and Prevalence of Amenorrhea

Amenorrhea is generally defined as the absence of menses for at least three or four menstrual cycles. There are two forms of amenorrhea, primary and secondary (Beals et al., 1999). Primary amenorrhea results in a delayed menarche. Two distinct situations mark the onset of primary amenorrhea. If a girl reaches the age of 14 years., and there is no emergence of secondary sex characteristics, or if no menstrual bleeding occurs by age 16 years. although there has been manifestation of secondary sex characteristics, either circumstance constitutes primary amenorrhea (Warren & Goodman, 2003). Secondary amenorrhea is the loss of menses for at least six months in women that have had at least one mense. The frequency of secondary amenorrhea among adult, female athletes may range from 66% to 3.4% as compared to approximately 2% to 5% in the general population (Bykowski, 1999; Loucks, 1990; Loucks & Horvath, 1985; Shangold, Rebar,

Wentz, & Schiff, 1990). Amenorrhea may be initiated abruptly or gradually. A gradual onset may begin with lengthened intervals between menses of 35 days or more (oligomenorrhea), as opposed to a more abrupt onset, where the athlete might simply cease menstruation for three or more cycles. Amenorrhea is most likely induced by disordered eating and/or excessive exercise in athletes (Teitz, 1997). These two features can combine to initiate athletic amenorrhea.

Athletic Amenorrhea

The onset of amenorrhea is attributed to imbalance in the hypothalamic-pituitary axis (Rickenlund et al., 2003; Warren & Shantha, 2000). The hypothalamic-pituitary axis, also known as the reproductive axis, is responsible for regulating a female's menstrual and reproductive functions. In amenorrheic athletes, when the axis is disrupted, it is, in all likelihood, attributed to insufficient energy availability. This, in turn, prompts the suppression of the pulsatile secretion of gonadotropin-releasing hormone (GnRH). The reproductive axis is a negative feedback system, thus, when a disruption occurs at one level, the rest of the axis will malfunction and homeostasis will cease (Warren & Goodman, 2003). In a normally functioning female, GnRH is secreted every 60-90 minutes. The purpose of GnRH is to send a signal to the pituitary gland to limit the secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). The diminished level of GnRH initiates a cascade of events that results in hypo-estrogenism, anovulation, and termination of the production of estradiol (Warren & Goodman). The lack of LH or an estradiol surge mid-cycle results in athletic amenorrhea.

Athletic amenorrhea has become the term that is used to refer to a host of hypothalamic-pituitary axis disorders (e.g., anovulation, primary and secondary amenorrhea, oligomenorrhea, retarded pubertal development, and atypical luteal phases) in the female athlete, of which the occurrence of these reproductive abnormalities can be as high as 79% (Constantini, 1994). Many of these abnormalities may go undetected. For example, the length of the luteal phase can be shortened, lessening the levels of available progesterone. All of this can happen with out a disruption of the normal menstrual cycle (Rome, 2003). Anovulation is another condition that may go unnoticed. Anovulation occurs when an oocyte is not released, resulting in release of estrogen without a corresponding release of progesterone (Rome).

In recent research, Cobb et al., (2003) documented the athletic amenorrhea occurrence. Cobb and colleagues investigated the menstrual status of 91 competitive female runners. This research revealed that out of 91 participants, 36% of the study sample experienced abnormal menstruation (26% were oligomenorrheic, and 10% were subjected to amenorrhea during the previous year) (Cobb et al.). This representative portion of the sample ran 18% more miles a week, had 45% fewer periods, and underwent menarche an average of 1.2 years later than the eumenorrheic females in the rest of the sample (Cobb et al.). Again, suppression of LH secretions from the pituitary gland is believed to be the cause of amenorrhea and anovulation in athletic women (Loucks, 1990; Loucks, Vaitukaitis, & Cameron, 1992).

Cobb et al. (2003), Constantini (1994), Loucks, & Horvath, (1985) have concluded that athletic amenorrhea is a consequence of reduced energy intake and excessive exercise intensity. There are several theories that have been hypothesized to

explain this phenomenon. The body composition theory was once thought to be the only cause of athletic amenorrhea (Warren & Goodman, 2003). The theory hypothesized that female athletes must have a body fat percent greater than 17% to 19% in order to initiate menarche with uniform cycles sustained when the body fat reached 22% and above (Frisch & Mc Arthur, 1974). Yet, there is no standard set point for body fat that relates to all female athletes. Another theory, the excessive exerciser theory, claimed that extreme exercise is the cause of disruption of the hypothalamic-pituitary axis. Although, research has not proven that excessive exercise alone suppresses LH secretion (Loucks, Verdun, & Heath, 1998). Concurrently, researchers now know that the body fat of an individual and exercise intensity are components of a complicated interplay of factors (intensive training, restrictive diet, decreased metabolic rate and prepubertal training) that result in amenorrhea (Sanborn, Albrecht, & Wagner, 1987; Loucks & Horvath, 1985). Evidently, the loss of fat sets up a convoluted milieu of endocrine and metabolic changes in the hypothalamic-pituitary axis that limits GnRH pulsitility, which in turn diminishes LH pulsitility (Bonen & Keizer, 1984; Warren & Goodman). Nonetheless, the decreased caloric intake of a disordered diet causes a reduction in the amount of nutrients, including the calcium that the athlete is consuming. Unfortunately, it is difficult to estimate dietary energy intake and exercise energy expenditure accurately (Otis, et al., 1997). This energy deficit in conjunction with excessive exercise behavior is believed to trigger the amenorrheic episode. There are additional features that can contribute to this occurrence.

Cortisol and athletic amenorrhea. Prior research has identified elevated cortisol levels as a mechanism in athletic amenorrhea (Loucks, 1989). Loucks discovered that amenorrheic athletes may have up to 25% greater levels of cortisol circulating in the body

as compared to eummenorrheic athletes and sedentary females. The negative feedback system maintaining endocrine homeostasis is a delicate balance of hormones. An elevation of cortisol or another mechanism, such as corticotrophin-releasing hormone (CRH) can disrupt the balance.

Corticotrophin-releasing hormone and athletic amenorrhea. There is also evidence of additional mechanisms that can initiate a disruption in the hypothalamic-pituitary axis. Elevated secretion of CRH inhibits the hypothalamic-pituitary axis by inhibiting the secretion of GnRH (Loucks, Mortola, Girton, & Yen, 1989). CRH is quite possibly responsible for the concurrent elevation in cortisol and prolactin which has been detected in female athletes (De Souza, Maguire, & Maresh, 1991).

Insulin-like growth factor-binding protein-1 (IGFBP-1) and athletic amenorrhea. There is increasing evidence that yet another mechanism might, increased serum levels of IGFBP-1, might have a part in disrupting the hypothalamic pituitary-axis (Laughlin & Yen, 1996). IGRBP-1 controls IGF-1 function. IGF-1, in turn, instigates early development of the follicle and LH pulse frequency (De Cree, 1998; Wilson, 1995). Thus, elevated IGFBP-1 may decrease the secretion of LH in female athletes that have amenorrhea (Wilson, 1995). Interestingly, IGF levels can be specific to the manner of activity that the female athlete engages in. IGF-1 levels tend to be lower in aerobic and endurance athletes as compared to anaerobic athletes (De Cree, 1998).

Leptin and athletic amenorrhea. The complete impact of leptin in amenorrheic athletes is unknown, but leptin is a regulator of basal metabolic rate (Kaufman, Warren, Wang, Heymsfield, & Pierson, 2002). Amenorrheic athletes commonly have a low metabolic rate coupled with irregular leptin secretion. Kaufman et al. discovered that

depressed leptin levels and metabolic rate is associated with reduced BMD. Kaufman et al. deduced that the three factors (leptin levels, BMD, and metabolic rate) may all be the result of inadequate nutritional intake and a restrictive caloric intake.

Overview of Athletic Amenorrhea

Athletic amenorrhea is most likely a form of hypothalamic amenorrhea, consisting of diminished levels of GnRH. GnRH is secreted from the hypothalamus (Trash & Anderson, 2000). GnRH stimulates the anterior pituitary gland to release follicle stimulating hormone (FSH) and luteinizing hormone (LH) into the circulatory system. The FSH and LH travel to the ovaries and act on the follicles.

Eventually the FSH and LH act on the cells surrounding the follicle and promote the production of estrogens, in this case primarily estradiol. Maturation of the follicle (release of the ovum) will result if the GnRH and resulting estradiol perform without alteration. This phenomenon is called the hypothalamic-pituitary axis. Without maturation of the follicle anovulation can occur (absence of ovulation). Chronic amenorrhea can lead to ovarian failure and infertility (Bonen & Keizer, 1984). Infertility is just one result of prolonged amenorrhea.

Amenorrhea and BMD

Another result of amenorrhea of particular relevance in the present study is loss of BMD. Women with amenorrhea have been found to have a 10% to 20% decrease in the BMD of the lumbar spine as compared to eumenorrheic women. Moreover, Cobb et al. (2003) found that oligomenorrheic and amenorrheic female runners exhibited a reduction of BMD in the lumbar spine of 5%, the total hip of 6%, and the whole body of 3% when controlling for weight, percent body fat, Eating Disorders Inventory (EDI) score, and age

of menarche. In this same study, females that were 115 pounds or less had fivefold-increased odds of experiencing osteopenia than females above 115 pounds (Cobb et al.). This is true even if the women resume normal menses. Estrogen seems to be a greater stimuli for bone reabsorption than calcium deficiency. Bone loss due to estrogen deficiency cannot completely be replaced by increased weight bearing exercise and calcium supplementation (Kiningham, Apgar & Schwenk, 1996). Clinical management of the disordered eating or eating disorder may be necessary.

Treatment and Intervention of Amenorrhea

Treating the athlete with disordered eating that is accompanied by amenorrhea requires a multi-faceted approach in which a psychologist, psychiatrist, a dietitian, a physician, and the team athletic trainer may be involved (Teitz, 1997). The initial step to prevention is to educate athletes and coaches about eating disorders and disordered eating. Experts agree that education of the team around the athlete (coach, trainer, parents, and sport related personnel) is also necessary. Yet, one investigation of 138 NCAA Division IA coaches revealed that just 44.5% had attended any kind of an educational program centered on eating disorders. Thus, education of the team around the athlete is paramount in recognizing the signs and symptoms before the athlete develops a full-blown eating disorder is crucial (Turk et al., 1999).

Intervention once a disordered behavior is identified is paramount. The risk to the athlete's BMD must be addressed immediately with calcium supplementation and resistance training. In some athletes estrogen replacement therapy is important to bring homeostasis to the endocrine system (Stone, 1999). The extent of treatment is generally determined by the age and length of time that the athlete has suffered from the disorder.

Clarks (1994) elaborated upon the case study of an amenorrheic dancer to highlight a treatment plan for amenorrhea. The athlete highlighted is a dancer with loss of menses and a caloric intake of approximately 1,000 calories per day. The initial goal was to educate the athlete about her pattern of disordered eating and the negative effects that her behavior has on performance and health. Reducing the athlete's preoccupation with food, gaining weight, and gaining body fat were crucial. Psychotherapy aided the implementation of this healthier ideal. Over time, the athlete's caloric intake was gradually increased to normal levels. In due course, she established a normal weight and regular menstrual cycle. The treatment plan required the athlete to make some changes to her diet and behavior. Throughout the plan she was required to eat a wider variety of food to include more nutrients, in addition to looking at food as a positive substance for her body. Keeping a daily journal of her eating habits and how the effect of those foods on her emotional status accomplished this process. The diary was then evaluated with the psychotherapist. The reduced caloric intake and heightened energy expenditure of athletes causes problems in performance (e.g. fatigue, muscle loss, and decreased anaerobic and aerobic power), but also is associated with amenorrhea and eventually osteoporosis in female athletes (Klossner, 2000).

There are many dangerous long-term physical consequences of an enduring eating disorder. The range of consequences can be as simple as reduced cognitive ability and as severe as morbidity. Amenorrhea can generally be a telltale sign of an eating disorder, although, disordered eating is only a component of a complicated interplay of factors that cause the athlete to cease menstruating. Amenorrhea can cause compromises in the BMD of the athlete, and eventually put her at risk for premature osteoporosis. Fortunately,

there is a positive effect of physical training on the BMD of female athletes

Physiological Effect of Training on BMD

The type of training of the female athlete seems to influence BMD. For example, research has shown that athletes who participate in sports that are more aerobic (e.g., running, rowing, etc.) in nature, tend to have an increased incidence of disrupting the hypothalamic-pituitary-axis due to overtraining, low body mass, low body fat, and energy restriction (Pettersen, Stalnacke, Ahlenius, Henrikkson-Larsen, & Lorentzon, 1999). Zanker, Cooke, Truscott, Oldroyd, & Jacobs (2004) discussed a case report of an amenorrheic runner. The athlete in this report embarked on a vigorous training regime at age 10 (before menarche). By age 11 she began restricting her diet, and by age 24 she was running 90-100 km, cycling 100-120 km, and 5-6 km of swimming per week. At age 24.8 she had still not reached menarche, and her DEXA values revealed a T-score of -2.8 in the proximal femur and -1.4 in the lumbar spine. This case represents an extreme episode of the FAT in that there proved to be virtually zero osteogentic benefits from the mechanical loading she experienced while running. Therefore, we can assume that aerobic exercise alone does not preservative effect on BMD.

Certain categories of training have a protective effect on bone. The osteogenetic response to the physical activity is localized to the bones that are being stressed (Fehling, Alekel, Clasey, Rector, & Stillman, 1995; Grimmston, Willows, & Hanley, 1993). Grimmston et al. (1993) indicates that the site-specific response of BMD is related to the particular nature of mechanical loading that the athlete undergoes. In fact, their research revealed that children who took part in impact loading sports (running, gymnastics, etc.) had greater BMD at the femoral neck than children who participated in active loading

sports (e.g., swimming).

Impact Loading Versus Active Loading

Athletes who partake in impact loading activities can produce ground reaction forces up to three times their body weight (Grimmston et al., 1993). Fehling et al., 1995, found that athletes who participated in sports that were intrinsically anaerobic or high impact in nature (e.g., volleyball and basketball), had superior BMD than athletes who participated in an active loading sport (swimming). Vainionpaa, Korpelainen, Leppaluoto, and Jamsa (2005) discovered that premenopausal women, aged 35 to 40 years., who performed high-impact exercise at least three times per week, improved their BMD in the femoral neck (1.1% vs. –0.4%; $p = 0.003$), intertrochanter (0.8% vs. -0.2%; $p = 0.029$), and total femoral (0.1% vs. -0.3%; $p = 0.006$) significantly over the non-exercise group during a 12 month period. In this investigation, there was also a significant improvement in trabecular bone measured at the distal ulna, distal radius, and ultradistal radius (7.3% vs. -0.06%; $p = 0.015$). The results of this investigation revealed that improvement was encountered at loaded sites versus non-weight bearing sites. Further, a meta-analysis executed by Wallace and Cumming (2000) established that high-impact exercise increased BMD especially at the femoral neck, a loaded site. The same fortifying impact is not experienced in female distance runners. Runners produce ground reaction forces that should have a protective effect on BMD. Yet, the cyclic disposition of running places excessive strain on the loaded bones which may increase bone turnover rates at the loaded sites elevating the rate of bone resorption (Mori & Burr, 1993; Wang & Salem, 2004).

In the aforementioned research of Fehling et al. (1995), swimmers had BMD

measurements similar to that of the control group (non-athletic females). The researchers attributed the difference in BMD to the manner of training and competition (e.g., mechanical loading) that is inherent to swimming. Swimming does tax the muscles, but it is performed in an almost zero gravity environment. No ground reaction forces are associated with swimming. The lack of impact exercise could have a negative result on swimmer's BMD.

High-impact exercise seems to have a positive effect on the BMD of female athletes and active exercisers; therefore, the repeated performance of specific exercises or long-term participation in a certain type of sport should also produce protective results. Sinaki, Canvin, Phillips, and Clarke (2004) suggest that there is possible a genetic link to BMD and exercisers and athletes that practice the same workouts repeatedly. According to their theory, athletes may be drawn to specific sports or exercisers might choose certain exercises due to their specific genetic predisposition to the sport or exercise. Individuals may have a predisposition to excel at a sport; yet, being predisposed to achieve may not translate to a conclusive consequence on BMD. Frost (1986) proposed that a minimum level of mechanical loading is required to impact BMD (Frost, 1986).

Minimal Effective Strain Stimulus

Frost (1986) coined the term "minimum effective strain stimulus (MESS)" to add definition to the principal of the necessity that a minimum level of mechanical loading is required to elicit change to BMD. Lanyon, Rubin and Baust (1986) and Rubin and Lanyon (1985) refined the nature of the stimulus placed upon the bone, by concluding that the magnitude of the strain and rate of strain placed on the bones are crucial components of mechanical loading. Mechanical loading can be facilitated via ground

reaction force or weight lifting/resistance activities. For further clarification, Dalsky et al. (1988) carried out a study on postmenopausal women and weight bearing activities. Sedentary women were mandated to perform certain weight bearing activities for 22 months. At the end of this period, the participants increased their BMD of the lumbar spine by seven percent. This effect was not maintained without continued weight bearing activity, and this is, perhaps, the most important finding of the Dalsky et al. investigation. After 12 months of inactivity and sedentary living, the participants lost all but one percent of their previous acquired BMD. This finding suggests that detraining or the lack of strain stimulus may impact BMD. This same detraining effect was found in athletes. Kudlac, Nichols, Sanborn, and DiMarco (2004) investigated bone loss in retired collegiate gymnasts (four years post retirement). Their findings suggest that there is a detraining effect on BMD. The BMD loss in the ex-gymnasts appeared to be site-specific. Both controls and ex-gymnasts showed significant decline in the femoral neck, greater trochanter and Ward's triangle (0.72% to 1.9% per yr), yet, the ex-gymnast exhibited further decline in the lumbar spine (0.87% per yr).

Another study dedicated to determining how high impact training can affect BMD was performed by Heinonen et al. (1996). Heinonen et al., (1996), performed an intervention on 98 sedentary premenopausal women (*M* age = 23.8, *SD* = 5.0 years.). The sample was divided into a control group ($n = 49$) and an experimental group ($n = 49$). The experimental group was submitted to 18 weeks of high-impact resistance training. BMD data was collected at the end of 18 weeks. The experimental group had significant improvement at the femoral neck as compared to the control group, although, the BMD at non-weight-bearing sites did not differ between groups. Heinonen and

colleagues concluded that the participants were with in their normal mechanical strain range, and therefore, there was not a significant enough stimulus to render change to their BMD. Resistance training can provide adequate stimulus to create bone improvement.

Resistance training can be accomplished via a series of different types of contractions done in conjunction with another (e.g., concentric and eccentric) or separately (e.g., isometric). An isometric contraction occurs when a muscle contracts, but the muscle does not lengthen or shorten. Concentric contractions transpire when the muscle that is contracted is shortened. Eccentric contractions, conversely, ensue as the muscle that is contracted is lengthened. An example of a concentric contraction followed by an eccentric contraction would be a bicep curl. The upward motion would be concentric and the downward action would be eccentric. It would seem that any type of resistance-training protocol would place a sufficient amount of strain to produce maximal strength gains. Yet, research has shown that optimal muscular tension emerges when eccentric contractions are practiced (Mahew, Roghstein, Finucane, & Lamb, 1995). Schroeder, Hawkins, & Jaque (2004) hypothesized that high intensity eccentric training would produce greater strength and BMD gains. The researchers randomly assigned cohort of 37 young women (*M* age = 24.3) to three groups (high-intensity eccentric [HRT], low-intensity eccentric [LRT], and control). Schroeder et al. findings propose that submaximal eccentric training is best for musculoskeletal modifications. Moreover, LRT actually influenced BMC and bone development, producing a significant gain (1.7%) of BMC at the spine, while HRT accelerated bone resorption. The work of Schroeder et al. provides another example of the negative musculoskeletal impact that overtraining can have on the athlete. Gravity and training have a positive impact on BMD.

Individuals are continually exposed to a gravitational pull that results in a slight amount of mechanical loading. When athletes add ground reaction forces and weight bearing activity, the result provides a positive impact on BMD. Conversely, individuals that are placed in a zero gravity (hypogravity) environment actually loose BMD due to the lack of gravitational pull principally in the spine and lower body regions (Shackelford et al., 2004). An example of this principal would come from research that has been conducted on astronauts. On a 28 day Skylab mission, the astronauts involved lost approximately 50 mg of calcium per day while in the weightless environment of the mission (Vose, 1974) Likewise, research that was performed on individuals that were obliged to continual bed rest or had no activity incurred loses in BMD (Giangregorio & Blimkie, 2002). In fact, research performed by Schneider (1984) on 90 healthy men who were submitted to 5 to 36 weeks of continual bed rest, found that the young men in their investigation lost approximately 200 mg of BMD per day and up to .5 percent per month. Another interesting finding in Schneider's research was that the majority of the BMD loss was found in the lower extremities. The work by Schneider, Vose, and Giangregorio and Blimkie, therefore, offer validity to Frost's MESS theory. Another researcher that has offered a theory on how strain and stress impact BMD is Julius Wolff.

Wolff's Law

Julius Wolff was a surgeon during the late 19th century (Forwood & Turner, 1995). Wolff theorized that bone, in itself, is a dynamic tissue whose architecture is determined by the function and stresses that the bone is submitted to. He noted that specifically trabecular bone is vulnerable to the mechanical stress that it is exposed to. He went further to hypothesize by stating that bone will respond to stress, in such a way,

that it will produce an anatomical structure that is best suited to counter the applied stress. In other words, bone will adapt to the strain that is exerted upon it, in that the areas that receive the greatest amount of stress will produce the maximum fortification. The theory that Wolff documented is commonly referred to as Wolff's Law (Wolff, 1891).

Other researchers have elaborated on Wolff's Law. Canter (1984), developed mathematical models to relate BMD preservation to the mechanical stress that bones are exposed to via discontinuous daily activities and the history of weight bearing activity (load history). Canter discovered that the degree of the loading is far more important than the loading cycle. Rubin and Lanyon (1984) found similar results in relation to the magnitude of load applied to the bone. Yet, Rubin and Lanyon added an additional discovery. They hypothesized that stress that is applied in a diverse nature further enhances BMD. This is an important finding that further supports the theory that the active loading of anaerobic sports has a maximal effect on BMD.

Salter further refined the principal established by Wolff. Salter (1970) confirmed that bone remodeling takes place in response to the mechanical stress and/or the lack of mechanical stress that it is subjected to. Basically, affirming that bone is formed specifically in sites that are subjected to mechanical loading, and is, therefore, resorbed from the sites that endure limited and/or no stress. Consequently, this researcher can surmise that there is a site-specific response to BMD according to the physical stress that the body is exposed to.

Site-specific Adjustments to BMD due to Training

BMD is typically superior in the areas that are subjected to repeated stress. Therefore, individuals that participate in the same sport should exhibit similar site-

specific responses to stress and mechanical loading. In fact, many sports are unilateral in nature. A number of studies have been conducted to document this phenomenon. For example, in 1994 Haapasalo and colleagues examined the side-to-side differences in the dominant arm in female squash athletes. Their results revealed a significant difference between the dominant and non-dominant arm. In a similar study, Montoye, Smith, Fardon, and Howley (1980) discovered that the ulna, radius and humerous of senior tennis players were significantly greater on the dominant limb. Likewise, Jones, Priest, Hayes, Tichnor, and Nagels (1977) observed that the tennis players in their study hand significantly greater cortical thickness in the dominant limbs. Calbet, Diaz Herrera, and Rodriquez (1999) focused on the asymmetric nature of volleyball to ascertain if there is a disparity between the skeletal tissue in dominant and non-dominant limbs. The research revealed that the dominant arm was approximately 3% heavier, had 4 percent more muscle, and had superior BMC at 9% and BMD at 7% ($P < 0.001$) values in the male, elite volleyball athletes as compared to the controls.

McClanahan et al. (2002) discovered that there was a disparity in the BMD in favor of the dominant arm in male and female college athletes that participated in a number of sports (e.g., baseball, basketball, football, golf, soccer, tennis, cross-country, track, and volleyball). The results revealed that the dominant arm had significantly greater BMD than the non-dominant arm. The results also proved that the sports that were unilateral in nature (e.g., tennis and baseball) produced BMD measurements in the dominant appendage that were more prominent. Though, the same side-to-side comparisons were not analogous for the lower extremities of the athletes in the same study.

Kontulainen, Sievanen, Kannus, Pasanen, and Vuori (2003) measured the influence of long-term impact loading on the humerous shaft and distal radius in 64 formerly ranked female tennis and squash players. The population of athletes was divided into two groups: (1) athletes who began their training on or before menarche (young starters, $n = 36$), and (2) athletes who began their training a minimum of one year after menarche (old starters, $n = 28$). The control group consisted of 27 sedentary women who were matched for age, height and weight. Results from pQCT and DEXA revealed a significant site-specific effect on the BMC of the dominant humeral shaft (a predominantly cortical bone) between the young (19%) and old starters (9%). This change was due to the variation in cross-sectional area in cortical bone between young (20%) and old starters (9%). There was no disparity between dominant and non-dominant sites in the cross-sectional area of the marrow cavity, suggesting that the discrepancy was due to periosteal growth (Kontulainen et al.). The bone strength index for the bone end between the two groups was 26% and 11% in favor of the young starters. At the distal radius, there were no significant differences between dominant and non-dominant sites for the controls, while the young starters had a 12% greater BMC in the dominant arm and a 5% greater trabecular density, and the old starters had a 6% superior BMC in the favored arm and a 7% larger trabecular density. There was no difference in cortical wall thickness between the distal radius of the dominant arm in young starters' and the non-dominant arm, but there was 6% dissimilarity between the same sites in the old starters'. Further, the total area of the dominant distal radius for the young starters was 11% greater than the opposing limb, yet, there was no variation discovered for the same site in the old starters'.

The distinction between young starters and old starters seems to suggest two findings: (1) a bone that is still in the growth process has enhanced capability to enhance bone mass, as seen in young starters, and (2) a bone that has ceased development in the epiphyseal area may not expand in size, yet, trabecular density may increase as seen in old starters (Forwood, 2001; Forwood & Burr, 1993; Kontulainen et al., 2003).

More recently, Ducher, Jaffre, Arlettaz, Benhamou, and Courteix (2005) examined the effect of lean tissue mass (LTM) on BMD. The results revealed that there was a significant difference between the BMD of the dominant and non-dominant forearm ($p < 0.0001$) favoring the dominant arm. Additionally, BMC and bone area correlated to grip strength in both the dominant and non-dominant forearms ($r = 0.81$-$0.84, p < 0.0001$). LTM and grip strength were both greater on the preferred forearm. This finding seems to bolster the notion of muscular involvement in the mechanical loading of bones (Ducher et al.)

When comparing the lower extremities, McClahahan et al. (2002) found that there was not a significant difference between left and right legs on BMD measurements among female athletes who participated in collegiate basketball, volleyball, golf, tennis, and track in their investigation. Lee et al. (1995) found no side-to-side, lower body divergence between females athletes that compete in basketball, soccer, and swimming, but they did find that the left lower limb (non-dominant) did illustrate a dissimilarity in volleyball athletes. Calbet et al. (1999) discovered variation between the BMC and BMD of the legs in elite, male volleyball athletes. Moreover, the left leg had a 4 percent greater BMC ($P < 0.05$) than the right (dominant). This result could be due to the approach steps for the volleyball attack (spike). The last step of the approach for a right handed athlete

would be off of the left leg and has the greatest magnitude in the momentum of the action. There were no inter-leg dissimilarities between the controls. Furthermore, Grimmston et al. (1993) suggest that there is a site-specific loading effect in regards to BMD.

Nevill, Burrows, Holder, Bird, and Simpson (2003) performed upper-body and lower-body comparisons of BMD on 49 female endurance runners that averaged 32.3 kilometers per week (*SD* = 17). None of the athletes were amenorrheic, although ten were oligomenorrheic. The investigators compared ten sites (left and right arm, left and right rib, thoracic and lumbar spine, pelvis, left and right leg, and hip). The results indicated that the left and right leg had significantly greater BMD than all other sites ($P < 0.001$). The lower extremity sites all produced mean values that exceeded the values for the upper body, and the BMD values declined the greater the distance was from legs. This finding suggests that the ground reaction forces encountered during endurance running builds BMD. Yet, the BMD values declined the further the site was from the legs, suggesting that the benefit of the exercise is most significant at the loaded site. Interestingly, none of the athletes were amenorrheic and only ten were oligomenorrheic, thus, there was possibly only a slight effect from hormonal influences. There could be other factors that effect site-specific changes in BMD.

Age related factors may affect BMD at certain sites. Mein, Briffa, Dhaliwal, and Price (2004) performed a longitudinal study on a cohort of 62 healthy Caucasian women. In the study, young women were subjected to 2-year placebo-controlled calcium intervention. Physical activity was also measured. The participants were tested at baseline (*M* age = 18.5 +/- 0.3 years.), after the 2-year intervention (~ 20 years.), and

approximately 7.4 years. (*M* age = 27.8 +/- 1.0 years.). The results indicated that there are site-specific changes that occur over time. At the lumbar spine, intertrochanter and whole body there was significant improvements in BMD (14.12%/decade). Conversely, there was a reduction in BMD at the femoral neck and trochanter. The differences that occurred coincide with a significant augmentation in area (2.7%/decade, $p < 0.001$) and BMC (7.2%/decade, $p < 0.001$) at the lumbar spine, an increase in BMC at the intertrochanter (2.4%/decade) and no change in area, a decline in BMC at the femoral neck (-2.8%/decade, $p < 0.001$) but no change in area, and at the trochanter bone area improved (2.8%/decade, $p < 0.012$) but BMC was unchanged. There was a positive association with physical activity at all BMD sites ($r = 0.254$, $p = 0.047$). These data suggest there is site-specific bone growth subsequent to linear development cessation.

The physiological effect of training can impact BMD in a number of significant manners. Physical activity, more specifically, training that forces weight bearing stimulates bone remodeling. Wolff was the first to recognize that bone adapts and fortifies to accommodate the repeated stress that is placed upon it. Further refining the theory of Wolff, Frost explained that there is a minimal level of stimulus necessary in order to influence BMD. Training which sanctions impact loading can generate stimulus that is a threshold great enough to initiate remodeling. Numerous investigations highlight the positive influence of high impact training on the weight bearing bones of athletes. Additionally, there appears to be a site-specific effect of loading on the load bearing sites of the skeletal structure of athletes. For example, athletes that participate in unilateral sports generally have greater BMD in the dominant appendage as compared to the non-dominant.

Summary

The primary focus of this review has been to underscore how training, as well as other factors, can impact the BMD of female athletes. The most drastic expression of bone injury is osteoporosis. Females are at a particular risk of developing osteoporosis mainly due to the hormonal fluctuations and bone geometry associated with aging and genetic structure. Female athletes are at an elevated risk of premature osteoporosis due to the nature of their training and societal pressures that open the door for entry into the FAT. The FAT is a collection of interconnected maladies (eating disorders, amenorrhea, and osteoporosis) that can influence the BMD of physically active females. Certain lifestyle interventions and varieties of training can offer a protective effect on the BMD of female athletes. In fact, there appears to be a site-specific effect on BMD to training.

Chapter III

Methodology

Participants

National Collegiate Amateur Athletic (NCAA) Division I-A female student-athletes (n=50) who attended a university in the southeastern U.S. during the 2004-2005 competitive season volunteered to engage in this study. The following intercollegiate sports were represented: volleyball (n=10), soccer (n=17), track (n=4), tennis (n= 5), golf (n=1), cheerleaders (n=5), softball (n=5), and volleyball/track (n=3). The population was multi-cultural with representatives from thirteen nations, with 66% of the sample listing the U.S. as their country of origin (n = 33), 6% listing Canada (n = 3), and 6% naming France (n = 3) respectively (see Table 1). The ethnicity was somewhat diverse with 78% (n = 39) reporting as Caucasian (see Appendix A). Interestingly, 12% (n = 6) identified themselves as "other". This was somewhat of a confounding factor. Although, many of the members of this group where from South American countries, they therefore, neither identify as Latino or Caucasian. The sample was divided into two groups, athletes who participated in a sport who used predominately the lower body (n=21) and athletes who participated in a sport that relied primarily on both the upper and lower body (n=29).

Approximately 42% of the athletes participated in sports that utilize predominately the lower body (n = 21) while 58% of the sample were members of team

athletics that employ a mixture of both the upper and lower body ($n = 29$). A majority of the sample, 88%, had been competing in their sport for at least six years (see Appendix A). The participants ranged in age from 18 to 35 ($M = 20.4$; $SD = 3.07$). Appendix A illustrates additional demographic information collected from the College Athlete Questionnaire. Table 2 reports the means and standard deviations for age, BMI, EAT-26, PBF, BMD, and grade point average (GPA). The data regarding GPA were recorded by the participants was not completely accurate. Many of the participants were either from foreign countries or were incoming freshmen; thus, a portion of the sample was unable to definitively report their GPA.

Study Design

This investigation was performed to discover descriptive information regarding the BMD of female athletes; therefore, the study design is descriptive.

Instrumentation

Data were collected in the Human Performance Lab on the campus of Middle Tennessee State University. Upon arrival at the testing site, each athlete read and signed an informed consent form and completed a battery of tests (See Appendix A). These tests included the a demographic questionnaire (See Appendix B), EAT-26; (Garner, Olmsted, Bohr, & Garfinkel, 1982) (See Appendix C), skinfolds at three sites (the abdomen, supraillium, and tricep) to measure percent body fat (PBF), height, weight, and four bone scans (right and left forearm and right and left femoral neck) on the dual-energy x-ray absorptiometry (DEXA). All of the data were collected in during one 20-minute session per participant.

Table 1

Nationality of Sample

Nationality	n	%
United States	33	66
Canada	3	6
Brazil	1	2
Argentina	1	2
Uruguay	1	2
Greece	1	2
France	3	6
Sweden	1	2
Scotland	1	2
Wales	2	4
England	1	2
Australia	2	4

Note. $N = 50$.

Demographic Questionnaire

The demographic information consisted of questions about the athlete's menstrual cycle function (primary and secondary amenorrhea, oligomenorrhea, and eumenorrhea),

age of menarche, dairy intake, year in school, sport, age, grade point average, exercise adherence outside of requirements, and history of stress fractures.

Eating Disorder Questionnaire

The Eat-26 is a Likert-type scale comprised of 26 items with responses ranging from 0-3. The test contains 3 subscales, dieting, bulimia and food preoccupation, and oral control, which have been shown to predict an eating disorder (Garner, Olmsted, Bohr, & Garfinkel, 1982). To score the EAT-26, each response is assigned a value according to the weight of the response given. Of the 26 questions, 25 are weighted from left to right. These items are positively scored and are assigned the following values, *always* = 3, *usually* = 2, *often* = 1, *sometimes* = 0, *rarely* = 0, and *never* = 0. Only one question is negatively scored (weighted from right to left), and this item is number 25. The negatively scored item is inversely weighed (Garner, 1993). The sum of all subscales produces the final score. Values above 20 indicate that there is a high likelihood of an eating disorder.

It is difficult to detect eating disorders in populations of adolescent or young women (Teitz, 1997). Young women may not answer questions honestly in order to hide their disordered eating regimen (Teitz, 1997). However, the EAT-26 has proven to be an effective tool when used as part of a two-stage screening process. According to surveys of young women, approximately 15 percent of the females tested scored above 20 on the Eat-26 (a positive score); (Garner & Garfinkel, 1979; Garner et al., 1982). Scoring 20 and above on the questionnaire reflects the presence of an eating disorder. Studies have indicated that those who achieve a score below 20 on the questionnaire do not have a diagnosed eating disorder. The likelihood that an individual would score below 20 on the

EAT-26 and still have an eating disorder is rare (King, 1989, 1991). Consequently, the EAT-26 produces few false negatives. The EAT-26 is accredited with an acceptable criterion-related validity due to its ability to accurately predict membership to a group at a significant level. Likewise, he reliability of the EAT-26 is high (α = 0.90 for the anorexia nervosa group).

Table 2

Characteristics of Participants

Characteristics	M	SD
Age	20.10	3.07
Height	65.25	2.86
Weight	144.10	22.12
Body Mass Index	23.66	2.95
Percent body fat	22.45	5.31
Grade point average	3.23	0.66
EAT-26	6.36	7.82
Dominant arm	0.60	0.06
Non-dominant arm	0.60	0.06
Dominant leg	1.00	0.14
Non-dominate leg	0.99	0.13

Note. N = 50.

Skinfold Measurement

Three skinfold measurements (SKF), of PBF, were obtained using Lange calipers (Cambridge Scientific Instruments). Lange calipers (LNG) are commonly used as field measures to determine PBF. Harpenden (HRP) calipers are the alternate calipers that exercise physiologists and exercise scientists frequently employ to predict PBF. Gruber et al. (1990) determined that a variance of 1.5 percent could be estimated when comparing the results of each type of caliper on the measurements extracted from female participants. The HRP calipers repeatedly produce a 1.5 percent lower estimate of PBF than the LNG calipers, and a 2.9 percent lower estimate than underwater weighing (UWW). The UWW has been considered the recognized model for measuring body density and PBF. The LNG calipers varied only 1.4 percent from the UWW. Gruber et al. (1990) estimate that the LNG calipers seems to be the more accurate and valid field measure for PBF.

The researcher extracted SKF at three sites (abdomen, supraillium, and tricep). To insure interreliability of the SKF, the researcher seized three measures at each, individual site. The average of the three trials was recorded as the final SKF value for each site. All the measurements were taken in one encounter by the primary investigator to provide additional reliability.

SKF measures the subcutaneous fat represented at the considered site. SKF works on two assumptions: (1) it is an accurate measure of subcutaneous fat, and (2) there is a correlation between total body fat and subcutaneous fat (Wagner & Heyward, 1999). Lohman (1992) analyzed a group of 153 men to determine the standard error of

estimation (SEE) in predicting BF with SKF. Lohman discovered the SEE to be 3.5 percent.

The following gender specific equation was utilized to predict the body density of the participants in the investigation: body density = 1.089733 – 0.0009245 (sum of three skinfolds) + 0.0000025 (sum of three skinfolds) 2 – 0.0000979 (age) (Jackson & Polluck, 1985). The original regression model was developed by Jackson and Polluck (1978). This formula evolved from measuring a sample of 308 men ages 18 to 61yrs., and then cross-validated with a similar population of 95 men. This equation utilized 7 SKF (chest, midaxillary, triceps, subscapula, abdomen, suprailium, and thigh; R = .90, SEE = 0.0078 g/cc) (Jackson & Polluck, 1978). Body density was then converted to body fat percent using the Siri (1961) equation: %BF = [(4.95/Db) - 4.50] x 100.

Additional Anthropometric Measurements

The height (measured in meters) and weight (measured in kilograms) of each athlete was measured to ascertain their body mass index (BMI) or Quetelet index, a field measurement that assesses weight in relationship to height. This measurement was employed to provide a reference point for relative size to PBF and BMD. BMI is calculated by dividing body weight in kilograms by height in meters squared (kg/m^2) (American College of Sports Medicine, 2000).

Bone Mineral Density Measurement

The DEXA is the most popular method of measuring bone mineral density (BMD). The DEXA Hologic QDR 4500W (S/N 49865) software version 11.2:5 was utilized to measure the athlete's BMD. Operating the DEXA requires a medical x-ray license. Data were collected by two faculty members who possess this license. The

Hologic QDR 4500W provided a superior system geometry, which utilized the fanbeam technology. The fanbeam detector emitted a large detector array, which enabled the scan to be clearer, take less time, and scan the measured area (Orwell & Bliziotes, 2003). Although the DEXA is the "gold standard" for measuring BMD, there is small chance of error. The estimated error of DEXA scans on samples of bone ranges from two to four percent (Kolta, Ravaud, Fechtenbaum, Dougados, & Roux, 1999; Kuiper, van Kuijk, Grashuis, Ederveen, & Schutte, 1996).

The reliability of the DEXA is contingent on several factors that may generate error. Some of the more common sources of error may arise with the machine, with the operator, or with the patients (Orwell & Bliziotes, 2003). Machine error may be due to variation wit the energy of the x-ray tube, however, if the unit is properly maintained, the error is approximately only 0.05%. The computer can also be inconsistent in determining bone edges. The operator can generate error by determining improper anatomic markers, or position the patient incorrectly. In addition, the patient can move after the operator has stabilized the position and cause inaccurate readings. Thus, reliability is optimal if the same operator performs the scans and positions the participant in the same manner for each scan. The scanner was calibrated before each testing session to insure reliability of the measure as well.

The DEXA is considered a safe procedure for both patient and operator. The DEXA does not require special adaptations to provide safety. For example, the scanning facility need not be shielded to protect the participants or the operator (Hologic, 1997). A dose of radiation provided during the course of a whole body scan (2.6 uSV), which requires approximately six minutes of scanning, is far less than the exposure that one

encounters when flying from New York to San Francisco round-trip (40 uSV) (Bezakova, Collins, & Beddoe, 1997). The participants in this investigation were participanted to four short, regional scans of the right and left forearm (radius and ulna) and the right and left femoral neck. Thus, the operator and participants were exposed to trace amounts of radiation.

Procedures

The Institutional Review Board (IRB) at Middle Tennessee State University (MTSU) approved this investigation (see Appendix D, E, and F). The researcher then sent a memo to the Assistant Athletic Director, and Senior Women's Administrator (SWA) to request access to the coaches and athletes in the Athletic Department (see Appendix G). The administrator granted permission to contact the coaches of all female sports. A memo explaining the relevance of the study was sent to each coach asking for authorization to include their athletes for the study (See Appendix H). Players who agreed to be a part of the study were scheduled for data collection according to the availability of the athletes, the x-ray technicians, and the scanning facility. The researcher allowed approximately 15-20 min. per athlete to collect the data. Upon arrival at the testing facility, each athlete was provided two copies of the informed consent form to read and sign. One copy remained with the participant and the other copy was returned to the researcher. After the informed consent form was signed, the athlete was requested to complete a questionnaire that obtained demographic information, and the EAT-26. Height and weight (BMI) was recorded. Next, three skinfold measurements were obtained followed by four scans on the DEXA. The x-ray technicians evaluated the scans and provided a final read out of the BMD measurements for each site.

Statistical Analysis

A multivariate analysis of variance (MANOVA) was utilized to determine differences between the type of sport and the preferred limb to upper and lower BMD. SPSS 11.5 for Windows: SPSS Chicago, Illinois, U.S.A (SPSS, 2002). The dependent variable was four levels of BMD of the preferred arm, non-preferred arm, preferred leg, and non-preferred leg. The independent variables, both categorical, consisted of the type of sport, predominately lower body sport or predominately upper and lower body sport. Four separate regression analysis were performed in order to determine the effect of the athlete's score EAT-26, the athlete's PBF, BMI, and whether or not the athlete had episodes of amenorrhea, had a main effect on each BMD measurement. The dependent variable for this equation was the four BMD measurements (dominant/non-dominant forearm and dominant/non-dominant femoral neck). The independent variables were the participant's score on the EAT-26, PBF, BMI, and whether or not the athlete had episodes of amenorrhea. The incidence of amenorrhea was a categorical independent variable (yes or no).

Chapter IV

Results

The Effect of Sport Type on Bone Mineral Density

A one-way multivariate analysis of variance (MANOVA) was performed on the BMD of the dominant and non-dominant forearm and femoral neck to determine if sport type influenced BMD. Table 1 lists the descriptive data for BMD. The dependent variable for the analysis was the T-scores for the BMD sites, while the independent variable was type of sport with two levels. The T-scores were utilized in this model to normalize the BMD measurements. There was a large discrepancy between the lower and upper body BMD measurements, because BMD measurements of the femoral neck are typically denser than the BMD of the forearm, resulting in high variance of BMD scores between the upper and lower body (see Table 3). Therefore, the data required normalization to find significant relationships in the analysis. According to WHO (1994), the T-score is the criterion reference for BMD by which all BMD diagnosis are ascertained.

Wilk's Lambda .810, $F(4, 45) = 2.64$, $p < .05$, indicated significant differences between the two sport types on the BMD. The Levene's Test was significant for the dominant/non-dominant femoral neck ($p < .01$), indication variance was not equal for the lower body BMD. Levene's test also indicated lack of significance for the upper body BMD. This indicated equal variance across the model for the upper body. An eta squared

was .19 indicated that 19% of the variance in the multivariate of BMD was explained by sport type. The sample size was too small to perform a post hoc test to determine which sport type (sports that make greater use of the lower body or sports that utilize the upper and lower body) contributed most to the BMD of this sample.

Table 3

Mean and Standard Deviation of Bone Mineral Density Data of Participants by Sport

Characteristics	BMD Measurements Forearm				BMD Measurements Femoral Neck			
	Dominant		Non-dominant		Dominant		Non-dominant	
	X	SD	X	SD	X	SD	X	SD
Volleyball	.600	.042	.607	.045	1.022	.092	1.024	.083
Softball	.558	.040	.541	.072	.949	.085	.942	.091
Soccer	.573	.053	.575	.050	.950	.079	.937	.077
Track	.630	.090	.623	.084	1.102	.067	1.081	.075
Volleyball/Track	.672	.063	.673	.036	1.281	.193	1.273	.188
Golf	.620	0	.590	0	.839	0	.871	0
Tennis	.605	.036	.587	.018	.913	.161	.873	.161
Cheerleading	.680	.065	.668	.067	1.030	.214	1.046	.126

Analysis of variance (ANOVA) was conducted on each BMD t-score to determine the effect of sport type on each of the BMD sites. Table 4 lists the *F* scores for each BMD site. The only significant finding for this model was for the non-dominant

femoral neck ($p < .05$). The eta squared for the non-dominant femoral neck was .102, indicating 10.2% of the variance of BMD of the non-dominant femoral neck was accounted for with sport type. Sport type was positively related to the BMD of the non-dominant femoral neck. Sport type was not related to the forearm (dominant/non-dominant), or to the dominant femoral neck. BMD of the non-dominant femoral neck is significantly higher than the BMD of the dominant femoral neck and dominant/non-dominant forearm.

Table 4

Multivariate and Univariate Analysis of *F* Scores For Bone Mineral Density

		ANOVA			
	MANOVA	Dominant Forearm	Non-dominant Forearm	Dominant Femoral Neck	Non-dominant Femoral Neck
Variable	F(4, 45)	F(1, 48)	F(1, 48)	F(1, 48)	F(1, 48)
Upper/Lower Body Sport	2.64*	4.22	3.03	2.17	4.55*

Note. *F* ratios are Wilk's approximation of *F*s. MANOVA = multivariate analysis of variance; ANOVA = univariate analysis of variance.

*$p < .05$.

Predictors of Bone Mineral Density

Four regression analyses were computed in order to calculate the result of the four main effects, PBF, EAT-26 score, episodes of amenorrhea, and BMI, on the dependent variable, the BMD measurements of the dominant/non-dominant forearm and femoral

neck. Only two participants (4%) produced a "positive score" (i.e., 20 or above) for the EAT-26 (Garner et al., 1982). Seven-teen athletes (34%) had undergone at least one episode of amenorrhea. Thus, this sample of athletes did not show a high prevalence of disruption of the hypothalamic-pituitary axis.

Bone Mineral Density from the Dominant Forearm

The full regression model consisted of four main effects and no interactions (see Table 5). There were two variables that did show significance, PBF and BMI ($p < .05$). The R^2 for the full model was .178. Episodes of amenorrhea and the EAT-26 score did not show significance, indicating that EAT-26 score, episodes of amenorrhea, PBF and BMI account for 17.8% of the variance in the BMD of the dominant arm.

For the second regression model, episodes of amenorrhea and the EAT-26 score were eliminated. BMI remained a significant indicator of BMD ($p < .05$), while PBF was not. The R^2 for step 2 was .126, indicating that 12.6 % of the variance in the BMD of the dominant forearm was explained by BMI and PBF. This results in a 5.25% decrease in the explanation of BMD of the dominant forearm by all four variables in the full model.

In the final regression model, PBF was removed. BMI was significant at ($p < .001$). The R^2 for step 3 was .429, which resulted in a R^2 change of .251. For BMD of the dominant arm in female collegiate athletes, BMI accounted for 42.9% of the variance. When controlling for the other variables in the full model, BMI was positively related to the BMD of the dominant forearm, while PBF, EAT-26 score, and episodes of amenorrhea were not significantly related to BMD of the dominant forearm.

Table 5

Summary of Regression Analysis for Variables that Predict the Bone Mineral Density in the Dominant Forearm of College-Aged Female Athletes

Variable	*B*	*SEB*	β	
Step 1				
Constant	0.436	0.071	0	***
Body fat percent	-0.005	0.002	-0.426	*
BMI	0.012	0.004	0.552	*
EAT-26	0.002	0.001	0.193	
Skipped menstrual cycle	-0.025	0.019	-0.188	
Step 2				
Constant	0.452	0.071		***
BMI	0.010	0.004	0.484	*
Body fat percent	-0.004	0.002	-0.349	
Step 3				
Constant	0.230	0.060		***
BMI	0.016	0.003	0.655	***

Note: $R^2 = .178$ for Step 1 ($p < .05$). $R^2 = .126$ for Step 2 ($p < .05$).

$R^2 = .429$ for Step 3 ($p < .001$).

According to intercorrelations of the four-predictor variables, episodes of amenorrhea, PBF, and BMI were significantly correlated ($p < .05$) to the BMD of the dependent variable (see Table 6). BMI and PBF were significantly related to one another ($p < .001$). EAT-26 and episodes of amenorrhea were also correlated to ($p < .05$).

Table 6

Intercorrelations for the Bone Mineral Density of the Dominant Forearm in College-aged Female Athletes and Predictor Variables

Variable	1	2	3	4
BMD of dominant forearm	.150	-.023	.246 *	.-.021
Predictor variable				
1. EAT-26		.239 *	.060	.073
2. Episodes of amenorrhea	.239 *		.149	-.084
3. BMI	.060	.149		.680 ***
4. Body fat percent	.073	-.084	.680 ***	

*$p < .05$. ***$p < .001$.

Bone Mineral Density from the Non-dominant Forearm

The results of the full regression model for the non-dominant forearm revealed an R^2 of .162. BMI and PBF were both significant predictors of BMD at the non-dominant forearm ($p < .05$), while episodes of amenorrhea and the EAT-26 score were not significant (see Table 7). EAT-26, episodes of amenorrhea, BMI, and PBF account for

16.2% of the variation in the BMD of the non-dominant forearm. Thus, the independent variables have only a small effect on BMD.

In the reduced regression model, BMI and PBF remained significant predictors of BMD ($p < .05$). Episodes of amenorrhea and the EAT-26 score were purged from the model to create a more parsimonious model. The R^2 for the reduced model was .147, indicating that 14.7% of the variation in the BMD of the non-dominant forearm was explained by the variables PBF and BMI. There was a .015 decrease in the R^2 for this model. Therefore, PBF and BMI explained 14.7% of the variance in the BMD of the non-dominant forearm. When controlling for EAT-26 score and episodes of amenorrhea, BMI was positively related and PBF is negatively related to the BMD of the non-dominant forearm of college-aged female athletes ($p < .05$). EAT-26 score and episodes of amenorrhea are not related to the BMD of the non-dominant forearm. The null hypothesis of no effect of BMI, PBF, EAT-26 score, and episodes of amenorrhea on the BMD of the non-dominant forearm was rejected for BMI and PBF. The null was accepted for EAT-26 score and episodes of amenorrhea, indicating that EAT-26 score and episodes of amenorrhea have no significant effect on the BMD of the non-dominant forearm.

The predictor variables were not significantly correlated with the BMD of the non-dominant forearm, but two intercorrelations among the predictor variables were significant (see Table 8). The relationship between EAT-26 score and episodes of amenorrhea ($p < .05$) were significantly correlated, and between PBF and BMI were ($p < .001$).

Table 7

Summary of Regression Analysis for Variables that Predict the Bone Mineral Density in the Non-dominant Forearm of College-Aged Female Athletes

Variable	*B*	*SEB*	β	
Step 1				
Constant	0.441	0.071		***
Body fat percent	-0.005	0.002	-0.463	*
BMI	0.012	0.004	0.562	*
EAT-26	0.001	0.001	0.074	
Skipped menstrual cycle	-0.017	0.019	-0.126	
Step 2				
Constant	0.450	0.070		***
BMI	0.011	0.004	0.515	*
Body fat percent	-0.005	0.002	-0.415	*

Note: $R^2 = .162$ for Step 1 ($p < .05$). $R^2 = .147$ for Step 2 ($p < .05$).

Bone Mineral Density from the Dominant Femoral Neck

The R^2 for the full regression model for the dominant femoral neck was .117 (see Table 9). BMI was the only significant main effect for this model. In the reduced regression model, PBF, EAT-26, and episodes of amenorrhea were eliminated, resulting in an R^2 change of .250. Thus, there was a 25% increase in of explaining BMD of the

dominant femoral neck by BMI. Therefore, 36.7% of the variance in the BMD of the dominant femoral neck was explained by BMI. When controlling for EAT-26, episodes of amenorrhea, and PBF, BMI was positively related to the BMD of the dominant femoral neck. EAT-26, episodes of amenorrhea, and PBF were not related.

Table 8

Intercorrlelations for the Bone Mineral Density of the Non-dominant Forearm in College-aged Female Athletes and Predictor Variables

Variable	1	2	3	4
BMD of non-dominant forearm	.043	.014	.233	-.065
Predictor variable				
1. EAT-26		.239 *	.060	.073
2. Episodes of amenorrhea	.239 *		.149	-.084
3. BMI	.060	.149		.680 ***
4. Body fat percent	.073	-.084	.680 ***	

*$p < .05$. ***$p < .001$.

The intercorrelations between the four main effects revealed a significant relationship between the predictor variable, BMI, and the BMD of the dominant femoral neck (see Table 10). Table also corroborates the significant relationship between EAT-26 and episodes of amenorrhea ($p < .05$), as well as BMI and PBF ($p < .001$).

Table 9

Summary of Regression Analysis for Variables that Predict the Bone Mineral Density in the Dominant Femoral Neck of College-Aged Female Athletes

Variable	*B*	*SEB*	β	
Step 1				
Constant	0.628	0.165		***
Body fat percent	-0.007	0.005	-0.280	
BMI	0.230	0.010	0.480	*
EAT-26	0	0.003	0.025	
Skipped menstrual cycle	-0.32	0.045	-0.108	
Step 2				
Constant	0.349	0.119		**
BMI	0.027	0.005	0.606	***

Note: $R^2 = .117$ for Step 1 ($p < .05$). $R^2 = .367$ for Step 2 ($p < .001$).

Bone Mineral Density from the Non-dominant Femoral Neck

Table 11 lists results of the main effects on the BMD of the non-dominant femoral neck. The full regression model generated an R^2 of .10. BMI was the only significant variable ($p < .05$) in the full model. After eradicating the non-significant main effects, the reduced regression model yielded an R^2 of .319; therefore, 31.9% of the variance in the BMD of the non-dominant femoral neck is explained by BMI. This finding reflects a 21.9% increase in the explanation of the BMD of the non-dominant neck by the BMI.

When controlling for EAT-26, episodes of amenorrhea, and PBF, BMI was significantly related to the BMD of the non-dominant femoral neck. EAT-26, episodes of amenorrhea, and PBF were not significantly related.

Table 10

Intercorrlelations for the Bone Mineral Density of the Dominant Femoral Neck in College-aged Female Athletes and Predictor Variables

Variable	1	2	3	4
BMD of dominant femoral neck	.008	-.007	.275 *	.057
Predictor variable				
1. EAT-26		.239 *	.060	.073
2. Episodes of amenorrhea	.239 *		.149	-.084
3. BMI	.060	.149		.680 ***
4. Body fat percent	.073	-.084	.680 ***	

*$p < .05$. ***$p < .001$.

Intercorrelations of the predictor variables indicated that none of the predictor variables were significantly correlated with BMD of the non-dominant femoral neck (see Table 12). The four main effects combined accounted for only 10% of the variance in BMD. There were significant relationships between, however, between EAT-26 score and episodes of amenorrhea ($p < .05$), and between BMI and PBF ($p < .001$).

Table 11

Summary of Regression Analysis for Variables that Predict the Bone Mineral Density in the Non-dominant Femoral Neck of College-Aged Female Athletes

Variable	*B*	*SEB*	β	
Step 1				
Constant	0.727	0.158		***
Body fat percent	-0.009	0.005	-0.354	
BMI	0.020	0.009	0.436	*
EAT-26	0.001	0.003	0.076	
Skipped menstrual cycle	-0.042	0.043	-0.150	
Step 2				
Constant	0.402	0.120		**
BMI	0.025	0.005	0.565	***

Note: $R^2 = .1$ for Step 1 ($p < .05$). $R^2 = .319$ for Step 2 ($p < .001$).

Additional Analysis

Two additional regression analyses were conducted to examine the relationship between BMI and BMD among athletes who participated in sports that used primarily upper/lower limbs or lower limbs. The models each contained four main effects, EAT-26, episodes of amenorrhea, BMI and PBF. The dependant variable was the mean score for BMD at all sites. The first model compared the four main effects to BMD among

athletes who participated in sports that utilize both the upper and lower body. Table 13 lists the results of model one. Model one was not significant, however, the main effect, BMI was a significant predictor of BMD in athletes who utilize both their upper and lower body in their sport ($p < .01$). The R^2 for this model was .17, indicating that only 17% of the variance in the BMD of upper/lower body athletes is explained by all four main effects. When controlling for the other variables in the model, BMI was significantly related to BMD.

Table 12

Intercorrlelations for the Bone Mineral Density of the Non-dominant Femoral Neck in College-aged Female Athletes and Predictor Variables

Variable	1	2	3	4
BMD of non-dominant Femoral Neck	.040	-.037	.177	-.040
Predictor variable				
1. EAT-26		.239 *	.060	.073
2. Episodes of amenorrhea	.239 *		.149	-.084
3. BMI	.060	.149		.680 ***
4. Body fat percent	.073	-.084	.680 ***	

*$p < .05$. ***$p < .001$.

Table 13

Summary of Regression Analysis for Variables that Predict the Bone Mineral Density in College-Aged Female Athletes Who Participate in Sports that Require the Upper and Lower Body

Variable	*B*	*SEB*	β	
Step 1				
Constant	-4.129	2.037		***
Body fat percent	-0.057	0.054	-0.294	
BMI	0.279	0.121	0.701	*
EAT-26	0.007	0.021	0.066	
Skipped menstrual cycle	-0.603	0.439	-0.289	

Note: $R^2 = .215$ ($p < .05$).

The second model compared the four main effects to BMD from athletes who perform using predominantly lower limbs. Table 14 lists the results from model two. Model two was not significant, but BMI was a significant predictor of BMD in lower body athletes ($p < .05$). The R^2 was .215. A variation of 21.5% in the BMD of females athletes can be explained by the main effects BMI, PBF, EAT-26, and episodes of amenorrhea. When controlling for EAT-26 score, episodes of amenorrhea, and PBF, BMI is significantly related the BMD of lower body athletes.

Table 14

Summary of Regression Analysis for Variables that Predict the Bone Mineral Density in College-Aged Female Athletes Who Participate in Sports that Require Predominantly Lower Body

Variable	*B*	*SEB*	β	
Step 1				
Constant	-4.129	1.055		***
Body fat percent	-0.060	0.033	-0.342	
BMI	0.176	0.061	0.558	**
EAT-26	0.017	0.017	0.141	
Skipped menstrual cycle	-0.301	0.286	-0.154	

Note: $R^2 = .170$ ($p < .01$).

The results of each coefficient table from the first and second models were plugged into the following regression formula: Y = B * X + A.or the following equation: BMD = BMI(x_1) + PBF(x_2) + EAT-26 score(x_3) + episodes of amenorrhea(x_4) + A. The main effects EAT-26, episodes of amenorrhea, and PBF were not significant in model one or model two; yet, the main effects, however, were utilized in the formula due to the original research questions. To find the BMI value for the upper/lower limb sport type and lower limb sport type, the mean values for BMI (M = 23.66), PBF (M = 22.45), EAT-26 (M = 6.36), and episodes of amenorrhea (M = .34) were multiplied by each

corresponding β from each regression model one and two. A T-score of -1 was inserted as the value of BMD. This value was chosen as the point at which low BMD becomes a great concern for athletes. According to WHO (1994) a T-score of -1 indicates the presence of osteopenia, the precursor to osteoporosis. The constant for BMD was 2, indicating that a T-score of 2 equates to normal BMD.

The results from the additional regression equation for model one uncovered a BMI value of 20.12. When controlling for PBF, EAT-26, and episodes of amenorrhea, athletes who utilize primarily both the upper and lower limbs in their sport, and who have a BMI of 20.12 or less will be more likely to experience osteopenia.

For the second model, the BMI value from the additional equation was 18.17. When controlling for PBF, EAT-26, and episodes of amenorrhea, female athletes who utilize predominantly the lower limbs in their sport that have a BMI of 18.17 or less will be more likely to experience osteopenia.

In conclusion, results from the MANOVA indicated a significant effect of sport type on BMD; although, the population was two limited to determine which sport type produced the significant effect. Results from the regression analysis indicate that there is no significant effect of EAT-26, episodes of amenorrhea, PBF and BMI on all four BMI sites. However, BMI was a significant predictor of BMD ($p < .05$; $p < .001$) at the sites measured in the female athletes in this population. PBF predicted BMD in the non-dominant forearm ($p < .05$), but was not a predictor of BMD at the other sites. Episodes of amenorrhea and EAT-26 scores were not significant predictors at any BMD site. All four variables were hypothesized to predict BMD, yet, BMI was the only variable that was a consistent predictor of BMD in each regression analysis. PBF was a significant

predictor of BMD at the non-dominant forearm exclusively. When the population was divided by sport type, only BMI was significantly related to the BMD of each sport type. EAT-26, episodes of amenorrhea, and PBF did not significantly predict BMD in either sport type.

Chapter 5

Discussion

Purpose Statement

The primary purpose of this study was to examine the extent to which BMD would be site-specific, that is, preferred appendages versus non-preferred and upper body versus lower body, among university Division I-A, female athletes. The secondary purpose of this study was to determine to what extent the EAT-26 score (an inventory for determining the presence of an eating disorder), BMI, PBF, and the episodes of amenorrhea contribute to the bone mineral status of university Division I-A female athletes.

This chapter is divided into four sections: (1) a summary of findings, (2) the design issues that influenced this investigation, (3) the conclusions that were ascertained from the data, and (4) the implications and future directions for this research.

Summary of Findings

The results of the current investigation supported the first hypothesis statement, the type of sport will effect the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, when controlling for episodes of amenorrhea, evidence of an eating disorder, PBF, and BMI. The second hypothesis was rejected, when controlling for BMI, past episodes of amenorrhea, PBF, limb preference and type of sport, evidence of an eating disorder will

predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes. PBF was significant for the forearm, thus, the third hypothesis, when controlling for BMI, evidence of an eating disorder, past episodes of amenorrhea, limb preference and type of sport, PBF will predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, was partially supported. BMI was found to be a significant predictor of BMD at each of the four sites measured, therefore, the fourth hypothesis, when controlling for the evidence of an eating disorder, PBF, past episodes of amenorrhea, limb preference and type of sport, BMI will predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, was supported in the current investigation. Finally, episodes of amenorrhea was not significant across each regression model, consequently, the fifth hypothesis, when controlling for the evidence of an eating disorder, PBF, limb preference and type of sport, and BMI past episodes of amenorrhea will predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, was rejected.

Type of Sport

The findings were inconclusive for sport type. There was a significant relationship found between type of sport and BMD, but there were too few participants to draw a conclusion as to which type of sport, upper/lower body or lower body, caused the significant affect. The power was moderate to low at .692. There was a significant interaction among type of sport and the BMD of the non-dominant femoral neck. This could indicate that the non-dominant leg of athletes may not receive the same bone

stimulating stress that the dominant leg does, thus, the leg has less BMD than the dominant leg. The results of this study indicated that sport type influences BMD. In this investigation, there appeared to be a site-specific response to the type of sport performed.

This investigation did not include a control sample to compare the means of BMD, but prior research on BMD and exercise has exposed a significant relationship between high-impact exercise and increased bone mass. As reviewed earlier, Vainionpaa et al.(2005) conducted a twelve-month intervention on a randomized sample of 120 premenopausal women, ages 35 to 40 years. The intervention sample was asked to perform high-impact exercises, three times per week. At the completion of the study, the exercise group improved their BMD in the femoral neck by 1.1%. and the intertrochanteric by .8%. The total lumbar did not improve. They conclude that high-impact exercise significantly increased the loaded bone areas of the lower body, but had little effect on the non-weight bearing bones. In their meta-analysis on the effect of resistance exercise on the femoral neck, Kelley and Kelley (2004) concluded that resistance exercise had little impact on the femoral neck in premenopausal women. This finding conflicts with the evidence from the current investigation.

For example, McClanahan and colleagues (2002) observed college athletes from a variety of sports (e.g., baseball, basketball, football, golf soccer, tennis, cross-country, track, and volleyball) to determine if there was a disparity between the athletes dominant and non-dominant forearms. BMD of the dominant arm was significantly higher than the BMD in the non-dominant arm, especially for athletes who participate in sports that are more unilateral in nature, such as, tennis and baseball. Interestingly, the authors did not

find the same side-to-side comparisons for the lower extremities, possibly due to the bilateral requirements on the lower limbs in most sports.

The upper and lower body sports included in the present investigation were predominantly bilateral in nature (e.g., golf, cheerleading, volleyball). While volleyball is a bilateral sport, these athletes tend to be more dependent on the dominant arm. Tennis and softball were the only unilateral sports incorporated in the present study, yet, the same site-specific significant results were not found due to a limited sample size.

Golf is a bilateral sport that requires total body movement. The non-dominant upper/lower limbs of the golf athlete were denser than the dominant limbs. Only one golf athlete was integrated into the study population. Clearly, more golf athletes are needed to draw conclusions, but findings of this investigation imply that the fortified BMD of the non-dominant sites is associated with the stress response that occurs during the drive. The golf swing requires a significant amount of force from the non-dominant side of the body to initiate and generate the momentum to carryout the swing. The non-dominant hip and arm instigate the initial power for the swing.

Cheerleading is also bilateral, yet, the data for cheerleaders revealed a varied outcome than the golf measurements. The cheerleaders had greater BMD in the dominant forearm, but the opposite was true for the lower body, that is, the dominant leg had slightly higher BMD than the non-dominant leg.

The results of this investigation did not reveal a marked differentiation in BMD between the dominant and non-dominant limbs of volleyball athletes and in athletes that participate in both volleyball and track. This result did not coincide with past research. For instance, Calbet et al. (1999) reported that the dominant arm of male volleyball

athletes was 3% heavier, had 4% larger muscle mass, 9% greater BMC, and had 7% superior BMD than did the controls. The athletes in the present investigation had uniform BMD scores for all four sites. The entire volleyball team (n = 13) was included in the present investigation. There was a balanced representation of the various positions. The sample was not laden with attackers and servers that would provide results similar to unilateral sports. Instead, the sample included setters and defensive specialist, as well as attackers. The inconclusive data for the volleyball athletes in the current investigation suggests that a position-to-position response to BMD accrual was evident because there was not a unilateral accrual pattern to BMD.

Another group of researchers examined women volleyball athletes in terms of the position that the athlete primarily plays. Shibata et al. (1999) studied the bilateral disposition of volleyball. There data revealed that athletes who were attackers and blockers had significantly higher BMD than the athletes whose position was strictly defensive, with jumping requirement. Repeated jumping provides the proper impetus to improve bone mass. Conversely, however, Shibata, Ohsawa, Watanabe, Miura, and Sato (2003) reported that the premenopausal women (n = 28) that practiced walking and jumping or walking exclusively, for one year showed no significant change in BMD of the femoral neck or other regions. It appears there may be a minimal strain threshold, which must be met in order to facilitate a response in BMD.

Data from the unilateral sports incorporated in this investigation displayed a strong relationship between the dominant forearm and augmented BMD. This same trend was found in the research of Ducher et al., who explored the relationship between the dominant and non-dominant forearms in 52 regional Canadian tennis players, 28 men and

24 women. The researchers observed a 20% larger BMC in the dominant arm in comparison to the non-dominant arm. There was a 6.5% greater difference of BMD, however, observed in favor of the dominant forearm. Grip strength and lean tissue mass also correlated positively to the dominant arm. Similar results were found in the work of Haapasalo et al. (1994), Jones et al. (1977), and Montoye et al. (1980), each of whom support the theory that the dominant forearm of athletes who participate in sports that are unilateral will possess greater BMD. Similar results were found among the tennis athletes in this present investigation. Not only did the tennis athletes have greater BMD of the dominant forearm, but also revealed larger BMD in the dominant femoral neck.

Softball was another unilateral sport that was represented in this current investigation. In the sample of softball athletes there was a difference among their dominant and non-dominant limbs. The dominant forearm possessed greater bone mass than the non-dominant. The non-dominant forearm is not usually subjected to the same amount of strain. The larger BMD found in the dominant arm of softball athletes in the current study could be due to the unilateral nature of the throw and the driving action from the trail arm that is needed to swing while batting. This same trend was not observed in the lower body of softball athletes. The non-dominant femoral neck had larger BMD than the dominant femoral neck. One reason for this result is that the non-dominant leg endures a greater amount of stress than the dominant leg during fielding and throwing action. The non-dominant hip also initiates the force to generate the swinging motion, an action that seems to provide sufficient impetus to improve BMD in these areas.

There was a stark contrast in the data of athletes that partake in lower body sports (e.g., soccer and track). The track and field athletes exhibited the second largest BMD measurements for the dominant (M = 1.10, SD = .07) and non-dominant femoral neck (M = 1.08 SD = .08) one of the highest values for the dominant (M = .63, SD = .09) and non-dominant forearm (M = .62, SD .08). Neither the dominant or non-dominant upper or lower body was dissimilar. All of the track and field athletes were jumpers (e.g., high jump, long jump, triple jump, etc.), therefore, the data for the femoral neck supports the theories on BMD acquisition (Frost, 1986; Lanyon et al., 1986; Wolff, 1891). Forearm measures could have been the result of the jumping motion that requires the use of the whole body, especially the momentum that the arms add to the lift.

The overall data for soccer athletes revealed low BMD values for both the upper and lower body. The upper body data demonstrated comparable values, although, the lower body BMD indicated a strong disparity between the dominant and non-dominant sides. The dominant femoral neck had greater BMD than did the non-dominant in female soccer athletes. This result suggests that the dominant leg undergoes superior and repeated strain causing larger BMD. As reviewed earlier, Wallace and Cumming (2000) discovered that high-impact exercise increases BMD at the loaded site. The upper body, in soccer athletes, is exposed to relatively little strain. Several of the soccer athletes tested for osteopenia in the forearm, highlighting the lack of high-impact training in the upper extremities of soccer athletes.

Valdimarsson, Alborg, Duppe, Nyquist, and Karlsson (2005) performed an eight year follow-up study on 66 Swedish female soccer players, including, 48 active players (M age = 18.2, SD 4.4) and 18 former players (M age = 43.2, SD = 6.2), and 64 age-

matched controls to determine if reduced training is associated with increased loss of BMD. Baseline BMD of active and retired players BMD was larger at the femoral neck at baseline (M = 1.13, SD = .19 g/cm^2) than controls (M = 1.00, SD =.13 g/cm^2). The annual gain of BMD in the leg was greater in the active players (M = .015, SD = .006 g/cm^2) than the control group (M = .007, SD = .012 g/cm^2). The investigators concluded that intense exercise post puberty is correlated with superior accrual of BMD. Conversely, the reduction or cessation of exercise is connected to an increased loss of BMD than in controls.

Overall, there was not a significant difference among the dominant and non-dominant appendages of the athletes in this sample. However, sport-to-sport BMD trends found from the data that suggests a site-specific adaptation of BMD in athletes as a function of sport type. For upper and lower body athletes in unilateral sports, there was a distinct differentiation between the dominant and non-dominant appendages. For the bilateral sport participants, however, the data did not express the same result across the sample, although, there was a sport-to-sport trend in BMD. Cheerleaders in this study did experience greater BMD in the dominant forearm as compared to the non-dominant forearm; yet, the volleyball athletes demonstrated no BMD differences for either the upper or lower sites. Collectively, the athletes that compete in both track and field and volleyball had the greatest BMD (M of dominant forearm = .67, SD = .06; M of non-dominant forearm = .67, SD = .04; M of dominant femoral neck = 1.28, SD = .19; M of non-dominant femoral neck = 1.27, SD = .19) in the population.

The secondary findings, in which BMI, and to a certain extent, PBF predicted BMD in athletes, did not support the hypothesis from this study. It was expected that

BMI, PBF, the score for the EAT-26, and episodes of amenorrhea would predict BMD. The EAT-26 and episodes of amenorrhea showed no significance in predicting BMD. PBF was seen as a significant predictor for the upper body, especially the non-dominant arm, but not the lower body sites. BMI was the only variable from this study that was a significant predictor for all four tested sites.

Body Mass Index

BMI was a significant predictor of BMD at the dominant/non-dominant femoral neck and forearm. In addition, BMI was the only significant main effect across all four regression equations. BMI for the dominant forearm was significant at $p < .001$, but only significant at $p < .05$ for the non-dominant forearm. PBF was also significant ($p < .05$) at the non-dominant forearm. For the femoral neck, dominant and non-dominant, BMI was significant at $p < .001$. In past studies, BMI was a predictor for BMD in college-aged women. Zanker and Swaine (1998) tested 33 distance runners (M age = 27.2, SD = 1.8) to determine the relationship between biochemical bone markers for turnover, nutritional status, and estradiol. They found that BMI was positively correlated with estradiol concentration. Zanker and Swaine concluded that there is a connection between low BMI and oestrogen deficiency, suggesting that, low BMI can be equated to reduced bone formation.

A set point, or level, at which BMI will impact BMD was suggested during a conference for gynecology at the University of Colorado, Dr. Stephen Scott addressed the topic of BMI and adolescent athletes. Scott (as cited in Jancin, 2001) found that BMI of 20 can predict BMD insufficiency in adolescent females. In addition, a BMI below 17.5 should cause a physician to take immediate action to cease the progression of damage to

the athlete's BMD. Hotta, Shibasaki, Sato, and Demura (1998) found that a BMI of 15 was a significant predictor of BMD of the spine. Results of the current investigation, indicated that BMD deficiency started when BMI 18.17 for lower body sports and 20.12 for upper and lower body sports. The mean BMI for the present study was 23.66 (*SD* = 2.95). These findings suggest that BMI can be used to predict an imbalance in bone remodeling. The imbalance in remodeling can be initiated by inappropriate eating habits a topic that was addressed in this study using the EAT-26 measure.

Eating Attitudes Test-26

One purpose of this study was to examine the effect of eating disorders on BMD. The present results indicated that only two individuals in this investigation produced a positive score on the EAT-26. Thus, effect of eating disorders on this sample was not significant, a finding that is anathema to the majority [elaborate here a bit] of the related literature. Only two members (4%) of the sample produced a positive score on the EAT-26. Conversely, the prevalence of eating disorders has been reported to be as high as 62% in female athletes (Nattiv & Lynch, 1994; Sundgot-Borgen, 1993; Yeager et al., 1993), although, Nattiv, Puffer, and Green (1997) determined that the there was a notable predominance of weight control behaviors in athletes that competed in sports where a reduced amount of body fat is considered optimal (e.g., track and cross-country) as compared to the controls. The sample consisted of 2,981 NCAA Division I athletes. Conversely, fewer than 4% of the athletes from the current study revealed a noteworthy disordered eating habit. Of the 2,981 athletes, Nattiv and colleagues reported that a total of 33% of the sample were females from 13 different sports. Sundgot-Borgen (1993) indicated comparable findings in sports that are aesthetic or require endurance.

Another finding from the current investigation provided evidence of the relationship between eating disorders and amenorrhea. The EAT-26 score was positively correlated with episodes of amenorrhea in all of the regression analyses ($p < .05$). In their study of NCAA athletes, Nattiv et al. (1997) found a similar correlation between pathogenic weight control behaviors and amenorrhea in Asian and Caucasian females. This evidence supports the principal of the female athlete triad, which was previously reviewed as an interrelated disorder that is comprised of eating disorders, amenorrhea, and osteoporosis. Previously discussed research indicates the relationship of eating disorders and amenorrhea (ACSM, 1997; Teitz, 1997; Trash & Anderson, 2000).

Amenorrhea

Amenorrhea was not a significant predictor of BMD in the current investigation. Although, 34% ($n = 17$) did experience at least one amenorrheic episode. Prior research by Constantini (1994), Nattiv et al. (1994), and Yeager et al. (1993) have indicated that the incidence of amenorrhea in the general population is between 2% to 5%, amenorrhea among female athletes is between 3.4% and 66%. A study by Dusek (2001) further solidified the amenorrheic prevalence in female athletes by investigating 72 female athletes ages 15-21 (10 volleyball players, 18 basketball players, 10 ballet dancers, and 34 runners) with a control sample of 96 non-athletes. Dusek found that the control group twice the rate of dysmenorrhea as compared to the athletes ($p < 0.001$). The prevalence of secondary amenorrhea in the athletes was three times greater than in the control group ($p = 0.037$), with runners (14/31) and long distance runners (11/17) having the highest frequency. There was also a larger incidence of primary amenorrhea in the sample of athletes as compared to the control (6/72 vs. 0/96, $p = 0.014$).

In another study Zanker and Swaine (1998) investigated 33 distance runners, 18 eumenorrheic, 9 amenorrheic, and six oligomenorrheic. The runners averaged 47.6 (*SD* = 22.4) km/week. A positive relationship between serum levels of bone formation markers, BMI, and estradiol was found among amenorrheic runners had. The researcher concluded that a reduced BMI along with estrogen deficiency were correlated with decreased bone formation in amenorrheic runners. Dusek's findings lend further credence to the relationship between BMI and dysfunction of the hypothalamic-pituitary axis on BMD in amenorrheic runners. Exercise intensity could have been one cause of the amenorrhea among female runners.

Exercise intensity has been theorized as a predictor of amenorrhea in female athlete (Warren & Goodman, 2003). In the present investigation, every athlete that reported experiencing episodes of amenorrhea also indicated that they exercised beyond what was required from their coach, (see Appendix A). Conversely, 82% ($n = 27$) of the general population that did not report having an episode of amenorrhea exercised beyond what was required. This indicates that exercise alone does not contribute to amenorrhea. The chronic energy deficit brought on by the exercise intensity, must contribute to the frequency of amenorrhea in athletes.

Percent Body Fat

In the present investigation, BMI correlated significantly with PBF at each BMD site ($p < .001$). PBF was also a significant predictor of BMD of the non-dominant forearm, but a weak predictor in the dominant forearm. PBF did not account for changes to the BMD of the femoral neck. Miller, Nickols-Richardson, Wootten, Ramp and Herbert (2004) found that PBF was not correlated with BMD in their investigation on the

relationship of BMD, body composition, and isokinetic strength in 76 young women. They did, however, conclude that there is an association between BMD and fat free mass. Miller et al. also reported that the relationship between muscular strength and BMD in the forearm was significant. It appears then that PBF may be negatively related to BMD at the forearm. As strength, muscle mass, and BMD increase, PBF decreases and results in more lean mass (LM) (Miller et al.). Evidently, there are site-specific adaptations to lean mass that can confound the result of PBF on the entire body. LM was not measured in this investigation, however, Mein et al. (2004) found that there was no association between PBF and BMD at most of the BMD sites measured, although LM was correlated with BMD.

The PBF of athletes that compete in sports that were both upper and lower body dominant ranged from 14.41% to 38.19%. The mean PBF for this group was 21.74% (*SD* = 5.36%). For athletes that participate in lower body sports, the PBF range was from 13.42% to 34.52%, with a mean percent of 23.42 (*SD* = 5.22%). The lowest PBF registered in this investigation was 13.42%, for one track and field athlete, who also had experienced at least one episode of amenorrhea. Frisch and McArthur, (1974) hypothesized that the PBF of females must exceed 17% to initiate menstruating and sustain a PBF of 22% to continuing menstruating. In the present study, nearly half of the population, 40% (n = 20) had a PBF above 22%. While, 24% (n = 12) a PBF less than 17%, 36% (n = 18) had a PBF between 18% and 22%. Of the athletes who reported episodes of amenorrhea, 35.2% had a PBF below 17% (n = 6), 23.5% (n = 4) had a PBF between 18% and 22%, and 41.2% (n = 7) had a PBF above 23%. These results indicate

that PBF cannot exclusively predict amenorrhea or BMD. PBF is only one component of the milieu of variables that affects BMD in female athlete.

Stress Fractures

Stress fractures are the result of diminished BMD. For competitive athletes who participate upper and lower body sports, 40% (n = 11) had experienced at least one stress fracture. Only two athletes (9.5%) from the lower body sports had suffered a stress fracture. Korpelainen, Orava, Karpakka, Siira, and Hulkko (2001) investigated several factors that were indicative of recurrent stress fractures among 31 athletes, 19 male and 12 female. The investigators found no association between BMI and the number of stress fractures. In the current investigation, however, there was a positive association between BMI the number of stress fractures (p = < .05). The research of Armstrong et al. (2004) on young military men and women during training support the current findings. Specifically, Armstrong et al. found no evidence of the FAT in the female recruits, but all 13 women experienced stress injury during the course of training. The researchers concluded that the drastic reduction in weight, which changes the recruits BMI value, in combination with acute exercise may be a significant risk factor for stress injury to bone among male and female military recruits. The current investigation did not find a clear link between the interrelated components of the FAT and BMD.

Limitations

The purpose of this investigation was to describe the effect of certain variables on BMD. As a result, the data collected were limited to describing the population. There were several limitations to the design and subject participation that resulted in limitations in this investigation. The scheduling of both the athletes and the lab presented problems

and the self-report method on the EAT-26 was a major concern. The participants comprised a sample of convenience; thus the sample size was far too limited to extrapolate a definitive set of conclusions. Further, the sample could have been tainted by volunteer bias. The population problems offered the principal questions in finding noteworthy conclusions.

Sample

The sample in this study included 50 female collegiate athletes representing seven sports, volleyball (n=10), soccer (n=17), track (n=4), tennis (n= 5), golf (n=1), cheerleadering (n=5), softball (n=5), and volleyball/track (n=3). Clearly, the size of the sample was low. There are several possible factors that were beyond the control of the investigator that caused this problem. First, access to the athletes was often difficult. Some of the coaches were extremely supportive of the investigation, and made the participation for their athletes mandatory. For example, if testing occurred during their team's competitive season, it was understandable that the coaches did not want to disrupt their athlete's training and concentration. Specifically, the softball and track and field teams were limited in their participation. The softball and track athletes who participated volunteered outside of their team responsibilities. Another limitation was volunteer bias. The participants were all volunteers, this was not a random sample of athletes, nor was it a balanced representation of the various sports that female collegiate athletes typically participate in. In addition, it would have been helpful to have larger representation from each of sports for sport-to-sport comparison purposes.

Another limitation was the composition of the study population was disproportionate between sport-type. The sports that make use of both the upper and

lower body (e.g., volleyball, tennis, golf, cheerleading, and softball) consisted of 29 athletes, while the sports that utilized predominately the lower body had only 21 members. A more uniform proportion of athletes from each type of sport would been desirable comparative purposes.

Another issue with the sample was the total amount of athletes who participated. A greater effect size would have increased the power for the MANOVA, as well as, the regression analyses. A larger total sample would have produced greater significance, especially in the MANOVA analysis.

Finally, because this was a sample of convenience, it did not include an emblematic inclusion of athletes who are known to be susceptible to the FAT. The FAT can increase the likelihood of developing serious deficiencies in BMD. This situation could have exposed the investigation to selection bias. All female collegiate sports were not represented. Athletes that participate in sports that require a lean body mass and/or are aesthetic in nature (e.g., gymnasts, distance runners, dancers, and divers) are known to have a greater likelihood of experiencing stress fractures (Nattiv & Lynch, 1994; Sundgot-Borgen, 1994). There were four athletes who participated only in track and field in this investigation; yet, none of the track and field athletes were distance runners. Distance runners and cross-country runners commonly experience stress fractures; yet, due to scheduling concerns, there was not a sufficient representation of runners in this sample (Nattiv & Armsey, 1997).

Scheduling

Scheduling was a problem for this investigation. Not only was there a concern with the athletes who were in season and accessible, there was also difficulty in

scheduling time on the DEXA along with radiology tech availability. Both were a hindrance to including enough athletes into the study. The professors who were licensed to perform the DEXA testing volunteered their time. Thus, the testing had to be performed when these professors were available. Often the time available for testing did not meet the time athletes were available. Consequently, the time spent with each athlete during the testing period was more limited than desirable. The athletes had to move through the testing quickly, which could have skewed the results of the EAT-26 inventory.

Eating Attitudes Test-26

The EAT-26 is a highly reliable and valid instrument to determine the presence of an eating disorder (Garner, 1983). Each participant was given limited instructions in completing the inventory. The participants were also required to self-report the answers. Prior research by Bauman and Koch (1984) has shown that participants don't accurately report the correct information on inventories. Adolescent and young women are especially prone to hiding their disordered eating regime (Teitz, 1997). This could be due to fear of being discovered by their coaches and friends. Only two participants produced results on the EAT-26 that would indicate an eating disorder. An additional issue was that 36% (n = 18) of the sample were from a foreign country, many of which did not speak English as their first language. There were cultural and language limitations in understanding pieces of the inventory. It would have been helpful to have read the questions on the inventory to each participant to insure that they understood the question.

Another possibility for the low scores on the EAT-26 could be the nature of the inventory. It is possible that athletes are more inclined to experience disordered eating,

or eating disorder not otherwise specified, as opposed to pathogenic weight control behaviors (APA, 1994). The EAT-26 may not have picked up the telltale signs of disordered eating. The pervasiveness of eating disorders ranges from 15% to 62% in female athletes, but this result was not discovered in this sample of college-aged female athletes (Nattiv & Lynch, 1994). Only 4% of the study population had a positive score on the EAT-26.

Conclusions and Future Directions

The type of sport, upper/lower body or predominantly lower, that a female athlete engages in does impact BMD. A significant relationship between limb preference and BMD was not observed across the sample of athletes, although, there was a strong sport to sport association relating BMD to the preferred forearm in unilateral sports and to the dominant leg in soccer. Conversely, the upper body in soccer athletes was relatively low. These finding suggests that superior bone remodeling takes place at the sites that receive the greatest amount of stress. Coaches, strength training specialists, and sports medicine personnel should be mindful of the discrepancy in bone remodeling in the non-weight bearing sites. The non-loaded sites can be targeted by additional resistance training to increase bone mass and avoid injury.

Of the four predictor variables (EAT-26 score, episodes of amenorrhea, BMI and PBF) that were hypothesized to impact BMD, only BMI was established as a significant predictor for all four sites tested. Yet, BMI can be an illusive measure to gauge in athletes. The BMI standards were established as norms for the average individual. Athletes have increased muscle mass for their relative height and weight. It is possible that an athlete who is high in muscle mass could have a BMI that exceeds the obesity

norm reference for her comparative size. PBF was significant for the forearms but not for either femoral neck. This investigation did not measure LM. There was little data to substantiate if PBF was significant because of the increased LM or if it was due to a chronic energy deficit that caused a reduction in overall body fat. Although, in all four regression analysis, PBF was negatively correlated with BMD, (e.g., PBF decreased BMD increased). The EAT-26 score and episodes of amenorrhea were not noteworthy indicators of BMD in this population. There seemed to be no connection between BMD and the FAT. This result did not coincide with previous research on the FAT. In fact, the results of the current investigation disproved the validity of the FAT. Obviously there is no single predictor that can be attributed to fortifying BMD. Instead, there seems to be a milieu of interrelated variables, which effect bone remodeling and BMD. Perhaps, the effect of the FAT will need to be further evaluated to determine if additional variables must be included in order to explain the BMD phenomenon. The results of this investigation are limited to this sample of collegiate athletes.

Recommendations

A comprehensive investigation on a large cohort of athletes ought to be carried out to determine the degree of impact sport-type, limb dominance, exercise intensity, PBF, LM, eating disorders, and amenorrhea have on the BMD of collegiate female athletes. The population should be composed of a representative sample of all collegiate sports, and stratified to provide an equal number of athletes with and without amenorrhea, disordered eating, and stress fractures. A more proficient method for determining the presence of an eating disorder or disordered eating must be acquired to

provide substantial results. The eating disorder inventory should be done in an interview fashion to insure proper understanding, and to help control for inaccurate self-reporting.

Additionally, an intervention study should be designed to determine if a certain training protocol should be administered to female collegiate soccer athletes to improve the BMD of their upper extremities.

Finally, more research needs to be executed to examine if the interrelated components of the FAT are predictors of BMD. It is quite possible that additional predictors will be added to the FAT in order to explain BMD issues in female athletes.

The results of the current investigation supported the first hypothesis statement, the type of sport will effect the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, when controlling for episodes of amenorrhea, evidence of an eating disorder, PBF, and BMI. The second hypothesis was rejected, when controlling for BMI, past episodes of amenorrhea, PBF, limb preference and type of sport, evidence of an eating disorder will predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes. PBF was significant for the forearm, thus, the third hypothesis, when controlling for BMI, evidence of an eating disorder, past episodes of amenorrhea, limb preference and type of sport, PBF will predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, was partially supported. BMI was found to be a significant predictor of BMD at each of the four sites measured, therefore, the fourth hypothesis, when controlling for the evidence of an eating disorder, PBF, past episodes of amenorrhea, limb preference and type of sport, BMI will predict

the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, was supported in the current investigation. Finally, episodes of amenorrhea was not significant across each regression model, consequently, the fifth hypothesis, when controlling for the evidence of an eating disorder, PBF, limb preference and type of sport, and BMI past episodes of amenorrhea will predict the BMD of the dominant and non-dominant forearm and dominant and non-dominant femoral neck of college-aged female athletes, was rejected.

The purpose of this study was to examine the effect of sport type on BMD, as well as to determine which variables, EAT-26, episodes of amenorrhea, PBF, and BMI, predict BMD in college-aged female athletes. The data indicated a sport-specific effect on BMD. This result fortifies the already existing literature on site-specific adjustments imposed on BMD by stress. The results of this study did not support the findings of previous research in regard to the predictors of BMD. There was little evidence of the direct relationship of the FAT to BMD; yet, BMI and PBF can be impacted by eating disorders, a component of the FAT. BMI was the only predictor that was significant at each BMD measurement; although, PBF was a significant indicator for the dominant and non-dominant forearm.

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Appendixes

Appendix A

College Athlete Questionnaire

Please read each question carefully, and circle the correct response. Your answers will be confidential.

1. What is your age?

   _____

2. What is your year in school?
   - Freshman
   - Sophomore
   - Junior
   - Senior
   - Fifth year (red shirt)

3. What is your ethnic background?
   - White/Caucasian
   - African American
   - Latino
   - American Indian
   - Asian
   - Other

4. Are you a scholarship athlete?
   - Yes
   - No

5. Which best describes your grade point average (GPA)? Please indicate your current GPA.

   _____

6. How often do you eat/drink dairy products?
   - Once a day
   - 2 – 3 times a day
   - More than 3 times daily
   - 2 times per week
   - Other, please list

7. Have you ever experienced a stress fracture?
   Yes
   No

8. Which best describes your menstrual cycle (period)?
   1 time in less than a 24 hour-period
   1 time every 24 – 29 days
   I have not had a period for the last two months
   I have not had a period for the last three to six months
   I have not had a period for the last year

9. Have you ever had episodes where you skipped regular menstrual cycles (periods)?
   Yes
   No

10. If yes, how long?
    1 – 3 months
    4 – 6 months
    7 + months

11. Do you currently take birth control pills?
    Yes
    No

12. Which hand do you prefer to throw or eat with?
    Right
    Left
    Both

13. Which do you prefer to kick with?
    Right
    Left
    Both

14. Which sport do you participate in?
    Volleyball
    Basketball
    Softball
    Soccer
    Tennis
    Track
    Cheerleaders
    Golf

15. How many years have you participated in your sport?
    1 – 5 years
    6 – 10 years
    11 +

16. Do you participate in physical exercise outside of the requirements of your sport?
    Yes
    No

17. How often do you participate in physical exercise outside of the requirements of your sport?
    1 – 3 hours per week
    4 – 9 hours per week
    More than 10 hours per week

18. When you participate in physical exercise outside of the requirements of your sport, at what intensity do you participate?
    Recreationally/at a mild intensity (less than 50% of your maximal heart rate)
    Moderate intensity (approximately 55-65% of your maximal heart rate)
    High intensity (approximately 7-=85% of your maximal heart rate)

Appendix A

Demographic Characteristics From College Athlete Questionnaire

Questions	n	%
Year in school		
Freshman	23	46
Sophmore	11	22
Junior	4	8
Senior	9	18
Fifth year (red shirt)	3	6
Ethnicity		
White/Caucasian	39	78
African American	7	14
LatinAmerican	1	2
American Indian	0	0
Asian	0	0
Other	3	6
Scholarship athlete		
Yes	47	94
No	3	6
Daily dairy intake		
Once a day	15	30
2-3 times a day	30	60
More than 3 times daily	1	2
Other	3	10
Stress fracture status		
Yes	13	26
No	36	73
Menstrual cycle description		
1 time in less than a 24 hour-period	5	10
1 time every 24-29 days	40	80
I have not had a period for the last two months	2	4
I have not had a period for the last three to six months	2	4
I have not had a period for the last year	1	2

(Appendix A continued)

Questions	n	%
Skipped menstrual cycles		
Yes	17	34
No	33	66
If yes, how long		
1-3 months	12	24
4-6 months	5	10
7 + months	0	0
Preferred hand		
Right	46	92
Left	2	4
Both	2	4
Preferred leg		
Right	45	90
Left	3	6
Both	2	4
Sport		
Volleyball	10	20
Basketball	0	0
Softball	5	10
Soccer	17	34
Track and field	4	8
Golf	1	2
Tennis	5	10
Cheerleading	5	10
Volleyball and track	3	6
Years of participation		
1-5 yrs	6	12
6-10 yrs	17	34
11 +	27	54
Exercise outside of requirement		
Yes	44	88
No	6	12

(Appendix A continued)

Questions	n	%
Exercise outside of requirement		
0 hours per week	5	10
1-3 hours per week	34	68
4-9 hours per week	10	20
More than 10 hours per week	1	2
Intensity of exercise		
No outside exercise	5	10
Recreational intensity	10	20
Moderate intensity	24	48
High intensity	11	22

Note. $N = 50$.

Appendix B

Informed Consent Form
for
Bone Mineral Density in College-Aged Athletic Women

You are invited to participate in this experiment to study bone mineral density in college-age, athletic women. The purpose of this research is to determine the bone mineral content (BMC) of college-aged, female athletes and to determine if there is a difference between your dominant and non-dominant forearm and hip content, and to determine what factors if any contribute to this problem. You are being invited to participate in this research since you are on a varsity sport at Middle Tennessee State University (MTSU). You may ask questions about anything you do not understand, before deciding whether or not to participate.

Please sign this informed consent form to indicate that you will voluntarily participate in this study. You will be asked to complete an inventory designed to evaluate your diet habits. In addition, you will have three skinfold measurements at the triceps, hip, and abdomen to determine the percent of your body weight this is fat. You will then experience four scans on the dual x-ray absorpitometry (DEXA), a procedure that measures bone density. A minimal dose of radiation will be used for this procedure. A DEXA certified technician will perform the scan. The entire process should take no more than 30 minutes.

The benefits of your participation include determining your BMC. This is important information because low BMC means that you may be at an increased risk for injury. Your participation in this study will assist in the development of a more accurate assessment procedure for determining athletes at risk for osteopenia, a bone density deficiency.

Your identity will remain confidential, except the investigators. In order to assure you that you are not identified the investigator will use random numeral codes when information is processed and recorded for documentation in all files. The data will be maintained by the investigator, locked in a file cabinet, and destroyed after five years. The results of this research study may be published (in a dissertation, presentation, and/or journal article), but your name/identity will not be revealed. As part of our service to you, you will receive the DEXA scan and other tests free of charge.

You may direct any questions or concerns that you have concerning the study to the principle investigators. You are free to forward the results of your screening to your primary care physician. The nature, demands, risks, and benefits of the project have been explained to you. You will be provided a photocopy of this consent form for your records.

The nature and purpose, the potential benefits, and possible risks associated with participation in this study have been explained to me, and all questions have been answered by the investigator. The above signature has been witnessed. The elements of the informed consent conform to the standards given by MTSU to protect the rights of human subjects.

Participants signature _____ Date_____

Print Name _____

Witness' signature _____ Date_____

Print Name _____

Signature of Investigator _____ Date_____

Investigator: Pamela Anderson, M.S., 615-898-5234

Appendix C

River Center Clinic Eating Disorders Program

The Eating Attitudes Test -26

The Eating Attitudes Test (EAT-26) is the screening test used for the 1998 National Eating Disorders Screening Program. The EAT is probably the most widely used standardized measure of symptoms and concerns characteristic of eating disorders (Garner, 1993; 1997). The EAT-26 alone does not yield a specific diagnosis of an eating disorder. Neither the EAT-26, nor any other screening instrument, has been established as highly efficient as the sole means for identifying eating disorders. However, studies have shown that the EAT-26 can be an efficient screening instrument as part of a two-stage screening process in which those who score at or above a cut-off score of 20 are referred for a diagnostic interview (King, 1989, 1991). This is the procedure that has been followed in the National Eating Disorders Screening Program.

Thank you for your interest in the Eating Attitudes Test (EAT). You have permission to use the EAT in your research and clinical work and there is no charge for this permission as long as you acknowledge the original publication (Garner et al., 1982) as a source.

Below is a copy of the EAT-26 as well as information on its scoring. I would appreciate you providing me with a copy of any reports or publication in which this instrument is used since it may serve as a useful resource for other researchers and clinicians.

Eating Attitudes Test (EAT-26)

	Always	Usually	Often	Sometimes	Rarely	Never	Score
1. Am terrified about being overweight	O	O	O	O	O	O	___
2. Avoid eating when I am hungry	O	O	O	O	O	O	___
3. Find myself preoccupied with food	O	O	O	O	O	O	___
4. Have gone of eating binges where I feel that I may not be able to stop	O	O	O	O	O	O	___
5. Cut my food into small pieces	O	O	O	O	O	O	___
6. Aware of the caloric content of foods							
7. that I eat	O	O	O	O	O	O	___
8. Particularly avoid foods with a high carbohydrate content	O	O	O	O	O	O	___
9. Feel that others would prefer if I ate more	O	O	O	O	O	O	___
10. Vomit after I have eaten	O	O	O	O	O	O	___
11. Feel extremely guilty after eating	O	O	O	O	O	O	___
12. Am preoccupied with a desire to be thinner	O	O	O	O	O	O	___
13. Take longer than others to eat my meals	O	O	O	O	O	O	___
14. Other people think that I am too thin	O	O	O	O	O	O	___
15. Am preoccupied with the thought of having fat on my body	O	O	O	O	O	O	___
16. Take longer than others to eat my meals	O	O	O	O	O	O	___
17. Avoid foods with sugar in them	O	O	O	O	O	O	___
18. Eat diet foods	O	O	O	O	O	O	___
19. Feel that food controls my life	O	O	O	O	O	O	___
20. Display self-control around food	O	O	O	O	O	O	___
21. Feel others pressure me to eat	O	O	O	O	O	O	___
22. Engage in dieting behavior	O	O	O	O	O	O	___
23. Feel uncomfortable after eating sweets	O	O	O	O	O	O	___
24. Engage in dieting behavior	O	O	O	O	O	O	___
25. Like my stomach to be empty	O	O	O	O	O	O	___
26. Enjoy trying new rich foods	O	O	O	O	O	O	___
27. Have the impulse to vomit after meals	O	O	O	O	O	O	___

Total Score ___

Scoring – for all items except #25, each of the responses receives the following value:

Always = 3
Usually = 2
Often = 1
Sometimes = 0
Rarely = 0
Never = 0

For item #25, the responses receive these values:
Always = 0
Usually = 0
Often = 0
Sometimes = 0
Rarely = 0
Never = 0

Appendix D

Human Participant Protections Education for Researc

Completion Certificate

This is to certify that

Pam Anderson

has completed the **Human Participants Protection Education for Research Teams** online course, sponsored by the National Institutes of Health (NIH), on 02/08/2005.

This course included the following:

- key historical events and current issues that impact guidelines and legislation on human participant protection in research.
- ethical principles and guidelines that should assist in resolving the ethical issues inherent in the conduct of research with human participants.
- the use of key ethical principles and federal regulations to protect human participants at various stages in the research process.
- a description of guidelines for the protection of special populations in research.
- a definition of informed consent and components necessary for a valid consent.
- a description of the role of the IRB in the research process.
- the roles, responsibilities, and interactions of federal agencies, institutions, and researchers in conducting research with human participants.

Appendix E

MIDDLE TENNESSEE STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
HUMAN SUBJECTS RESEARCH REVIEW FORM

Documentation of IRB Training (attach appropriate documentation):
Internet training certificate ☒ Workshop certificate ☐ Registration for workshop ☐

Request for Expedited Review ☒ Full Review☐

Investigator(s) name(s) Pamela S. Anderson

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Investigator Status (For each investigator)
☐Faculty/Staff
☒ Graduate Student
☐ Undergraduate Student
☐ Other

If the principal investigator is a student, list name department and local telephone of faculty supervisor. Please note that THE FACULTY SUPERVISOR MUST INDICATE KNOWLEDGE AND APPROVAL OF THIS PROPOSAL BY SIGNING THIS FORM.

Faculty Supervisor Name Dr. Mark Anshel

Faculty Supervisor e-mail: manshel@mtsu.edu

Address & Telephone 898-2812

Social Security #

Source of funding for project none

Expected starting date for project 2/21/05

Is this project expected to continue for more than one year?
☐ Yes ☒No Anticipated Completion Date 06/05

Approval for projects is valid for one year only. Investigators must request a continuation of the approval yearly if the activity lasts more than one year. Only two continuations will be granted for a given project. After three years, the project must be resubmitted.

PROJECT DESCRIPTION

- The following information is required for all projects.
- Limit your answers to the space provided. (Further information may be attached to supplement this description, but not to replace it)
- Attach copies of all questionnaires, testing instruments or interview protocols; include any cover letters or instructions to subject.

DESCRIPTION
Provide a **BRIEF** description, in **LAYMAN'S TERMS**, of the proposed research:

The research project is designed to examine predictors of bone mineral content among college-age Division I female athletes. Osteoporosis is a serious health problem that can begin to show symptoms as early as the late teens and early twenties. This study will examine two research questions: 1) To determine the extent to which bone mineral density in Division I A female athletes, ages 18-25 yrs., differs as a function of site (upper vs. lower limb; dominant vs. non-dominant limb), and 2) To determine the degree to which selected measures, specifically, eating disorders/disordered eating, Body Mass Index, amenorrhea, and total lean mass can predict the bone mineral status of the athletes.

METHOD (check all that apply)
☒ QUESTIONNAIRE ☐ OBSERVATION ☐ TEST
☐ INTERVIEW ☐ FILES ☐ TASK
☐ TREATMENT
☒ OTHER DEXA

NUMBER OF SUBJECTS:

~50-75

SUBJECT POPULATION (check all that apply)
☒ ADULT ☐ MINOR
☐ PRISONER ☐ MENTALLY RETARDED
☐ MENTALLY ILL ☐ PHYSICALLY ILL
☐ DISABLED ☐ OTHER
Specify:

SUBJECT SELECTION
Are subjects to be drawn from the Psychology subject pool? ☐ Yes ☒ No

- If yes, a completed sample sign-up sheet must be submitted.
- If no, describe how subjects will be selected for participation in this project and any payment to be received by the subject:

NOTE: If the subjects are to be drawn from an institution or organization (e.g., hospital, social service agency, prison, school, etc.) which has the responsibility for the subjects, then documentation of permission from that institution must be submitted to the Board before final approval can be given.

CONFIDENTIALITY
Specify steps to be taken to guard the anonymity of subjects and/or the confidentiality of their responses. Indicate what personal identifying indicators will be kept on subjects. Specify procedures for storage and ultimate disposal of personal information. Federal guidelines require all study related documents (documentation of informed consent, surveys, study notes, data analysis, and all study-related correspondence) be kept for at least 3 years.

The participants will be asked to sign an informed consent (see attached) that will explain the protocol for keeping all acquired information private and secure. Each participant will receive a random number. The number will be used to organize information by subject. All test information and forms will be kept in a locked cabinet in the researchers office.

CONSENT

Specify how subjects will be informed of the following: a) the nature of their participation in the project, b) that their participation is voluntary and that they may withdraw at any time without repercussions, and c) that their responses are confidential. (If a consent form is being used, attach a copy. If presented orally, a copy of presentation must be submitted.)

All this information is contained on the consent form. Each participant will read and sign the consent form, and receive a photo copy for their records (see attached form).

ADDITIONAL PROCEDURAL INFORMATION

INDICATE BELOW WHETHER YOUR PROJECT INVOLVES ANY OF THE FOLLOWING. FOR EACH ITEM CHECKED, PROVIDE THE REQUESTED INFORMATION IN THE ADDITIONAL PROCEDURAL INFORMATION SECTION BEGINNING ON PAGE 5

- ☐ A) Risk (p. 5)
- ☐ B) Minors as subjects (p. 5)
- ☐ C) Psychological intervention (p. 6)
- ☐ D) Deception (p. 6)
- ☐ E) Physiological intervention (p. 7)
- ☐ F) Biomedical procedures (p. 7)

SEE THE PAGE INDICATED FOR A MORE DETAILED DESCRIPTION OF THESE CATEGORIES

SIGNATURES

The **Principal Investigator** must sign this form.

I certify that 1) the information provided for this project is accurate, b) no other procedures will be used in this project, and c) any modifications in this project will be submitted for approval prior to use.

Signature of Investigator **Date** 3/7/05

If the P.I. is a student, his/her **Faculty Supervisor** must also sign this form.

I certify that this project is under my direct supervision and that I am responsible for insuring that all provisions of approval are complied with by the investigator.

Signature of Faculty Supervisor **Date** 2/7/05

Description of Research

Statement of the Problem

- There is a dearth of research on the sport-specific impact on bone health in female athletes.
- Female athletes are at an increased risk of developing preliminary signs and symptoms of osteoporosis.
- Osteoporosis is a major health concern for women.

- The research project is designed to examine predictors of bone mineral content among college-age, Division I, female athletes. Osteoporosis is a serious health problem that can begin to show symptoms as early as the late teens and early twenties. This study will examine the following research question: what effect does type of sport and dominance have on BMD in the upper body and lower body of college-aged, female athletes. In this investigation the researcher will also consider the extent to which the female has an eating disorder, the number of stress fractures experienced, her body fat percent, her height and weight, and whether she has had episodes of amenorrhea.

Procedures

- Seek approval from the Internal Review Board at MTSU.
- Seek approval from the athletic director to gain access to the athletes and coaches at MTSU.
- Send a letter to all female athletic coaches at MTSU for approval to use their athletes.
- Data collection times will be scheduled. Teams will be scheduled together.
- Each athlete will receive a packet of study information.
- The packet will contain: two informed consent forms, the EAT-26, a scantron sheet, and the demographic questionnaire.
- The athletes will sign the informed consent form and be given a copy for their records.
- Next, the athletes will fill out a demographic questionnaire and the EAT-26.
- All athletes will be submitted to four DEXA scans and three skinfold measurements.
- Finally, the participant's height and weight will be recorded to determine BMI.
- All forms will be collected and returned to the researcher.
- The questionnaires will be coded and scored.
- The DEXA results will be coded and compiled with the questionnaire data.
- The participants will be grouped according to sport type.
- Sports that require predominantly lower body strength (soccer, track, and cross-country)
    - Sports that require both upper and lower body strength (volleyball, softball, basketball, tennis and golf).

Appendix F

Institutional Review Board
P.O. Box 124
Middle Tennessee State University
Murfreesboro, Tennessee 37132
Office: (615) 898-5005

MIDDLE TENNESSEE STATE UNIVERSITY

February 21, 2005

Pamela Anderson, Graduate Student
402 Kingwood Drive
Murfreesboro, TN 37129

Dr. Mark Anshel, HPER Department
Protocol Title: Bone Mineral Density in College Aged Female Athletes
Protocol Number: 05-142

Dear Ms. Anderson:

The MTSU Institutional Review Board, or representative of the IRB, has reviewed your research proposal identified above. It has determined that the study poses minimal risk to subjects and qualifies for an expedited review under 45 CFR 46.110 and 21 CFR 56.110.

Please note that any unanticipated harms to subjects or adverse events must be reported to the Office of Sponsored Programs at (615) 898-5005.

Approval is granted for one (1) year from the date of this letter for 75 subjects

You will need to submit an end-of-project report to the Office of Research and Sponsored Programs upon completion of your research.

Please note that any change to the protocol must be submitted to the IRB before implementing this change.

Sincerely,

Reuben Kyle

Reuben Kyle, IRB Member

cc: Sponsored Programs

Appendix G

MEMORANDUM

February 22, 2005

To: Diane Turnham
Assistant Athletic Director / Senior Women's Administrator
Middle Tennessee State University

From: Pam Anderson
Doctoral Student Middle Tennessee State University
Department of Health and Human Performance

RE: Request to have access to female athletes for dissertation research

Dear Ms. Turnham,

I would like to request permission to include the female intercollegiate athletes at this university to participate in the research for my dissertation. The title of the work is *Bone Mineral Density in College-Aged Female Athletes*. My emphasis will be looking at the trends in bone density in athletes that participate in sports that are either lower body dominant or who utilize both upper/lower body. A secondary emphasis will be to examine if the established predictors of body mass index, percent body fat, episodes of amenorrhea, and the presence of an eating disorder have an impact on predicting bone mineral density in the sample of female athletes at MTSU.

The testing protocol should only take 15-20 minutes per individual. Each athlete would be required to fill out three forms: (1) an informed consent, (2) a questionnaire of demographic data, and (3) an eating disorders inventory. Additionally, the athletes will each have three skinfolds taken to establish their percent body fat and height and weight. Finally, the athlete will be submitted to four scans on the dual energy x-ray absorpitometry (DEXA) to establish their bone mineral density score at both femoral necks and forearms. The scans emit far less radiation than dental x-rays; therefore, the athlete will receive no harm from participating.

I will begin testing the athletes during the spring semester (end of March – April) of 2005. Can I get your endorsement on this endeavor so that I may contact the coaches of female sports at MTSU?

Thank you for your consideration in this matter,

Pam Anderson, M. S.

Appendix H

MEMORANDUM

Date: February 25, 2005

To: Matt Peck, Stephany Smith, Rachel Short, Aston Rhoden, Leigh Podlesny, Dean Hayes, and Randy Holden

From: Pam Anderson (Doctoral student at MTSU)

Re: Participation in a research study

Dear coaches

I am in the process of working on my dissertation. I have an interest in physical problems that are unique to female athletes. I have chosen a dissertation topic that compliments this interest. The name of my investigation is *Bone Mineral Density in College-Aged Female Athletes*. As you know, stress fractures can be quite common in female athletes. What we are also discovering is that females are having issue with their bone mineral content (BMC) due to hormonal fluctuations, exercise intensity, etc. Unfortunately, this condition does not only limit the athlete's participation, but may also place them at risk for developing osteoporosis later in life. My desire is to add to the body of knowledge regarding female athletes and BMC. Therefore, I am contacting you to ask for access to your athletes. I would need about 20 minutes of the athlete's time. Basically, each athlete would undergo 4 dual energy x-ray aborpitometry (DEXA) scans (the scans provide no harm to the athlete), have three skinfolds taken, record their height and weight, and have them fill out an eating disorders inventory and a demographic questionnaire. Everything would be kept confidential. Athletes would be given the results of the data after it has been processed. The DEXA information would be quite valuable for them to possess. Would you mind encouraging your athletes to be involved? I can provide more detailed information about the investigation upon request.

Thanks for your attention to this matter. You can contact me at 482-7437 or by email, ppamisue@comcast.net.

Pam Anderson, M. S.