EFFECTS OF A 10-WEEK STRENGTH TRAINING INTERVENTION AMONG COMMUNITY-DWELLING FEMALES WITH EATING DISORDERS

by

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AMONG COMMUNITY-DWELLING

EATING DISORDERED FEMALES

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The purpose of this study was to determine the effects of a 10-week resistance training intervention on bone mineral density (BMD), body composition (BC), strength, depression, and eating-disordered tendencies in community-dwelling females with anorexia nervosa (AN) or eating disorder not otherwise specified (EDNOS). The sample included 14 females, ages 17 years to 35 years, who were randomly assigned to an experimental or a control group. Participants were required to have a minimum body mass index (BMI) of 14 kg/m^2 and a clinical diagnosis of AN or EDNOS. All participants completed a preparticipation screening questionnaire. In addition, participants completed a risk stratification questionnaire, the Beck Depression Inventory-II (BDI-II), the Eating Disorders Inventory–3 (EDI-3), and the Incorporating More Physical Activity and Calcium in Teens Food Frequency Questionnaire (IMPACT FFQ) pre- and posttest. Body composition using skinfold calipers, estimated 1 repetition max (1-RM) of chest and legs, and BMD measures of the forearm, hip, and spine using dual energy x-ray absorptiometer (DXA) were also assessed pre- and post-intervention. Participants in the experimental group completed a 10-week, supervised, and progressive strength training program utilizing Nautilus strength training machines and free weights. Exercises targeted each major muscle group (leg press, lunges, lat pulldown, bench press, bicep curl, triceps extension, military press, and abdominal curls). Using a repeated measures analysis of variance (ANOVA), it was determined that there were no differences in body fat percentage, BDI-II scores, or EDI-3 scores between the

experimental and control groups following the intervention. Using a multivariate analysis of variance (MANOVA), it was also determined there were no differences between the experimental and control groups on lumbar spine, hip, and forearm BMD. However, there was a significant increase ($p < .001$) in chest and leg strength of the experimental group from pretest to posttest, but not for the control group. In addition, there were clinically relevant increases in BMD, as well as changes in body fat percentage and psychological measures between the two groups. These data show that community-dwelling women with eating disorders can participate in a supervised resistance training exercise program and experience positive physical and psychological results.

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CHAPTER 1

INTRODUCTION

An eating disorder is a term used for several psychophysiological illnesses that include behavior intended to control weight. The disorder can have profound effects on the physical and psychological health of the individual. Several clinically recognized eating disorders have been identified, including anorexia nervosa (AN) and bulimia nervosa (BN). In addition, eating disorder not otherwise specified (EDNOS) is another recognized disorder which can include some traits associated with AN or BN, but does not meet all of the characteristics of either disorder (Klein & Walsh, 2003).

The prevalence of eating disorders has risen over the past five decades (Academy for Eating Disorders [AED], n.d.c.; Kohn & Golden, 2001). At any given time, over 10% of females in their late teen or early adult years report symptoms of eating disorders (AED, n.d.c.). Anorexia nervosa disproportionately affects women, and is the third most common chronic illness of adolescent females (AED, n.d.c.; Fisher et al., 1995; Kreipe, 1995; National Institute of Mental Health [NIMH], 2001; Wakeling, 1996).

Anorexia nervosa has the highest mortality rate of any psychiatric disorder (Kohn & Golden, 2001). Crude mortality rates for this disorder are estimated to range between 5% and 10%, and there is an annual mortality of 0.5% per year (Klein & Walsh, 2003; Kohn & Golden). Nearly 50% of all deaths among individuals diagnosed with AN are

due to suicide, while 25% of deaths among this population are the result of medical complications (Kohn & Golden).

Numerous factors appear to be involved in the origin of disordered eating behavior, although there is no known direct cause (Klein & Walsh, 2003). However, an interaction among several psychological, biological, and environmental risk factors is likely to contribute to the development of the disorder (Fisher et al., 1995). Individuals diagnosed with AN often also have comorbid disorders such as depression, obsessionality, and irritability (Casper, 1998). Major depression is the most common comorbid psychiatric disorder in individuals with AN (Bulik, 2002). Furthermore, depression can occur before or after onset of AN (Bulik). In addition to mood disturbances, a variety of physiological consequences, ranging from mild to severe, affect individuals with AN.

Malnutrition in an individual with AN can lead to problems ranging from minor physiological disturbances to potentially life-threatening conditions. Chronic eating disorders can affect all systems of the body, including the cardiovascular system, nervous system, organ systems, and musculoskeletal system (AED, n.d.a). Endocrine and gastrointestinal system disturbances, hypothermia, short stature, menstrual dysfunction, and osteopenia are all complications of extended malnutrition (Fisher et al., 1995). Some of the most visible physiological changes occurring in persons with AN include alterations in body composition (BC).

Individuals with AN often have body composition changes such as loss of fat-free mass (FFM) as well as fat mass (Kerruish et al., 2002; Polito, Cuzzolaro, Raguzzini,

Censi, & Ferro-Luzzi, 1998; Shephard, 1991). While the extent of muscle mass lost partially depends on the amount of exercise performed by the patient, up to 2/3 of skeletal muscle may be lost by the acute phase of AN (Heymsfield, Olafson, Kunter, & Nixon, 1979; Shephard). Lean mass is lost from visceral tissue such as the kidneys, liver, and spleen in addition to muscles (Davies, Von Dobeln, Fohlin, Freychuss, & Thoren, 1978; Fohlin, 1977). Refeeding can restore weight and body mass index, however, the changes in fat mass (FM) and FFM vary with weight restoration among persons with AN.

Refeeding and weight restoration of an individual with AN can result in significant changes in BC. Lean mass accounts for less than 1/2 of the weight regained by persons with AN, although there is large inter-individual variability (Falk, Halmi, & Tyron, 1985; Forbes, Kreipe, Lipinski, & Hodgman, 1984; Polito et al., 1998). Fat mass can range from 21%-77% of the total weight gained by a person with AN, averaging over 50% (Orphanidou, McCargar, Birmingham, & Belzberg, 1997; Polito et al., 1998; Probst et al., 2001; Scalfi et al., 2002). Although a large percentage of the weight that is regained during refeeding consists of FM, persons who have recovered from AN continue to have a lower body fat percentage than age- and sex-matched controls (Frey et al., 2000). Another component of BC that can be greatly affected by AN is bone mineral density (BMD).

Significant loss of BMD, which can lead to osteopenia and osteoporosis, is a common occurrence among women suffering from eating disorders (Baker, Roberts, & Towell, 2000; Brooks, Cavalier, & Howat, 1999; Carruth & Skinner, 2000; Daee et al., 2002; Heer, Mika, Grzella, Heussen, & Herpertz-Dahlmann, 2004; NIMH, 2001). This

loss of BMD may be partially due to the fact that development of AN is most common during adolescence and early adulthood, which is a crucial time for bone growth (Carmichael & Carmichael, 1995; Misra & Klibanski, 2002). Development of an eating disorder at this time can impair bone development as well as compromise peak mineralization in bone mass, leading to decreased peak height (Carmichael & Carmichael; Misra & Klibanski; NIH, 2005a).

Osteoporosis in the lumbar vertebrae occurs in half of all women diagnosed with AN, leading to a significantly higher likelihood of nontraumatic fractures than healthy controls (Baker et al., 2000). This increased fracture risk can continue for years after diagnosis (Lucas, Melton, Crowsen, & O'Fallon, 1999; Morris et al., 2004; Vestergaard et al., 2002). Furthermore, a long-term concern in premenopausal woman with osteopenia is that once menopause ensues, it is more difficult to prevent osteoporosis and fractures (Brooks et al., 1999).

Reduced lumbar spine BMD has been consistently found in groups of adolescent and adult females with AN (Bachrach, Katzman, Litt, Guido, & Marcus, 1991; Biller et al., 1989; Brotman & Stern, 1985; Davies et al., 1990; Lennkh et al., 1999; Misra et al., 2004). Reduced BMD of the hip (Treasure, Fogelman, & Russell, 1986), femur (Hartman et al., 2000; Lennkh et al.) and whole body (Bachrach, Guido, Katzman, Litt, & Marcus, 1990) has also been documented in this population. A relationship between low BMD and the length of time since diagnosis has been reported by many researchers; however, some published reports (Wong, Lewindon, Mortimer, & Shepherd, 2001) have indicated that BMD is not significantly reduced in the early stages of AN.

Improvement in BMD among individuals diagnosed with AN is possible, although researchers have reported conflicting results. A significant number of adult and adolescent females with AN continue to be osteopenic after weight recovery and resumption of menses, even though BMD may have improved (Herzog et al., 1993; Iketani, Kiriike, Nakanishi, & Nakasuji, 1995; Rigotti, Neer, Skates, Herzog, & Nussbaum, 1991). However, other researchers (Bolton, Patel, Lacey, & White, 2005; Orphanidou et al., 1997; Zipfel et al., 2001) have found substantial increases in BMD after short-term weight restoration. Despite these increases, a large percentage of recovered patients continued to have persistent low bone mass. The contradictory research examining restoration of BMD among persons with AN and the uncertainty regarding the relationship between weight recovery and bone density among this population warrants additional research. Components of weight recovery that may also affect BMD include proper nutrition as well as nutritional supplements.

Bone mineral density can improve in individuals with AN who have had weight recovery, even without resumption of menses, indicating that nutritional factors such as diet and calcium supplementation play an important role in the BMD status of those with AN (Bachrach et al., 1991; Heer et al., 2004; Misra & Klibanski, 2002). The effect of calcium supplementation on BMD in individuals with AN is of interest, as skeletal growth requires a positive calcium balance until peak bone mass is attained (Fisher et al., 1995). If the onset of an eating disorder occurs in adolescence, calcium intake may be significantly compromised (Fisher et al.). However, some researchers have found that calcium supplementation does not appear to improve BMD in individuals diagnosed with

AN, possibly due to metabolic abnormalities associated with the disorder (Abrams et al., 1993; Bachrach et al., 1990; Klibanski, Biller, Schoenfeld, Herzog, & Saxe, 1995; Rigotti, Neer, Skates, Herzog, & Nussbaum, 1991). In contrast, Brooks et al. (1999) found that calcium supplementation and exercise increased appendicular bone density in a case study of a 41-year-old woman with AN. Clearly, additional research needs to be done in order to assess the effect of calcium on preservation or restoration of bone mass among individuals with AN. In order for low BMD and the many comorbid complications that occur in conjunction with AN to be properly treated, an interdisciplinary intervention approach is needed.

The treatment team for individuals diagnosed with AN or EDNOS frequently includes a medical doctor, psychologist or psychiatrist, nutritionist, and occasionally, a social worker (Kohn & Golden, 2001). Depending upon the severity of the illness, treatment may be attempted on either an outpatient or an inpatient basis. Goals of treatment for individuals with AN include restoration of weight in addition to addressing physiological complications that have occurred as a result of the disorder (Fisher et al., 1995; Kohn & Golden). Psychotherapy and medication are normally attempted after physical and psychological effects of weight loss are reversed (Klein & Walsh, 2003). In addition, exercise is reduced or stopped until a medically safe body weight is reached (Kohn & Golden).

Once a safe body weight is reached, properly supervised exercise may be attempted. The American College of Sports Medicine (ACSM) recommends a variety of activities be prescribed for individuals with AN, specifically emphasizing activities with

low energy demand (2000). An exercise prescription involving resistance training results in less energy expenditure and could be a beneficial addition to treatment for individuals with AN (Brooks et al.; Chantler, Szabo, & Green, 2005). Strength training for patients with AN should not only provide possible functional and strength gains, but also psychological benefits (Chantler et al., 2005).

Little research exists on the effects of resistance training interventions on physiological and psychological variables among inpatient females with AN, and no data are available on outpatients (Szabo & Green, 2002). Variations in caloric intake, hormonal factors, and level of supervision during an exercise intervention will differ between inpatients and outpatients (Szabo & Green). Recently, Szabo and Green (2002) examined the effects of an 8-week resistance training program on BC and psychological well-being of inpatient participants with AN. Participants were divided into three groups: an exercise group comprised of patients with AN, an exercise group of healthy age- and sex-matched controls, and a control group of females with AN. Body weight, resistance bands, and light dumbbells were utilized to exercise all of the major muscle groups. The two groups who participated in the exercise intervention experienced significant increases in body mass, BMI, body fat percentage, FM, and lean body mass (LBM). Scores on the Beck Depression Inventory (BDI) significantly decreased in the AN non-exercising group, while neither exercising group showed a significant change in BDI score. Total Eating Disorder Inventory (EDI) scores significantly decreased in both of the AN groups, but not in the non-AN group. The authors concluded that the resistance training program did not offer an advantage for participants over non-participants, but was not detrimental

for participants with AN. Possible explanations for the results offered by the researchers included the short intervention time and the possibility that the non-intervention AN group was furtively exercising.

Chantler et al. (2005) also examined the effects of an 8-week light resistance training program among inpatients with AN. Patients with AN who took part in the intervention were compared to patients with AN who did not exercise and participants without known EDs who also participated in the exercise program. The program utilized light dumbbells, resistance bands, and body weight to work each of the major muscle groups. Following the resistance training program, the exercisers with AN significantly increased peak torque of the knee extensors and flexors as well as the elbow flexors. The authors concluded that although decreased muscle strength and function are common in persons with AN, a resistance training program in combination with refeeding can in individuals with AN.

Research is needed to fully explore the effects of resistance training on community-dwelling individuals with AN and EDNOS. Preliminary inpatient studies have shown promising results, however, these cannot be generalized to an outpatient population. Due to the dearth of research regarding this topic, the potential clinical effects of strength training on physiological and psychological factors in outpatients are unknown. Furthermore, the implications of such a program as an addition to a comprehensive eating disorder treatment program needs additional investigation.

Purpose

The purpose of this study was to determine the effects of a 10-week resistance training intervention on BMD, BC, strength, depression, and eating-disordered tendencies in community-dwelling females with AN or EDNOS.

Hypotheses

1. Following the resistance training intervention, there would be greater improvements in BMD of the hip, forearm, and lumbar spine, decreases in body fat percentage, and increases in chest and leg strength in the experimental group than in the control group.
2. Following the resistance training intervention, there would be greater decreases in scores on the Beck Depression Inventory–II and the Eating Disorders Inventory-3 in the experimental group than the control group.

Definition of Terms

1RM: One repetition max. The maximal amount of weight that can be moved through a full range of motion in a controlled manner with good posture a single time (ACSM, 2006).

Amenorrhea: Absence of the menstrual cycle for 3-6 consecutive months (Bemben, Buchanan, Bemben, & Knehans, 2004).

Anorexia nervosa (AN): An eating disorder characterized by refusal to maintain minimally normal body weight for height and age, intense fear of gaining weight or becoming fat, body image disturbance, and amenorrhea in females (American Psychological Association [APA], 1994).

Bone mineral density (BMD): Measure of the weight of bone in grams (g) divided by the area in centimeters square (cm^2).

Eating disorder not otherwise specified (EDNOS): Can include characteristics of AN, BN, and/or BED, but does not fit the strict DSM-IV criteria for these disorders (Klein & Walsh, 2003).

Oligomenorrhea: Irregular menstrual cycle for 39-90 days (Bemben et al., 2004).

Resistance training: Activities such as weight lifting that enhance muscular strength and endurance (ACSM, 2006).

Basic Assumptions

The following assumptions were made in this investigation:

1. All participants were honest and accurate when completing the questionnaires.
2. The resistance training program was followed by the participants in the manner prescribed and to the best of their ability.
3. All participants continued with their regular medical and psychological care during the course of the investigation.

Delimitations of the Study

1. Participants were females between the ages of 16 years and 35 years who were diagnosed with AN or EDNOS.
2. Participants in the control group continued with their regular physical activity program, which did not include a prescribed resistance training component.

Significance of the Study

Anorexia nervosa and EDNOS are grave illnesses with severe psychological and physiological consequences. Although extensive retrospective and observational research exists, the unique population and sensitive nature of these disorders have limited intervention studies. Furthermore, interventions on outpatients with AN or EDNOS are rare. Resistance training exercise is one such intervention that may significantly effect the various comorbidities often associated with these disorders.

Further investigation is warranted due to the lack of scientific research addressing the effects of exercise, specifically resistance training, on the psychological and physiological systems of outpatient individuals with AN and EDNOS. The positive effects of exercise within all body systems of healthy individuals have been extensively reported, however, these results are not generalizable to persons with eating disorders. The commonly associated comorbidities of cachexia (weight loss, muscle wasting, and loss of appetite associated with anorexia), body composition abnormalities, low BMD, and personality disorders are factors that need to be taken into account when observing exercise among this population.

Increasing both muscle mass and BMD in females recovering from EDs may drastically change the risk profiles of these individuals for developing osteoporosis and connective tissue degeneration. In addition, previous research has shown that resistance training can reduce depressive symptoms and eating disordered tendencies in persons with AN. However, this research has been conducted on inpatient participants with AN, and cannot be generalized to outpatients.

Finally, a supervised resistance training program can educate the participants with regard to an exercise modality that should be incorporated as a lifelong activity to prevent the deleterious effects of the eating disorder from which they are recovering. Resistance training has the potential to benefit both inpatient and outpatient individuals with AN and EDNOS. Interventions such as this may then be used in the future as a potential part of treatment for women recovering from eating disorders.

CHAPTER II

REVIEW OF LITERATURE

In this chapter, an overview of eating disorders is presented including definitions, diagnosis, prevalence, epidemiology, etiology, psychological and physiological consequences, and treatments. Research on BMD and BC are discussed, as these are physiological variables that may be altered through an exercise intervention among a population with eating disorders. The chapter ends with an overall summary and a review of the purpose of the study.

Eating Disorders

An eating disorder is a persistent disturbance of eating behavior or behavior intended to control weight that significantly impairs psychosocial functioning or physical health and is not secondary to another psychiatric disorder or general medical condition (Fairburn, 2001). Anorexia nervosa and BN are the most widely recognized eating disorders (Klein & Walsh, 2003). The prominent trait of AN is the refusal to maintain a minimally normal body weight for height and age, while BN is characterized by recurrent episodes of binge eating followed by behaviors to avoid weight gain such as use of laxatives or self-induced vomiting (Romano, 2005). Other recognized eating disorders include binge eating disorder (BED), characterized by recurrent episodes of binge eating without compensatory behaviors, and EDNOS, which may include characteristics of AN,

BN, and/or BED, but does not fit the strict DSM-IV criteria for these disorders (Klein & Walsh).

Definitions and diagnosis of AN. Features of AN include weight loss, increased physical activity, amenorrhea, and psychological disturbances (Romano, 2005). The principal characteristics of AN include an unyielding pursuit of thinness as well as a refusal to maintain body weight at a minimally acceptable standard for height and age (Klein & Walsh, 2003). This standard is defined in the DSM-IV as 85% of expected weight (APA, 1994). However, if the patient meets all other diagnostic criteria, but weighs between 85%-90% of expected weight, a diagnosis of AN is still acceptable (Klein & Walsh). Furthermore, Noordenbos et al. (2002) reported that even obese individuals can have AN. Another critical feature of the disorder is an extreme fear of gaining weight or becoming fat (Fisher et al., 1995). In addition, people with AN often display disturbed perceptions of their own body size and shape (Romano). Finally, post-menarchal females with AN are amenorrheic (Klein & Walsh).

There are two clinical subtypes of AN recognized in the DSM-IV. These subtypes are the restricting (ANR) and the binge-eating/purging (ANBP) types (APA, 1994). People with ANR lose weight primarily through excessive exercise or fasting, while individuals with ANBP attempt to decrease weight through behaviors such as laxative abuse or self-induced vomiting (Klein & Walsh, 2003; Kohn & Golden, 2001). There can be some crossover between the two subtypes, and it is unclear whether these are two lasting subgroups, or different phases in the same illness (Eddy et al., 2002). In addition to crossover between the two subtypes of AN, it is also common to see crossover

between AN and BN. Up to 40% of people with AN have symptoms of BN and 10% to 30% cross over from one disorder to the other at some time during the course of their illness (Golden & Sacker, 1984; Strober, Freeman, & Morrell, 1997).

Epidemiology of AN. The prevalence of eating disorders has risen over the past five decades (AED, n.d.c.; Kohn & Golden, 2001). At any given time, over 10% of females in their late teen or early adult years report symptoms of EDs (AED, n.d.c.). Anorexia nervosa disproportionately affects women, at a ratio of 10-20 females to 1 male (Klein & Walsh, 2003). Between 0.5% and 1% of late adolescent or adult women meet the criteria for the diagnosis of AN and between 0.5% and 3.7% will experience AN in their lifetime (AED, n.d.c.; Klein & Walsh; National Institutes of Mental Health [NIMH], 2001). However, because many cases are undiagnosed, the true prevalence rate of AN is likely higher (Misra & Klibanski, 2002). The age at diagnosis is bimodal at ages 14 years and 18 years, and the high incidence (approximately 2%) of AN among girls 10 years to 19 years of age makes the disorder the third most common chronic illness of adolescent females following obesity and asthma (Fisher et al., 1995; Kreipe, 1995; Wakeling, 1996).

The mortality rate from AN is the highest for any psychiatric disorder (Kohn & Golden, 2001). Approximately half of people diagnosed with AN recover, while 33% improve slightly, and 20% remain chronically ill (AED, n.d.b.). Crude mortality rates for AN are estimated to be between 5% and 10%, and there is an annual mortality of 0.5% per year (Klein & Walsh, 2003; Kohn & Golden). A distinct decrease in mortality among females with AN occurs after 10 years of onset (Deter & Herzog, 1994; Kohn & Golden).

Among males, the highest period of mortality is within 2 years of diagnosis (Kohn & Golden). Suicide accounts for approximately half of all deaths among individuals diagnosed with AN (Kohn & Golden). Medical difficulties, including complications from refeeding, account for 25% of all deaths among people with AN (Kohn & Golden).

Anorexia nervosa has been described as a disorder that primarily affects Caucasian, middle- to upper-class females; however, this description is no longer completely accurate (Fisher et al., 1995; Kohn & Golden, 2001). Migrant populations are at equal or increased risk of developing AN and the prevalence of EDs is increasing in non-Western and non-English speaking countries (Kohn & Golden). In addition, EDs in the United States are increasingly seen in young women of various ethnic groups including Hispanics, Native Americans, Blacks, and Asians (Crago, Shisslak, & Estes, 1996).

Many people exhibit partial forms of AN, but do not meet the strict diagnostic criteria for the disorder (Klein & Walsh, 2003; Kohn & Golden, 2001). Individuals who do not meet the criteria for AN are often categorized at EDNOS, the most common diagnosis among people seeking treatment for eating disorders (Turner & Bryant-Waugh, 2004). Persons included in this diagnosis are those that induce vomiting to maintain bodyweight in a normal range, those who binge and purge regularly but not at a frequency of twice weekly, those who purged but do not binge, those that have lost weight but have been amenorrheic for less than 3 months, and those that have not yet lost 15% of their body weight (Fisher et al., 1995; Kohn & Golden).

Etiology of AN. Multiple factors appear to be involved in the genesis of disordered eating behavior. While several risk factors have been identified, there is no known direct cause (Klein & Walsh, 2003). Risk factors associated with developing AN include being female, perfectionism and eagerness to please, difficulty communicating negative emotions, difficulty resolving conflict, low self esteem, fear of maturation, family history of ED, identity conflicts, depression, poor body image, and being teased (Fairburn & Harrison, 2003: Kohn & Golden, 2001; Kreipe, 1995). Anorexia nervosa has a low incidence rate, making it difficult to study prospectively (Klein & Walsh). In addition, many more people have risk factors than develop AN (Klein & Walsh). An interaction of several psychological, biological, and environmental risk factors is likely to contribute to the development of the disorder (Fisher et al., 1995). Furthermore, the risk factors that lead to dieting behavior and disordered eating appear to be different from those that maintain the behavior once it becomes established (Klein & Walsh).

The unique characteristics, rising prevalence, and high mortality rates make AN and EDNOSR an important topic for study. These eating disorders are psychiatric illnesses that have physiological manifestations. However, despite the obvious physical symptoms, persons with eating disorders can also have additional psychological comorbidities.

Psychological Consequences of Eating Disorders

A distorted body image is one of the most significant perceptual changes among individuals with AN (Fisher et al., 1995; Romano, 2005). Individuals with AN-induced cachexia often believe that they are overweight (Klein & Walsh, 2003). Occasionally, a

person with AN will acknowledge being thin or underweight, but insists that a particular body part is fat (Klein & Walsh). In addition, persons with AN may not accept that their weight loss is life threatening (Klein & Walsh). These perceptual changes are often seen in conjunction with neuropsychological disturbances among individuals who are in the acute phase of AN.

A variety of neuropsychological disturbances are seen in persons who are severely ill with AN, including difficulties with learning, visuospatial abilities, problem solving, motor speed, concentration, attention, and memory (Fisher et al., 1995; Klein & Walsh, 2003). Katzman, Christensen, Young, and Zipursky (2001) found that enlarged cerebral ventricles and diffusely atrophic frontal lobes may be associated with some of these abnormalities. Elevations in cortisol levels associated with a severely underweight state may also be associated with some of these neurological deficits (Laessle, Fischer, Fichter, Pirke, & Krieg,1992). It is not clear if weight restoration resolves the neuropsychological abnormalities seen in severe cases of AN (Fisher et al.; Klein & Walsh). Whatever the cause, the presence of cognitive deficits in acute cases of AN may contribute to the irrational preoccupation with body weight and food, in addition to the difficulty in treating this population with psychotherapy (Klein & Walsh).

In addition to perceptual and neuropsychological changes, mood disturbances are also common in acute cases of AN (Klein & Golden, 2003). Depression, obsessionality, and irritability are psychological disturbances frequently seen comorbidly with AN (Casper, 1998). However, research has shown that weight restoration often decreases these symptoms (Klein & Golden).

Depression. Clinical observations of individuals with AN often reveal depressive symptoms, and major depression is the most common comorbid psychiatric disorder in those with AN (Bulik, 2002). Up to 90% of persons diagnosed with AN have been reported to display depressive symptoms during the acute phase (Kaye, 1997). Symptoms of clinical depression are commonly seen in hospitalized AN patients, with restricting individuals being less depressed on traditional scales than individuals with ANBP (Casper, 1998). In addition, depression can occur before or after onset of AN (Bulik).

Although depressive symptoms can improve with refeeding and weight restoration, some researchers have shown that depression may persist even after recovery from AN. Holtkamp, Muller, Heussen, Remschmidt, and Herpertz-Dahlmann (2005) reported that 17 individuals with at least 3 years of complete recovery from AN had significantly higher levels of depression when compared to age- and sex-matched controls. Toner, Garfinkel, and Garner (1988) found a 40% lifetime history of depression in a 5- to 14-year follow-up of 47 individuals previously diagnosed with AN compared to a 12% rate in controls. An even higher rate of 68% lifetime history of depression was found by Halmi et al. (1991) in a 10-year follow-up of 62 persons with AN compared with 21% in the control group. In contrast, Pollice, Kaye, Greeno and Weltzin (1997) found that depression symptoms improved with refeeding in AN patients; however, mild to moderate depressive symptoms persisted after long-term weight restoration.

Anorexia nervosa is associated with a variety of comorbid psychological disorders. Researchers are unclear whether many of these illnesses can be reversed with

treatment and refeeding of a person with AN. In addition to psychological disturbances, AN can also cause a myriad of physical complications among persons with the disorder.

Physiological Consequences of Eating Disorders

Long-term, eating disorders can affect the majority of organ systems, in addition to the musculoskeletal and nervous systems, leading to various complications including death (AED, n.d.a.). Features of malnutrition are among the most notable consequences of AN, and laboratory and physical abnormalities are seen, most of which are consistent with reduced metabolic expenditure (Klein & Walsh, 2003; Kohn & Golden, 2001).

Reduced energy intake in an individual with AN can lead to complications ranging from minor physiological to potentially life-threatening conditions. Minor changes such as hair and skin abnormalities, generalized muscle weakness, and muscle wasting, are common in this population (Fisher et al., 1995; Kohn & Golden, 2001). Extended malnutrition can lead to more serious and long-term complications in individuals with AN, such as endocrine and gastrointestinal system disturbances, hypothermia, short stature, menstrual dysfunction, and osteopenia (Fisher et al.). Life-threatening conditions due to reduced energy intake in persons with AN are often due to cardiovascular disturbances (Fisher et al.).

The majority of clinical pathology seen in AN are due to cardiovascular disturbances (Giannini, Newman, & Gold, 1990). Cardiac output is reduced and congestive heart failure may occur, although rarely, during refeeding (Klein & Walsh, 2003). Mass of the heart is reduced, leading to hypotension and reduced pulse rate (Fisher et al., 1995). Hypotension, with or without postural changes, is seen in 60% of

patients diagnosed with AN (Giannini et al.; Kohn & Golden, 2001). Marked bradycardia is present in up to 90% of individuals with AN, with resting heart rates of under 50 beats per minute (Giannini et al.; Kohn & Golden). In addition, EKG abnormalities are common, often including atrial flutter or fibrillation (Giannini et al.; Klein & Walsh). Individuals with acute cases of AN may have right bundle branch block or premature ventricular contractions (Giannini et al.). Arrhythmias may also occur, specifically when electrolyte imbalances are present (Klein & Walsh).

Electrolyte imbalances are seen in over 40% of individuals with AN (Giannini et al., 1990). These imbalances are often due to dehydration (Kohn & Golden, 2001). Calcium and potassium imbalances, in particular, can cause a primary danger to the heart (University of Maryland Medical Center [UMMC], 2002). Most end-stage anorectic patients have severe potassium deficiency, and starvation ketosis leads to metabolic acidosis in many patients (Giannini et al.).

Adolescents with AN face additional physical consequences of the eating disorder. Specific physiologic features of adolescents with EDs include the potential for pubertal delay or interruption of maturation (Fisher et al., 1995). This slowed sexual development can affect maturational changes in lean and FM, height velocity, and the onset of menses (Fisher et al.).

Body composition. Body composition changes seen in individuals with AN include a substantial loss of FFM as well as fat mass (Kerruish et al., 2002; Polito et al., 1998; Shephard, 1991). In addition, when compared to a healthy population, AN patients have a significantly lower weight and body mass index (Kerruish et al.; Polito et al.).

Lean mass accounts for 15%-45% of the total decrease in body mass (Shephard). However, not all components of FFM are affected in the same way. Up to 2/3 of skeletal muscle may be lost by the acute phase of AN, but the extent of muscle loss partially depends on the amount of exercise performed by the patient (Heymsfield et al., 1979; Shephard). Furthermore, muscle mass of the upper extremities tends to atrophy more rapidly than the lower extremities or total body muscle mass (Keys, Brozek, Henschel, & Taylor, 1950). Lean mass is lost not only from muscle, but also from visceral tissues, such as the kidneys, liver, and spleen (Davies et al., 1978; Fohlin, 1977). Body mass index and weight can be restored with refeeding, however, the changes in FM and FFM vary with weight restoration among persons with AN.

Refeeding of patient with AN can result in significant changes in BC. Fat-free mass accounts for less than 1/2 of the weight regained by patients with AN, although there is large interindividual variability, depending upon the refeeding diet, patient age, duration of illness, and whether or not the individual had been allowed to participate in moderate intensity exercise (Falk et al., 1985; Forbes et al., 1984; Polito et al., 1998). Forbes et al. found that 64% of weight regained by 12 individuals with AN was FFM, while, more recently, Polito et al. found that muscle mass per kg of body weight in patients with AN was approximately 13% below that of controls after weight restoration. Due to the relatively small percentage of FFM that is regained by people who are recovering from AN, the amount and distribution of FM during weight restoration is reviewed in much of the literature.

Fat mass represents anywhere from 21%-77% of the total weight gained by the patient during recovery from AN, averaging over 50% (Grinspoon et al., 2001; Orphanidou, McCargar, Birmingham, & Belzberg, 1997; Polito et al., 1998; Probst et al., 2001; Scalfi et al., 2002). Some researchers have reported that the increase in FM is not evenly distributed throughout the body. A significant increase in trunk adiposity with weight restoration in anorexics has been found in several studies (Grinspoon et al., 2001; Iketani, Kiriike, Nagata, & Yamagami, 1999; Misra et al., 2003; Scalfi et al., 2002). In addition, Scalfi et al. reported that skinfold measurements at abdominal and bicep sites, as well as waist-to-hip ratio, were greater in weight-restored anorexics than in healthy controls. In contrast, Orphanidou et al. (1997) found that although FM was the largest component of weight gained by 26 females with AN, there was no preferential fat disposition in any one area. Regardless of the deposition pattern and percentage of FM regained with weight restoration, a recent study by Frey et al. (2000) found that individuals formerly treated for AN still had a lower body fat percentage than age- and sex-matched controls after 10 years. Another component of body composition that can be profoundly affected by malnutrition in an individual with AN is BMD.

Bone mineral density. Significant loss of BMD, which can lead to osteopenia and osteoporosis among women suffering from EDs, is a common occurrence (Baker et al., 2000; Brooks et al., 1999; Carruth & Skinner, 2000; Daee et al., 2002; Heer et al., 2004; NIMH, 2001). Not only is low BMD common in this population, but it occurs early in the course of the disease (NIH, 2005a).

Osteopenia and osteoporosis are commonly defined with the use of *t*-scores and *z*-scores (Misra & Klibanski, 2002). A *t*-score is used to compare current bone density to peak bone density in order to estimate the extent of bone loss in individuals diagnosed with eating disorders (Misra & Klibanski). However, the *t*-score is not an accurate way to determine BMD in adolescents as they are still accumulating bone mass, therefore, the *z*-score is used (Misra & Klibanski). A *z*-score is a measure of bone density in terms of standard deviations above or below the mean bone density for age and sex (Misra & Klibanski). Osteopenia is defined as a BMD *t*-score of between 1 standard deviation (*SD*) and 2.5 *SD*s below normative values in adult women and a BMD *z*-score of more than 2 *SD*s below normative values in children and adolescents (Misra & Klibanski; Rencken, Chesnut, & Drinkwater, 1996). Osteoporosis is defined as a BMD *t*-score of more than 2.5 *SD*s below normative values in adult women and a BMD *z*-score of less than 2 *SD*s in children and adolescents (Misra & Klibanski; Rencken et al.).

With each decrease of 1 *SD* of BMD, fracture risk doubles (Misra & Klibanski, 2002). Because of this increase in fracture risk, evaluation of BMD is recommended for individuals diagnosed with AN (Misra & Klibanski). Bone loss in AN can occur at all skeletal sites, and involves both cortical and trabecular bone, increasing the risk for fractures at the spine and the hip.

Factors that contribute to bone loss include menopause, low body weight, family history of low bone mass, amenorrhea, medications such as oral contraceptives, low calcium intake, excessive exercise or lack of exercise, smoking, excessive alcohol use, and high cola consumption (Baker et al., 2000; Carruth & Skinner, 2000; NIH, 2005a).

Due to the large number of factors that can affect BMD, it is reasonable to conclude that osteoporosis in females with EDs is not caused by the same physiological mechanisms in each instance and may be associated with the interaction of several variables (Baker et al.). However, several specific factors are thought to contribute to low BMD among people with AN.

Hormonal imbalances that negatively affect BMD are common in individuals with AN (NIH, 2005a). Females with low body weight often stop producing estrogen, leading to amenorrhea and losses in BMD (NIH, 2005a). In addition, individuals with AN frequently produce excessive amounts of the adrenal hormone cortisol, which is also known to stimulate bone loss (NIH, 2005a). Furthermore, decreases in growth factors and growth hormone, low body weight, malnutrition, calcium deficiency, and extended duration of illness can all contribute to bone loss in females with AN (NIH, 2005a).

Carmichael and Carmichael (1995) presented one of the first comprehensive evaluations of BMD in eating disordered patients. Bone mineral density was measured using dual energy x-ray absorptiometer (DXA). In addition, markers for bone formation and resorption, and mineral and metabolic markers were measured concurrently via serum and urine samples. Participants included 58 Caucasian females, aged 16 years to 46 years, with diagnoses of either AN, BN, or a combination of anorexia and bulimia (AB). Bone mineral density of the lumbar spine, both absolute values and z-scores, were lower in participants with AN compared to those with AB or BN. High levels of cortisol, often associated with lower BMD, occurred more often in participants with AN. In addition, participants with AN had a greater incidence and duration of secondary

amenorrhea. No association was found between BMD and body mass index, estrogen deficiency, or markers of bone turnover.

More recently, Turner et al. (2001) studied predictors of low bone density in young adolescent females with AN and other dieting disorders. Although individuals with AN were more malnourished, their BMD was similar to that of other dieting patients. Participants with low BMD in this study had often not achieved menarche or had longer duration of secondary amenorrhea, had lower estrogen levels, lower LBM, and had dieted longer. The researchers concluded that regardless of a clinical diagnosis, adolescents with eating disorders have an increased risk of low BMD when the behavior begins early in puberty.

Half of women suffering from AN have osteoporosis in the lumbar vertebrae and are significantly more likely to experience nontraumatic fractures than women who do not have AN (Baker et al., 2000). In addition to persons with AN, women who have been diagnosed with other eating disorders, including EDNOS have also been found to have significantly lower BMD than controls (Baker et al.; Morris et al., 2004). This increased risk of fracture can continue for decades after diagnosis of the eating disorder (Lucas et al., 1999; Morris et al., 2004; Vestergaard et al., 2002). The period of the greatest danger of osteoporosis and related fractures is later in life, when even previously healthy females also have an increased risk of fractures (Morris et al.). A long-term concern in premenopausal woman with osteopenia is that once menopause ensues, it is more difficult to prevent osteoporosis and fractures (Brooks et al., 1999).

The development of AN is most common during adolescence and early adulthood, which is a crucial time for bone growth (Carmichael & Carmichael, 1995; Misra & Klibanski, 2002). Maximum bone mass accrual normally occurs between the ages of 11 years and 14 years in girls, and 25% of peak bone mass accrual occurs in the 2 years surrounding peak height velocity (Misra & Klibanski). Development of an eating disorder at this time can not only compromise peak mineralization of bone mass, but also impair bone development if occurring during the adolescent growth spurt (Carmichael & Carmichael; Misra & Klibanski; NIH, 2005a). In addition to compromised BMD and delays in normal bone maturation, EDs that manifest during osteogenesis in adolescence and early adulthood can also lead to prevention of peak height (Carmichael & Carmichael; NIH, 2005a).

Early research examining the relationship between loss of BMD and EDs showed a relationship between reduced lumbar spine BMD and AN (Rigotti, Nussbaum, Herzog, & Neer, 1984). Since this report, reduced lumbar spine BMD has been consistently found in groups of adolescent and adult females with AN (Bachrach et al., 1991; Biller et al., 1989; Brotman & Stern, 1985; Davies et al., 1990; Lennkh et al., 1999; Misra et al., 2004). Some researchers have also shown reduced BMD of the hip (Treasure et al., 1986), femur (Hartman et al., 2000; Lennkh et al.), and whole body (Bachrach et al., 1990). Although the majority of research published shows a relationship between reduced BMD and AN among females diagnosed with the disorder, some researchers have found that BMD is not significantly reduced in the early stages of AN.

The relationship between AN and BMD in adolescents was studied by Wong et al. in 2001. Participants included 24 adolescent females (mean age = 14 years) who had been diagnosed with AN in the previous 12 months (mean months of diagnosis = 7.6). Total body and lumbar spine BMD, as well as bone mineral content (BMC) and BC, were measured using a Lunar DPX-L densitometer. Although there was a slight decrease in lumbar spine BMD, neither total body nor lumbar spine BMD were significantly decreased in the participants when compared to age- and sex-matched controls. While the results from this study do not show significant losses in BMD among adolescents recently diagnosed with AN, research has shown a decline in BMD with increased duration of an ED. Early and aggressive interventions in individuals with AN may prevent loss of BMD and possibly allow for achievement of normal peak bone mass and height, decreasing complications from loss of bone later in life.

Recovery of BMD. Several researchers have explored factors that may lead to BMD improvement among individuals with AN or those that are recovering from the disorder with conflicting results. Some studies have indicated that although improvement in bone mass can occur with weight recovery and resumption of menses, a significant number of adult and adolescent females with AN continue to be osteopenic (Herzog et al., 1993; Iketani et al., 1995; Rigotti et al., 1991). Furthermore, Klibanski et al. (1995) found that low BMD persisted in half of women with AN who were weight recovered after 18 months. Women who did not recover weight within this time period had a further decrease in BMD. Hartman et al. (2000) examined lumbar spine and femoral neck BMD in 19 women previously diagnosed with AN who were weight recovered for

an average of 21 years and found that femoral BMD was still significantly lower when compared to controls. In contrast, Orphanidou et al. (1997) found substantial increases in BMD after short-term weight restoration. In addition, Zipfel et al. (2001) found an increase in BMD and BMD *t*-scores at the lumbar spine and a decrease in the prevalence of osteopenia and osteoporosis after weight recovery in individuals with AN. However, a large percentage of these patients continued to have persistent low bone mass.

Recently, Bolton, Patel, Lacey, & White (2005) found that among anorexic patients who regained weight during treatment, total body BMC and lumbar spine BMD increased significantly. Therefore, research examining restoration of BMD among anorexics is contradictory and uncertainty exists regarding the relationship of complete and sustained clinical recovery and bone density. In addition to weight recovery, estrogen therapy has also been studied as a possible treatment for low BMD among individuals with AN.

Although estrogen replacement has been shown to be effective in increasing BMD among athletes with amenorrhea, this form of therapy has not been as successful in treating low bone mass among women with AN (Hergenroeder et al., 1997; Misra & Klibanski, 2002). Seeman, Szmukler, Formica, Tsalamandris, and Mestrovic (1992) found that women diagnosed with AN who had taken oral contraceptives for 30 months had higher lumbar BMD than those who did not receive contraceptives, but values were still lower than those of controls. In contrast, Grinspoon et al. (2000) found no difference in BMD at any site with or without estrogen supplementation in women with AN. Kibanski et al. (1995) also found no improvement in BMD among females with AN who

received estrogen supplementation. The results did, however, suggest that estrogen therapy may prevent further bone loss in individuals of very low body weight. A recent study by Munoz et al. (2002) examined BMD in 22 females with AN before and after treatment with estrogen and progestin, and found no improvement in BMD with this supplemental therapy. In addition to treatment with hormonal supplements such as estrogen, nutritional supplements such as calcium and vitamin D have also been examined as predictors of BMD in individuals with eating disorders.

The effect of calcium supplementation on BMD in individuals with AN is inconclusive. Skeletal growth requires a positive calcium balance until peak bone mass is attained (Fisher et al., 1995). Calcium intake may be significantly compromised when the onset of an eating disorder occurs during adolescence, a critical time for bone mineralization (Fisher et al.). However, some researchers have found that calcium supplementation does not appear to improve BMD in individuals diagnosed with AN (Bachrach et al., 1990; Klibanski et al., 1995; Rigotti et al., 1991).

The non-significant impact of calcium supplementation on preservation or restoration of bone mass in AN patients may be due to metabolic abnormalities as opposed to inadequate intake. Soyka, Grinspoon, Levitsky, Herzog, and Klibanski (1999) examined calcium and vitamin D intake among 19 girls with AN and 19 healthy controls and found that fewer controls met the criteria for intake of these two nutrients. Abrams et al. (1993) found abnormal calcium metabolism as well as decreased absorption and increased excretion among adolescents with AN.

In contrast, Brooks et al. (1999) found that calcium supplementation along with exercise increased appendicular bone density in a case study of a 41-year old woman with AN. The participant performed heavy resistance training 3 times per week, high impact exercise in the form of jumping daily, and supplemented 800 mg elemental calcium daily. Although the BMD of the intertrochanteric region, proximal femur, and total femur increased, due to the study design, it is impossible to tell whether the increase in BMD was due to the exercise, calcium supplementation, or a combination of the two. Clearly, additional research needs to be done in order to assess the effect of calcium on preservation or restoration of bone mass among individuals with AN.

Nutrition-dependent factors and nutrition status are also important in the loss of BMD that occurs in persons with AN (Misra & Klibanski, 2002). The profound effects of nutrition status on bone metabolism have been seen in healthy people, with a 50% reduction in rates of bone formation after short-term fasting over a four day period (Grinspoon, Baum, Kim, Coggins, & Klibanski, 1995). Bone mineral density can improve in individuals with AN who have had weight recovery, even without resumption of menses, indicating that nutritional factors play an important role in the BMD status of those with AN (Bachrach et al., 1991; Heer et al., 2004; Misra & Klibanski).

Relationship among body composition, weight, and BMD. Researchers have found a relationship between BC or BMI/weight and BMD among eating disordered individuals. Grinspoon et al. (2000) found that weight was a significant factor associated with BMD in individuals diagnosed with AN at all skeletal sites. Wong et al. (2004) reported that duration of illness and low BMI were significant predictors of reduced

BMD in Chinese patients with AN. An investigation by Misra et al. (2004) indicated that higher weight, LBM, FM, and BMI were important positive predictors of BMD while age at menarche was negatively related to BMD. Out of all of these factors, LBM was the single most important predictor of BMD at most sites measured, contributing to the variability of BMD of the lumbar spine (28%), hip (34%), femoral neck (43%), and total body (23%). As researchers have shown that LBM predicts BMD, monitoring of BC and BMI are important for individuals with AN in order to predict risk of low BMD and the complications this can cause.

Anorexia nervosa and the associated malnutrition can lead to a variety of physiological consequences, ranging from minor to life-threatening. These physiological outcomes, as well as comorbid psychological disturbances of AN, are addressed with a multidisciplinary approach in treatment.

Treatment for Eating Disorders

Interdisciplinary intervention is common for people with fully diagnosed EDs (Fisher et al., 1995; Kohn & Golden, 2001). Medical, psychological, and nutritional needs of the individual are addressed during treatment and a team approach is often utilized. The treatment team for individuals diagnosed with AN frequently includes a medical doctor, psychologist or psychiatrist, nutritionist, and occasionally, a social worker (Kreipe, 1995). Regular contact among the team members is essential to the recovery of the patient. Involvement of the family and school personnel is desirable for children and young adolescents with AN still living at home (Kohn & Golden).

Children and adolescents with early or mild eating disorders may be treated on an outpatient basis through education and minor behavioral interventions, often by the primary care physician without outside assistance (Kohn & Golden, 2001). A body weight goal and weight loss limit are established by the physician based upon stage of pubertal development, prior growth pattern, age, and height (Kreipe, 1995). Education for the patient involves information regarding basic nutritional requirements and the dangers of eating disorders (Fisher, 1995). Preoccupation with body weight is addressed and monitored by the physician and daily weighing is discouraged (Kohn & Golden).

Individuals with moderate or already established eating disorders require interdisciplinary intervention (Kohn & Golden, 2001). Patients that fit this profile include those who weigh less than 85% of ideal body weight, those that are eating less than 1,000 calories per day, and/or individuals who are binging and purging (Kreipe, 1995). Outpatient management with the interdisciplinary team is often necessary for these individuals (Kohn & Golden). Hospitalization is needed for individuals with AN when weight loss continues despite outpatient treatment or when weight loss has been so rapid or prolonged that life-threatening complications are imminent (Fisher et al., 1995). In addition, it is expected that the sooner in the weight loss cycle the patient is hospitalized, the more complete the recovery (Fisher et al.).

Whether the treatment occurs in an outpatient or inpatient setting, the goals for the patient remain the same (Fisher et al., 1995). The goals of medical intervention for individuals with AN are to promote weight gain, reverse medical complications of malnutrition, and for females, to promote spontaneous resumption of menses (Kohn &

Golden, 2001). Physiological aspects of the intervention, such as nutritional therapy and body weight gain, are essential for psychotherapy to be successful (Fisher et al., 1995; Kohn & Golden). In addition, exercise is reduced or stopped until a medically safe body weight is reached (Kreipe, 1995). Females who are amenorrheic for more than 6 months should also have BMD checked, and if found to be osteopenic or osteoporotic, estrogen therapy may be recommended (Kohn & Golden).

The goals of psychological interventions include the development of coping skills, increasing self-esteem, and focusing on issues that led to the development of the eating disorder (Kohn & Golden, 2001). Family therapy is also an important part of treatment, as the whole family is affected when one member has an eating disorder, specifically if the patient is young (Fisher et al., 1995; Kohn & Golden). In addition to patient and family therapy, medications are also used in the management of eating disorders.

In AN, selective serotonin reuptake inhibitors (SSRIs) are used when there is comorbid obsessive compulsive disorder (OCD) or depression (Kohn & Golden, 2001). However, these medications have little or no effect on the symptoms of very low body weight inpatients (Attia, Haiman, Walsh, & Flater, 1998; Ferguson, LaVia, Crossan, & Kaye, 1999). Once weight is restored in AN patients, pharmacological interventions may be more effective. An additional, if controversial, component of treatment that has the potential to affect the recovery progress among individuals with AN is exercise.

Exercise as a component of treatment for eating disorders. Physical activity as part of a treatment program has the potential to be problematic for patients with AN, who

may use exercise in a purgative fashion for energy expenditure (Brooks et al., 1999; McGilley & Pryor, 1998). Patients with AN have compromised health due to inadequate diet and overactivity can aggravate the state of undernutrition, leading to physical complications (Beumont, Arthur, Russell, & Touyz, 1994). However, supervised, regulated exercise may be beneficial during treatment for individuals with AN (Beumont et al.).

Properly supervised exercise and counseling can help the individual with AN increase body awareness as well as alleviate fears of gaining fat instead of muscle (Beumont et al., 1994). In addition, a structured exercise program can reduce discomforts associated with eating such as bloating and distention (Beumont et al.). Furthermore, participation in appropriate exercise can also help to teach persons with AN kinesthetic awareness (Beumont et al.). Appropriate exercise for persons with AN may be weight-bearing activities such as resistance training that may improve physiological and psychological variables in this population.

Weight-bearing exercise such as resistance training has been shown to increase lean muscle mass, BMD, and body image in healthy women. Therefore, this type of activity may help patients with eating disorders regain weight while experiencing healthy physiological and psychological changes (Carmichael & Carmichael, 1995; Grinspoon et al., 2001; Iacopino et al., 2003; Layne & Nelson, 1999; Lohman et al., 1995; Orphanidou et al., 1997; Polito, et al., 1998; Vuori, 1995). In healthy women without eating disorders, weight-bearing exercise is often part of the treatment plan for increasing BMD as well as in the prevention and treatment of osteoporosis (Brooks et al., 1999;

Carmichael & Carmichael, 1995; Iacopino et al., 2003). Resistance training may be useful in increasing BMD by effectively overloading the bone to induce modeling, which is the process by which bones experience gains in mass as well as modifications in shape (Brooks et al.; Layne & Nelson; Shaw & Witzke, 1998;Vuori, 1995). Unlike aerobic activity, resistance training results in less energy expenditure, increases lean body mass and strength, and therefore could be a beneficial addition to treatment for patients with eating disorders (Brooks et al.; Chantler et al., 2005).

The American College of Sports Medicine (ACSM, 2000) recommends a variety of activities for people with AN, specifically emphasizing activities with low energy demand. The purpose of prescribing exercise for persons suffering from EDs includes educating participants about BC, increasing LBM, BMD, and body image as well as using the exercise program as a means of behavioral modification (Sundgot-Borgen, Rosenvinge, Bahr, & Schneider, 2002). In addition, an exercise prescription involving resistance training for individuals with AN should not only provide functional and strength gains, but also psychological benefits without increasing metabolic demand on the participant to the extent that there are BMI, fat, and body mass losses (Chantler et al., 2005).

Limited research exists on the effect of resistance training interventions on BMD, BC, strength, and depression among inpatient females with AN, and no data are available on outpatients (Szabo & Green, 2002). Response to resistance training among inpatients may differ from outpatient females with AN due to variations in caloric intake, hormonal factors, and level of supervision (Szabo & Green). Recently, Szabo and Green examined

the effect of resistance training on BC and psychological well-being of inpatient females with AN. Groups that participated in the study included individuals with AN who took part in an exercise program, individuals with AN who did not exercise, and persons without AN who also engaged in the exercise program. The individuals who did not have AN were recruited from the community and age-matched to the hospital sample. The average age of the exercisers with AN was 20.1 years and the average age of the control group with AN was 21 years. The resistance training program consisted of two alternating schedules and included of a variety of exercises targeting the major muscle groups of the body. Body weight and elastic bands were utilized for the lower body, while 2.5 kg dumbbells were used for the upper body exercises. The 8-week program was the same for both of the exercising groups.

At the conclusion of the intervention, several significant changes were seen among the groups. The exercising groups both experienced significant increases in body mass, BMI, body fat, FM, and LBM. Scores on the Beck Depression Inventory (BDI) significantly decreased in the control group with AN, while neither exercise group showed a significant change in BDI scores. Total Eating Disorder Inventory (EDI) scores significantly decreased in both of the groups of patients with groups, but not in the healthy exercising group. As the majority of the changes did not differ significantly between either inpatient group, the changes may have been due to other components of the treatment program. The authors concluded that the resistance training program did not offer an advantage for participants over non-participants, however it did not appear to be detrimental for inpatients in the exercise group. The authors cite a short intervention

time, and the possibility that the control group with AN was furtively exercising as possible explanations for the results of the study.

Chantler et al. (2005) examined the effects of an 8-week light resistance training program on muscular strength of the knee and elbow extensors of hospitalized patients with AN. The participants with AN who took part in the intervention were compared to patients with AN who did not exercise and healthy participants who also followed the exercise program. Following the resistance training program, the exercisers with AN significantly increased peak torque of the knee extensors and flexors as well as the elbow flexors. The control group of inpatients and the healthy exercisers did not experience an increase in peak torque after the program. The authors concluded that although metabolic changes and muscle myopathy can lead to decreased muscle strength and function in persons with AN, appropriate refeeding combined with a resistance training program may contribute to strength gains.

The studies by Chantler et al. (2005) and Szabo and Green (2002) have several limitations. The small sample sizes and inpatient status of the participants limit generalizability of the findings. In addition, the resistance training programs were not progressive and utilized body weight, bands, and light dumbbells as opposed to weight machines and heavy dumbbells. More research is needed to fully explore the effects of resistance training on individuals with AN. These preliminary studies show promising results in terms of safety as well as physiological and psychological benefits that may be experienced by person with eating disorders due to this type of intervention.

Overall Summary and Conclusions

Anorexia nervosa is an illness with severe psychological and physiological consequences. Research addressing the effects of this disorder on mental and physical body systems is vast; however, due to the unique population and sensitive nature of the disorder, many of the published studies to date are retrospective or observational. Little research exists exploring the effects of interventions on physiological and psychological variables in persons with AN, and no data are available on outpatients. One such intervention that has the potential to have positive effects on the various comorbid disorders seen with AN is exercise.

The lack of scientific research addressing the effects of exercise, specifically resistance training, on psychological and physiological systems of outpatient individuals with AN emphasizes the need for further investigation. Researchers have reported the positive effects of exercise on BC, BMD, strength, and depressive symptoms among healthy populations. Although these positive effects of exercise are widely known and accepted, these findings cannot be generalized to individuals with AN, many of whom have specific complications involving muscle wasting, excessive gain of FM with refeeding, osteopenia and osteoporosis, and depression.

Therefore, the purpose of the present study was to determine the effects of a 10-week resistance training intervention on BMD, BC, strength, and depression in community-dwelling females with AN and EDNOSR. It is hypothesized that, following the resistance training intervention, there will be greater improvements in BMD of the hip, forearm, and lumbar spine, decreases in body fat percentage, and increases in chest

and leg strength in the experimental group than in the control group. It is also hypothesized that, following the resistance training intervention, there will be greater decreases in scores on the Beck Depression Inventory–II and the Eating Disorders Inventory-3 in the experimental group than the control group.

CHAPTER III

METHODOLOGY

Participants

Outpatient females who were clinically diagnosed by a medical doctor, psychologist, or psychiatrist with AN or EDNOSR (meeting all criteria for AN and oligomenorrheic at diagnosis), aged 16 years-35 years ($N = 14$), were recruited through local eating disorder specialists. Participants were required to have a minimum BMI of 14 kg/m^2, based upon previous research by Szabo and Green (2002) and Beumont et al. (1994), as well as a recommendation from their physician to be involved. Participants who were pregnant or who thought they may be pregnant were excluded from participation. Participants were randomly assigned to either the experimental or the control group. Persons assigned to the control group had the option to participate in the exercise program following the 10-week intervention.

Instrumentation

Demographic and risk stratification questionnaire. A risk stratification questionnaire was completed pre- and post-intervention. The questionnaire includes items based upon ACSM (2006) risk stratification guidelines in addition to items added by the principal investigator regarding menstrual status, duration of eating disorder, current medications, and supplements (see Appendix A).

Eating Disorders Inventory-3. The Eating Disorders Inventory-3 (EDI-3) was used to assess tendencies of disordered eating (Garner, 2006). The EDI-3 provides normative information for eating disordered females aged 13 years through 53 years old (Garner). The Inventory consists of 91 questions organized into 12 scales, including 3 eating disorder specific scales (Garner). The 3 eating disorder specific scales include the Drive for Thinness (DT) scale, the Bulimia (B) scale, and the Body Dissatisfaction (BD) scale. These 3 scales are equally weighted and comprise the Eating Disorder Risk Composite (EDRC), which supplies a comprehensive measure of weight and eating concerns (Garner). Eating Disorder Risk Composite *t*-scores on the EDI-3 were analyzed for the purposes of this study. Scores can range from under 45 to over or equal to 57 for the EDRC. Scores under or equal to 45 represent the low clinical range, scores from 46 to 56 represent the typical clinical range, and scores equal to or over 57 represent the elevated clinical range (Garner). The EDRC reliability ranges from .90 to .97 across the three diagnostic groups (ANR, ANBP, and EDNOS) used in the current study (Garner). The 3 Eating Disorder Risk Scales that make up the EDRC have reliabilities in the high .80s to the low .90s across the groups (Garner). The EDI-2 has been validated for use in eating disordered populations (Garner, Olmstead, & Polivy, 1983).

Beck Depression Inventory-II. The Beck Depression Inventory–II (BDI-II) was used to assess depression (Beck, Steer, & Brown, 1996). The BDI-II is a 21-item scale used to assesse intensity of depression in normal and clinical populations (Beck et al.). Scores range from 0 to 63. Scores from 0-12 represent the minimal range of depression, 14-19 represent mild depression, 20-28 fall into the moderate depression range, and

scores from 29-63 are considered in the severe range (Beck et al., 1996). Total scores on the BDI-II were analyzed for the purposes of this study.

The BDI-II is a revision of the BDI, which has been validated for use in individuals diagnosed with eating disorders. The BDI has an average internal consistency of .87 (Beck, Steer, & Garbin, 1988). Principal factor analysis done by Pulos (1996) investigated whether the BDI was consistent with a measure of depression in individuals with eating disorders. The results suggested that a single factor was present, with all items correlated with the factor; therefore, the BDI appears to be an adequate measure of depression in persons with eating disorders.

Preparticipation screening questionnaire. The American Heart Association (AHA)/ACSM Health/Fitness Facility Preparticipation Screening Questionnaire was used to assess cardiovascular risk factors and symptoms (ACSM, 2006). The questionnaire defines a broad scope of chronic disease that may be aggravated by exercise (ACSM, 2006). The questionnaire was used by the principal investigator to assess any potential health problems that needed to be addressed prior to the intervention. The questionnaire is divided into two sections. The first section includes questions involving history and symptoms of cardiovascular disease as well as other health issues. The second section contains questions regarding specific cardiovascular risk factors. Information from this questionnaire was used in order to inform the principal investigator of any potential health problems.

Incorporating More Physical Activity and Calcium in Teens Food Frequency Questionnaire (IMPACT FFQ). The IMPACT FFQ was designed to evaluate nutrition and enhance future bone health in adolescents (Hoelscher, Day, Kelder, Hergenroeder, Ward, 2003). The questionnaire was used to assess average caloric intake and average calcium intake. All participants were asked to record dietary intake pre- and post-intervention. The FFQ was analyzed using ESHA professional nutrition analysis software and database, version 7.7 for Windows. Calcium was reported in average milligrams per day. Calories were reported in average kilocalories per day.

Anthropometric measures. Height was measured to the nearest 0.5 cm using a stadiometer (SECA model 222, Seca Gmbh & Co. Kg., Hamburg, Germany). During the measurement, the participant stood barefoot on level ground with heels together and weight evenly distributed. Participants' heads were positioned so that the line of vision was horizontal and the sagittal plain of the head was vertical. Weight was measured in hospital scrubs without shoes after voiding to the nearest 0.1 kg using a digital scale (SECA model 770, Vogel & Halke, Hamburg, Germany). All participants stood backwards in order to avoid viewing weight. Height and weight were used to calculate BMI. Body mass index was calculated using weight in kilograms divided by height in meters squared.

Body composition. Skinfold sites (abdominal, triceps, chest, midaxillary, subscapular, suprailiac, and thigh) were measured using Lange skinfold calipers (Beta Technology Incorporated, Cambridge, Maryland) by the principal investigator. Although DXA is often used for body composition measurements, it has been found to

overestimate body fat percentage in lean subjects (Fogelholm, Kukkonen-Harjula, Sievänen, Oja, & Vuori, 1996). Furthermore, Probst et al. (2001) reported that body fat estimation by skinfold appeared to be as accurate as underwater weighing in individuals with AN. Serial measurements were taken on the right side of the body a minimum of two times. Any measurements that were not within 1 mm for a specific site were remeasured a third time. The closest two measurements for each site were averaged together and used in the equation. Body density was calculated using 7-site skinfold equation for women (Jackson & Pollock, 1985). Body fat percentage was calculated from estimates of body density using a population-specific formula (Heyward & Stolarczyk, 1996).

Bone mineral density. Lumbar spine, hip, and forearm BMD were measured using a Holgic QDR 4500W elite dual energy x-ray absorptiometer (QDR 4500W Elite Windows Version 11.2; Wattham, MA). The manufacturer's standard protocol was followed by a licensed technician. Lumbar spine BMD was assessed by imaging L1-L4, which includes the body of the vertebra, the pedicles, lamina, spinous process, and transverse process. Hip and forearm scans were performed on the participant's dominant side. The hip scan included the femoral neck, trochanteric region, bone shaft, and Ward's triangle. The forearm scan included the radius, the ulna, and the first row of carpal bones. The DXA was calibrated daily using a spine and step phantom assuring that all calibrations fell within appropriate ranges. All participants wore hospital scrubs and removed all metal objects from their bodies in order to avoid false images on the scan.

Muscular strength. Chest and leg strength estimated 1RMs were estimated (Heyward, 1998). Participants warmed up by completing 5-10 repetitions of the exercise at 40%-60% of the estimated 1RM. A starting point for women who do not usually lift weights is generally 30% to 50% of body weight. Participants then rested for 1 minute and statically stretched the muscle group. The rest and stretch were followed by 3-5 repetitions of the exercise at 60%-80% of the estimated 1RM. Participants rested for 1 minute, then weight was conservatively increased. Participants rested, weight was increased until repetitions to fatigue did not exceed 10. The amount of weight lifted as well as the number of repetitions was then used to estimate 1-RM using the Brzycki (1993) regression equation.

Procedures

Approval was gained by the University Institutional Review Board (see Appendix B). All participants were informed of the procedures of the study and signed an informed consent form (see Appendix C). Minors signed a child assent form and parents completed a parental informed consent form (see Appendix D and E, respectively). All forms and questionnaires, as well as all anthropometric, BC, BMD, and strength measures were completed during a visit to the University Exercise Science Lab pre- and post-intervention. All participants were assigned a number using a random number generator and were randomly assigned to either the experimental or the control group.

Intervention. The experimental group participated in a program of resistance training at a frequency of three times per week, as recommended by the ACSM (2006). Based upon data from Beverly et al. (1989), Chantler et al. (2005), and Szabo and Green

(2002), it was decided that an intervention period of 10 weeks should be sufficient to observe changes in BMD, strength, BC, and psychological measures. All training sessions during the 10-week intervention were in group format and supervised by the principal investigator, who holds a Master of Science in Exercise Science, is a certified ACSM Health/Fitness Instructor, and is a certified American Heart Association CPR Instructor. Training sessions took place at the University or the Young Men's Christian Association (YMCA), depending on where the participant lived. The resistance training program utilized Nautilus strength training machines and dumbbells, and included exercises designed to work each major muscle group (leg press, lunges, lat pulldown, bench press, bicep curl, triceps extension, military press, and abdominal curls). The leg and bench press was initially performed at 70% of the participants' estimated 1RM. All exercises were performed at a weight that could be lifted for two sets of 10-12 repetitions. As the repetitions became easier, resistance was increased (based upon the progressive resistance principle). Upper body exercises were increased in 2.5 pound increments for machines and 2 to 3 pound increments for free weights. Leg press was increased in 5 pound increments. Once 13 repetitions could be completed, resistance was increased, causing repetitions to initially decrease until the participant experienced increased in strength. Participants in the experimental group also continued with their treatment program during the course of the intervention.

The control group continued with their treatment program prescribed by their physician, psychologist, or psychiatrist during the course of the study. Each participant was contacted weekly by the principal investigator.

Posttest measures. After the 10-week prescribed exercise intervention, participants in both the experimental and control group each had their height, weight, BMD and BC remeasured, using the same instrumentation and procedures as used in the pretest. In addition, participants again completed the EDI-3, the BDI-II, the IMPACT FFQ, and answered questions regarding menstrual status, medications, and supplements.

Data Analyses

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 15.0. Physical descriptive data were reported using means and standard deviations. Correlation was used to confirm that calcium intake was not related to any of the dependent variables for either the experimental or control group. Welch independent samples *t*- tests were conducted to determine if experimental or control group dependent variable scores differed by menstrual status; the scores did not differ. Therefore, neither calcium nor menstrual status were controlled for. A 2x2 repeated measures analyses of variance (ANOVA) were used to determine if there were differences in BC and scores on the EDI-3 and BDI-II between the experimental and control groups. Multivariate analyses of variance (MANOVA) were used to determine if there were differences between the experimental and control groups on lumbar spine, hip, and forearm density, as well as chest and leg strength. Effect sizes were reported and an alpha of .05 was used to determine the significance of each test.

CHAPTER IV

RESULTS

The following hypotheses were tested in this investigation:

1. Following the resistance training intervention, there will be greater improvements in BMD of the hip, forearm, and lumbar spine, decreases in body fat percentage, and increases in chest and leg strength in the experimental group than in the control group.
2. Following the resistance training intervention, there will be greater decreases in scores on the Beck Depression Inventory–II and the Eating Disorders Inventory -3 in the experimental group than the control group.

Description of Participants

Data were collected from 14 outpatient females with eating disorders. There was 100% compliance to the intervention within the full sample. Participants were randomly assigned to the experimental group and the control group. A group assignment list for 30 potential participants was randomly generated with a ratio of 2 (experimental) to 1 (control). However, the final sample size contained 14 participants with 7 participants being randomly placed in the experimental group and 7 being randomly placed in the control group.

Individuals participating in the current study had a mean age of 25.6 years (± 5.0 years). Furthermore, mean height for the full sample was 1.63 kg (± .05). Additional

descriptive statistics for the full sample are presented in Table 1, while descriptive statistics for participants in the experimental group and participants in the control group are presented in Tables 2 and 3, respectively.

Findings of ANOVA and MANOVA Analyses

Correlation was used to confirm that calcium intake was not related to any of the dependent variables, thus, it was not necessary to control for this variable. Welch independent samples *t*-tests were conducted to determine if experimental or control group dependent variable scores differed by menstrual group; the scores did not differ. Thus, menstrual status was not controlled. Therefore, 2x2 repeated measures ANOVA and repeated measures MANOVA were used in the analyses. An alpha of .05 was used to determine significance of each test.

Differences between the experimental and control groups on percentage of body fat, the EDI-3, and BDI-II were determined using two-way repeated measures ANOVAS (see Table 4). It was hypothesized that the experimental group would have lower percent body fat percentage than the control group following the intervention. There was a significant difference in body fat percentage from pretest to posttest among the entire sample. However, the change in body fat percentage over time was similar within the experimental and control groups and the average percent body fat was similar between the groups. The hypothesis was not supported; therefore, participants in the experimental group did not have greater changes from pretest to posttest in body fat percentage after the intervention than participants in the control group.

Table 1

Descriptive Statistics of Study Variables for Total Sample (N = 14)

Variable	Pretest		Posttest	
	M	*SD*	*M*	*SD*
Weight (kg)	56.96	7.09	57.42	7.67
BMI (kg/m^2)	21.52	2.89	21.65	3.18
Body fat (%)	15.10	6.37	20.18***	8.03
Bone mineral density (g/cm^2)				
Lumbar spine	0.995	0.118	0.999	0.105
Hip	0.877	0.100	0.874	0.093
Forearm	0.517	0.041	0.521	0.036
Calcium intake (mg/day)	982.07	399.27	1094.07	464.53
Muscle strength (est. 1RM)				
Chest	72.39	22.56	84.00***	21.10
Leg	370.50	116.75	448.00***	131.79
Psychological test scores				
EDI-3 EDRC *t*-score	48	11	46	16
BDI-II	23	12	18	14

Note. BMI = body mass index; BM = body mass; Est. 1RM = estimated 1 repetition max; EDI-3 EDRC = Eating Disorders Inventory-3 Eating Disorder Risk Composite; BDI-II = Beck Depression Inventory-II.

*** $p < .001$: Significant difference in mean pretest and posttest scores.

Table 2

Descriptive Statistics of Study Variables for Experimental Group (n = 7)

Variable	Pretest		Posttest	
	M	*SD*	*M*	*SD*
Weight (kg)	55.59	5.74	56.06	6.72
BMI (kg/m^2)	20.95	2.59	21.07	3.14
Body fat (%)	12.32	4.82	16.71	6.63
Bone mineral density (g/cm^2)				
Lumbar spine	1.012	0.140	1.026	0.123
Hip	0.861	0.104	0.867	0.098
Forearm	0.501	0.030	0.507	0.025
Calcium Intake (mg/day)	867.43	337.95	1067.71	308.79
Muscle strength (est. 1RM)				
Chest	70.82	14.44	92.20**	19.06
Leg	387.59	140.49	526.68**	97.65
Psychological test scores				
EDI-3 EDRC *t*-score	47	13	43	21
BDI-II	24	11	17	13

Note. BMI = body mass index; BM = body mass; est. 1RM = estimated 1 repetition max; EDI-3 EDRC = Eating Disorders Inventory 3 Eating Disorder Risk Composite; BDI-II = Beck Depression Inventory II.

** $p < .01$: Significant difference in mean pretest and posttest scores.

Table 3

Descriptive Statistics of Study Variables for Control Group (n = 7)

Variable	Pretest		Posttest	
	M	*SD*	*M*	*SD*
Weight (kg)	58.33	8.45	58.83	8.80
BMI (kg/m^2)	22.10	3.26	22.32	3.36
Body fat (%)	17.87	6.84	23.56	8.28
Bone mineral density (g/cm^2)				
Lumbar spine	0.977	0.100	0.973	0.084
Hip	0.892	0.102	0.880	0.095
Forearm	0.532	0.047	0.5436	0.042
Calcium Intake (mg/day)	1096.7	447.8	1120.6	608.9
Muscle strength (est. 1RM)				
Chest	73.97	29.81	76.81	21.52
Leg	353.42	95.48	370.11	117.47
Psychological test scores				
EDI-3 EDRC t-score	50	10	50	10
BDI-II	21	13	19	16

Note. BMI = body mass index; BM = body mass; 1RM = 1 repetition max; EDI-3 EDRC = Eating Disorders Inventory-3 Eating Disorder Risk Composite; BDI-II = Beck Depression Inventory-II.

Further, no statistically significant changes from pretest to posttest on EDI-3 and BDI-II scores were found for the entire sample (see Table 4). Consequently, hypothesis 2 was not supported; participants in the experimental group did not experience greater changes on EDI-3 and BDI-II scores from pretest to posttest than participants in the control group.

Chest and leg strength differences from pretest to posttest between the experimental and control groups were determined by MANOVA. There was a significant main effect of time on change in strength measures. Therefore, the average chest and leg strength for the entire sample changed over the time of the intervention, $F(2, 11) = 59.75$, *Pillai's trace* = .92, observed power = 1.0, $p < .001$. The main effect of group on leg and chest strength measures was not significant. Hence, when examining the groups individually, the average chest and leg strength was not significantly different between the two, $F(2, 11) = 1.58$, *Pillai's trace* = .22, observed power = .27, $p = .25$. However, as predicted, there was a significant interaction between time and group. Therefore, the chest and leg strength averages significantly increased for the experimental group but not for the control group, $F(2, 11) = 36.10$, *Pillai's trace* = .87, observed power = 1.00, $p < .001$. Thus, this part of the hypothesis was supported (see Figures 1 and 2).

Univariate tests were conducted as follow-up analyses for the MANOVAs. As predicted, the ANOVA results indicated that time was a significant predictor of change in both chest and leg strength for the entire sample from pre- to posttest. On average, the

Table 4

Univariate Analyses for Quantitative Study Variables

Variable	*F*	Obs. Power	omega^2	*p*
Body fat (%)				
Time	42.37	1.00	.60	< .001***
Group	3.09	.37	.07	.10
Body fat * Group	.70	.43	0.00	.42
Muscle strength (est. 1RM)				
Time				
Chest	27.60	1.00	.49	< .001***
Leg	34.18	1.00	.54	< .001***
Group				
Chest	.284	.08	.00	.60
Leg	2.56	.31	.14	.14
Time * Group				
Chest	16.16	.96	.35	.00**
Leg	21.11	.99	.42	.00**
Psychological test scores				
EDI-3 EDRC t-score				
Time	.71	.12	.00	.42
Group	.35	.09	.00	.56
EDI-3 * Group	.61	.11	.00	.45

Table 4 (continued).

Variable	*F*	Obs. Power	omega^2	*p*
BDI-II				
Time	2.32	.29	.00	.15
Group	0.01	.05	.00	.91
BDI * Group	1.10	.16	.00	.32

Note. All degrees of freedom values are 1, 12. BMI = body mass index; BM = body mass; 1RM = 1 repetition max; EDI-3 EDRC = Eating Disorders Inventory-3 Eating Disorder Risk Composite; BDI-II = Beck Depression Inventory-II; Obs. power = observed power.

** $p < .01$, *** $p < .001$.

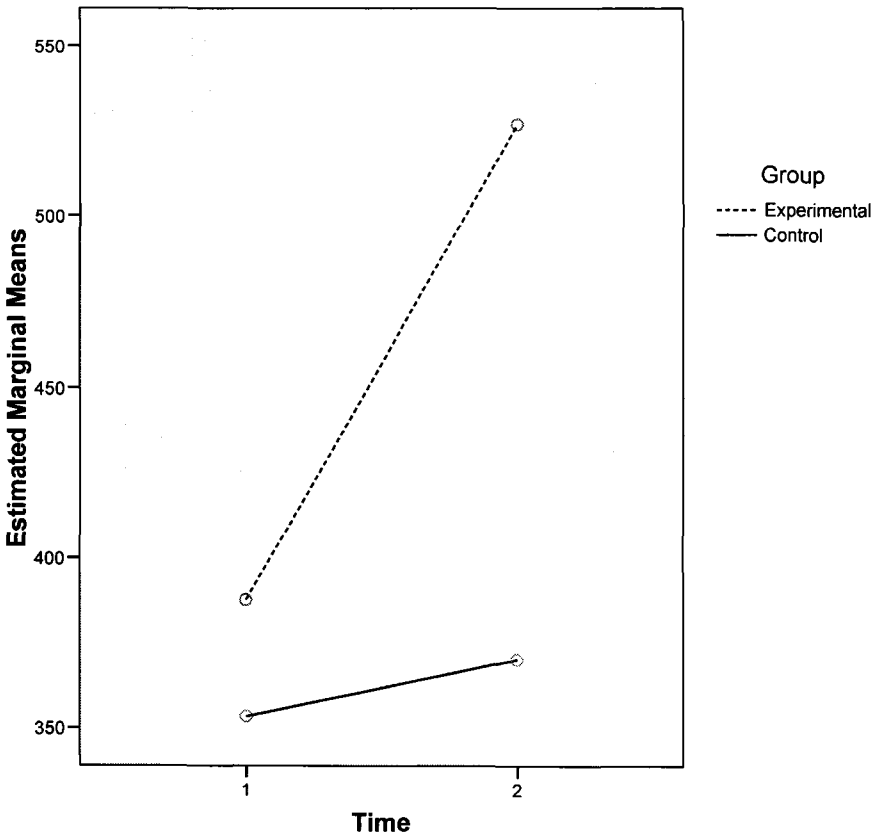

Figure 1. Mean Estimated Leg 1RM

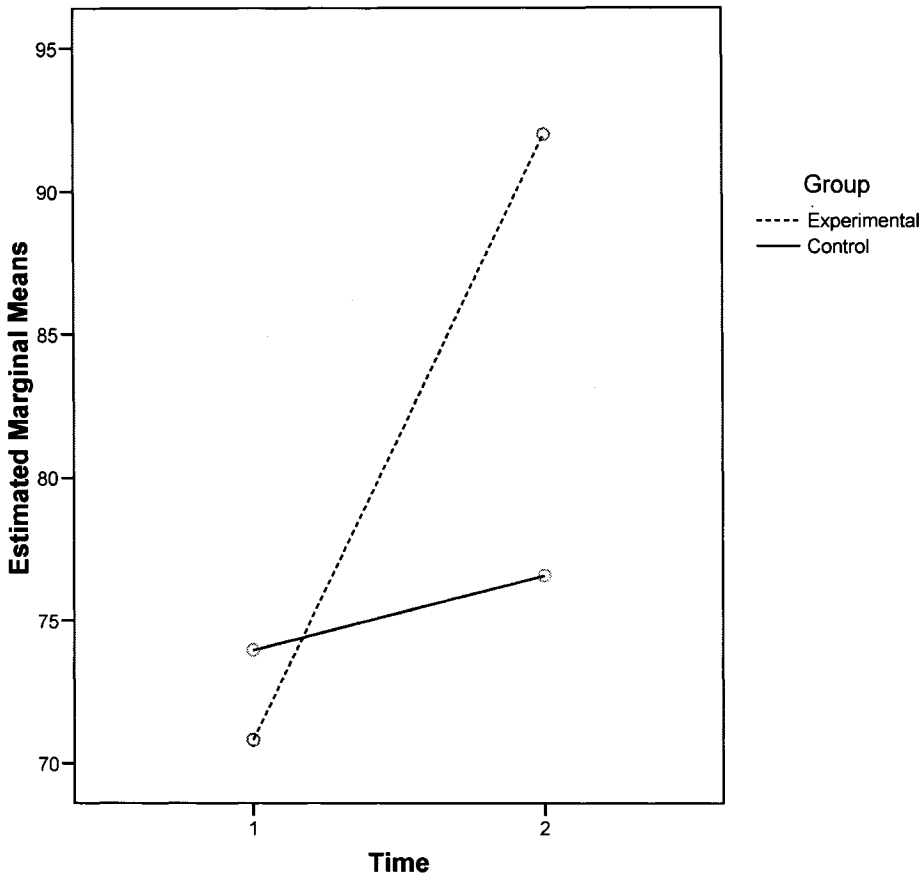

Figure 2. Mean Estimated Chest 1RM

experimental and control groups had similar leg and chest strength scores (i.e. the test of group was not significant). Both chest and leg strength improved for the experimental group but not for the control group.

Differences in lumbar spine, hip, and forearm BMD between the experimental and the control groups from pretest to posttest were determined by MANOVA. The average spine, hip, and forearm BMD values were not significantly different between the groups, F (2, 11) = 2.79, *Pillai's trace* = .46, observed power = .48, p = .10. Furthermore, BMD did not change from pretest to posttest, F (3, 10) = .79, *Pillai's trace* = .19, observed power = .36, p = .53. Further, there was no interaction, indicating that the experimental group did not experience greater improvement in BMD of the spine, hip, and forearm from pre- to posttest than the control group, F (3, 10) = 1.31, *Pillai's trace* = .28, observed power = .174, p = .32. Thus, the hypothesis was not supported.

CHAPTER V

DISCUSSION

Anorexia nervosa is a psychological illness that affects millions of adolescent and adult women. Various physiological and physiological consequences appear comorbidly with AN and EDNOS, including loss of BMD, loss of FM and FFM, a reduction in strength, and a greater incidence of depression. Resistance training has been shown in many populations to increase BMD, lean mass, strength, and depressive symptoms. Although studies exist regarding the effects of cardiovascular exercise on physiological and psychological comorbidities in females with EDs, limited research exists regarding the effects of resistance training on these variables.

The purpose of the present study was to determine the effects of a 10-week resistance training intervention on BMD, BC, strength, depression, and eating disordered tendencies in community-dwelling females with EDs. The training program consisted of supervised sessions, 1 hour in length, three times per week. Each major muscle group of the body was worked with a combination of free weights and conventional strength training machines. Participants completed 2 sets of 8-12 repetitions for each prescribed exercise. Once a weight could easily be lifted over 12 times, the resistance was increased. All participants completed each training session and no adverse outcomes or events occurred during the study.

Description of Sample

The sample included 14 women receiving outpatient treatment for either AN or EDNOS with AN tendencies. Sample sizes in other intervention studies are often low, typically between 7 to 15 participants per group (Chantler et al., 2005; Lear, Pauly, & Birmingham, 1999; Szabo & Green, 2002; Thien, Thomas, Markin, & Birmingham, 2000). Researchers often conduct retrospective studies for ease of access to information on large numbers of people in this special population.

Participants in the current sample were all clinically diagnosed by a physician, psychiatrist, or psychologist as meeting the criteria for an ED, as defined by DSM-IV. The full sample consisted of 5 participants diagnosed with ANR (35%), 3 participants diagnosed with ANBP (21%), 2 participants diagnosed with EDNOS/AN (14%), and 4 participants with dual diagnoses of ANR and ANBP (28%). The breakdown of percentages represented in the current study is comparable to other exercise intervention research in this population. For example, participants in an exercise program designed by Calogero and Pedrotty (2004) had diagnoses of ANR (32%), ANBP (13%), and EDNOS (20%). The remaining 35% were diagnosed with BN.

Individuals participating in the current study had a mean age of 25.6 years (± 5.0 years). Average ages of research participants with AN or EDNOS/AN tendencies range from 20 years - 27 years for inpatients (Bolton et al., 2005; Goebel, Schweiger, Krüger, & Fichter, 1999; Iketani et al., 1995). In addition, the average number of years since diagnosis among participants in the current study was 5.8 years (± 4.2 years). Likewise, the number of years since diagnosis in other exercise intervention studies in this

population ranged from 6.2 years ± 7.2 years to 8.3 years ± 8.5 years (Calogero & Pedrotty, 2004).

Furthermore, of the 14 participants, 50% had regular menstrual cycles, while the other 50% were either oligo- or amenorrheic. While up to 90% of females with AN and 45% of females with ENDOS/AN have been shown to have either oligo- or amenorrhea, fewer outpatients suffer from these disorders (Goebel et al., 1999). This may be due to the medical stability and/or higher body mass of outpatients, which has been linked to a resumption of menses (Iketani et al., 1995). Menstrual status was not correlated with any of the dependent variables in the experimental or control group in the current study.

Anthropometrics. The average weight of participants in the current study was 56.96 kg ± 7.09 kg. Among the experimental group, the mean body weight was 55.59 kg ± 2.59 kg, while the control group's average body weight was 58.33 kg ± 8.45 kg. Community-dwelling adolescents with AN have been reported to have mean body weights of 46.4 kg ± 5.9 kg (Misra et al., 3003). In contrast, Touyz, Lennerts, Arthur, and Beumont (1993) performed an exercise intervention on inpatients, which had an experimental group with an average weight of 39.07 kg ± 3.14 kg and a control group with an average weight of 37.17 kg ± 4.27 kg. Individuals who require inpatient treatment for AN are often emaciated, while outpatients have generally gained enough weight to be medically stable. It is important to note that although one of the criteria of DSM-IV diagnosis of AN describes an unwillingness to maintain 85% of ideal body weight, individuals who are in outpatient therapy have often regained enough weight to be considered medically stable, and are often of normal weight (Giordano, 2005).

Noordenbos, Oldenhave, Muschter, & Terpstra (2002) found that people of all weights can have AN, from thin to obese. Therefore, despite weight gain while in the process of recovery, the initial diagnosis of AN can still exist (McIntosh et al., 2004; Noordenbos et al.).

Percent body fat for the sample fell between reported average body fat percentages found in outpatients and that of inpatients. Misra et al. (2004) reported that average body fat percentage among community-dwelling adolescents with AN was 18.5% ± 5.1%. In contrast, participants in an inpatient exercise intervention by Szabo and Green (2005) described a mean body fat percentage of 10.0% ± 2.2% for the experimental group and a mean body fat percentage of 12.2% ± 2.7% in the control group. Mean body fat percentage of the full sample in the present intervention was 15.10% ± 6.37%. Further, the mean body fat percentages for the experimental and control groups were 12.32% ± 4.82% and 17.87% ± 6.84%, respectively.

Participants in the current study also had similar BMI values to those in other exercise intervention studies. The average BMI of the full sample in the current investigation was 21.5 kg/m^2 ± 2.9 kg/m^2. The mean BMI of the experimental and control groups were 21.0 kg/m^2 ± 2.6 kg/m^2 and 22.1 kg/m^2 ± 3.3 kg/m^2, respectively. Body mass index values for both inpatients and outpatients in past exercise interventions have ranged from 15.1 kg/m^2 ± 1.6 kg/m^2 to 20.5 kg/m^2 ± 5.0 kg/m^2 (Calogero & Pedrotty, 2004; Thien et al., 2000). Body fat percentage and BMI of the current sample were comparable to those of participants in previous exercise interventions as well as community-dwelling females with AN. The higher mean values for weight among the

current participants are likely due to the age of the participants in addition to weight stabilization necessary for outpatient status.

Bone mineral density. Individuals who participated in the current exercise intervention had an average pre-test hip BMD of 0.86 g/cm^2 ± 0.10 g/cm^2, lumbar spine BMD of 1.01 ± 0.14 g/cm^2, and forearm BMD of 0.50 g/cm^2 ± 0.03 g/cm^2. The control group had average pre-test hip BMD values of 0.89 g/cm^2 ± 0.10 g/cm^2, lumbar spine BMD values of 0.97 g/cm^2 ± 0.10 g/cm^2, and forearm BMD values of 0.53 g/cm^2 ± 0.05 g/cm^2. Goebel et al. 1999 reported lumbar spine BMD of ANR, ANBP, and EDNOS/AN inpatients as 1.00 g/cm^2 ± 0.13 g/cm^2, 0.95 g/cm^2 ± 0.12 g/cm^2, and 1.06 g/cm^2 ± 0.12 g/cm^2, respectively. Similar lumbar spine values have been reported by researchers in numerous other studies (Baker et al., 2000; Bolton et al., 2005). Although forearm measurements are not taken as frequently as hip and spine measurements in persons with low BMD, the location was included in the current study, as it has been indicated as a common fracture site (Lucas et al., 1999). Overall, the pre-test BMD values of the participants were comparable to other research samples.

Nutrition information. Experimental group participants in the current study consumed an average of 1,638 kcal ± 515 kcal per day, while the control group had an average daily intake of 2,381 kcal ± 959 kcal. The daily caloric intake by the experimental group closely resembles intake of community-dwelling adolescents with AN as reported by Misra et al. (2006). These authors found that community-dwelling adolescents with AN consumed significantly less calories than healthy controls. Calories consumed by the females with AN in their study averaged 1,649 kcal ± 110 per day as

compared to 1,970 kcal ± 91 consumed by the healthy adolescents. Caloric intake of participants in Szabo and Green's (2002) resistance training investigation averaged 2,500 kcal per day. However, it is important to note that these participants were residents at an impatient treatment center where food intake was regulated.

Furthermore, calcium intake by the experimental group in the current investigation averaged 867 mg/day ± 338 mg/day per day, and 1,096 mg/day ± 448 mg/day per day for by the control group. Recommended daily calcium intake is between 800mg/day and 1,500 mg/day for women over the age of 18 years (NIH, 2005b). Many specialists recommend a daily calcium intake of 1,300 mg/day to 1,500 mg/day for females with AN (Golden, 2003).

Misra et al. (2006) described calcium intake by adolescents with AN (1,446 mg/day ± 99 mg/day) that was significantly higher than calcium intake by healthy controls (1,060 mg/day ± 92 mg/day) due to supplement use. Without calcium supplementation, the average intake was 981 mg/day ± 88 mg/day, closely resembling the intake values of participants in the current study. Calcium supplements were not taken by any participants in the current sample, nor was calcium intake correlated with any of the variables explored in the current study.

Overall, average caloric intake by the participants in the current study fell between caloric intakes by inpatients and caloric intake by community-dwelling females with AN. Furthermore, average calcium intake was just within the recommended daily allowance, but also representative of intake by other outpatient females with AN.

Psychological test scores. The average BDI-II score for the current sample was 23 ± 12. Goebel et al. (1999) reported average BDI scores of inpatients with EDs upon admission which ranged from 21 ± 13 to 26 ± 12 for ANR, ANBP, and EDNOS/AN. The EDI-3 EDRC *t*-score for the current sample averaged 48 ± 11, which falls within the typical clinical range. EDI scores are reported in a variety of ways in ED studies, including raw score per scale, total raw score, and *t*-scores. The EDI scores of the participants in this intervention are within the typical range of other research samples (Dare, Chania, Eisler, Hodes, & Dodge, 2000; Bizeul, Brun, & Rigaud, 2003; Garner et al., 1983). Bizeul et al. reported that depression influences EDI scores. Indeed, among the current sample, there was a significant correlation between BDI-II and EDI-3 scores ($r = .68, p = .008$). The current sample exhibited similar scores on the BDI-II and the EDI-3 to those reported by Bizeul et al. Therefore, the participants in the present study are similar to participants in other research as well comparable to individuals in ED treatment with respect to depression and eating disordered tendency measures.

Summary. Based on the above information, the current sample is representative of females with AN in both the research population and the general population with respect to sample size, age, years since diagnosis, pretest BMD values, caloric and calcium intake, and psychological test scores. However, the participants in the present study had slightly higher anthropometric measures when compared to participants in inpatient studies.

Bone Mineral Density Results

In the present study, participants in the experimental group did not have statistically higher BMD of the hip, forearm, or spine than the control group following the intervention. Post-intervention, individuals in the experimental group had an average hip BMD of 0.86 g/cm^2 ± 0.10 g/cm^2, lumbar spine BMD of 1.02 g/cm^2 ± 0.12 g/cm^2, and forearm BMD of 0.51 g/cm^2 ± 0.03 g/cm^2. In contrast, the control group had hip, lumbar spine, and forearm BMD values of 0.88 g/cm^2 ± 0.10 g/cm^2, 0.97 g/cm^2 ± 0.08 g/cm^2, and 0.54 g/cm^2 ± 0.04 g/cm^2, at posttest, respectively.

There are a few possibilities why significance was not attained. The sample size may have been too small resulting in low to moderate statistical power. In addition, the posttest scans performed on the participants in the experimental group may have taken place during a time of bone remodeling. During this process, osteonal canals are temporarily much larger than inactive osteons (Rauch & Schoenau, 2001). Relative cortical bone volume is smaller when intracortical remodeling activity is high, thus leading to a decrease in BMD (Rauch & Schoenau). Finally, DXA technology does not discriminate between cortical and trabecular bone, which have different rates of remodeling (Rauch & Schoenau). Quantitative computed tomography (QCT) has the ability to measure both cortical and trabecular bone at any site of the skeleton, regardless of bone shape, and may be able to distinguish differences in the phases of remodeling between both of these compartments (Rauch & Schoenau). QCT may be beneficial in future research to determine the rates of remodeling of both cortical and trabecular bone.

Although the BMD changes at each site did not reach statistical significance, the percentage change in BMD at each site between the groups can offer information regarding the clinical significance of the intervention. The experimental group had an increase in lumbar spine BMD of 1.50% ± 2.4%, an increase in hip BMD of .90% ± 2.12%, and an increase in forearm BMD of 1.03% ± 3.38%. In contrast, the control group showed an average change of 0.00% ± 1.82% in lumbar spine BMD, a decrease of 1.07% ± 1.88% in hip BMD, and an average increase of .94% ± 3.38% in forearm BMD. Therefore, there was an overall difference between the groups of 1.5% in BMD values of the lumbar spine, 2.05% in hip BMD values, and 0.09% in forearm BMD values. The percentage change shown between these two groups has implications for BMD values of the participants as they age and possibly recover from the ED. An increase in BMD greater than 1.0% over a period of 9 months is considered clinically significant, considering that the average rate of loss after attainment of peak BMD in early adulthood is 0.5-1.0% per year (Shaw & Witzke, 1998; Shaw & Snow, 1995).

Fracture risk. The development of osteopenia and osteoporosis in individuals with EDs is believed to be a result of two major mechanisms. First, due to the early onset of many EDs, peak bone mass is often not realized. Peak bone mass achieved at the end of puberty or by early adulthood is a major determinant of future BMD, and therefore, fracture risk later in life (Lucas et al., 1999; Zipfel, Herzog, Beumont, & Russell, 2000). Furthermore, there is premature and increased bone damage in people diagnosed with EDs due to nutritional and hormonal influences (Zipfel et al.).

In AN, there is low bone turnover due to decreased bone formation and increased bone resorption. Thus, an increase in bone formation along with a decrease in bone resorbtion is necessary to increase BMD in these individuals (Lennkh et al., 1999; Treasure & Serpell, 2001). This imbalanced pattern leads to rapid and significant bone loss, often within 6 months to 1 year of disease onset (Grinspoon et al., 2000; Lennkh et al.). Fracture risk among these individuals increases for each standard deviation decrease in BMD (Vestergaard et al., 2003).

The criteria for osteopenia is a *t*-score between -1 and -2.5 in adult women. Osteopenia was seen in at least one site in 10 out of the 14 study participants. Participants in the current study had average *t*-scores of -0.55 ± .84 for the hip and -0.54 ± 1.15 for the spine. In addition, the average *t*-score for forearm BMD among the participants was -1.11 ± .74, therefore increasing fracture risk. Furthermore, the average time since diagnosis among the current sample was 5.75 years ± 4.19 years. Fractures typically occur 7 years -10 years after diagnosis of AN and are responsible for a significant percentage of the hospitalization for chronic osteoporosis (Jagielska et al., 2002; Zipfel et al., 2000). Because 71% of the current sample met the criteria for osteopenia during the intervention, the chance of fracture among these individuals is likely if they follow the typical pathological course of AN or EDNOS/AN.

Unfortunately, fracture risk remains high for people in recovery for EDs. Vestergaard et al. (2003) found in a Danish nationwide register study of persons with EDs that fracture risk among people diagnosed with AN increased 160% after diagnosis. Furthermore, this risk of fracture remained more than 10 years afterwards. Similarly,

Hartman et al. (2000) examined BMD in 19 women who had been fully recovered from AN for an average of 21 years and found that femur BMD was still significantly less than that of healthy controls. In addition, Lucas et al. (1999) indicated that forearm, spine, and hip fractures occurred an average of 24 years, 25 years, and 38 years, respectively, after diagnosis. Cumulatively, the incidence of any fracture 40 years after the diagnosis of AN was 57%. These results suggest that even with full clinical recovery from AN, BMD is not restored to a normal level.

Mechanics of bone overload. Bone responds to alterations in mechanical forces. These mechanical loads may be the only type of stimuli capable of inducing modeling in mature bone (Shaw & Witzke, 1998). In addition, magnitude of mechanical force has been shown to be more important for skeletal modeling than the number of repetitions or load cycles (Shaw & Witzke).

Resistance training is one strategy to increase BMD by means of mechanical force as well as reduce the risk of fractures in persons diagnosed with osteopenia and osteoporosis. Researchers vary on length of time and type of mechanical stress to place on the bone in order to invoke modeling. A common consensus among researchers is that several months are needed before changes in BMD due to a resistance training intervention can be detected in a healthy population (Friedlander, Genant, Sadowski, Byl, & Glüer, 1995; Lohman et al., 1995; Snow-Harter, Bouxsein, Lewis, Carter, Marcus, 1992; Warner & Shaw, 2000).

However, the typical length of time required for change in BMD does not necessarily apply to persons who are already deficient. A number of exercise

intervention studies have demonstrated significant gains in BMD of people with osteopenia and osteoporosis (e.g. Heinonen et al., 1996; Kemmler et al., 2004; Preisinger et al., 1996). Due to the lower BMD of these individuals, they have potential for increased skeletal adaptation due to mechanical loading, as they are capable of greater gains in a shorter period of time (Warner & Shaw, 2000). Beverly et al. (1989) conducted a study of 99 post-menopausal women, 30 with a fractured forearm. The participants were asked to squeeze a tennis ball three times a day for 6 weeks using their uninjured arm. Bone mineral content increased significantly as a result of this exercise. Based on these data, an intervention period of 10 weeks should be sufficient to generate change in BMD. As documented, the current 10 week training protocol resulted in clinically relevant changes in BMD. This is a promising outcome deserving of further research.

Body Composition

Body fat percentage changed significantly ($p < .001$) for the entire sample in the current study, increasing 5.05% from pre- to posttest. However, when the analysis was run at the group level, the strength-training intervention did not result in a significantly different changes in body fat percentage between the experimental and control groups. The lack of significance at the group level may reflect a decrease in power due to the sample size. However, though not statistically significant, the increase in body fat percentage within each group has clinical implications, as a main goal of treatment for individuals with AN and EDNOS/AN is an increase in body mass, including FM and FFM.

Generally, exercise interventions focus on aerobic exercise, which commonly changes BC due to a decreased amount of FM. However, traditional aerobic interventions are not appropriate for this population. The few interventions with both inpatients and outpatients in this population that have shown changes in BC often contained a combination of stretching, yoga, mind-body exercises, and light aerobic exercise (Calogero & Pedrotty, 2004; Thien et al., 2000). Even less research exists on the effects of resistance training as an exercise intervention on BC among persons with EDs. Generally, participating in a resistance training program increases body mass and FFM, resulting in a decrease in body fat percentage (Kraemer, Volek, & Fleck, 1998; 2000). These BC changes have been seen among a healthy population in short-term resistance training programs ranging from 6 weeks – 24 weeks (Kraemer & Fleck, 1987).

Changes in BC due to an exercise intervention in a population with EDs have been seen in as little as 8 weeks. Unlike the effects in a healthy population, researchers have shown that individuals with EDs gain fat in addition to FFM and total body mass after participating in a resistance training program (Chantler et al., 2005; Szabo & Green, 2002; Thien et al., 2000). The experimental group in the current study showed an increase in body fat percentage of 4.39%, while the control group gained an average of 5.59% body fat. As both groups continued with their regular treatment, this gain in body fat percentage may be due to further normalization of weight and caloric intake among the participants. Many researchers have found that individuals with AN tend to gain a significant amount of fat with weight restoration (Boschi et al., 2003; Grinspoon et al.,

2001; Iketani et al., 1999; Orphanidou et al., 1997; Polito et al., 1998; Salisbury, Levine, Crow, & Mitchell, 1995; Scalfi et al., 2002).

Szabo and Green (2002) examined the effects of an 8 week strength training program on inpatient females with AN. The study consisted of three groups of 7 participants each: exercisers with AN, exercisers without AN, and a control group of females with AN. Exercisers with AN, as well as the control group with AN, experienced significant increases in body mass, BMI, body fat percentage, FM, and FFM. The experimental and control groups in the current study also experienced increases in body mass and body fat, however, the changes did not reach statistical significance. In contrast with Szabo and Green, LBM also decreased. This reduction in LBM may have been due to the low caloric intake of the participants. Individuals participating in the strength training program had an average daily caloric intake of 1,638 kcal (± 51). Conversely, the group of inpatient exercisers with AN in the study by Szabo and Green had a daily caloric intake of 2,500 kcal. It is possible that the participants in the current study were not consuming enough calories to support an increase in LBM due to the resistance training program.

Cardiovascular exercise was controlled for in the present study by randomization of participants. Individuals were instructed to continue with their regular routine if they exercised aerobically. If the participants were not currently performing any cardiovascular exercise at the time of the intervention, they were instructed not to begin. Therefore, any decrease in FM associated with cardiovascular activity should have been controlled. In addition, participants were not asked to record cardiovascular exercise due

to the concern that this request would have them focus on an activity that is often used in a negative fashion for weight loss among this population.

The impact of the intervention on BC in the experimental group of the current study can be seen in the amount of body fat that was gained in comparison to LBM. Both the groups experienced a loss of LBM. However, the individuals who took part in the strength training program lost 4% of LBM, while the control group that was not strength training lost 6% of LBM. Therefore, participants in the experimental group were able to maintain 2% more of their LBM than participants in the control group. In addition, even with a caloric deficit, it is important to note that participation in this exercise program did not lead to weight loss in the experimental group.

The results of the current study support previous exercise intervention research on females with ED that has shown that participation in strength training can lead to an increase in body mass as well as FM. Consequently, a resistance training intervention can be used in conjunction with refeeding and therapy to increase body weight to an acceptable level in inpatients, as well as continuing to help promote healthy BC in outpatients.

Muscular Strength

In the current study, participants in both groups experienced changes in estimated 1 RM values for chest strength and leg strength. The average chest and leg strength was significantly different between the groups. Therefore, this part of the hypothesis is supported.

Participants in the experimental group experienced an average increase of 30% in estimated chest 1RM, compared to an average increase of 3.8% in the control group. Furthermore, the average estimated leg 1RM increased by 35.9% in the experimental group, while leg strength in the control group increased by 4.7%. These increases in strength suggest that the design of the resistance training program in the current study provided sufficient overload.

Loss of muscle mass is one of the most prominent physical consequences of people with AN. This atrophy, which leads to proximal muscular weakness, is most commonly caused by skeletal muscle myopathy associated with atrophy of type 2 muscle fibers (Essén, Fohlin, Thorén, & Saltin, 1981; McLoughlin et al., 1998; 2000). Lindboe, Askevold, and Slettebø (1982) found that, when compared with healthy controls, type 1 muscle fibers were reduced by 46% and type 2 fibers by 75% in young women with AN.

The muscle myopathy associated with reduced type 2 muscle fibers has been shown to impair muscular strength among females with severe AN. Strength test values, such as maximal voluntary contraction force, are significantly less than predicted values in women with AN (Einerson, Ward, & Hanson, 1988; McLoughlin et al., 1998). Researchers are unsure if malnutrition, as seen in AN, affects the nerve fibers innervating the muscle fibers, or the muscle fibers directly (Lindboe et al., 1982). However, McLoughlin et al. (2000) reported that myopathy in AN is not secondary to neuropathy. Resistance training programs, as the one performed in the current study, have the potential to increase muscular strength among persons with EDs, thereby increasing ease

of activities of daily living as well as increasing load on the skeleton, possibly leading to an increase in BMD.

Chantler et al. (2005) demonstrated that inpatient females with AN can experience increases in strength after an 8-week strength training intervention. Participants exercised 2 times a week, for 1 hour each session, under supervision of a trained person. The exercising participants with AN experienced increases in peak torque of the knee extensors and flexors as well as the elbow flexors and extensors, suggesting that muscles of these individuals were able to respond to the training stimulus. Chantler et al. concluded that the increases in strength were due to the daily 2,500 kcal caloric intake of the participants. However, the increases in strength measures that participants in the current study experienced are in contradiction of the conclusions of Chantler et al. (2005). Despite significantly lower caloric intake by the experimental group in the current study, increases in strength were observed.

Overall, participants in the experimental group gained an average total body mass of .43 kg, while individuals in the control group gained an average of .49 kg. In addition, an increase in body fat percentage and a decrease in percentage of LBM were seen in both groups. As such, the increase in muscular strength in the control group is likely not from muscle hypertrophy, but rather from neuromuscular adaptations. Specifically, it has been documented that gains in strength during interventions of shorter duration (4 to 20 weeks), are due to neural adaptations related to learning, the ability to recruit prime movers, and coordination (ACSM, 2006; Powers & Howley, 1998). The results of the

current investigation indicate that resistance training can lead to increases in strength in females with AN despite lower caloric intake.

Depression

The strength training intervention did not have a significant effect on BDI-II scores of participants in the current investigation. Scores on the BDI-II range from 0-63, with 0-13 representing minimal depression, 14-19 indicating mild depression, 20-28 representing moderate depression, and 29-63 indicating severe depression. Of the 7 participants in the experimental group, 1 was classified as minimally depressed, 1 was classified as mildly depressed, 3 were classified as moderately depressed, and 2 fell into the severely depressed category. The participants in the control group had similar initial categorizations. After the intervention, scores on the BDI-II for participants in the experimental group improved for 4 participants, 2 participants had the same scores as pre-intervention, and 1 participant's score worsened. Individuals in the control group were comparable on posttest BDI scores as well, with 3 participants improving, 2 participants' scores remaining the same and 2 participants experiencing a worsening of scores. These findings support results described by Szabo and Green (2002), who reported that their intervention group of inpatient females with AN also experienced a non-significant decrease in BDI scores following a resistance training program.

Major depression is the most common comorbid psychiatric disorder in those with AN (Bulik, 2002). It has been reported that anywhere from 50% to 90% of people clinically diagnosed with an ED meet the clinical criteria for major depression (Herzog, 1984; Geller et al., 1998; Kaye, 1997). In addition, when individuals with EDs have been

compared with healthy controls, the rates of depression are higher in clinical groups (Powers, Schocken, & Boyd, 1998). Exercise has been shown to have positive effects on various psychological disturbances, including depression (Bosscher, 1993; Scully, Kremer, Meade, Graham, & Dudgeon, 1998).

Studies investigating the effects of exercise on depressive symptoms commonly use aerobic activity as the intervention. Various researchers have reported that depression is reduced in both men and women in all adult age groups, in community-dwelling and experimental samples, after periods of aerobic exercise (Scully et al., 1998). In addition, it has been reported that the antidepressant effect is greatest among clinical samples (Scully et al.).

Studies regarding the effect of anaerobic exercise among healthy individuals have had conflicting results. Martinsen (1990) found that non-aerobic exercise, such as resistance training, was as effective as antidepressant medication in treatment of depressive symptoms in a 1 year program. Furthermore, participants who continued to exercise after termination of the program had lower depression scores than sedentary controls. North et al. (1990) reported that both aerobic and non-aerobic exercise were effective antidepressants. The greatest improvements in depression from both aerobic and anaerobic exercise were found after an average of 17 weeks of participation, although effects have been reported from 4 weeks on (Scully et al., 1998). Furthermore, it was suggested that the greater the number of exercise sessions, the greater the decrease in depressive symptoms. However, Scully et al. concluded that additional research needed

to be done focusing on the benefits of anaerobic exercise, given that the results of many studies have not indicated a positive effect.

As improvements in depressive symptoms occur on average 17 weeks after beginning aerobic and/or anaerobic exercise, the intervention utilized in the current study may not have been long enough to result in a significant decrease in scores. In addition, studies (Folkins & Sime, 1981) have indicated no significant effect on depression due to strength training interventions. Furthermore, anorexia often presents with various comorbities in addition to depression. Therefore, it is possible that additional confounding variables were present during the course of the study, inhibiting improvements in depressive symptoms.

However, the decrease in scores on the BDI-II by 4 out of the 7 participants in the experimental group is of clinical importance. A reduction in depressive symptoms may allow individuals in treatment to address additional issues essential for earlier recovery. Further research, perhaps with a larger sample size and a longer intervention time, may show additional decreases in depressive symptoms participants.

Eating Disordered Tendencies

In the current study, participants in the experimental group did not experience significantly greater decreases in EDI-3 *t*-scores than those in the control group. However, the decrease in *t*-scores may be of clinical significance. The *t*-score of interest represents the EDRC, which is comprised of three subscales: Drive for Thinness, Bulimia, and Body Dissatisfaction. A score of 42 or under represents the low clinical range, 43 to 55 represent the typical clinical range, and scores over 56 denote an elevated

clinical range. The overall average EDI-3 EDRC *t*-score for the intervention group decreased from 47 ± 12 to 43 ± 21 following the intervention. These results support findings by Szabo and Green (2002), who also found a general, but non-significant, decrease in all of the EDI sub-scale scores among inpatients participating in a strength training intervention. It is important to note that improvements in inpatient EDI scores may not necessarily correlate with improvements in scores of outpatients due to differences in length of time in treatment, years since diagnosis, and the autonomy associated with participating in an outpatient program. The increase or stability of the scores in the current sample indicate that a resistance training program may not have deleterious effects on ED symptomology and could in fact improve symptoms over time.

The effects of strength training on body dissatisfaction in healthy females with body image issues have also been studied. Body image is often disturbed in persons with EDs and body dissatisfaction is a subscale of the EDI-3 EDRC. In 2004, Depcik and Williams examined the impact of a weight training intervention on body-image-disturbed college females. After 13 weeks of training, 41% of the participants experienced improvements in body image so great that they were no longer classified as body-image-disturbed. Williams and Cash (2001) found that a 6 week circuit training program improved body satisfaction and evaluations of physical appearance in college students. Clearly, participating in a resistance training program has the potential to improve eating disordered tendencies, including body image, among both inpatient and outpatient groups. Additional research with larger samples of outpatients is encouraged.

Implications of the Study and Overall Conclusions

Participants in the current study had 100% adherence to the resistance training intervention. In addition, no ill effects such as weight loss, injury, or increase of deleterious psychological issues occurred during the study. Furthermore, significant increases in strength as well as clinically relevant increases in BD and BMD were found. In addition, clinically significant decreases in eating disordered symptoms and depression were observed.

This study demonstrates that participation in a supervised resistance training program can have valuable clinical significance regarding the treatment of outpatient females with EDs. Although outpatients do not always conform to all components of a treatment program, support by the rest of the treatment team to begin resistance training exercise may make the individual more compliant with other components of treatment. In addition, participation in this type of exercise program can be a first step in teaching an individual healthy exercise habits. Due to the lower caloric expenditure of strength training, there is less of a chance that the individual will use this mode of exercise in a purgative fashion. Furthermore, the positive physiological and psychological results from such a program may expedite recovery and possibly hinder relapse.

A need exists for additional studies examining the effects of weight training on BMD, BC, strength, depression, and eating disordered tendencies in both inpatient and outpatient females diagnosed with AN and EDNOS/AN. To our knowledge, this study is the first of its kind. Further investigation is warranted to determine the optimal length of the program, the number of days per week, the percentage of estimated 1RM to lift, and

the number of sets and repetitions for each exercise that would improve the variables examined in the current study to a beneficial level for each individual. After a period of time, even with the stimulus of mechanical loading, increases in BMD and strength will plateau (Warner & Shaw, 2000). Therefore, investigating the proper maintenance workout program to keep BMD values at the highest level possible is also recommended. Future research is also necessary to determine how long the positive effects last after cessation of structured resistance training, as once the loading stimulus is removed, BMD and strength decrease. Investigations of the length and structure of a strength training program necessary to maintain or improve psychological health are also encouraged. For example, investigation of specific subscales of the EDI-3 EDRC, rather than overall *t*-test scores, may be beneficial; specifically changes in the Body Dissatisfaction subscale. Finally, treatment for EDs will be advanced by determining the effectiveness of using strength training in conjunction with inpatient and outpatient treatment programs.

There is an crucial need for effective interventions to treat the growing number of people who suffer from EDs. For some individuals, the ability to participate in any exercise during treatment can lead to increased autonomy and compliance with the program. Furthermore, physical training may be beneficial in helping to increase strength, BMD and FFM, thereby increasing the ability to perform activities of daily living, decreasing the risk of osteopenia and osteoporosis, and increasing overall body mass. Resistance training may also assist in the development and maintenance of a positive body image, as well as reduce rates of depression and eating disordered

tendencies. In addition, participation in an exercise program during treatment may be an effective way of teaching persons with EDs lifelong healthy patterns of physical activity.

The current study demonstrated that community-dwelling females with EDs can participate in resistance training exercise with no deleterious physiological or psychological effects. The significant increases in strength, the clinically relevant increases in BMD and body fat percentage, and the deceases in depression and eating-disordered tendencies shown in this study address several issues that are commonly focused on during treatment. While exercise among patients with AN has generally been a sensitive subject and rarely allowed during treatment, this investigation shows that the addition of an appropriate exercise program can be a beneficial addition to the course of recovery in an outpatient program.

REFERENCES

Abrams S. A., Silber, T. J., Esteban, N. V., Vieira, N. E., Stuff, J.E., Meyers, R., et al. (1993). Mineral balance and bone turnover in adolescents with anorexia nervosa. *The Journal of Pediatrics, 123*, 326-331.

Academy for Eating Disorders. (n.d.a.). *Consequences of eating disorders.* Retrieved May 14, 2005 from, http://www.aedweb.org/eating_disorders/consequences.cfm

Academy for Eating Disorders. (n.d.b.). *Course and outcomes of eating disorders.* Retrieved May 14, 2005 from, http://www.aedweb.org/eating_disorders/outcomes.cfm

Academy for Eating Disorders. (n.d.c.). *Prevalence of eating disorders.* Retrieved May 14, 2005 from, http://www.aedweb.org/eating_disorders/prevalence.cfm

American College of Sports Medicine. (2000). *ACSM's guidelines for exercise testing and prescription* (6th ed.). Philadelphia: Lippincott, Williams & Wilkins.

American College of Sports Medicine. (2006). *ACSM's guidelines for exercise testing and prescription* (7th ed.). Philadelphia: Lippincott, Williams & Wilkins.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Attia, E., Haiman, C., Walsh, B. T., & Flater, S. R. (1998). Does fluoxine augment the inpatient treatment of anorexia nervosa? *American Journal of Psychiatry, 155,* 548-551.

Bachrach, L. K., Guido, D., Katzman, D., Litt, I. F., & Marcus, R. (1990). Decreased bone density in adolescent girls with anorexia nervosa. *Pediatrics, 86,* 440-447.

Bachrach, L., Katzman, D., Litt, I., Guido, D., & Marcus, R. (1991). Recovery from osteopenia in adolescent girls with anorexia nervosa. *Journal of Clinical Endocrinology and Metabolism, 72,* 602-606.

Baker, D., Roberts, R., & Towell, T. (2000). Factors predictive of bone mineral density in eating-disordered women: A longitudinal study. *The International Journal of Eating Disorders, 27*, 29-35.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory – II manual.* San Antonio: Harcourt Assessment.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*, 77–100.

Bemben, D., Buchanan, T., Bemben, M., & Knehans, A. (2004). Influence of type of mechanical loading, menstrual status, and training season on bone density in young women athletes. *Journal of Strength and Conditioning Research, 18*, 220-226.

Beumont, P., Arthur, B., Russell, J., & Touyz, S. (1994). Excessive physical activity in dieting disorder patients: Proposals for a supervised exercise program. *International Journal of Eating Disorders*, *15*, 21-36.

Beverly, M. C.., Rider, T. A., Evans, M. J., & Smith, R. (1989). Local bone mineral response to brief exercise that stresses the skeleton. *British Medical Journal*, *299*, 233-235.

Biller, B., Saxe, V., Herzog, D., Rosenthal, D., Holzman, S., & Klibanski, A. (1989). Mechanisms of osteoporosis in adult and adolescent women with anorexia nervosa. *Journal of Clinical Endocrinology and Metabolism, 68,* 548-554.

Bizeul, C., Brun, J. M., & Rigaud, D. (2003). Depression influences the EDI scores in anorexia nervosa patients. *European Psychiatry, 18,* 119-123.

Bolton, J. G. F., Patel, S., Lacey, J. H., & White, S. (2005). A prospective study of the changes in bone turnover and bone density associated with regaining weight in women with anorexia nervosa. *Osteoporosis International, 16,* 1955-1962.

Boschi, V., Siervo, M., D'Orsi, P., Margiotta, N., Trapanese, E., Basile, F. et al. (2003). Body composition, eating behavior, food-body concerns and eating disorders in adolescent girls. *Annals of Nutrition & Metabolism, 47,* 284-293.

Brooks, E. R., Cavalier, D. S., & Howat, P. M. (1999). Calcium supplementation and exercise increase appendicular bone density in anorexia: A case study. *Journal of the American Dietetic Association, 99,* 591-594.

Brotman, A. W., & Stern, T. A. (1985). Osteoporosis and pathologic fractures in anorexia nervosa. *American Journal of Psychiatry, 124,* 495-496.

Brzycki, M. (1993). Strength testing – Predicting one-rep max from reps-to-fatigue. *Journal of Physical Education, Recreation, and Dance, 64,* 88-90.

Bulik, C. M. (2002). Anxiety, depression, and eating disorders. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity* (pp. 194-195). New York: The Guilford Press.

Calogero, R. M., & Pedrotty, K. N. (2004). The practice and process of healthy exercise: An investigation of the treatment of exercise abuse in women with eating disorders. *Eating Disorders, 12*, 273-291.

Carmichael, K. A., & Carmichael, D. H. (1995). Bone metabolism and osteopenia in eating disorders. *Medicine, 74*, 254-268.

Carruth, B. R., & Skinner, J. D. (2000). Bone mineral status in adolescent girls: Effects of eating disorders and exercise. *Journal of Adolescent Health, 26*, 322-329.

Casper, R. C. (1998). Depression and eating disorders. *Depression and Anxiety, 8,* S96-S104.

Chantler, I., Szabo, C. P., & Green, K. (2005). Muscular strength changes in hospitalized anorexic patients after an eight week resistance training program. *International Journal of Sports Medicine, 26,* 1-6.

Crago, M., Shisslak, C. M., & Estes, L. S. (1996). Eating disturbances among American minority groups: A review. *International Journal of Eating Disorders, 19,* 239-248.

Daee, A., Robinson, P., Lawson, M., Turpin, J. A., Gregory, B., & Tobias, J. D. (2002). Psychologic and physiologic effects of dieting in adolescents. *Southern Medical Journal, 95*, 1032-1042.

Dare, C., Chania, E., Eisler, I., Hodes, M., & Dodge, E. (2000). The eating disorder inventory as an instrument to explore change in adolescents in family therapy for anorexia nervosa. *European Eating Disorders Review, 8*, 369-383.

Davies, C. T. M., Von Dobeln, W., Fohlin, L., Freychuss, U., & Thoren, C. (1978). Total body potassium, fat-free weight and maximal aerobic power in children with anorexia nervosa. *Acta Paediatrica Scandinavica, 67*, 229-234.

Davies, K., Pearson, P., Huseman, C., Greger, N., Kimmel, D., & Recker, R. (1990). Reduced bone mineral in patients with eating disorders. *Bone, 11*, 143-147.

Depcik, E., & Williams, L. (2004). Weight training and body satisfaction of body-image-disturbed college women. *Journal of Applied Sport Psychology, 16,* 287-299.

Deter, H. C., & Herzog, W. (1994). Anorexia nervosa in a long term perspective: Results from the Heidelberg-Mannheim study. *Psychosomatic Medicine, 56,* 20-27.

Eddy, K. T., Keel, P. K., Dorer, D. J., Delinsky, S. S., Franko, D. L., & Herzog, D. B. (2002). Longitudinal comparison of anorexia nervosa subtypes. *International Journal of Eating Disorders, 31*, 191-201.

Einerson, J., Ward, A., & Hanson, P. (1988). Exercise responses in females with anorexia nervosa. *International Journal of Eating Disorders, 7,* 253-260.

Essén, B., Fohlin, L., Thorén, C., & Saltin, B. (1981). Skeletal muscle fiber types and sizes in anorexia nervosa patients. *Clinical Physiology, 1,* 395-403.

Falk, J. R., Halmi, K. A., & Tyron, W. W. (1985). Activity measures in anorexia nervosa. *Archives of Psychiatry, 42,* 811-814.

Fairburn, C. G. (2001). Atypical eating disorders. In C. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive textbook* (2nd ed.). New York: Guilford Press.

Fairburn, C., & Harrison, P. (2003). Eating disorders. *The Lancet, 361*, 407-416.

Ferguson, C. P., LaVia, M. C., Crossan, P. J., & Kaye, W. H. (1999). Are serotonin selective reuptake inhibitors effective in underweight anorexia nervosa? *International Journal of Eating Disorders, 25,* 11-17.

Fisher, M., Golden, N. H., Katzman, D. K., Kreipe, R. E., Rees, J., Schebendach, J., et al. (1995). Eating disorders in adolescents: A background paper. *Journal of Adolescent Health, 16,* 420-437.

Fogelholm, G. M., Kukkonen-Harjula, T. K., Sievänen, H. T., Oja, P., & Vuori, I. M. (1996). Body composition assessment in lean and normal-weight young women. *British Journal of Nutrition, 75,* 793-802.

Fohlin, L. (1977). Body composition, cardiovascular and renal function in adolescent patients with anorexia nervosa. *Acta Paediatrica Scandinavica, 268,* 7-20.

Folkins, C., & Sime, W. (1981). Physical fitness training and mental health. *The American Psychologist, 36,* 373-389.

Forbes, G. B., Kreipe, R. E., Lipinski, B. A., & Hodgman, C. H. (1984). Body composition changes during recovery from anorexia nervosa: Comparisons of two dietary regimes. *American Journal of Clinical Nutrition, 40,* 1137-1145.

Frey, J., Hebebrand, J., Muller, B., Ziegler, A., Blum, W. F., Remschmidt, H., et al. (2000). Reduced body fat in long-term follow-up female patients with anorexia nervosa. *Journal of Psychiatric Research, 34,* 83-88.

Garner, D. M. (2006). *Eating Disorder Inventory – 3 professional manual.* Lutz, FL: Psychological Assessment Resources, Inc.

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional Eating Disorders Inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2,* 15-34.

Geller, J., Johnston, C., Madsen, K., Goldner, E. M. Remick, R. A., & Birmingham, C. L. (1998). Shape- and weight-based self-esteem and the eating disorders. *International Journal of Eating Disorders, 24,* 285-298.

Giannini, A. J., Newman, M., & Gold, M. (1990). Anorexia and bulimia. *American Family Physician, 41*, 1169-1177.

Giordano, S. (2005). Risk and supervised exercise: The example of anorexia to illustrate a new ethical issue in the traditional debates of medical ethics. *Journal of Medical Ethics, 31,* 15-20.

Goebel, G., Schweiger, U., Krüger, R., & Fichter, M. M. (1999). Predictors of bone mineral density in patients with eating disorders. *International Journal of Eating Disorders, 25*, 143-150.

Golden, N. H. (2003). Osteopenia and osteoporosis in anorexia nervosa. *Adolescent Medicine, 14,* 97–108.

Golden, N., & Sacker, I. M. (1984). An overview of the etiology, diagnosis and management of anorexia nervosa. *Clinical Pediatrics, 23*, 209-214.

Grinspoon, S., Baum, H., Kim, V., Coggins, C., & Klibanski, A. (1995). Decreased bone formation and increased mineral dissolution during acute fasting in young women. *Journal of Clinical Endocrinology and Metabolism, 80,* 3628-3633.

Grinspoon, S., Thomas, L., Miller, K., Pitts, S., Herzog, D., & Klibanski, A. (2001). Changes in regional fat distribution and the effects of estrogen during spontaneous weight gain in women with anorexia nervosa. *American Journal of Clinical Nutrition, 73,* 865-869.

Grinspoon, S., Thomas, E., Pitts, S., Gross, E., Mickley, D., Miller, K., et al. (2000). Prevalence and predictive factors for regional osteopenia in women with anorexia nervosa. *Annals of Internal Medicine, 133,* 790-794.

Halmi, K., Eckert, E., Marchi, P., Sampugnaro, V., Apple, R., & Cohen, J. (1991). Comorbidity of psychiatric diagnoses in anorexia nervosa. *Archives of General Psychiatry, 48,* 712-718.

Hartman, D., Crisp, A., Rooney, B., Rackow, C., Atkinson, R., & Patel, S. (2000). Bone density of women who have recovered from anorexia nervosa. *International Journal of Eating Disorders, 28,* 107-112.

Heer, M., Mika, C., Grzella, I., Heussen, N., & Herpertz-Dahlmann, B. (2004). Bone turnover during inpatient nutritional therapy and outpatient follow-up in patients with anorexia nervosa compared with that in healthy control subjects. *American Journal of Clinical Nutrition, 80,* 774-782.

Heinonen, A., Kannus, P., Sievanen, H., Oja, P., Pasanen, M., Rinne, M., et al. (1996). Randomized controlled trail of effect of high-impact exercise on selected risk factors for osteoporotic fractures. *The Lancet, 348,* 1343-1348.

Hergenroeder, A., Smith, E., Shypailo, R., Jones, L., Klish, W., & Ellis, K. (1997). Bone mineral changes in young women with hypothalamic amenorrhea treated with oral contraceptives, medroxyprogesterone, or placebo over 12 months. *American Journal of Obstetrics And Gynecology, 176,* 1017-1025.

Herzog, W., Minne, H., Deter, C., Leidig, G., Schellberg, D., Wüster, C., et al. (1993). Outcome of bone mineral density in anorexia nervosa patients 11.7 years after first admission. *Journal of Bone and Mineral Research, 8,* 597-605.

Heymsfield, S. B., Olafson, R. P., Kunter, M. H., & Nixon, D. W. (1979). A radiographic method of quantifying protein-calorie undernutrition. *American Journal of Clinical Nutrition, 32,* 693-702.

Heyward, V. H. (Ed.). (1998). *Advanced fitness assessment and exercise prescription.* Champaign, IL: Human Kinetics.

Heyward, V. H., & Stolarczyk, L. M. (Eds.). (1996). *Applied body composition assessment.* Champaign, IL: Human Kinetics.

Hoelscher, D. M., Day, R. S., Kelder, S. H., Hergenroeder, A. C., Ward, J. L. (March 2003). Implementation and evaluation of a school-based osteoporosis prevention program: Incorporating More Physical Activity and Calcium in Teens (IMPACT) Study. *Abstract presented at the Annual Meeting of the Society of Behavioral Medicine.*

Holtkamp, K., Muller, B., Heussen, N., Remschmidt, H., & Herpertz-Dahlmann, B. (2005). Depression, anxiety, and obsessionality in long-term recovered patients with adolescent-onset anorexia nervosa. *European Child & Adolescent Psychiatry, 32,* 693-702.

Iacopino, L., Siani, V., Melchiorri, G., Orlandi, C., De Luna, A., Cervelli, V., et al. (2003). Body composition differences in adolescent female athletes and anorexic patients. *Acta Diabetologica, 40*, S180-S182.

Iketani, T. Kiriike, N., Nagata, T., & Yamagami, S. (1999). Altered body fat distribution after recovery of weight in patients with anorexia nervosa. *International Journal of Eating Disorders, 26,* 275-282.

Iketani, T., Kiriike, N., Nakanishi, S., & Nakasuji, T. (1995). Effects of weight gain and resumption of menses on reduced bone density in patients with anorexia nervosa. *Biological Psychiatry*, *37*, 521-527.

Jackson, A. S., & Pollock, M. L. (1985). Practical assessment of body composition. *The Physician and Sportsmedicine, 13*, 76-90.

Jagielska, G., Wolanczyk, T., Komender, J., Tomaszewicz-Libudzic, C., Przedlacki, J., & Ostrowski, K. (2002). Bone mineral density in adolescent girls with anorexia nervosa. *European Child & Adolescent Psychiatry, 11*, 57-62.

Katzman, D. K., Christensen, B., Young, A. R., & Zipursky, R. B. (2001). Starving the brain: Structural abnormalities and cognitive impairment in adolescents with anorexia nervosa. *Seminars in Clinical Neuropsychiatry, 6,* 146-152.

Kaye, W. H. (1997). Anorexia nervosa, obsessional behavior, and serotonin. *Psychopharmacology Bulletin, 33,* 335-344.

Kemmler, W., Lauber, D., Weineck, J., Hensen, J., Kalender, W., & Engelke, K. (2004). Benefits of 2 years of intense exercise on bone density, physical fitness, and blood lipids in early postmenopausal osteopenic women. *Archives of Interval Medicine, 164,* 1084-1091.

Kerruish, K. P., O'Connor, J., Humphries, I. R. J., Kohn, M. R., Clarke, S. D., Briody, J. D., et al. (2002). Body composition in adolescents with anorexia nervosa. *American Journal of Clinical Nutrition, 75,* 31-38.

Keys, A., Brozek, J., Henschel, A., & Taylor, H. L. (1950). *The biology of human starvation.* Minneapolis: University of Minnesota Press.

Klein, D. A., & Walsh, B. T. (2003). Eating disorders. *International Review of Psychiatry, 15*, 205-216.

Klibanski, A., Biller, B., Schoenfeld, D., Herzog, D., & Saxe, V. (1995). The effects of estrogen administration on trabecular bone loss in young women with anorexia nervosa. *The Journal of Clinical Endocrinology and Metabolism*, *80*, 898-904.

Kohn, M., & Golden, N. H. (2001). Eating disorders in children and adolescents. *Pediatric Drugs, 3*, 91-99.

Kraemer, W. J., & Fleck, S. J. (1987). *Designing resistance training programs* (2nd ed.). Champaign, IL: Human Kinetics Publishers.

Kraemer, W. J., Volek, J. S., & Fleck, S. J. (1998). Chronic musculoskeletal adaptations to resistance training. In J. L. Roitman, T. LaFontaine, & M. Wegner (Eds.), *ACSM's resource manual for guidelines for exercise testing and prescription* (p. 182). Philadelphia: Lippincott, Williams & Wilkins.

Kraemer, W. J., Volek, J. S., & Fleck, S. J. (2000). Chronic musculoskeletal adaptations to resistance training. In J. L. Roitman, & T. LaFontaine (Eds.), *ACSM's resource manual for guidelines for exercise testing and prescription* (p. 180). Philadelphia: Lippincott, Williams & Wilkins.

Kreipe, R. E. (1995). Eating disorders among children and adolescents. *Pediatric Review, 16,* 370-379.

Laessle, R. G., Fischer, M., Fichter, M. M., Pirke, K. M., & Krieg, J. C. (1992). Cortisol levels and vigilance in eating disorder patients. *Psychoneuroendocrinology, 17,* 475-484.

Layne, J. E., & Nelson, M. E. (1999). The effects of progressive resistance training on bone density: a review. *Medicine & Science in Sports & Exercise, 31*, 25-30.

Lear, S. A., Pauly, R. P., & Birmingham, C. L. (1999). Body fat, caloric intake, and plasma leptin levels in women with anorexia nervosa. *International Journal of Eating Disorders, 25*, 283-288.

Lennkh, C., de Zwaan, M., Bailer, U., Strnad, A., Nagy, C., El-Giamal, N., et al. (1999). Osteopenia in anorexia nervosa: Specific mechanisms of bone loss. *Journal of Psychiatric Research, 33,* 349-356.

Lindboe, C. F., Askevold, F., & Slettebø, M. (1982). Changes in skeletal muscles of young women with anorexia nervosa. *Acta Neuropathologica, 56,* 299-302.

Lohman, T., Going, S., Pamenter, R., Hall, M., Boyden, T., Houtkooper, L., et al. (1995). Effects of resistance training on regional and total bone mineral density in premenopausal women: A randomized prospective study. *The Journal of Bone and Mineral Research, 10*, 1015-1024.

Lucas, A., Melton, L., Crowson, C., & O'Fallon, W. (1999). Long-term fracture risk among women with anorexia nervosa: a population-based cohort study. *Mayo Clinic Proceedings, 74*, 972-977.

McGilley, B. M., & Pryor, T. L. (1998). Assessment and treatment of bulimia nervosa. *American Family Physician, 57*, 2743-2751.

McIntosh, V. V. W., Jordan, J., Carter, F. A., McKenzie, J. M., Luty, S. E., Bulik, C. M. et al. (2004). Strict versus lenient weight criterion in anorexia nervosa. *European Eating Disorders Review, 12*, 51-60.

McLoughlin, D. M., Spargo, E., Wassif, W. S., Newham, D. J., Peters, T. J., Lantos, P. L., et al. (1998). Structural and functional changes in skeletal muscle in anorexia nervosa. *Acta Neuropathologica, 95*, 632-640.

McLoughlin, D. M., Wassif, W. S., Morton, J., Spargo, E., Peters, T. J., & Russell, G. F. M. (2000). Metabolic abnormalities associated with skeletal myopathy in severe anorexia nervosa. *Nutrition, 16*, 192-196.

Misra, M., Aggarwal, A., Miller, K. K., Almazan, C., Worley, M., Soyka, L. A., et al. (2004). Effects of anorexia nervosa on clinical, hematologic, biochemical, and bone density parameters in community-dwelling adolescent girls. *Pediatrics, 114,* 1574-1583.

Misra, M., & Klibanski, A. (2002). Evaluation and treatment of low bone density in anorexia nervosa. *Nutrition in Clinical Care, 5,* 298-308.

Misra, M., Soyka, L. A., Miller, K. K., Grinspoon, S., Levitsky, L. L., & Klibanski, A. (2003). Regional body composition in adolescents with anorexia nervosa and changes with weight recovery. *American Journal of Clinical Nutrition, 77,* 1361-1368.

Misra, M., Tsai, P., Anderson, E. J., Hubbard, J. L., Gallagher, K., Soyka, L. A., Miller, K. K., et al. (2006). Nutrient intake in community-dwelling adolescent girls with anorexia nervosa. *American Journal of Clinical Nutrition, 84,* 698-706.

Morris, J., Tothill, P., Gard, M., McPhail, K., Hannan, J., Cowen, S., et al. (2004). Reduced bone mineral density in bulimia as well as anorexia nervosa. *European Eating Disorders Review, 12,* 71-78.

Munoz, M. T., Morande, G., Garcia-Centenara, J. A., Hervas, F., Pozo, J., & Argente, J. (2002). The effects of estrogen administration on bone mineral density in adolescents with anorexia nervosa. *European Journal of Endocrinology, 146,* 45-50.

National Institutes of Health. (2005a, June). *What people with anorexia nervosa need to know about osteoporosis.* Retrieved June 15, 2007 from, http://www.niams.nih.gov/ bone/hi/anorexia_nervosa.htm

National Institutes of Health. (2005b, September 23). *Dietary supplement fact sheet: Calcium.* Retrieved June 14, 2007 from, http://.ods.od.nih.gov/factsheets/calcium_pf.asp

National Institutes of Mental Health. (2001). *Eating disorders: Facts about eating disorders and the search for solutions.* Retrieved May 1, 2005 from, http://www.nimh.nih.gov/publicat/eatingdisorders.cfm

Noordenbos, G., Oldenhave, A., Muschter, J., & Terpstra, N. (2002). Characteristics and treatments of patients with chronic eating disorders. *Eating Disorders, 10,* 15-29.

North, T. C., McCullagh, P., & Tran, Z. V. (1990). Effect of exercise on depression. *Exercise and Sports Science Review, (18),* 379-415.

Orphanidou, C., McCargar, L., Birmingham, C., & Belzberg, A. (1997). Changes in body composition and fat distribution after short-term weight gain in patients with anorexia nervosa. *The American Journal of Clinical Nutrition, 65,* 1034-1041.

Polito, A., Cuzzolaro, M., Raguzzini, A., Censi, L., & Ferro-Luzzi, A. (1998). Body composition changes in anorexia nervosa. *European Journal of Clinical Nutrition, 52,* 655-662.

Pollice, C., Kaye, W. H., Greeno, C. G., & Weltzin, T. E. (1997). Relationship of depression, anxiety, and obsessionality to state of illness in anorexia nervosa. *The International Journal of Eating Disorders, 21,* 367-376.

Powers, S. K., & Howley, E. T. (1998). *Exercise physiology: Theory and application to fitness and performance* (6th Ed.). Philadelphia: Lippincott Williams & Wilkins.

Powers, P. S., Schocken, D. D., & Boyd, F. R. (1998). Comparison of habitual runners and anorexia nervosa patients. *The International Journal of Eating Disorders, 23,* 133-143.

Preisinger, E., Alacamlioglu, Y., Pils, K., Bosina, E., Metka, M., Schneider, B., et al. (1996). Exercise therapy for osteoporosis: results of a randomized controlled trial. *British Journal of Sports Medicine*, *30*(3), 209-212.

Probst, M., Goris, M., Vandereycken, W., & Van Coppenolle, H. (2001). Body composition of anorexia nervosa patients assessed by underwater weighing and skinfold-thickness measurements before and after weight gain. *The American Journal of Clinical Nutrition*, *73*, 190-197.

Pulos, S. (1996). Validity of the Beck Depression Inventory with eating disorder patients. *Educational and Psychological Measurement, 56*, 139-142.

Rauch, F., & Schoenau, E. (2001). Changes in bone density during childhood and adolescence: an approach based on bone's biological organization. *Journal of Bone and Mineral Research, 16,* 597-604.

Rencken, M. L., Chesnut, C. H., & Drinkwater, B. L. (1996). Bone density at multiple skeletal sites in amenorrheic athletes. *Journal of the American Medical Association, 276*, 238-241.

Rigotti, N. A., Neer, R. M., Skates, S. J., Herzog, D. B., & Nussbaum, S. R. (1991).The clinical course of osteoporosis in anorexia nervosa: A longitudinal study of cortical bone mass. *Journal of the American Medical Association, 265,* 1133-1138.

Rigotti, N., Nussbaum, S. R., Herzog, D. B., & Neer, R. M. (1984). Osteoporosis in women with anorexia nervosa. *The New England Journal of Medicine, 311,* 1601-1605.

Romano, S. J. (2005). Anorexia nervosa. In D. J. Goldstein (Ed.), *The management of eating disorders and obesity* (pp. 64-65). Totowa, NJ: Humana Press Inc.

Salisbury, J. J., Levine, A. S., Crow, S. J., & Mitchell, J. E. (1995). Refeeding, metabolic rate, and weight gain in anorexia nervosa: A review. *International Journal of Eating Disorders, 17,* 337-345.

Scalfi, L., Polito, A., Bianchi, L., Marra, M., Caldara, A., Nicolai, E., et al. (2002). Body composition changes in patients with anorexia nervosa after complete weight recovery. *European Journal of Clinical Nutrition, 56,* 15-20.

Scully, D., Kremer, Meade, M. M., Graham, R., & Dudgeon. (1998). Physical exercise and psychological well being: a critical review. *British Journal of Sports Medicine, 32,* 111-120.

Seeman, E., Szmukler, G., Formica, C., Tsalamandris, C., & Mestrovic, R. (1992). Osteoporosis in anorexia nervosa: the influence of peak bone density, bone loss, oral contraceptive use, and exercise. *Journal of Bone and Mineral Research, 7,* 1467-1474.

Shaw, J. M., & Snow, C. (1995, September). Osteoporosis and physical activity. *PCPFS Research Digest, 2*, Series 2, Number 3. Retrieved June 1, 2007, from http://www.fitness.gov/osteoporosis.pdf

Shaw, J. M., & Witzke, K. A. (1998). Exercise for skeletal health and osteoporosis prevention. In J. L. Roitman & T. LaFontaine (Eds.), *ACSM's resource manual for guidelines for exercise testing and prescription* (pp. 288-293). Philadelphia: Lippincott, Williams & Wilkins.

Shephard, R. J. (1991). Pathological disturbances of body composition. In G. W. Lasker, C. G. N. Mascie-Taylor, & D. F. Roberts (Eds.), *Body composition in biological anthropology* (pp. 226-241). Cambridge: Cambridge University Press.

Snow-Harter, C., Bouxsein, M. L., Lewis, B. T., Carter, D. R., & Marcus, R. (1992). Effects of resistance and endurance exercise on bone mineral status of young women: A randomized exercise intervention trail. *Journal of Bone and Mineral Research, 7,* 761-769.

Soyka, L. A., Grinspoon, S., Levitsky, L. L., Herzog, D. B., & Klibanski, A. (1999). The effects of anorexia nervosa on bone metabolism in female adolescents. *The Journal of Clinical Endocrinology and Metabolism, 84*, 4489-4496.

Strober, M., Freeman, R., & Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse and outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders, 22,* 339-360.

Sundgot-Borgen, J., Rosenvinge, J. H., Bahr, R., & Schneider, L. S. (2002). The effect of exercise, cognitive therapy, and nutritional counseling in treating bulimia nervosa. *Medicine & Science in Sports & Exercise, 34*, 190-195.

Szabo, C. P., & Green, K. (2002). Hospitalized anorexics and resistance training: Impact on body composition and psychological well-being. A preliminary study. *The Journal of Eating and Weight Disorders, 7,* 293-297.

Thien, V., Thomas, A., Markin, D., & Birmingham, C. L. (2000). Pilot study of a graded exercise program for the treatment of anorexia nervosa. *International Journal of Eating Disorders, 28,* 101-106.

Toner, B. B., Garfinkel, P. E., & Garner, D. M. (1988). Affective and anxiety disorders in the long term follow-up of anorexia nervosa. *International Journal of Psychiatry in Medicine, 18,* 357-364.

Touyz, S. W., Lennerts, W., Arthur, B., & Beumont, P. J. V. (1993). Anaerobic exercise as an adjunct to refeeding patients with anorexia nervosa: Does it compromise weight gain? *European Eating Disorders Review, 1,* 177-182.

Treasure, J., Fogelman, I., & Russell, G. (1986). Osteopenia of the lumbar spine and femoral neck in anorexia nervosa. *Scottish Medical Journal, 31*, 206-207.

Treasure, J., & Serpell, L. (2001). Osteoporosis in young people. *Eating Disorders, 24*, 359-370.

Turner, H., & Bryant-Waugh, R. (2004). Eating disorder not otherwise specified (EDNOS): Profiles of clients presenting at a community eating disorder service. *European Eating Disorders Review, 12*, 18-26.

Turner, J., Bulsara, M., McDermott, B., Byrne, G., Prince, R., & Forbes, D. (2001). Predictors of low bone density in young adolescent females with anorexia nervosa and other dieting disorders. *International Journal of Eating Disorders, 30,* 245-251.

University of Maryland Medical Center. (2002). How serious is anorexia nervosa? Retrieved June 28, 2006, from, http://www.umm.edu/patiented/articles/how_serious_anorexia_nervosa_000049_5.htm

Vestergaard, P., Emborg, C., Støving, R., Hagen, C., Mosekilde, L., & Brixen, K. (2002). Fractures in patients with anorexia nervosa, bulimia nervosa, and other eating disorders - a nationwide register study. *The International Journal of Eating Disorders, 32,* 301-308.

Vuori, I. (1995). Exercise and physical health: Musculoskeletal health and functional capabilities. *Research Quarterly for Exercise and Sport, 66,* 276-286.

Wakeling, A. (1996). Epidemiology of anorexia nervosa. *Psychiatry Research, 62,* 3-9.

Warner, S. E., & Shaw, J. M. (2000). Estrogen, physical activity, and bone health. *The Journal of Physical Education, Recreation, & Dance, 71,* 19-29.

Williams, P., & Cash, T. (2001). Effects of a circuit weight training program on the body images of college students. *The International Journal of Eating Disorders, 30,* 75-82.

Wong, J. C. H., Lewindon, P., Mortimer, R., & Shepherd, R. (2001). Bone mineral density in adolescent females with recently diagnosed anorexia nervosa. *International Journal of Eating Disorders, 29,* 11-16.

Wong, S., Au, B., Lau, E., Lee, Y., Sham, A., & Lee, S. (2004). Osteoporosis in Chinese patients with anorexia nervosa. *International Journal of Eating Disorders, 36,* 104-108.

Zipfel, S., Herzog, W., Beumont, P. J., & Russell, J. (2000). Osteoporosis. *European Eating Disorders Review, 8,* 108-116.

Zipfel, S., Seibel, M. J., Lowe, B., Beumont, P. J., Kasperk, C., & Herzog, W. (2001). Osteoporosis in eating disorders: A follow-up study of patients with anorexia and bulimia nervosa. *Journal of Clinical Endocrinology and Metabolism, 86,* 5227-5233.

APPENDICES

Appendix A

Risk Stratification Questionnaire

RESISTANCE TRAINING PRE-TEST QUESTIONNAIRE

Participant Number:_____

Health Questions:

1. What year were you diagnosed with an eating disorder by a MEDICAL DOCTOR, PSYCHOLOGIST, or PSYCHIATRIST?

2. How long have you been in treatment for your eating disorder?

3. Which eating disorder(s) were you diagnosed with? *(Please circle)*

Anorexia/ Binge-Eating Purging Type Anorexia Restricting Type

EDNOS (please explain):_____

_____

_____

4. Have you ever been diagnosed with osteopenia and/or osteoporosis? If yes, when?

5. Have you been under any treatment for osteopenia/osteoporosis? If yes, what has your treatment consisted of?

6. What medications are you currently taking? (*Please specify name of medicine, purpose, and dose*)

7. Are you currently pregnant, or is there a possibility that you may be pregnant?

8. When was your last menstrual period?

9. Do you have regular menstrual periods? (Please describe: each month, every other month, not for the past 6 months, never, etc.)

Any additional questions or comments:

Thank you for your participation!

Appendix B

University Institutional Review Board Letter

Office of Compliance
Business and Aerospace Building S245
Middle Tennessee State University
Murfreesboro, Tennessee 37132
Office: (615) 494-8918 • Fax: (615) 904-8020
www.mtsu.edu/~research/compliance.html

MIDDLE TENNESSEE STATE UNIVERSITY

July 7, 2006

Protocol Title: Effects of a 12-week strength training intervention among community dwelling eating disordered females
Protocol Number: 06-281
gls2d@mtsu.edu

Dear Gina Sobrero Evans,

The MTSU Institutional Review Board has reviewed the research proposal identified above. Approval is granted for one (1) year from the date of this letter for 40 participants.

Please note that any unanticipated harms to participants or adverse events must be reported to the Office of Compliance at (615) 494-8918.

You will need to submit an end-of-project report to the Office of Compliance upon completion of your research. You will need to submit a progress report before requesting an extension.

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

According to MTSU Policy, a researcher is defined as anyone who works with data or has contact with participants. Anyone meeting this definition needs to be listed on the protocol and needs to provide a certificate of training to the Office of Compliance. If you add researchers to an approved project, please forward an updated list of researchers and their certificates of training to the Office of Compliance before they begin to work on the project.

All research materials must be retained by the PI or faculty advisor (if the PI is a student) for at least three (3) years after study completion and then destroyed in a manner that maintains confidentiality and anonymity.

Sincerely,

William Langston
MTSU Institutional Review Board

Appendix C

Informed Consent

Principal Investigator: Gina S. Evans
Study Title: Effects of a 10-Week Strength Training Intervention Among Community-Dwelling Eating Disordered Females
Institution: Middle Tennessee State University

Name of participant: _____ Age: _____

The following information is provided to inform you about the research project and your participation in it. Please read this form carefully and feel free to ask any questions you may have about this study and the information given below. You will be given an opportunity to ask questions, and your questions will be answered. Also, you will be given a copy of this consent form.

Your participation in this research study is voluntary. You are also free to withdraw from this study at any time. In the event new information becomes available that may affect the risks or benefits associated with this research study or your willingness to participate in it, you will be notified so that you can make an informed decision whether or not to continue your participation in this study.

For additional information about giving consent or your rights as a participant in this study, please feel free to contact Tara Prairie at the Office of Compliance at (615) 494-8918.

1. **Purpose of the study:**
   You are being asked to participate in a research study because you have been identified by an expert as having an eating disorder. The purpose of the study is to determine the effects of a 10-week resistance training intervention on bone mineral density (BMD), body composition (BC), muscle strength, depression, and eating disordered tendencies in young women with eating disorders.

2. **Description of procedures to be followed and approximate duration of the study:**
   The pre- and posttests will occur at the Human Performance Lab at Middle Tennessee State University. The testing procedures will include two 120 minute visits to MTSU. If you agree to participate, you will have your height, body mass, body composition, upper and lower body strength, and bone mineral density measured. You will also be asked to complete the Beck Depression Inventory-II, the Eating Disorders Inventory 3, the Impact Food Frequency Questionnaire, and the AHA/ACSM Health/Fitness Facility Preparticipation Screening Questionnaire, in addition to a health history questionnaire.

   A number of procedures will be used to collect data in this project. Total body mass will be measured using a digital scale. All participants will stand backwards during this measure. Height will be measured with a stadiometer. Body composition will be measured using skinfold calipers. Chest and leg strength will be measured on a universal strength training machine. Lumbar spine, hip, and forearm BMD will be measured using a DXA scan. The DXA scan involves laying comfortably on a cushioned tabletop while the arm of the machine passes over the body. If you are pregnant or think that you may be pregnant, please inform the technician or investigator immediately. You may wish to skip this scan or ask questions regarding this scan and the benefits and risks during pregnancy.

   If you are chosen for the experimental group, you will participate in a resistance training program supervised by the primary investigator for up to 10 weeks. The program will consist of exercises for the entire body and will meet three days per week for 45-60 minute sessions. Training will take place at either MTSU or at a designated site in Nashville, depending upon where you live. If you are chosen for the control group, you will continue your normal care with your physician and/or counselors and will return for post testing after 10 weeks. At the conclusion of the study, you will have an opportunity to participate in the same intervention as persons chosen for the experimental group.

3. **Expected costs:**
   The expected costs include your cost of transportation to Murfreesboro for the pre- and posttests as well as transportation to the training sessions, to be held either in Murfreesboro or at a designated site in Nashville.

4. **Description of the discomforts, inconveniences, and/or risks that can be reasonably expected as a result of participation in this study:**
   The above procedures pose little health risk. With any resistance program there is an elevated risk of injury to the muscles or bones. Unusual cardiovascular events can occur in persons with known or undiagnosed heart disease. When beginning any resistance training program, minor muscle soreness can be expected. This initial soreness may last up to 10 days. However, a slow progression following American College of Sports Medicine guidelines will be followed in order to minimize risks to the greatest extent possible.

   There are no known psychological or social consequences to performing a DXA scan. Physiological risks associated with a scan are minimal. The DXA scan is noninvasive and pain-free. The radiation associated with a DXA scan is extremely low. Normal exposure to background radiation at sea level is 8.0 μSV per day. The dose produced by a lumbar spine DXA scan is 4.0 μSV, a hip DXA scan is 1.3 μSV, and a forearm DXA scan is 0.05 μSV (a combined effective dose value of 5.35 μSV). For comparison, the dose associate with a dental bitewing x-ray is 60.0 μSV.

   Transportation and travel time to and from the testing and training sites is a reasonable inconvenience that can be expected as a result of participation in this study.

5. **Unforeseeable risks:**
   Because this treatment is investigational, meaning non-FDA approved, there may be unknown or unforeseeable risks associated with participation.

6. **Compensation in case of study-related injury:**
   If you are injured as a result of this study, I am unable to provide you with compensation.

7. **Anticipated benefits from this study:**
   a) The potential benefits to science and humankind that may result from this study are: Interventions such as this may be used in the future as a possible part of treatment for women recovering from eating disorders in order to improve body composition, decrease the risk of osteopenia and osteoporosis, decrease depressive symptoms, and educate regarding the proper use of daily physical activity for health.

   b) If you participate in the experimental (resistance training) group, the potential benefits to you from this study are increased muscle mass and bone mineral density. Increasing muscle mass and bone mineral density in females recovering from eating disorders could drastically change your risk profile for developing osteoporosis and connective tissue degeneration. Resistance training has also been shown to have positive effects on depressive symptoms. In addition, the supervised resistance training program will educate you with regard to an exercise that should be incorporated as a lifelong activity to prevent the deleterious effects of the eating disorder from which you are recovering. You will also benefit by receiving free body composition analysis, bone mineral density analysis, and a supervised training program.

   c) If you participate in the control group, you will continue with your current treatment from your physician and/or mental health professional. You will receive free body composition analysis and bone mineral density analysis. At the conclusion of the study, you will be offered

the opportunity to participate in the same strength training intervention as the experimental group.

8. **Alternative treatments available:**
   Available alternative approaches for possibly increasing bone mineral density include hormonal and pharmacological approaches, which are administered by invasive means (injection and orally). Resistance training is a noninvasive alternative to increasing bone mineral density. There are no known alternative approaches for increasing muscle mass and improving body composition among this population other than proper exercise and nutrition. Participating in this study should not replace the care you are already receiving from your physician and/or mental health professional.

9. **Compensation for participation:**
   You will not receive monetary compensation for participating in this study.

10. **Circumstances under which the Principal Investigator may withdraw you from study participation:**
    If the investigators determine that you are at physical or psychological risk during the course of the study, you may be withdrawn. Any necessary steps will be taken in order to assure your safety if you are found to be at physical or psychological risk.

11. **What happens if you choose to withdraw from study participation:**
    There will be no penalty to you if you chose to withdraw from study participation. If you do not complete the study, your data will not be used for the purposes of this research and will be destroyed.

12. **Contact Information.** If you should have any questions about this research study or possibly injury, please feel free to contact **Gina Evans** at **615-898-5545 or gls2d@mtsu.edu** or my Faculty Advisor, **Dr. Jennifer L. Caputo** at **615-898-5547**.

13. **Confidentiality.** All efforts, within reason, will be made to keep the personal information in your research record private but total privacy cannot be promised. Your information may be shared with the MTSU University Institutional Review Board or your referring physician or counselor if you or someone else is in danger or if we are required to do so by law.

STATEMENT BY PERSON AGREEING TO PARTICIPATE IN THIS STUDY

I have read this informed consent document and the material contained in it has been explained to me verbally. I understand each part of the document, all my questions have been answered, and I freely and voluntarily choose to participate in this study.

_____ _____

Date Signature of patient/volunteer

Consent obtained by:

_____ _____

Date Signature

_____

Printed Name and Title

Appendix D

Child Assent Form

Principal Investigator: Gina S. Evans
Study Title: Effects of a 10-Week Strength Training Intervention Among Community-Dwelling Eating Disordered Females
Institution: Middle Tennessee State University

Name of participant: _____ Age: _____

The following information is provided to inform you about the research project and your participation in it. Please read this form carefully and feel free to ask any questions you may have about this study and the information given below. You will be given an opportunity to ask questions, and your questions will be answered. Also, you will be given a copy of this consent form.

Your participation in this research study is voluntary. You are also free to withdraw from this study at any time. In the event new information becomes available that may affect the risks or benefits associated with this research study or your willingness to participate in it, you will be notified so that you can make an informed decision whether or not to continue your participation in this study.

For additional information about giving consent or your rights as a participant in this study, please feel free to contact Tara Prairie at the Office of Compliance at (615) 494-8918.

1. **Purpose of the study:**
   You are being asked to participate in a research study because you have been identified by an expert as having an eating disorder. The purpose of the study is to determine the effects of a 10-week resistance training intervention on bone mineral density (BMD), body composition (BC), muscle strength, depression, and eating disordered tendencies in young women with eating disorders.

2. **Description of procedures to be followed and approximate duration of the study:**
   The pre- and posttests will occur at the Human Performance Lab at Middle Tennessee State University. The testing procedures will include two 120 minute visits to MTSU. If you agree to participate, you will have your height, body mass, body composition, upper and lower body strength, and bone mineral density measured. You will also be asked to complete the Beck Depression Inventory-II, the Eating Disorders Inventory 3, the Impact Food Frequency Questionnaire, and the AHA/ACSM Health/Fitness Facility Preparticipation Screening Questionnaire in addition to a health history questionnaire.

   A number of procedures will be used to collect data in this project. Total body mass will be measured using a digital scale. All participants will stand backwards during this measure. Height will be measured with a stadiometer. Body composition will be measured using skinfold calipers. Chest and leg strength will be measured on a universal strength training machine. Lumbar spine, hip, and forearm BMD will be measured using a DXA scan. The DXA scan involves laying comfortably on a cushioned tabletop while the arm of the machine passes over the body. If you are pregnant or think that you may be pregnant, please inform the technician or investigator immediately. You may wish to skip this scan or ask questions regarding this scan and the benefits and risks during pregnancy.

   If you are chosen for the experimental group, you will participate in a resistance training program supervised by the primary investigator for up to 10 weeks. The program will consist of exercises for the entire body and will meet three days per week for 45-60 minute sessions. Training will take place at either MTSU or at a designated site in Nashville, depending upon where you live. If you are chosen for the control group, you will continue your normal care with your physician and/or counselors and will return for post testing after 10 weeks. At the conclusion of the study, you will have an opportunity to participate in the same intervention as persons chosen for the experimental group.

3. **Expected costs:**
   The expected costs include your cost of transportation to Murfreesboro for the pre- and posttest s as well as transportation to the training sessions, to be held either in Murfreesboro or at a designated site in Nashville.

4. **Description of the discomforts, inconveniences, and/or risks that can be reasonably expected as a result of participation in this study:**
   The above procedures pose little health risk. With any resistance program there is an elevated risk of injury to the muscles or bones. Unusual cardiovascular events can occur in persons with known or undiagnosed heart disease. When beginning any resistance training program, minor muscle soreness can be expected. This initial soreness may last up to 10 days. However, a slow progression following American College of Sports Medicine guidelines will be followed in order to minimize risks to the greatest extent possible.

   There are no known psychological or social consequences to performing a DXA scan. Physiological risks associated with a scan are minimal. The DXA scan is noninvasive and pain-free. The radiation associated with a DXA scan is extremely low. Normal exposure to background radiation at sea level is 8.0 μSV per day. The dose produced by a lumbar spine DXA scan is 4.0 μSV, a hip DXA scan is 1.3 μSV, and a forearm DXA scan is 0.05 μSV (a combined effective dose value of 5.35 μSV). For comparison, the dose associate with a dental bitewing x-ray is 60.0 μSV.

   Transportation and travel time to and from the testing and training sites is a reasonable inconvenience that can be expected as a result of participation in this study.

5. **Unforeseeable risks:**
   Because this treatment is investigational, meaning non-FDA approved, there may be unknown or unforeseeable risks associated with participation.

6. **Compensation in case of study-related injury:**
   If you are injured as a result of this study, I am unable to provide you with compensation.

7. **Anticipated benefits from this study:**
   a) The potential benefits to science and humankind that may result from this study are: Interventions such as this may be used in the future as a possible part of treatment for women recovering from eating disorders in order to improve body composition, decrease the risk of osteopenia and osteoporosis, decrease depressive symptoms, and educate regarding the proper use of daily physical activity for health.

   b) If you participate in the experimental (resistance training) group, the potential benefits to you from this study are increased muscle mass and bone mineral density. Increasing muscle mass and bone mineral density in females recovering from eating disorders could drastically change your risk profile for developing osteoporosis and connective tissue degeneration. Resistance training has also been shown to have positive effects on depressive symptoms. In addition, the supervised resistance training program will educate you with regard to an exercise that should be incorporated as a lifelong activity to prevent the deleterious effects of the eating disorder from which you are recovering. You will also benefit by receiving free body composition analysis, bone mineral density analysis, and a supervised training program.

   c) If you participate in the control group, you will continue with your current treatment from your physician and/or mental health professional. You will receive free body composition analysis and bone mineral density analysis. At the conclusion of the study, you will be offered

the opportunity to participate in the same strength training intervention as the experimental group.

8. **Alternative treatments available:**
   Available alternative approaches for possibly increasing bone mineral density include hormonal and pharmacological approaches, which are administered by invasive means (injection and orally). Resistance training is a noninvasive alternative to increasing bone mineral density. There are no known alternative approaches for increasing muscle mass and improving body composition among this population other than proper exercise and nutrition. Participating in this study should not replace the care you are already receiving from your physician and/or mental health professional.

9. **Compensation for participation:**
   You will not receive monetary compensation for participating in this study.

10. **Circumstances under which the Principal Investigator may withdraw you from study participation:**
    If the investigators determine that you are at physical or psychological risk during the course of the study, you may be withdrawn. Any necessary steps will be taken in order to assure your safety if you are found to be at physical or psychological risk.

11. **What happens if you choose to withdraw from study participation:**
    There will be no penalty to you if you chose to withdraw from study participation. If you do not complete the study, your data will not be used for the purposes of this research and will be destroyed.

12. **Contact Information.** If you should have any questions about this research study or possibly injury, please feel free to contact **Gina Evans** at **615-898-5545 or gls2d@mtsu.edu** or my Faculty Advisor, **Dr. Jennifer L. Caputo** at **615-898-5547**.

14. **Confidentiality.** All efforts, within reason, will be made to keep the personal information in your research record private but total privacy cannot be promised. Your information may be shared with the MTSU University Institutional Review Board or your referring physician or counselor if you or someone else is in danger or if we are required to do so by law.

STATEMENT BY PERSON AGREEING TO PARTICIPATE IN THIS STUDY

I have read this assent document and the material contained in it has been explained to me verbally. I understand each part of the document, all my questions have been answered, and I freely and voluntarily choose to participate in this study.

_____ _____

Date Signature of patient/volunteer

Consent obtained by:

_____ _____

Date Signature

_____

Printed Name and Title

Appendix E

Parental Informed Consent

Principal Investigator: Gina S. Evans
Study Title: Effects of a 10-Week Strength Training Intervention Among Community-Dwelling Eating Disordered Females
Institution: Middle Tennessee State University

Name of participant: _____ Age: _____

Name of parent: _____

The following information is provided to inform you about the research project and your child's participation in it. Please read this form carefully and feel free to ask any questions you may have about this study and the information given below. You will be given an opportunity to ask questions, and your questions will be answered. Also, you will be given a copy of this consent form.

Your child's participation in this research study is voluntary. Your child is free to withdraw from this study at any time. In the event new information becomes available that may affect the risks or benefits associated with this research study or your child's willingness to participate in it, you will be notified so that you and your child can make an informed decision whether or not to continue your participation in this study.

For additional information about giving consent or your child's rights as a participant in this study, please feel free to contact Tara Prairie at the Office of Compliance at (615) 494-8918.

1. **Purpose of the study:**
   Your child is being asked to participate in a research study because she has been identified by an expert as having an eating disorder. The purpose of the study is to determine the effects of a 10-week resistance training intervention on bone mineral density (BMD), body composition (BC), muscle strength, depression, and eating disordered tendencies in young women with eating disorders.

2. **Description of procedures to be followed and approximate duration of the study:**
   The pre- and posttests will occur at the Human Performance Lab at Middle Tennessee State University. The testing procedures will include two 120 minute visits to MTSU. If your child agrees to participate, she will have her height, body mass, body composition, upper and lower body strength, and bone mineral density measured. Your child will also be asked to complete the Beck Depression Inventory-II, the Eating Disorders Inventory 3, the Impact Food Frequency Questionnaire, and the AHA/ACSM Health/Fitness Facility Preparticipation Screening Questionnaire in addition to a health history questionnaire.

   A number of procedures will be used to collect data in this project. Total body mass will be measured using a digital scale. Your child will stand backwards during this measures so as not to view the results. Height will be measured with a stadiometer. Body composition will be measured using skinfold calipers. Chest and leg strength will be measured on a universal strength training machine. Lumbar spine, hip, and forearm BMD will be measured using a DXA scan. The DXA scan involves laying comfortably on a cushioned tabletop while the arm of the machine passes over the body.

   If your child is chosen for the experimental group, she will participate in a resistance training program supervised by the primary investigator for up to 10 weeks. The program will consist of exercises for the entire body and will meet three days per week for 45-60 minute sessions. Parents or guardians will not be allowed to train with or observe the training of their children. Training will take place at either MTSU or at a designated site in Nashville, depending upon where you live. If your child is chosen for the control group, she will continue her normal care with her physician and/or mental health professional and will return for post testing after 10 weeks. At the

conclusion of the study, your child will have an opportunity to participate in the same intervention as persons chosen for the experimental group.

3. **Expected costs:**
   The expected costs include your cost of transportation to Murfreesboro for the pre- and posttests as well as transportation to the training sessions, to be held either in Murfreesboro or at a designated site in Nashville.

4. **Description of the discomforts, inconveniences, and/or risks that can be reasonably expected as a result of participation in this study:**
   The above procedures pose little health risk. With any resistance program there is an elevated risk of injury to the muscles or bones. Unusual cardiovascular events can occur in persons with known or undiagnosed heart disease. When beginning any resistance training program, minor muscle soreness can be expected. This initial soreness may last up to 10 days. However, a slow progression following American College of Sports Medicine guidelines will be followed in order to minimize risks to the greatest extent possible.

   There are no known psychological or social consequences to performing a DXA scan. Physiological risks associated with a scan are minimal. The DXA scan is noninvasive and pain-free. The radiation associated with a DXA scan is extremely low. Normal exposure to background radiation at sea level is 8.0 μSV per day. The dose produced by a lumbar spine DXA scan is 4.0 μSV, a hip DXA scan is 1.3 μSV, and a forearm DXA scan is 0.05 μSV (a combined effective dose value of 5.35 μSV). For comparison, the dose associate with a dental bitewing x-ray is 60.0 μSV.

   Transportation and travel time to and from the testing and training sites is a reasonable inconvenience that can be expected as a result of participation in this study.

5. **Unforeseeable risks:**
   Because this treatment is investigational, meaning non-FDA approved, there may be unknown or unforeseeable risks associated with participation.

6. **Compensation in case of study-related injury:**
   If your child is injured as a result of this study, I am unable to provide her with compensation.

7. **Anticipated benefits from this study:**
   a) The potential benefits to science and humankind that may result from this study are: Interventions such as this may be used in the future as a possible part of treatment for women recovering from eating disorders in order to improve body composition, decrease the risk of osteopenia and osteoporosis, decrease depressive symptoms, and educate regarding the proper use of daily physical activity for health.

   b) If your child participates in the experimental (resistance training) group, the potential benefits to her from this study are increased muscle mass and bone mineral density. Increasing muscle mass and bone mineral density in females recovering from eating disorders could change your child's risk profile for developing osteoporosis and connective tissue degeneration. Resistance training has also been shown to have positive effects on depressive symptoms. In addition, the supervised resistance training program will educate your child with regard to an exercise that should be incorporated as a lifelong activity to prevent the deleterious effects of the eating disorder from which she is recovering. Your child will also benefit by receiving free body composition analysis, bone mineral density analysis, and a supervised training program.

c) If your child participates in the control group, she will continue with her current treatment from your physician and/or mental health professional. Your child will receive free body composition analysis and bone mineral density analysis. At the conclusion of the study, your child will be offered the opportunity to participate in the same strength training intervention as the experimental group.

8. **Alternative treatments available:**
   Available alternative approaches for possibly increasing bone mineral density include hormonal and pharmacological approaches, which are administered by invasive means (injection and orally). Resistance training is a noninvasive alternative to increasing bone mineral density. There are no known alternative approaches for increasing muscle mass and improving body composition among this population other than proper exercise and nutrition. Participating in this study should not replace the care your child is already receiving from your physician and/or mental health professional.

9. **Compensation for participation:**
   Your child will not receive monetary compensation for participating in this study.

10. **Circumstances under which the Principal Investigator may withdraw you from study participation:**
    If the investigators determine that your child is at physical or psychological risk during the course of the study, she may be withdrawn. Any necessary steps will be taken in order to assure your child's safety if she is found to be at physical or psychological risk.

11. **What happens if you choose to withdraw from study participation:**
    There will be no penalty to your child if she chooses to withdraw from study participation. If your child does not complete the study, her data will not be used for the purposes of this research and will be destroyed.

12. **Contact Information.** If you or your child should have any questions about this research study or possibility of injury, please feel free to contact **Gina Evans** at **615-898-5545 or gls2d@mtsu.edu** or my Faculty Advisor, **Dr. Jennifer L. Caputo** at **615-898-5547**.

15. **Confidentiality.** All efforts, within reason, will be made to keep the personal information in your research record private but total privacy cannot be promised. Your child's information may be shared with the MTSU University Institutional Review Board or her referring physician or mental health professional if she or someone else is in danger or if we are required to do so by law.

STATEMENT BY PERSON AGREEING TO PARTICIPATE IN THIS STUDY

I have read this informed consent document and the material contained in it has been explained to me verbally. I understand each part of the document, all my questions have been answered, and I freely and voluntarily choose to allow my child to participate in this study.

_____ _____

Date Signature of parent/guardian

Consent obtained by:

_____ _____

Date Signature

_____

Printed Name and Title