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# Bone mineral density in resistance trained premenopausal females

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# BONE MINERAL DENSITY IN RESISTANCE TRAINED PREMENOPAUSAL FEMALES

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A thesis presented to the faculty of
the School of Health Sciences
and Human Performance
at Ithaca College

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In partial fulfillment of the
requirements for the degree of
MASTER OF SCIENCE

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by
Stephen B. Hess
September 1994

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## DEDICATION

This thesis is dedicated to my parents, Michael and Geraldyne for their love and care, and my wife Alicia, for her support and dedication.

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Thesis Advisor: Dr. B. A. Keller

Ithaca College

School of Health Sciences and Human Performance

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**CERTIFICATE OF APPROVAL**

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**MASTER OF SCIENCE THESIS**

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This is to certify that the Master of Science Thesis of

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submitted in partial fulfillment of the requirements for the degree of Master of Science in the School of Health Sciences and Human Performance at Ithaca College has been approved.

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# BONE MINERAL DENSITY IN RESISTANCE TRAINED PREMENOPAUSAL FEMALES

by

Stephen B. Hess

An Abstract

of a thesis submitted in partial fulfillment
of the requirements for the degree of
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**ABSTRACT**

The purpose of this study was to compare the effects of chronic resistance training on bone mineral density (BMD) in resistance trained and sedentary premenopausal females. Thirteen pairs of resistance trained (R, age: $\underline{M}$ = 24.4 ± 1.04) and sedentary (S, age: $\underline{M}$ = 22.8 ± 1.31) subjects were matched for weight and height. Percent fat determined by densitometry was 18% for R and 27.4% for S. Measures of upper body and lower body strength were 34% and 42% higher in R, respectively ($p < 0.01$). BMD was measured by dual energy x-ray absorptiometry (DEXA) at the lumbar spine (L1-L4), femoral neck, forearm (radius and ulna), and whole body. A paired t-test revealed 4% - 10% differences in BMD between the matched pairs at the ulna, radius, and whole body ($p < 0.05$). Spine and femoral neck BMD for R was 4% and 8% greater respectively than S, however this was not statistically significant. Pearson product correlation coefficients for BMD and strength for the R group indicated significant correlations ($p < 0.05$) between strength measures and BMD. However, no significant correlations between strength measures and BMD were observed in the S group. This study extends findings of increased BMD following weight bearing, endurance type exercise to include chronic resistance exercise as a modality that might increase BMD in females.

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## INTRODUCTION

Osteoporosis continues to be the prevalent metabolic bone disease in western industrialized societies, despite generally adequate nutrition and health care (Martin and Houston, 1987). This disease causes much pain and suffering among those who are affected by it (Purvis, 1990). Osteoporosis expenditures represent a sizeable portion of the health care dollars attributed to women in this country. In 1986, the cost of debilitating hip fractures totalled 5.2 billion dollars (Kleerekoper and Avioli, 1990).

Osteoporosis occurs when normal mineralization of the bone is reduced to levels that increase the risk of fracture in the absence of trauma, or, in response to relatively minor trauma. While bone mineralization and histology may be within normal limits, a decrease in bone mass and pathological fractures characterize the presence of osteoporosis.

This disease is most commonly associated with postmenopausal women. The etiology of osteoporosis remains somewhat elusive and understanding of the disease to date is limited. We do know that the degree to which bone mass is maximized prior to menopause, and the rate at which bone is lost seem to be related to the development and progression

of osteoporosis (Sanborn, 1990).

The majority of research completed in the study of bone indicates that proper nutrition is essential for normal bone growth and maintenance of bone mineral content (BMC). Recent research has also indicated the importance of weight-bearing activity and exercise as a prophylactic means of maintaining or minimizing the decrease in age related bone demineralization (Recker, 1984; Marcus and Cann, 1985; Whalen and Carter, 1982). Researchers agree that the most effective way to combat osteoporosis is to increase bone mineral density (BMD) at a young age and maintain these increases into old age through exercise, nutrition, and supplementation (Dalsky, 1987; Sanborn, 1990).

Exercise-related studies have generally examined the impact of weight-bearing activity (e.g., walking, dancing, running) on BMD. Little attention has focused on the effects of resistance training on BMD. Block et al. (1989) reported a 9% higher bone density in males who engaged in a generalized resistance training program compared to sedentary counterparts. However, the prophylactic significance of using this exercise modality in females, who are at greatest risk for osteoporosis, is unclear.

The purpose of this investigation will be to compare

BMD of the hip, spine, and forearm in premenopausal resistance trained (R) and sedentary (S) females who are matched for body weight and height. The first hypothesis of this study will be that BMD is significantly greater in premenopausal R when compared to S. Another problem will be to evaluate the relationship between strength and BMD, in these subjects. Therefore, additional hypotheses for this study are related to predicting a strong positive relationship between BMD and muscular strength in R subjects.

## REVIEW OF RELATED LITERATURE

The purpose of this study is to compare bone density in R and S premenopausal females. This chapter is a review of the relevant literature and is organized into the following sections: (1) physiology of bone formation, (2) measurements of bone density, (3) osteoporosis: an overview, (4) exercise and bone density, (5) relationship between muscle mass, strength, and bone density, (6) supplementation and bone density, and (7) summary.

### Physiology of Bone Formation

Bone is continually deposited by cellular structures known as osteoblasts. Osteoblasts are found on the outer surface of the bone and in the bone cavities. There is a small amount of on-going osteoblastic activity in all living bones (on about 4% of all surfaces in adult bone), therefore, bone formation is a constant process (Guyton, 1987).

Living bone undergoes a continuous absorption in the presence of osteoclasts, which are normally active at any time on 1% of the surface of bone. Osteoclast activity is largely influenced by parathyroid hormone. Parathyroid hormone has two separate effects (i.e., phases) on bone

while causing absorption of calcium and phosphate. The rapid phase probably occurs when already existing bone cells are activated to promote calcium and phosphate absorption; this phase usually takes minutes. Parathyroid hormone converts 25-hydroxycholecalciferol to 1,25 dihydroxycholecalciferol (the active form of vitamin D) thereby determining the functional effects of vitamin D in the body. It specifically affects calcium absorption in the intestines and thereby influences the formation of bone. The slow phase of bone absorption usually takes several days or even weeks and it results from the proliferation of osteoclasts. This is followed by increased osteoclastic reabsorption of the bone itself, not merely absorption of calcium phosphate salts from the bone. Following significant osteoclastic reabsorption, there is secondary stimulation of osteoblasts. While the slow phase is marked by both osteoclast and osteoblast proliferation the predominant effect of parathyroid hormone is that of bone absorption (Guyton, 1987).

Histologically, bone absorption occurs immediately adjacent to the osteoclasts. The osteoclasts send out villous like projections toward the bone, and from these villi two types of substances are secreted; 1) proteolytic

enzymes, released from the lysosomes of the osteoclasts and 2) several acids, including citric acid and lactic acid. These enzymes digest the organic matrix of the bone, while the acids dissolve the bone and form a solution of bone salts. The "villi" are also capable of engulfing whole fragments of bone salt and collagen (phagocytosis) (Guyton, 1987). Bone deposition and absorption occur simultaneously. The rate of bone deposition and absorption are comparable, so that the total mass of bone remains constant. Osteoclasts usually exist in large masses. They act on the bone for about three weeks and create a tunnel that may be as large as one millimeter in diameter. At the end of this time the osteoclasts disappear and the area is invaded by osteoblasts. Osteoblasts deposit bone for several months in successive layers on the inner surfaces of the cavity until the tunnel is filled. The deposition of new bone continues until the bone begins to encroach on the blood vessels supplying the area. The only remaining area from the original cavity is the Haversian canal. Each new area of bone deposited in this way is called an osteon.

Bone remodeling is a cyclic process which replaces the mineralized bone matrix while maintaining the gross architecture and size of bone. Bone remodeling occurs to

maintain mineral homeostasis and to prevent the accumulation of microfractures or fatigue damage. Individuals can experience excessive mineral loss in mature bone resulting from remodeling. This occurs when reabsorption exceeds formation, thus, adversely affecting the structural integrity of the bone (Dalsky, 1990)

The strength of a bone is largely determined by the amount of force to which it is subjected. Thus, a bone increases in diameter and density (within genetically determined ceilings) when subjected to heavy loads. The shape of a bone is also determined by stress patterns which influence the areas where bone is deposited and absorbed. Finally, new organic matrix is needed as the old organic matrix degenerates. In this manner, the integrity of the bone is maintained (Dalsky, 1989).

## Measurements of Bone Density

The four radiographic techniques currently used for non-invasive assessment of bone density are: Single-photon absorptiometry (SPA), dual-photon absorptiometry (DPA), quantitative-computed tomography (QCT), and DEXA.

SPA is used to quantify BMD measurements in the appendicular skeleton. This technique is relatively fast, inexpensive, and has a low radiation dose of only 5 to 13

mrems. This method can not determine BMD in the vertebral column and femur.

DPA is a modification of SPA. It uses a dual-energy radionuclide source. DPA is used to measure BMD in the lumbar spine (L2-L4), the proximal femur, and the total body. This technique is precise, accurate and fairly inexpensive.

A modification of DPA is DEXA. Unlike DPA, DEXA uses a filtered x-ray source, providing shortened scanning times, reduced radiation exposure, improved precision, and higher resolution images than DPA.

QCT may also be used to measure BMD in the spine. Unfortunately, QCT is limited in scope. It scans only a small area of purely trabecular bone measured from the center of the vertebral body. The accuracy and precision of QCT is not as good as DPA and exposes subjects to as much as 500 times the radiation of DPA (Sanborn, 1990).

## Osteoporosis: An Overview

Osteoporosis is a metabolic bone disease in which the resistance of bone tissue to fracture is impaired as the volume of mineralized trabecular and cortical bone is decreased within the periosteal envelope, compromising the architectural integrity of the bone (Albanese, 1978). This

disease not only causes physical disorders, but emotional problems as well (Aloia, 1989). The direct and indirect cost for treating osteoporosis ranges from 7 to 10 billion dollars annually. In today's society the crippling symptoms of osteoporosis have almost become synonymous with old age. Individuals with this debilitating disease may suffer one or more of the following common disorders: The dowager's hump (demineralization of the spine resulting in a disfiguring hump on the superior part of the spine), loss of height, and painful, often debilitating fractures of the spine and hip (Purvis, 1990). Approximately, 24 million Americans are affected by osteoporosis, and each year 1.3 million American females over the age of 40 fracture one or more of their bones. Out of those who sustain hip fractures, 12-20% die and many live in chronic pain for the duration of their lives. The U.S. Bureau of Census predicts that Americans who live to be older than 65 will double from 12% in 1988 to 24% in 2020. It seems that osteoporosis will become an even greater problem in the years to come (Sanborn, 1990).

Cohn, Vaswani, Zanzi, and Ellis (1976) considered osteoporosis to be a condition which was no different from the changes observed in the natural aging process but varied only in degree. They suggested that osteoporosis may be an

acceleration of the normal aging process, but found that this disease can occur at any age. Disuse osteoporosis can affect any individual in the population, not just the elderly. It occurs when there is a period of immobilization or inactivity in which the rate of bone reabsorption exceeds the rate of bone formation. Others have speculated that bone loss due to inactivity occurred largely in the trabecular portion of the bone because of the greater surface-to-volume ratio of trabecular bone (Brewer, Meyer, Keele, Upton, and Hagan, 1983). They believe that some compact bone is also lost.

Changes in bone due to aging generally involve both compact and trabecular bone. Diminution of compact bone begins in females prior to menopause (about age 40) at the rate of about three percent per decade. The decrease in compact bone seems to occur in two phases. The first phase, which constitutes approximately one-third of the total bone loss, is the slow loss of bone. This loss occurs at approximately three percent per decade and begins after peak bone mass has been attained. The second phase, following menopause, is responsible for two-thirds of bone loss, and occurs at a rate of nine percent per decade.

Mazess (1981) found that females lose trabecular bone

at a rate of six to eight percent per decade beginning in young adulthood (20-40 years). These findings suggest that the majority of bone loss in the trabecular portion of the bone occurs prior to menopause. These results imply that the long standing belief that females lose a large percentage of trabecular bone after menopause may be misleading. Sanborn (1990) suggested that skeletal sites susceptible to osteoporotic fractures usually consist of trabecular bone rather than compact cortical bone because of the larger surface area of trabecular bone. However, more research is needed to determine what portion of bone loss is due to trabecular or cortical bone.

Abramson and Delagi (1960) concluded that osteoporosis has many causes of which disuse is only one. There is not enough evidence to suggest that osteoporosis can be reversed, therefore, therapeutic efforts should be directed toward prevention. Disuse osteoporosis results in a loss of bone mineral content due to the absence of mechanical force on bone. These losses are unlimited and can only be reversed with mobilization. Mobilization of a limb requires mechanical force. The architecture of a mature bone is largely determined by the mechanical stress it receives, within a genetically determined limit. The reabsorption and

formation of bone are indirectly affected by changes in strain and stress on the bone cell. Bone growth is stimulated by these external factors and further influenced by hormonal and nutritional milieus (Dalsky, 1989).

## Exercise and Bone Density

Exercise appears to slow bone loss and it may even reverse it. The majority of studies have concluded that weight-bearing exercise is a beneficial mechanism in combatting bone loss.

Saville and Whyte (1969) studied the effects of running on muscle and bone hypertrophy in rats. The exercise period consisted of 24 alternating 15 min intervals of running and rest per day. Each rat ran approximately 2000 m/day, five days a week. Following training, the wet weights of the cleaned humeri, femora, and tibiae/fibulae were greater at any given body weight in the exercised group. The hind limb muscle mass of the exercised animals was also greater than the control group at any given weight. Moreover, it was shown that bone and muscle hypertrophy were in exact proportion to each other. Saville and Whyte (1969) suggested that these findings could be generalized to young children. Children who increase their bone and muscle size at an early age may protect themselves against osteoporosis

and related fractures later in life.

In older women, exercise appears to inhibit bone loss rather than increase bone deposition. Exercise in postmenopausal women appears to be primarily preventative, as opposed to therapeutic, with respect to bone loss (Rickli and McManis, 1990). It seems that the intensity, frequency and duration of exercise needed to maintain musculoskeletal fitness is extensive. In fact, it is harder to induce musculoskeletal improvements than it is to improve cardiorespiratory fitness (Aloia, 1978).

Stillman, Lohman, Slaughter, and Massey (1986) compared active, moderately active and inactive females, ages 30 to 85. They found a significant difference in BMC, measured by SPA, between the active group and the two less active groups even when age and menstrual status were considered ($p < 0.05$). A higher BMC in the mid-shaft of the radius was seen in females with higher levels of physical activity than in those who were less active. These results indicate that high levels of physical activity may be a factor in reducing age-related bone loss.

Similarly, Brewer et al. (1983) compared active and inactive females between the ages of 20-49 with diverse physical activity levels. The active group, including

marathon runners, had a greater BMC at the mid-shaft of the radius ($p < 0.05$). Findings in these studies and others (Chow, Harrison, Brown, & Hajek, 1986) suggest that physical activity may reduce the amount of bone loss in postmenopausal females. These studies imply however, that improvements in bone density occur only with intense training.

Wolman, Faulmann, Clark, Hesp, and Harries (1991) compared BMC of the femoral mid-shaft of elite, female athletes. The subjects included 67 elite, female athletes comprised of 21 runners, 36 rowers, 10 dancers, and 13 eumenorrheic, sedentary females. The mean BMC of the runners was 1.51 g/cm. This was significantly greater than the rowers, dancers, and sedentary controls whose values were 1.43, 1.39, and 1.40 g/cm, respectively. There was no significant difference in BMC between rowers, dancers and the sedentary group. These findings suggested that the type of exercise, may be the most important factor in determining BMC. Similarly, Risser, Lee, LeBlanc, Poindexter, and Schneider (1990) found athletes in weight-bearing activities to have greater BMD compared to inactive females and female swimmers. Lumbar BMD was $1.31 \pm 0.03$ $g \cdot cm^{-2}$ for volleyball players, $1.26 \pm 0.04$ $g \cdot cm^{-2}$ for basketball players, $1.05 \pm$

0.03 $g \cdot cm^{-2}$ for swimmers, and 1.18 ± 0.03 $g \cdot cm^{-2}$ for non-athletes ($p < 0.05$). The lumbar BMD of the swimmers was lower than that of the inactive group and amenorrheic runners. These results further strengthen the argument that the type of physical activity is the most important factor for increasing BMD.

A three year study by Smith, Reddan, and Smith (1981) studied the effect of physical activity and calcium on BMC in aged females (age $\underline{M}$ =81). BMC and width of the radius was determined by SPA at two sites. Four groups were formed: a control group, a drug group (who received 0.7 g of calcium supplementation and 400 IU of vitamin D a day), a physical activity group (who participated in 30 minutes of light to mild physical activity (e.g., walking, running in place three times a week), and a physical activity with drug group. BMC decreased significantly by 3.29% in the control group while the physical activity group increased 2.29% ($p < 0.05$) and the drug group increased 1.58% ($p < 0.07$) during the study. Failure to achieve significance with physical activity plus drug use may be due to the fact that individuals randomly placed in this group were older and did not seem as motivated as other participants. The authors were unable to evaluate the physical characteristics of each

group until the three year double blind study was completed and the drug code broken. Results indicated that the increased BMC in both the drug and physical activity group were similar in magnitude. The increase in BMC in the physical activity group was in the absence of any type of calcium supplementation. They received no vitamin D or any type of drug which could alter BMC, and their diet did not vary from the control group who continued to lose bone. The mechanical stress due to physical activity may be responsible for increased BMC. The study indicates that bone loss in the aged may be reversed and maintained through physical activity or calcium and vitamin D supplementation. The combination of physical activity and supplements did not significantly improve BMC in this elderly female population. However, the physically active group who did not take any type of supplementation were the only group to have significant increases in BMC.

A study conducted by Jacobson, Beaver, Grubb, Taft, and Talmage (1984) compared college athletes and older athletic females to sedentary, aged-matched controls. Mean BMD for both college and older athletes were significantly higher than their aged matched controls ($p < 0.02$). The findings in this study imply that regular physical activity reduces

the rate of "normal" bone loss that accompanies age.

Exercise affects BMC in females in two different ways. Exercise can have a direct action on the bone, for example, the bone is affected when mechanical stress is placed on it through activities such as walking or jogging. The type and intensity of exercise can often influence how the bone responds to the particular stress. The second way exercise affects bone is by modifying the function of the hypothalamic pituitary-ovarian axis, leading to reduced estrogen status (Wolman et al., 1991). Wolman et al. concluded that reduction in estrogen levels, due to increases in hypothalamic-pituitary-ovarian axis activity, is often caused by overtraining. A reduction in estrogen level may lead to a decrease in BMC. To avoid reduction in estrogen levels exercise patterns should be modified.

Block, Smith, Friedlander, and Genant (1989) evaluated 27 studies that were designed to determine if exercise prevents osteoporosis. The results were obtained from both cross-sectional and longitudinal studies. The 17 cross-sectional studies all indicated that elite athletes have greater BMD when compared to sedentary controls. When comparing recreational athletes, elite athletes, and sedentary controls, the results tended to be inconsistent.

The ten prospective studies that evaluated the effect of exercise on BMD in recreational athletes and sedentary controls yielded conflicting results. Only one study found an overall positive response in compact BMD at the radial site, and only one study showed a significant increase in lumbar BMD among the exercisers. Block et al. (1991) concluded that the majority of studies have serious methodological flaws. Most of the studies did not employ a randomized design, sample sizes were generally too small, and most studies were too short in duration. Block and colleagues do not make recommendations on the type, duration, and frequency of exercise needed to enhance bone density. Current information suggests that exercise may have only limited value in affecting BMD. Further investigation using greater methodological rigor are important if the relationship between exercise and osteoporosis is to be accurately determined.

The results of research concerning exercise and BMC is so confusing that some clinicians hesitate to give a specific exercise prescription to prevent osteoporosis. However, when exercise is recommended, a comprehensive program including aerobics and strength conditioning is usually prescribed. Most clinicians are able to justify

their prescription of exercise because additional benefits in areas such as weight control and reduction of heart disease are so well established (Munnings, 1992).

**Inactivity**

Disuse osteoporosis may affect portions of the body or the entire body. This disease is due to immobilization or inactivity and may occur at any age (Jenkins & Cochran, 1969).. This point was illustrated when three healthy males were restricted to bed rest for periods of 30-36 weeks (Donaldson, Hulley, Vogel, Hattner, Bayers and McMillan, 1970). Measured calcium loss during the entire period averaged 4.2% of estimated total body calcium. The mechanism for the loss of bone mineral during bed rest is uncertain. It is believed that bone loss is caused by inadequate mechanical stress on the bones or absence of tension applied to bone by muscle. The reaccumulation of mineral in the central oscalcis following reambulation was similar to the rate of loss during bed rest.

Although osteopenia is generally considered to be fully reversible, this has not previously been documented. Jenkins and Cochran (1969) found that osteoporosis produced by disuse of the femur after distal thigh amputation is characterized by extensive periosteal and subperiosteal bone

reabsorption. The bone reabsorption in the femur of male subjects occurred over a short period of time. These findings imply that bone reabsorption occurs quickly in the absence of mechanical stress. This was further demonstrated by Mazess and Whedon (1983) who studied astronauts subjected to a gravity free environment. The subjects lost bone at a monthly rate of about 4% for trabecular bone and 1% for cortical bone.

Sanborn (1990) stated that aging osteoporosis may be related to an overall decrease in physical activity. With aging, stressors placed on the femur from the vertical axis of the body are reduced, as the elderly often decrease the frequency of total use of an extremity. One way to overcome this may be to increase activity level, ideally participating in cardiovascular activities supplemented by resistive type weight training for at least 45 minutes three times a week.

**Weight Training**

Block et al. (1989) examined bone density among male athletes engaged in weight-bearing or non weight-bearing activities. They compared the two groups to an inactive control group. Group one consisted of 20 males who played on a national championship varsity water polo team. The

players were of Olympic caliber, had played for at least two years on a collegiate team, and had considerable previous water polo experience. They practiced nine months per year, six days per week, and at least two hours per day. These individuals also participated in some type of weight bearing activities which did not exceed 20% of their total physical activities. The second group of males were engaged in a recreational weight lifting program. They participated in some type of strength program for at least 12 months prior to the study. The third group served as an inactive control group. There were no differences for any of the BMC measures between active groups. The results showed an 18% higher BMC at the spine and a 9% higher BMC at the hip in both active groups when compared to the inactive group. Although these findings are remarkable, few empirical studies have been undertaken to evaluate the effects of weight-bearing versus non weight-bearing activities on BMC. This study illustrated that active subjects have a higher BMC at the spine and hip.

Chow et al. (1985) studied the effect of cardiovascular and strength training on BMC in postmenopausal females. Their results were expressed as calcium bone index (CBI). CBI is calculated as the ratio of the subject's calcium

value and the estimated mean value for normal subjects of the same size, based on height and arm span. Results showed that both cardiovascular and strength trained postmenopausal females had significantly greater CBI than inactive females ($p < 0.01$). This study indicated that physical activity minimizes the loss of bone in postmenopausal females, however, the optimal frequency, duration, and intensity of specific exercise required for maintaining or increasing BMC are unknown. Many studies have looked at the effect of exercise on bone mass, yet relatively little is still known regarding the best type of exercise to enhance bone density (such as aerobic exercise versus strength exercise).

Most research to date has examined the effects of aerobic activity on BMC. Some studies examined the effects of resistance training on BMC, but usually in comparison to aerobic exercise, or as a component of a general exercise program. Few studies have evaluated the effects of resistance training only on BMC, and have uncovered some interesting results.

Twelve males (19-40 years) participated in a year long resistance training program (free weights and/or exercise machines), two or more times a week for 45 minutes or longer, to determine the effect of muscle hypertrophy on

bone density (Colletti, Edwards, Gordon, Shary, and Bell, 1989). They were compared to age-matched controls. BMD was measured at the lumbar spine, trochanter, and femoral neck by DPA and at mid-radius via SPA. Results showed that the resistance trained group had a higher BMD at the lumbar spine (1.35 ± 0.03 vs. 1.22 ± 0.02 $g \cdot cm^{-2}$, $p < 0.01$), trochanter (0.99 ± 0.04 vs. 0.86 ± 0.02 $g \cdot cm^{-2}$, $p < 0.01$), and femoral neck (1.18 ± 0.03 vs. 1.02 ± 0.02 $g \cdot cm^{-2}$, $p < 0.01$). However, resistance training did not increase BMD of the mid-radius. The results of this study seem surprising as resistive trained subjects showed increases in BMD at all measured sites except the mid-radius. Colletti and associates speculated that resistance training is associated with an increase in BMD at certain sites on the axial skeleton.

Similar to Colletti et al. (1989), Rockwell and others (1990), examined the effects of a nine month weight training program on BMD of premenopausal females and inactive controls. Unlike the Colletti et al. study, the exercise group experienced a non-significant decline in lumbar spine bone density by 2.90% at 4.5 months and 3.96% at 9 months ($p < 0.01$) whereas the control group experienced no change over the nine month period. The decreased BMC of the exercise

group may be attributed to the short duration of the study. It may be that the initial effects of the resistance training program served to establish a new basal state of bone turnover, and that studies over a longer period are needed to evaluate the ultimate effect on bone mass.

Another study was conducted to determine the effects of weight lifting on BMD in premenopausal females (Gleeson, Protas, Le Blanc, and Schneider, 1990). A group of 68 premenopausal females participated in a 12 month exercise program and were compared to a group of 38 sedentary females. The groups were matched by age, BMI, and activity levels. The exercise group participated three times per week for 30 minutes at a prescribed intensity (20 repetitions at 60% of one repetition maximum). There was a non-significant increase in lumbar BMD in the weight lifting group (0.81%) while the control group had a non-significant decrease of 0.5%. A paired t-test was used to determine posttraining group differences between exercises and controls. The test revealed a significantly higher lumbar BMD in the weight lifting versus control group following training. However, Gleeson et al. (1990) concluded that moderate resistance training may not be the answer for osteoporosis. They believed that the time and effort

necessary to sustain this type of exercise program far exceeds the small benefits that one can hope to gain.

The results of Rockwell et al. (1990) and Gleeson et al. (1990) indicate that resistance training may not be the most appropriate type of exercise to increase BMD. It is possible that the nine and 12 month training period used in these studies were not long enough to significantly increase BMD. In both studies, however, there were significant increases in strength. In light of these results, it appears that strength gains are not necessarily correlated to increases in BMD.

Notelowitz, Martin, and Tesar (1991) compared ten females who strength trained and took estrogen supplements (TS) with ten females who took estrogen supplements (ES) only. The TS group increased their BMC by 8%. BMC of the ES group remained unchanged. Although the sample size was not large, the results imply that strength training combined with estrogen supplementation may improve BMC.

Menkes and colleagues (1993) studied the effects of strength training on BMD in middle aged and older males. Eighteen previously untrained males between 50 and 70 years of age were resistance trained for 16 weeks, three times per week. Their results were compared to seven inactive

controls of similar age and weight. BMD of the exercise group increased by 3.8% in the femoral neck and 2.0% in the lumbar spine ($p < 0.05$). The exercise group had increased regional BMD at all sites, however, the differences were not significant. The small changes occurring in BMD following a 16 week training period, led to the suggestion that males between the ages of 50-70 may not experience large changes in BMD if they participate in a short term resistance training program.

## Relationship Between Muscle, Strength, Size, and Bone Density

Improving muscular strength appears to be effective in the protection against osteoporotic fractures. It has been suggested that increased muscular strength not only has a direct physiological effect on bone but may also improve stability which serves to prevent the likelihood of falling, thereby reducing the risk of fracture (Munnings, 1992). However, future research is needed to establish if a relationship exists between BMc and strength training.

In a cross-sectional study on BMC and strength in inactive subjects, Sinaki, Opitz, Heinz, and Wahner (1974) found no significant correlation between age related loss of BMC and change in muscle strength. Results of this study

indicate that there is no relationship between decreases in strength (presumably due to inactivity) and decreases in BMC.

The relationship between bone density and strength is not yet fully understood. It is well known that heavy resistance training does increase strength, although the effects of strength training on bone density are not clear. In cross-sectional studies of males, premenopausal females, postmenopausal females, and rats, increases following resistance training were reported in strength and bone density, yet the authors failed to report the correlation between strength and bone density (Saville & Whyte, 1969; Chow et al., 1986; Colletti et al., 1989; Gleeson et al., 1990).

It is well documented that strength increases (even in older subjects) within 6-8 weeks, whereas bone remodeling is estimated to take a minimum of 4-6 months (Rickli & McManis, 1990). Many of the studies to date have failed to report a significant relationship between strength and BMC. In most cases, it is likely that the resistance training regimens were too short in duration to elicit significant bone mineral adaptations. Further research is warranted in which training of durations longer than 6-12 months should be

employed to study the influence of resistance training on bone remodeling.

Heavy resistance training is known to cause skeletal muscle hypertrophy in humans (Cureton, Collins, Hill, and McElhannon, 1988). Doyle, Brown, and LaChance (1970) proposed that the weight of a muscle reflects the forces that it exerts on the bone to which it is attached. The mechanical stress applied to bone is related to the architecture of that bone. The more pressure applied, the greater the bone formation within a genetically determined ceiling. Muscle weight, therefore, is an important determinant of bone mass.

Meema (1966) did a study to determine the changes in muscle mass and BMC with aging. The BMC in the proximal end of the shaft of the right radius and diameter of the forearm muscle mass were evaluated from a roentgenogram of the right upper extremity. No significant differences were found in the muscle diameters of the premenopausal and postmenopausal females. Conversely, a 19% greater BMC was found in the premenopausal group when compared to the postmenopausal group. It was concluded that the difference in BMC was not related to a difference in muscle diameter. Consequently, it appears that the progression of osteoporosis, as

indicated by a decrease in BMC in the postmenopausal group was associated with the onset and extent of menopause not muscle size.

However, Doyle et al. (1970) explained that posttreatment fractures are often experienced in patients suffering from severe renal failure and are treated by hemodialysis. The majority of these patients have severe myopathy. The myopathy itself may be an important determinant of bone loss that precedes these fractures. Similarly, severe osteomalacia patients often suffer extreme muscle atrophy. After the osteomalacia is cured, the concentration of bone mineral in the periphery often remains low, suggesting that reduced bone mass may be due in part to the myopathy. Lastly, Doyle et al. reminded us that anabolic steroids are used to inhibit further bone loss in osteoporotic patients and limit further deterioration via promoting muscle hypertrophy.

Saville and Whyte (1969) evaluated muscle hypertrophy and BMC in exercising rats. The authors hypothesized that muscle hypertrophy was associated with an increase in BMC. They found that rats who participated in running had comparable increases in muscle size and BMC. The authors are confident that these findings can be generalized to

humans, particularly adolescents.

Munnings (1992) believes that inactivity may cause a decrease in BMC. A lack of intervention may lead to more serious problems such as osteoporosis. According to Munnings, losses in BMC can be avoided with the appropriate combination of physical activity, estrogen replacement, and calcium supplementation.

## Supplementation and Bone Density

Calcium supplementation is valuable in slowing age-related bone loss. Calcium plays an integral part in the bone building process, however, estrogen must be available to prevent bone loss (Munnings, 1992). Without adequate amounts of estrogen, the true benefits of calcium can not be realized because bone loss often exceeds bone formation (Heaney, 1982).

### Calcium

Dietary calcium is a vital part of skeletal health. Peak bone mass appears to be associated with calcium intake from early childhood to young adulthood. The recommended dietary allowance of calcium has recently been increased to 1200 mg/day through the age of 24. Recommended calcium intake in the adult and elderly remain controversial. The range varies from 800 mg/day to 1500 mg/day, and calcium

intake should be achieved through a well-balanced diet. If this is not possible, supplementation may be advised (Sanborn, 1990).

Calcium supplementation in a group of elderly women (age $\underline{M}$ =81) prevented further bone loss and actually increased bone density over a period of 36 months (Smith et al., 1981). The group in this study who received supplementation experienced a decrease in bone width while BMC increased by 1.50% ($p < 0.07$) resulting in an increase of BMC to width ratio. The increase in BMC following calcium supplementation may have resulted in a change of negative calcium balance to near neutral or positive balance, via enhanced intestinal absorption. This important finding has large implications for the elderly population at risk of suffering osteoporosis. An increase in BMC due to calcium supplementation may reduce the risk of fractures in the elderly.

Extreme disuse of bone often leads to the secretion of calcium. There is also reambulation of calcium in the oscalcis of the bone following long periods of inactivity (Donaldson et al., 1970). Donaldson et al. looked at the effects of 30-36 weeks of bed rest on BMC in three healthy males. Calcium loss during the entire period averaged 6.2%

of estimated total body calcium. Similarly, Manzke, Chestnut, Wergedal, Baylink, and Nelp (1976) found a group of osteoporotic patients continued to lose BMC as they aged. The authors speculated that maximum BMC is determined at skeletal maturity and followed by an inevitable decrease in BMC. The results of these two studies imply that disuse and aging may have the same severe effect on BMC in the skeleton.

Researchers investigated the relationship between spinal trabecular bone density and the intake of dietary calcium in amenorrheic and estrogen repleated athletes. Sixty-seven elite female athletes participated in this study of which 25 were amenorrheic, 27 were eumenorrheic, and 15 were taking oral contraceptives. QCT was used to measure BMC. Calcium intake and estrogen supplementation were determined through a questionnaire. The mean bone density of amenorrheic athletes (168 $mg \cdot cm^{-3}$) was significantly lower than the eumenorrheics (211 $mg \cdot cm^{-3}$) and oral contraceptive group (215 $mg \cdot cm^{-3}$, $p < 0.01$). Researchers found a positive linear relationship between calcium intake and trabecular bone density indicating that trabecular bone density does increase with calcium supplementation. However, factors which may determine calcium intake such as energy intake and

expenditure, were not measured. Therefore, it is possible that the relationship between increased calcium intake and BMD in young women is not as strong as the results imply (Wolmon, Clarke, McNally, Harries, and Reeve, 1992).

Heaney (1982) suggested that all individuals should incorporate calcium supplementation with physical activity to attain the highest peak bone mass prior to old age. Thereafter, one should try to maintain peak bone mass for as long as possible.

Sales of calcium supplementation increased by more than 300% from 1983 to 1987. However, the majority of epidemiologic studies have failed to support the hypothesis that increased calcium ingestion improved bone density (Martin and Houston, 1987). Anderson (1990) suggested that adolescents and young adults may enhance their BMD through physical activity, sufficient intake of calcium and other nutrients especially protein, phosphorus, and vitamin D. Physical activity increases the efficiency of intestinal absorption and improves skeletal uptake of calcium. Ingestion of vitamin D increases the blood level of minerals, notably calcium and phosphorus, permitting bone formation and maintenance (Hamilton, Whitney, and Sizer, 1988).

**Estrogen**

Decrease in bone mass is most often linked to a lack of estrogen. There is marked acceleration of bone loss following menopause that may be limited by estrogen supplementation (Oyster et al., 1983). Drinkwater et al. (1984) compared femoral BMC in amenorrheic and eumenorrheic college-aged female athletes. A significantly higher lumbar BMC of 16% was observed in the eumenorrheic group. Bone density of the eumenorrheic group was similar to age predicted levels, and bone density of the amenorrheic group was below the fracture threshold of 0.965 $gm \cdot cm^{-3}$. Low BMD in amenorrheic female athletes is linked to low estrogen levels. The exact relationship of estrogen and BMD has not yet been determined. It is believed that the effects of estrogen on bone are indirect, due to the fact that there are no estrogen receptors on bone.

A follow-up study was completed to determine BMC after resumption of menses in amenorrheic athletes. It was found that six of the seven subjects had a 6.2% increase in vertebral bone density 14.4 months after the resumption of menses. It is not known at this time whether the BMC of these subjects will reach normal levels, however, the reported increase in BMC is encouraging. These results

imply a positive relationship between estrogen levels and bone density (Drinkwater, Nilson, Ott, and Chestnut, 1986).

Mazess (1981) speculated that estrogen replacement clearly inhibits postmenopausal loss of compact bone, however, estrogen protection apparently decreases with long-term treatment. Estrogen works chiefly on the menopause-induced rapid phase of compact bone loss but not on the slower phase. The effects of estrogen on BMC requires further research to clarify this relationship.

Buchanan (1988) reported no relationship between the level of estrone, a form of estrogen, and vertebral bone density ($r = 0.19$) in 30 young females of similar weight. Contrary to this, Dalsky (1990) found estrogen deficient postmenopausal women to have low lumbar BMD and increased incidence of fracture of the vertebrae and proximal femur. To further clarify the relationships of estrogen, exercise and calcium, longitudinal research would be wise to monitor diet and diet supplements at frequent intervals throughout the study (Drinkwater et al., 1986).

## Summary

Research has been unable to determine the single, most effective means of increasing bone mass. The information acquired from studies on BMD report conflicting results,

which make it difficult to develop a recommendation for combatting bone loss. The majority of researchers suggest that BMD is increased through the interaction of physical activity, calcium supplementation, and estrogen availability. According to some of the research, the most effective means of maintaining BMD is to achieve the maximum amount of BMD possible at a young age through activity and diet, and minimize loss at an older age through activity, diet, supplementation, and hormone intervention.

The type of activity best suited for enhancing bone density has not been determined. The use of exercise may have only limited value in affecting BMD. The widespread recommendation for the prophylactic use of exercise should await further validation studies using better methodology to acquire more accurate results. There is some evidence suggesting that weight-bearing exercise increases BMD, however, very few studies have examined the impact of resistance training on BMD over a long period of time. Resistance training may an be important factor in increasing BMD via the mechanical forces imparted on bone. Drinkwater et al. (1986) strongly suggested that future longitudinal studies on the effects of exercise and supplementation on BMD should monitor exercise, diet, and supplements at

frequent intervals. It is clear that more research is needed on factors that influence BMD if osteoporosis is to be eliminated.

## METHODOLOGY

This chapter outlines the methods and procedures to be used in this study. A detailed description of the (a) selection of subjects, (b) testing schedule, (c) bone density evaluation, (d) strength testing, (e) body composition, (f) maximum oxygen consumption, and (g) statistical analyses are presented in this section.

### Selection of Subjects

Twenty-six premenopausal females between the ages of 18-32 will be recruited to participate in this study. Subjects who meet the criteria for R or S will complete an informed consent document (Appendix A). Classification of subjects will be determined following completion of a medical history questionnaire (Appendix B) and a physical activity questionnaire (Appendix C). Criteria for inclusion as a R subject will include; 1) regular participation in a weight training program for the previous three or more years, 2) average frequency of weight training of three or more times per week, 3) average length of training session of at least one hour, 4) no more then three consecutive months off weight training in the past three years. R subjects

will not have used anabolic steroids at any time prior to the study. For a S subject to be included in the study she must have been inactive (has not participated in physical activity more than once a week) for the previous year. Menstrual status for both R and S will be determined using the medical history questionnaire prior to acceptance into the study. No subject will be admitted into the study if she has missed two consecutive menstrual cycles or if her cycle is irregular. Dietary habits and vitamin or mineral supplementation for both R and S will also be determined through the medical history questionnaire.

**Testing Schedule**

Subjects will report to the Department of Nuclear Medicine at the State University of New York Health Science Center for bone density analysis on the first day of testing. The second and third day of testing will be in the Exercise Physiology Laboratory in Hill Center at Ithaca College. During the first session, BMD will be measured using DEXA. The second day of testing will include analysis of body composition by hydrostatic densitometry and measurement of strength. On the third day of testing, maximum oxygen consumption

($VO_2$max) will be measured.

## Bone Density Evaluation

BMD will be measured by DEXA with the Hologic QDR-1000/W (Waltham, MA). Orwoll and Oviatt (1991) evaluated the consistency of eight Hologic QDR 1000/W machines to analyze BMD of the lumbar spine and hip. They reported a variability between scanners of only 0.7%. The long term precision of the eight scanners tested differed by only 0.43%. The differences at each anatomical site were 1.1% for the spine, 1.2% for the femoral neck, 1.3% for the trochanter, and 2.4% for Ward's triangle. These data were obtained by testing subjects who periodically visited the clinic (Orwoll and Oviatt, 1991).

## Position of Subject for Spine Scan

Positioning for the spine scan will be as follows: The subject will be supine on the scanning table with lower legs and feet elevated approximately two inches on a cushion, thus creating a 135 degree angle at the knee. This placement ensures that the spine will be parallel and between the scan limit lines on the table. The scanner arm will be positioned one to two inches proximal to the xiphoid process and will scan to one to

two inches below the anterior iliac crest.

Scanning will begin at mid L5 such that a small amount of the pelvis will be visible at the bottom corners of the scan image. If the pelvis is not visible in the scan image, the scan will be repeated after the subject is repositioned. Bone density will be determined for L1-L4.

**Position of Subject for Hip Scan**

The subject will be supine on the scanning table with the right foot and knee stabilized such that the hip is positioned midway between the scan limit lines. The scan will begin at approximately two inches below the level of the femoral head. The areas which will be scanned include the femoral head, and the greater and lesser trochanters of the right hip.

**Position of Subject for Forearm Scan**

The subject will be seated on a chair next to the scanner such that the entire length of the forearm and hand will be placed on the long axis of the table. The forearm will be stabilized in position by having the subject press gently against the edge of a foam block. The entire length of the subject's forearm and hand will be in contact with the block. The non-dominant

forearm will be scanned from the ulnar styloid to the olecranon process of the ulna.

**Position of Subject for Whole Body Scan**

The subject will be supine on the scanning table with the head positioned below a lucite block located at the proximal end of the scanning table. The body will be aligned with the longitudinal axis of the table with the hands completely pronated and resting on the scanning table.

**Strength Testing**

Strength will be tested using a Universal Gym (Cedar Rapids, IA). Upper body strength will be determined using a bench press and shoulder press. Lower body strength will be assessed using leg press, leg extension, and leg curl. Maximum strength will be defined as a one repetition maximum contraction (1 RM) that will be determined in the following manner: 1) The subject will complete a warm up routine prior to each strength measure. The routine will consist of two sets of 15-20 repetitions followed by static stretching. 2) Subjects will estimate their 1 RM and the starting point will then be set at approximately 10% less then this estimation. 3) Weight increments

will be increased by 1 to 4.5 kg after each successfully completed repetition. Following each successful repetition the subject will be given a two minute rest. 4) The 1 RM will be determined as the highest weight that is lifted successfully (through a full range of motion).

**Leg Press**

The subject will be seated in the leg press apparatus with the knees flexed at approximately 110 degrees. The subject will forcefully extend her legs against a resistance to within five degrees of complete knee extension. The weight will be increased 4.4 or more kg following each successfully completed repetition.

**Leg Curl**

The subject will be prone with the leg curl apparatus positioned approximately two inches proximal to her ankle joint. The legs will be fully extended at the knee joint. The subject will forcefully flex her knees against a resistance to at least 90 degrees of flexion. The weight will be increased 2.2 or more kg following each successfully completed repetition.

**Leg Extension**

The subject will be seated with the knees flexed at 90 degrees. The subject will forcefully extend both knees against a resistance to within five degrees of complete knee extension. The weight will be increased 2.2 or more kg following each successfully completed repetition.

**Bench Press**

For the bench press, the subject will be supine on a bench with the chest aligned directly below the bench press handles. While grasping the handles at shoulder width, the subject will forcefully extend her elbows against a resistance to within five degrees of complete elbow extension. The weight will be increased one or more kg following each successfully completed repetition.

**Shoulder Press**

The subject will be seated on a bench such that the acromion process is aligned with the handles of the shoulder press apparatus. While grasping the handles, the subject will forcefully flex her shoulders against a resistance to within five degrees of complete shoulder flexion and elbow extension. The weight will

be increased one or more kg following each successfully completed repetition.

## Body Composition

Hydrostatic densitometry will be used to determine the percentage of body fat. To determine underwater weight, the subject will be seated in a chair suspended in a four foot water tank. The subject will flex her trunk to bring her head toward her knees and completely submerge. At the same time she will exhale to residual volume (RV). The subject will then hold her breath for 3-5 seconds while in a bent forward position. This procedure will be completed eight to ten times. The last three trials will be used to calculate body volume. Underwater weight will be measured via a load cell (Transducers, INC, Cerritos, CA) interfaced with an IBM computer. Percent fat will be determined using the equation of Siri (1961). Hydrostatic weight will be corrected for RV, which will be determined using the oxygen dilution procedure (Wilmore, 1969) immediately prior to hydrostatic weighing. Two trials of RV will be performed with a five minute time period between each trial. If the two trials differ by more than 150 ml, a third trial will be completed. An average of the

two closest trials will be used in the calculation of body volume.

**Maximum Oxygen Consumption**

$VO_2$max will be determined using a continuous treadmill protocol. Subjects will run at a self-selected pace determined during a three minute warm-up stage (0% grade) at the beginning of the test. Each stage thereafter will be two minutes long. The speed will remain constant throughout the test and the grade will be increased 2.5% each stage. The test will be terminated upon volitional exhaustion of the subject.

The three criteria which will be used to determine the attainment of $VO_2$max are; 1) maximum heart rate (MHR) within minus 10 bpm of age predicted MHR (220 - age), 2) respiratory exchange ratio (RER) greater than 1.10, 3) leveling off in $VO_2$ between the final two workloads. To determine if the subject will satisfy the leveling off criterion, the difference in $VO_2$ between the final two workloads must be smaller than the differences between any two successive, previous workloads, excluding the first and second workload.

Oxygen consumption will be measured via open circuit spirometry using the SensorMedics 2900 metabolic measurement system (SensorMedics, Yorba Linda, CA). Oxygen consumption will be calculated and printed every 20 seconds.

## Statistical Analysis

Descriptive statistics will be used to analyze and define the physical characteristics of the groups. Paired t-tests will be employed to compare the two groups on height, weight, body composition, strength, $VO_2max$, and BMD of the spine, hip, forearm, and whole body. Pearson product-moment correlation will be used to assess the relationship among variables. Statistical significance will be established at the 0.05 level for all analyses.

## REFERENCES

Abramson, A. S., & Delagi, F. D. (1961). Influence of weight-bearing and muscle contraction on disuse osteoporosis. Archives of Physical Medicine and Rehabilitation, 42, 147-151.

Aloia, J. F. (1989). Osteoporosis: A guide to prevention and treatment. Champaign, IL: Leisure press.

Aloia, J. F., Cohn, S. H., Babu, T., Abesamis, C., Kalici, N., & Ellis, K. (1978). Skeletal mass and body composition in marathon runners. Metabolism, 27(12), 1793-1796.

Anderson, J. B. (1990). Dietary calcium and bone mass through the lifecycle. Nutrition Today, March/April.

Bell, D. G., & Jacobs, I. (1990). Muscle fiber area, fiber type and capilarization in male and female body builders. Canadian Journal of Sport Science, 15(12), 115-119.

Block, J. E., Friedlander, A. L., Brooks, G. A., Steiger, P., Harrison, A. H., & Genant, H. K. (1989). Determinants of bone density among athletes engaged in weight-bearing and non weight-

bearing activity. Journal of Applied Physiology, 67(3), 1100-1105.

Block, J. E., Smith, R., Friedlander, A., & Genant, H. K. (1989). Preventing osteoporosis with exercise: A review with emphasis on methodology. Medical Hypothesis, 30, 9-19.

Brewer, V., Meyer, B.M., Keele, M. S., Upton, J., & Hagan, R. D. (1983). Role of exercise in prevention of involutional bone loss. Medicine and Science in Sports and Exercise, 15(6), 445-449.

Buchanan, J. R., Myers, C, Lloyd, T., Leuenberger, P., & Demers, L. M. (1988). Determinants of peak trabecular bone density in women: the role of androgens, estrogen, and exercise. Journal of Bone and Mineral Research, 3, 673-680.

Chow, R. K., Harrison, J. E., Brown, C. F., & Hajek, V. (1986). Physical fitness effect on bone mass in postmenopausal women. Archives of Physical Medicine and Rehabilitation, 67, 231-234.

Cohn, S. H., Vaswani, A., Zanzi, I., & Ellis, K. J. (1976). Effect of aging on bone mass in adult women. American Journal of Physiology, 230(1),

143-148.

Colleti, L. A., Edwards, J., Gordon. L., Shary, J., & Bell, N. H. (1989). The effects of muscle-building exercise on bone mineral density of the radius, spine, and hip in young men. Calcification Tissue Internation, 45, 12-14.

Cureton, K. J., Collins, M. A., Hill, D. W., & McKllhannon, F. M. (1988). Muscle hypertrophy in men and women. Medicine and Science in Sports and Exercise, 20(4), 338-344.

Dalen, N., & Olsson, K. E. (1974). Bone mineral content and physical activity. Acta Orthopedic Scandenavica, 45, 170-174.

Dalsky, G. (1989). The role of exercise in the prevention of osteoporosis. Comprehensive Therapy, 15, 30-37.

Dalsky, G. (1990). Effect of exercise on bone: permissive influence of estrogen and calcium. Medicine and Science in Sports and Exercise, 22, 281-285.

Donaldson, C. L., Hulley, S. B., Vogelm, J. M., Hattner, R. S., Bayers, T. H., & McMilllan, D. E. (1970). Effect of prolonged bed rest on bone

mineral. Metabolism, 19(12), 1071-1084.

Doyle, D., Brown, T., & Lachance, C. (1970). Relation between bone mass and muscle weight. Lancet, 1, 391-393.

Drinkwater, B. L., Bruemner, B., Chestnut, C. H. (1990). Menstrual history as a determinant of current bone density in young athletes. Journal of American Medical Association, 263, 545-548.

Drinkwater, B. L., Nilson, K., Chestnut, C. H., Bremner, W. J., Shainholtz, S., & Southworth, M. B. (1984). Bone mineral content of amenorrheic and eumenorrheic athletes. The New England Journal of Medicine, 311(5), 277-281.

Drinkwater, B. L., Nilson, K., Otts, S., & Chestnut, C. H. (1986). Bone mineral density after resumption of menses in amenorrheic athletes. Journal of American Medical Association, 256(3), 380-382.

Gleeson, P. B., Protas, E. J., LeBlanc, A. D., Schneider, V. S., & Evans, H. J. (1990). Effects of weight lifting on bone density in premenopausal women. Journal of Bone and Mineral Research, 5(2), 153-158.

Gray, H. (1974). Gray's Anatomy, 189.

Guyton, A. C. (1987). Human physiology and mechanism of disease (4th ed.). Philadelphia: WB Saunders Company.

Hamilton, E., Whitney, E., & Sizer, F. (1988). Nutrition (4th ed.). New York: West Publishing Company.

Heaney, R. P. (1982). Diet and exercise [summary], presented under the auspices of the Florida Medical Association and the American Medical Association, (pg. 1106). Journal of American Medical Association, 247(8).

Huddleston, A. L., Rockwell, D., Kulund, B. N., & Harrison, R.B. (1980). Bone mass in lifetime tennis athletes. Journal of American Medical Association, 244, 1107-1109.

Jacobson, P. C., Beaver, W., Grubb, A., Taft, T.N., & Talmage. R.V. (1984). Bone density in women: college athletes and older athletic women. Journal of Orthopaedic Research, 2, 328-332.

Jenkins, D. P., & Cochran, T. H. (1969). Osteoporosis: The dramatic effect of disuse of an extremity. Clinical Orthopaedics and Related

Research, 64, 128-134.

Kleerekoper, M, anad Avioli, L. V. Evaluation and Treatment of Postmenopausal Osteoporosis, In: Favus, M. J. (Ed.) Primer on Metabolic Bone Diseases and Disorders of Mineral Metabolism, 1st ed. 151-154, American Society for Bone and Mineral Research, Kelseyville, CA., 1990.

Manzke, E., Chestnut, C. H., Wergedal, J. E., Baylink, D. J., & Nelp, W. B. (1975). Relationship between local and total bone mass in osteoporosis, Metabolism, 24(5), 605-615.

Marcus, R., Cann, C., Madvig, P., Minkoff, J., Goddard, M., Bayer, M., Martin, M., Gaudiani, L., Haskell, W., & Genant, H. (1985). Menstrual function and bone mass in elite women distance runners. Annals of Internal Medicine, 102, 158-163.

Martin, A. D., & Houston, C. S. (1987). Osteoporosis, calcium, and physical activity. Canadian Medical Association, Journal, 136, 587-593.

Mazess, R. B. (1982). On aging bone loss. Clinical Orthopaedics and Related Research, 165, 239-252.

Mazess, R. B., Howard, S. B., Bisek, J. P., Hanson, J. (1990). Dual energy x-ray absorptiometry for

total body and regional bone-mineral and soft-tissue composition. American Journal of Clinical Nutrition, 51, 1106-1112.

Mazess, R. B., Peppler, W. W., Chesney, R. W., Lange, T. A., Lindgren, U., & Smith, E. (1984). Does bone measurement on the radius indicate skeletal status? Concise communication. Journal of Nuclear Medicine, 25, 281-288.

Mazess, R. B., & Whedon, G. D. (1983). Immobilization and bone. Calcification. Tissue Innovation, 35, 265-267.

Meema, H. E. (1966). Menopausal and aging changes in muscle mass and bone mineral content. The Journal of Bone and Joint Surgery, 48(6), 1138-1144.

Menkes, A., Mazel, S., Redmond, A., Koffler, K., Libanat, C. R., Gundberg, C. M., Zizic, T. M., Hagberg, J. M., Pratley, R. E., & Hurley, B. F. (1993). Strength training increases regional bone mineral density and bone remodeling in middle-aged and older men. Journal of Applied Physiology, 74(5), 2478-2484.

Munnings, F. (1992). Osteoporosis: What is the role of exercise? The Physical and Sports Medicine,

20(6), 127-138.

Nilsson, B. E., & Westlin, N. E. (1971). Bone density in athletes. Clinical Orthopaedics and Related Research, 77, 179-183.

Nutter, J. (1986). Physical activity increases bone density. National Strength and Conditioning Association Journal, 8(3), 67-69.

Oyster, N., Morton, M., & Linnell, S. (1983). Physical activity and osteoporosis in post-menopausal women. Medicine and Science in Sports and Exercise, 16(1), 44-50.

Purvis, A. (1990, December 5). When bones are brittle. Time, p. 82.

Raisz, L. G., & Smith, J. (1987). Osteoporosis in Martin T. J. Clinical Endocrinology of Calcium Metabolism, (pp. 201-248). New York: Marcel Dekker Inc.

Recker, R. R. (1984). The role of calcium in bone health. Nutritional News, 47(2), 5-6.

Rickli, R. E., & McManis, B. G. (1990). Effects of exercise on bone mineral content in postmenopausal women. Research Quarterly for Exercise and Sport, 61(3), 243-249.

Riggs, B. L., & Melton, L. J. (1986). Involutional osteoporosis. New England Journal of Medicine, 314, 1676-1686.

Risser, W. L., Lee, E. J., Leblanc, A., Poindexter, H. B. Risser, J. H., & Schneider, V. (1990). Bone density in eumenorrheic female college athletes. Medicine and Science in Sports and Excercise, 22(5), 570-574.

Rockwell, J. C., Sorensen, A. M., Baker, S., Leahey, D., Stock, J. L., Michaels, J., & Baran, D. T. (1990). Weight training decreases vertebral bone density in premenopausal women: A prospective study. Journal of Clinical Endocrinology and Metabolism, 71(4), 988-993.

Sanborn, C. F. (1990). Exercise, calcium, and bone density. Sports Science Exchange, 2(24).

Saville, P. D., & Whyte, M. P. (1969). Muscle and bone hypertrophy. Positive effect of running exercise in the rat. Clinical Orthopaedics and Related Research, 65, 81-88.

Sinakai, M., Opitz, J. L., & Wahner, H. W. (1974). Bone mineral content: Relationship to muscle strength in normal subjects. Archives of Physical

Medicine and Rehabilitation, 55, 508-512.

Siri, W. E. (1956). Gross composition of the body. In: Advances in Biological and Medical Physics, Vol. IV, (JH Lawrence, CA Tobias, Eds.). New York: Academic Press.

Smith, E. L., Reddan, W., & Smith, P. E. (1981). Physical activity and calcium modalities for bone mineral increase in aged women. Medicine and Science in Sports and Exercise, 13(1), 60-64.

Stillman, R. J., Lohman, T. G., Slaughter, M. H., & Massey, B. H. (1986). Physical activity and bone mineral content in women aged 30 to 85 years. Medicine and Science in Sports and Exercise, 18(5), 576-580.

Wilmore, J. H. (1969). A simplified method for determination of residual lung volume. Journal of Applied Physiology, 27, 96.

Wolman, R. L., Clark, P., McNally, E., Harries, M. G., & Reeves, J. (1992). Dietary calcium as a statistical determinant of spinal trabecular bone density in amenorrhoeic and oestrogen-replete athletes. Bone and Mineral, 17, 415-423.

Wolman, R. L., Faulmann, L., Clark, P., Hesp, R.,

Harries, M. G. (1991). Different training patterns and bone mineral density of the femoral shaft in elite, female athletes. Annals of the Rheumatic Diseases, 50, 487-489.

# BONE MINERAL DENSITY IN RESISTANCE TRAINED PREMENOPAUSAL FEMALES

A Research Manuscript

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## INTRODUCTION

Osteoporosis is a metabolic bone disease that occurs when normal mineralization of the bone is reduced to levels that increase risk of fracture in the absence of trauma or in response to relatively minor trauma. This occurs as a result of a decrease in volume of mineralized trabecular and cortical bone within the periosteal envelope, compromising the architectural integrity of the bone (Albanese, 1975). This disease causes physical disorders, emotional problems, and is a huge economic burden on society (Aloia, 1989; Purvis, 1990). Approximately 24 million Americans are affected by osteoporosis, and each year 1.3 million American women over the age of 40 fracture one or more bones (Sanborn, 1990). There is no known cure for osteoporosis, however researchers generally agree that this disease can be prevented by maximizing BMD at a young age through physical activity and diet. Experts also believe that it is possible to minimize loss at an older age through activity, diet, vitamin/mineral supplementation, and hormonal intervention (Dalsky, 1989; Sanborn, 1990).

Recent research has demonstrated the significance of weight bearing activity as a prophylactic means of maintaining BMD or minimizing the decrease in age related

bone demineralization (Recker, 1984; Marcus & Cann, 1985). The majority of exercise related studies have examined the impact of weight bearing activity (e.g., walking, dancing, running) on BMD. Little attention has focused on the effects of mechanical stress from resistance training on BMD. Resistance training may also be an important factor in maximizing BMD and maintaining bone mass throughout the aging process (Chow et al., 1986; Block et al., 1989; Menkes et al., 1993). According to authorities, the increase in BMD that may follow resistance training is believed to occur when the strain on bone elicits a biochemical signal thought to be mediated by an electrical field possibly arising from the piezo-electric effect (Brighton et al., 1985). It is proposed that a transient electrical potential occurs across the bone due to bending and loading of the bone. The piezo-electric field acts as a pulsed electric field that induces bone cell activity, leading to increased bone deposition at points of compressional stress (Lanyon and Hartman, 1977).

Some however, have failed to substantiate increases in BMD with resistance training (Rockwell et al., 1990; Gleeson et al., 1990). It is possible that short-term training used in some of these previous studies did not allow enough time to increase BMD. Therefore, the purpose of this

investigation was to compare BMD in chronically resistance trained and sedentary premenopausal females.

## METHODS AND PROCEDURES

### Subjects

Twenty six premenopausal females between the ages of 18-32 yrs participated in this study. Subjects were assigned to either a resistance trained (R) group ($n$ = 13) or sedentary (S) group ($n$ = 13). Criteria for inclusion as an R subject included; 1) regular participation in a weight training program for the previous three or more years, 2) average frequency of weight training of three or more times per week, 3) average length of training session of at least one hour, and 4) no more then three consecutive months off weight training in the past three years. R subjects did not use anabolic steroids at any time prior to the study. Criteria for S subjects required participation in physical activity to be less than two times per week for the previous year. The type of activity in which S subjects participated could not include resistance training. Menstrual status for both R and S was determined from a questionnaire (Appendix C) prior to acceptance into the study. No subject participated in the study if they had missed more than two consecutive menstrual cycles or if their cycles were

irregular. Oral contraceptives were used by six subjects in R for 1.7 ± 2.7 yrs, and by eight subjects in S for 1.6 ± 3.0 yrs. Pairs of R and S subjects were matched for weight (diff = 0.87 kg, $p > 0.05$) and height (diff = 5.4 cm, $p = 0.02$). Written informed consent was obtained from each subject prior to participating in the study (Appendix A).

## Bone Mineral Density

BMD was measured using dual energy x-ray absorptiometry (DEXA) (Hologic QDR-1000/W, Waltham, MA). Using this technique, Orwell and Oviat (1991) reported a variability of 0.7% between scanners. The anatomical sites scanned included the spine (L1-L4), femoral neck, forearm (radius total and ulna total), and whole body with BMD expressed in $g \cdot cm^{-2}$. Subject positioning was consistent with manufacturer's guidelines.

## Strength Testing

Strength was tested using a Universal Gym (Cedar Rapids, Iowa). One repetition maximum (1 RM) strength was determined for the bench press, shoulder press, leg press, leg extension, and leg curl exercises. The 1 RM was determined in the following manner: 1) The subject warmed up before every strength assessment by performing two sets of 15-20 repetitions with a weight that was approximately 50%

less than their self-estimated 1 RM, followed by static stretching; 2) The first attempt for both the R and S subjects was approximately 10% less than their self-estimated 1 RM; 3) Weight increments were increased by 1 to 4.5 kg after each successfully completed repetition until the weight could no longer be lifted. Following each successful repetition the subject was given a 2 minute rest period; 4) A 1 RM was determined as the heaviest weight lifted prior to failure. The 1 RM was determined within 4-6 trials.

For subsequent statistical analyses, upper body (UB) strength was calculated as the sum of the highest bench press and shoulder press scores. Lower body (LB) strength was determined by summing the highest leg press, leg extension, and leg curl scores.

**Body Composition**

Hydrostatic densitometry was used to determine percent body fat (%Fat). Underwater weight was measured with a load cell (Transducers, INC, Cerritos, CA) interfaced via an A/D converter with an IBM compatible computer, and programmed to calculate %Fat using the Siri (1956) equation. Body composition was calculated after correction for residual volume. Residual volume was measured on land, in a

seated position, out of water using the oxygen dilution procedure (Wilmore, 1969) immediately prior to hydrostatic weighing.

**Maximum Oxygen Consumption**

$VO_2$max was determined using a continuous treadmill protocol. Subjects ran at a self selected pace determined during a three minute warm-up stage (0% grade) at the beginning of the test. Each stage thereafter was 2 min with speed remaining constant and grade increasing 2.5% each stage. The test was terminated upon volitional exhaustion of the subject. Oxygen consumption was measured via open circuit spirometry using the SensorMedics 2900 Metabolic Measurement System (Yorba Linda, CA).

Three criteria were used to determine the validity of the $VO_2$max test and included; 1) maximum heart rate (MHR) within minus 10 bpm of age predicted MHR (220-age), 2) respiratory exchange ratio (RER) greater then 1.10, and 3) plateau in $VO_2$max with an increase in workload. A plateau in $VO_2$max was achieved if the difference in the two consecutive workloads was smaller than the mean difference between any two successive, previous workloads, excluding the first and second workload. All subjects met two or more of the criteria for achieving $VO_2$max.

## Statistical Analysis

It was assumed that the procedure of matching R and S subjects would effectively remove within pair variability in weight and height, and the resultant influence on BMD. Based on this assumption, a paired t-test was used to compare the physical characteristics of the groups. In addition, a paired t-test was used to compare strength, $VO_2$max, and BMD of the matched pairs. Pearson product-moment correlation coefficients were determined to evaluate the relationships among variables. Statistical significance was established at the 0.05 level.

## RESULTS

The physical and training characteristics for the R and S groups are shown in Table 1. The R group had trained for the previous 4.3 yrs (range = 3-8 yrs). While the groups varied little in age and weight, the S group was 5.2 cm shorter and considerably higher in %Fat (27.4 vs 18.1%) than the R group. The R group had a lean body mass that was 15% greater than the S group. Table 2 includes $VO_2$max and strength measures for both groups. $VO_2$max was higher and strength was 23 to 76% greater in the R group ($p < 0.01$). The composite scores of UB and LB strength were 34% and 42% higher respectively, in the R group ($p < 0.01$).

The BMD measurements for spine (L1-L4), femoral neck, forearm, and whole body are displayed in Table 3. BMD was significantly higher in the R subjects for the ulna (10%), radius (8%) and whole body (5%) measures ($p < 0.05$). Although R had a BMD at the femoral neck that was 8% higher than S, the difference approached, but did not attain statistical significance ($p = 0.058$). Likewise, a 4% higher spine BMD in R was not statistically different from S ($p = 0.12$). While others have reported smaller non-significant differences at the spine following resistance training (Colleti et al., 1989; Gleeson et al., 1990; Rockwell et al., 1990), it is likely that the coefficient of variation of 7-8% for the present subjects would have been reduced with a larger sample size, and possibly yielded significant differences at this site.

Pearson product moment correlations for BMD, $VO_2max$, strength, and body composition for the R group indicated significant correlations ($p < 0.05$) between strength measures and BMD (Table 4). Significant correlations were found between UB strength and BMD for ulna total ($r = 0.45$) and radius total ($r = 0.41$). Likewise, LB strength correlated significantly with BMD at the spine ($r = 0.47$), femoral neck ($r = 0.39$), ulna total ($r = 0.52$,) and radius

total (r = 0.49). No significant correlations, however, were observed in the S group for UB or LB strength and BMD (Table 5).

## DISCUSSION

In the present study, significantly higher BMD in R compared to S subjects at the forearm (ulna total, radius total) and whole body are attributed to the practice of heavy resistance training that differentiated these groups. Studies have shown with few exceptions that individuals exposed to high levels of physical activity have a greater BMD than less active individuals, regardless of the sites measured (Brewer et al., 1983; Block et al., 1987; Block et al., 1989). It has been suggested that weight-bearing activities are an effective means to enhance and maintain bone density (Wolman et al., 1992). However, little research has been done to differentiate the most effective types of weight-bearing activities necessary to enhance bone density. The results of the present study support the postulate that strength training is an effective activity for increasing BMD in premenopausal females.

Previous studies on other populations support the findings of the present study. Conroy et al. (1993) reported greater BMD in male junior olympic weight lifters

**TABLE 1**

**Subject Characteristics for Resistance Trained and Sedentary Groups**

| | Resistance trained (n = 13) | Sedentary (n = 13) |
|---|---|---|
| | **Mean (± SD)** | **Mean (± SD)** |
| Age (yrs) | 24.4 ± 1.04 | 22.8 ± 1.31 |
| Height (cm) | 171.3 ± 5.46 | 166.1 ± 7.38** |
| Weight (kg) | 61.2 ± 8.14 | 60.8 ± 9.63 |
| %Fat | 18.1 ± 4.47 | 27.4 ± 8.52** |
| LBM | 110.2 ± 16.17 | 95.5 ± 9.85** |
| Training (yrs) | 4.3 ± 1.54 | 0.0 ± 0.00** |

Values are mean ± SD

LBM = lean body mass

**$p < 0.01$

**TABLE 2**

**Dependent t-test for $VO_2$max and strength measures for R and S matched pairs**

| | Resistance trained (n = 13) | Sedentary (n = 13) | |
|---|---|---|---|
| | **Mean (± SD)** | **Mean (± SD)** | **% Diff** |
| $VO_2$max ($ml \cdot kg^{-1} \cdot min^{-1}$) | 48.2 ± 6.55 | 35.8 ± 5.43** | 35 |
| Bench Press (kg) | 51.3 ± 12.16 | 35.8 ± 8.29** | 43 |
| Shoulder Press (kg) | 39.1 ± 6.32 | 31.7 ± 5.01** | 23 |
| Leg Press (kg) | 185.3 ± 33.46 | 140.6 ± 25.94** | 32 |
| Leg Curl (kg) | 16.1 ± 3.67 | 9.1 ± 3.15** | 76 |
| Leg Extension (kg) | 96.2 ± 11.42 | 59.8 ± 12.50** | 61 |
| Upper body | 90.4 ± 17.71 | 67.4 ± 12.96** | 34 |
| Lower body | 297.6 ± 42.07 | 209.5 ± 37.40** | 42 |

Values are mean ± SD

Upper body = sum of bench press, shoulder press (kg)

Lower body = sum of leg press, leg curl, leg extension (kg)

$\% \text{ Diff} = \frac{R-S}{S}$

**$p < 0.01$

**TABLE 3**

**Dependent t-test between R and S matched pairs for BMD of the spine, hip, forearm, and, whole body**

| | Bone Mineral Density ($g \cdot cm^{-2}$) | | | |
|---|---|---|---|---|
| | Resistance trained (n = 13) | Sedentary (n = 13) | | |
| | Mean (± SD) | Mean (± SD) | % Diff | p |
| Spine (L1-L4) | 1.092 ± 0.092 | 1.049 ± 0.078 | 4 | 0.126 |
| Femoral neck | 0.923 ± 0.103 | 0.855 ± 0.101 | 8 | 0.058 |
| Ulna total | 0.545 ± 0.050 | 0.496 ± 0.042 | 10 | 0.021* |
| Radius total | 0.586 ± 0.045 | 0.545 ± 0.042 | 8 | 0.023* |
| Whole body | 1.139 ± 0.075 | 1.080 ± 0.086 | 5 | 0.028* |

Values are mean ± SD

$\% \text{ Diff} = \frac{R-S}{S}$

*$p < 0.05$

**TABLE 4**

Pearson product-moment correlation coefficients for BMD, strength, $VO_2max$, and body composition for R subjects (n=13)

| BMD site | %Fat | LBM | $VO_2max$ | UB | LB |
|---|---|---|---|---|---|
| Spine | .43* | .53** | .16 | .48* | .47* |
| Femoral neck | .35 | .41* | .30 | .41* | .39* |
| Ulna total | .53** | .57** | .36 | .45* | .52** |
| Radius total | .52** | .57** | .41* | .41* | .49* |
| Whole body | .26 | .71** | .24 | .56** | .50** |

*$p < 0.05$

**$p < 0.01$

**TABLE 5**

**Pearson product-moment correlation coefficients for BMD, strength, $VO_2max$, and body composition for S subjects (n=13)**

| BMD site | %Fat | LBM | $VO_2max$ | UB | LB |
|---|---|---|---|---|---|
| Spine | -.15 | .52* | -.13 | .51 | .47 |
| Femoral neck | -.52 | .26 | .33 | .25 | .30 |
| Ulna total | -.41 | .63* | .20 | .31 | .44 |
| Radius total | .56* | .62* | .45 | .23 | .33 |
| Whole body | .45 | .77** | .28 | .51 | .49 |

*$p < 0.05$

**$p < 0.01$

at the lumbar spine and proximal femur. Similarly, Menkes et al. (1993) reported significant increases of 2% and 3.8% in BMD at the spine and femoral neck respectively, in older males (age: $\underline{M}$ = 59 yrs) who resistance trained for 16 weeks.

There are some studies, however, which contradict the present results. Rockwell et al. (1990) and Gleeson et al. (1990) reported little or no change in the BMD of females who resistance trained for 9 and 12 months, respectively. It is possible that differences in BMD development between these studies and those finding positive results may be related to training volume. Conroy et al. (1993) tested subjects that had trained for an average of 2.7 ± 1.4 yrs which was substantially longer than the training periods used by either Rockwell et al. (1990) or Gleeson et al. (1990). The increased BMD reported in the rather brief, longitudinal study conducted by Menkes et al. (1993) may be related to the level of training intensity. Menkes et al. trained their subjects at a resistance of 85% of 1 RM, whereas Gleeson et al. (1990) used 60% of 1 RM. Therefore, the large differences in BMD observed between these studies may be due to either the length or intensity of training (i.e., training volume).

In the present study, R subjects had resistance trained

for at least three years. Although intensity of training sessions was not measured directly in the 13 R subjects, they reported participating in activities that required heavy resistance training of approximately 1-3 hours/day, 3-6 days/week. It is plausible that the relationship between training volume and BMD may be directly related to the effect of repeated and prolonged contractile activity on properties of bone development. Only exposure to a sufficient quantity and intensity of contractile stimuli may lead to measurable changes in bone density.

In the present study, measures of upper body and lower body strength were 34% and 42% higher, respectively, in the R group versus the S group. The R group also had 13% more lean body mass than the S group. It is well accepted that the volume of resistance training will dictate the degree of strength development. Recently, Conroy et al. (1993) argued that the mechanical loading of the bone during heavy resistance training is also the factor which accounts for an increase in BMD. This seems logical given the piezo-electric effect of a pulsed electrical field on bone cell activity as previously described by Lanyon and Hartman (1977). Given this common positive effect of resistance training upon both strength and BMD it might be speculated

that mechanical loading should lead to a spurious relationship between measures of strength and BMD in those who are chronically resistance trained. This argument is furthered when the correlational data from the present study are considered.

Many significant, albeit moderate, relationships ($r = 0.39 - 0.56$) between BMD and measures of strength in R subjects were observed in the present study. Remarkably, significant correlations ($r = 0.41 - 0.45$) where seen between UB strength and BMD measures in the upper body (i.e., ulna and radius) while LB strength measures were also significantly correlated ($r = 0.39 - 0.47$) with BMD at lower body sites (i.e., spine L1-L4 and femoral neck). Interestingly, in R subjects, LB strength was related to upper body BMD (i.e., ulna and radius) and UB strength was also related to lower body BMD (i.e., spine and femoral neck). This may be due to the need to regularly involve the lower body in load support and/or stabilization in upper body resistance training and the upper body in load support and/or stabilization in lower body resistance training. In contrast, the S group showed no significant relationships between strength and BMD.

It is postulated that chronic resistance training

drives both strength and BMD upward, ultimately leading to a positive, yet spurious, relationship between these variables. In the absence of training, the variability in BMD and strength is likely directed by a number of other factors, which do not necessarily have a common effect on these variables. For example, genetics and diet, which were not accounted for in the present study probably affect both BMD and strength but not necessarily in any common and consistent fashion which would cause a relationship between these variables. In the data set from this study, mechanical loading (i.e., chronic resistance training) may have caused an increase in both strength and BMD. It should be noted, however, strength did not likely cause an increase in BMD, and BMD did not increase strength. If this hypothesis is correct, then a group of subjects who do not chronically train, but who are genetically endowed with great strength should display no substantial relationship between strength and BMD. Accordingly, it is speculated that mechanical loading causes a spurious relationship between strength and BMD. The S group of the present study displayed no significant correlations between strength and BMD because they do not expose themselves to extensive mechanical loading.

It is difficult to determine a cause and effect relationship between resistance training and BMD in this cross-sectional study. These data, however, do provide reason to suggest that future longitudinal studies should examine the effect of resistance training on BMD in premenopausal females. Increasing BMD prior to the cessation of menses and maintaining BMD following menses through exercise, diet, hormonal intervention and vitamin/mineral supplementation is often considered a necessary measure for preventing the onset of osteoporosis (Dalsky, 1989; Sanborn, 1990). Longitudinal studies are required to determine how important a protective effect resistance training may have on osteoporosis. Furthermore, the effects of training intensity and duration should be examined in both pre and postmenopausal females. In fact, it would be prudent to examine specifically the impact of a resistance training program on BMD and osteoporosis if that program is instituted in the years that are predicted to immediately precede the onset of menses.

In summary, this is the first study to report site specific differences in BMD between resistance trained and sedentary premenopausal females for ulna, radius and whole body measures. Differences which did not achieve

statistical significance were also observed at the lumbar spine and femoral neck. An additional finding in this study was the existence of a significant relationship between strength and BMD only seen in resistance trained subjects. It is believed that this relationship is driven by a common effect of intense contractions over a prolonged period (i.e., chronic resistance training) on both strength and BMD. These findings support the argument that resistance training can be used as an effective tool to enhance BMD in premenopausal females.

**REFERENCES**

Albanese, A. A., Edelson, A. H., Woodhull, M. L., & Wein, E. H. (1975). Problems of bone health in elderly. New York State Journal of Medicine, Feb, 326-333.

Aloia, J. F. (1989). Osteoporosis: A guide to prevention and treatment. Champaign, IL: Leisure press.

Block, J. E., Smith, R., Black, D., Ettinger, B., Genant, H. K. (1987). Does exercise prevent osteoporosis? Journal of American Medical Association, 257, 3115-3117.

Block, J. E., Friedlander, A. L., Brooks, G. A., Steiger, P., Harrison, A. H., & Genant, H. K. (1989). Determinants of bone density among athletes engaged in weight-bearing and non weight-bearing activity. Journal of Applied Physiology, 67(3), 1100-1105.

Brewer, V., Meyer, B. M., Keele, M. S., Upton, J., & Hagan, R. D. (1983). Role of exercise in prevention of involutional bone loss. Medicine and Science in Sports and Exercise, 15(6), 445-449.

Brighton, C. T., Katz, M. J., Goll, S. R., Nichols C. E., & Pollack, S. R. (1985). Prevention and treatment of sciatic denervation disuse osteoporosis in the rat tibia with capacitatively coupled electrical stimulation. Bone, 6, 87-97.

Chow, R. K., Harrison, J. E., Brown, C. F., & Hajek, V. (1986). Physical fitness effect on bone mass in postmenopausal women. Archives of Physical Medicine and Rehabilitation, 67, 231-234.

Colleti, L. A., Edwards, J., Gordon. L., Shary, J., & Bell, N. H. (1989). The effects of muscle-building exercise on bone mineral density of the radius, spine, and hip in young men. Calcification Tissue Internation, 45, 12-14.

Conroy, P., Kraemer, W. J., Maresh, C. M., Fleck, S. J., Stone, M. H., Fry, A. C., Miller, P. D., & Dalsky, G. P. (1993). Bone mineral density in elite junior olympic weightlifters. Medicine and Science in Sports and Exercise, 25(10), 1103-1109.

Cureton, K. J., Collins, M. A., Hill, D. W., & McKllhannon, F. M. (1988). Muscle hypertrophy in men and women. Medicine and Science in Sports and Exercise, 20(4), 338-344.

Dalsky, G. (1990). Effect of exercise on bone: permissive influence of estrogen and calcium. Medicine and Science in Sports and Exercise, 22, 281-285.

Doyle, D., Brown, T., & Lachance, C. (1970). Relation between bone mass and muscle weight. Lancet, 1, 391-393.

Gleeson, P. B., Protas, E. J., LeBlanc, A. D., Schneider, V. S., & Evans, H. J. (1990). Effects of weight lifting on bone density in premenopausal women. Journal of Bone and Mineral Research, 5(2), 153-158.

Lanyon, L. E., Hartman, W. (1977). Strain related electrical potentials recorded in vitro and in vivo. Calcification Tissue Reserve, 22, 315-327.

Marcus, R., Cann, C., Madvig, P., Minkoff, J., Goddard, M., Bayer, M., Martin, M., Gaudiani, L., Haskell, W., & Genant, H. (1985). Menstrual function and bone mass in elite women distance runners. Annals of Internal Medicine, 102, 158-163.

Menkes, A., Mazel, S., Redmond, A., Koffler, K., Libanat, C. R., Gundberg, C. M., Zizic, T. M., Hagberg, J. M., Pratley, R. E., & Hurley, B. F.

(1993). Strength training increases regional bone mineral density and bone remodeling in middle-aged and older men. Journal of Applied Physiology, 74(5), 2478-2484.

Purvis, A. (1990, December 5). When bones are brittle. Time, p. 82.

Recker, R. R. (1984). The role of calcium in bone health. Nutritional News, 47(2), 5-6.

Rockwell, J. C., Sorensen, A. M., Baker, S., Leahey, D., Stock, J. L., Michaels, J., & Baran, D. T. (1990). Weight training decreases vertebral bone density in premenopausal women: A prospective study. Journal of Clinical Endocrinology and Metabolism, 71(4), 988-993.

Sanborn, C. F. (1990). Exercise, calcium, and bone density. Sports Science Exchange, 2(24).

Siri, W. E. (1956). Gross composition of the body. In: Advances in Biological and Medical Physics, Vol. IV, (JH Lawrence, CA Tobias, Eds.). New York: Academic Press.

Wilmore, J. H. (1969). A simplified method for determination of residual lung volume. Journal of Applied Physiology, 27, 96.

Wolman, R. L., Clark, P., McNally, E., Harries, M. G., & Reeves, J. (1992). Dietary calcium as a statistical determinant of spinal trabecular bone density in amenorrhoeic and oestrogen-replete athletes. Bone and Mineral, 17, 415-423.

## Appendix A

Informed Consent for the Research Study

Bone Mineral Density in Resistance

Trained Premenopausal Females

## Appendix A

## INFORMED CONSENT

The purpose of this study is to evaluate the effect of long term, regular resistance training on bone mineral content, body composition and aerobic capacity in females. The results of this study will directly impact the consideration of regular, heavy resistance training as a means to promote bone growth and combat the development of osteoporosis.

PROCEDURE: This study will include three days of testing. Each subject will report to the Department of Nuclear Medicine at the SUNY Health Science Center on the first day of testing. The second and third day of testing will be in the Exercise Physiology laboratory in Hill Building. The first test session will last approximately 55 minutes. The second test session will last approximately 90 minutes. The third test session will last approximately 60 minutes.

1. Your bone mineral content will be measured using dual energy x-ray absorptiometry (DEXA). Radiation from the DEXA procedure is less than 5 mRems. As a means of comparison, radiation exposure from a standard chest x-ray is 20-50 mRems and 300 mRems from full mouth x-rays. DEXA has an error rate less than 1% as compared to other alternative procedures for measuring bone density with error rates of 30-50%. You will assume a supine position on a table and the DEXA scanner will scan over the length of your body.
2. Your height and weight will be measured.
3. Your body volume will be measured by a water immersion test. You will be seated in a chair suspended in a four foot water tank. You will exhale air and submerge. You will hold your breath for 3-5 seconds while in a bent forward position. This procedure will be repeated 6-10 times with ample time between. The chair is balanced so your sitting position is maintained throughout the test.
4. You may use a snorkel if you wish. A nose clip and ear plugs can also be worn. You may raise your face out of the water at any time. The procedure is similar to sitting in a bath tub with the water level up to your neck. You then lean forward to submerge your head

(Initials_____)

while you are weighed.

5. The volume of your lungs will also be measured prior to the water test. You will sit in a chair and breathe into the spirometer for 6-8 normal breaths. A nose clip is worn. The procedure takes about 15 seconds, and is done twice. The lung volume is needed in the calculation of your body composition.

6. To measure strength, you will complete five strength measures using a Universal Gym. You will sit in the Universal leg press apparatus. The knee will be passively flexed at approximately 110 degrees. You will actively extend your legs against a resistance to within five degrees of complete knee extension. The weight will be increased ten or more lbs following each successfully completed repetition until the weight can no longer be lifted. You will then be prone with the leg curl apparatus positioned approximately two inches proximal to your ankle joint. Your legs will be fully extended at the knee joint. You will forcefully flex your knees against a resistance to at least 90 degrees of flexion. The weight will be increased five or more lbs following each successfully completed repetition until the weight can no longer be lifted. You will then be seated on the leg extension apparatus with both knees flexed at 90 degrees. You will forcefully extend both knees against a resistance to within five degrees of complete knee extension. The weight will be increased five or more lbs following each successfully completed repetition until the weight can no longer be lifted. You will then be supine on the bench press apparatus with the chest aligned directly below the bench press handles. You will grasp the handles at shoulder width and forcefully extend the elbows against a resistance to within five degrees of complete elbow extension. The weight will be increased 2.2 or more lbs following each successfully completed repetition until the weight can no longer be lifted. You will then be seated on a bench such that the acromion process is aligned with the handles of the shoulder press apparatus. While grasping the handles, you will forcefully flex your shoulders against a resistance to within five degrees of complete shoulder flexion and elbow extension. The weight will be increased 2.2 or more lbs following each

(Initials_____)

successfully completed repetition until the weight can no longer be lifted.

7. Maximal oxygen consumption will be determined with a treadmill running test. You will be familiarized with treadmill running prior to the test. You will begin the test by walking or jogging slowly on the treadmill at a comfortable pace on a level grade. After two minutes the treadmill grade will be increased slightly and every three minutes thereafter. This simulates uphill running. You will run until you feel you can no longer continue. Throughout the test you will wear a nose clip and breathe through a mouthpiece. Your exhaled air will be analyzed to determine your oxygen consumption. The treadmill test evaluates your current level of cardiovascular fitness. You will experience a feeling of overall fatigue, especially in your legs. This discomfort may persist for up to one hour after the test, and there may be some residual leg soreness for as long as 48 hours.

DISCOMFORTS OR RISKS: Exposure to low dosages of radiation (<5 mRem) equivalent to one-tenth of a commonly performed chest X-ray. Subjects may experience some dryness in the mouth from breathing through the mouth piece during exercise on the treadmill. Leg fatigue may be experienced following the treadmill test and some leg soreness may persist for up to 48 hours. Slight muscle soreness following strength testing is possible but is not common with the isotonic mode of strength testing. In general, there is little risk associated with body composition tests. In rare cases, a subject may swallow a small amount of water if they inhale instead of exhale during submersion. No long lasting pain or discomfort is expected as a result of all testing associated with this study. If fact, you should generally find the testing interesting.

BENEFITS:

a. Exposure to scientific research.
b. Contribution to the advancement of scientific knowledge.
c. An appraisal of the subject's bone mineral content, body composition, muscle strength and maximal oxygen consumption, which is the best indicator of cardiovascular fitness.
d. Written copy of subject's results.

(Initials_____)

QUESTIONS AND ANSWERS: The investigator is willing to answer any questions concerning the testing and results, please call:

Stephen Hess, Hill Center 274-1288
or
Dr. B. Keller (274-1683), Dr. G.A. Sforzo (274-3359)

WITHDRAWAL: The subject is free to withdraw consent and discontinue participation in this research study at any time without prejudice. The investigator would appreciate prior notice of a subject's intent to withdraw.

CONFIDENTIALITY: The subject is assured that all data will be kept confidential for use only in this investigation. No names will be used in the report or in any publications which may result from this investigation.

I have read and understand the Informed Consent Document and hereby give my consent for participation in the investigation as described above. I acknowledge that I am 18 years of age or older.

Signed: _____ Date_____
Subject Signature

## Appendix B

Medical History Questionnaire for

the Research Study

Bone Mineral Density in Resistance

Trained Premenopausal Females

Appendix B

MEDICAL HISTORY QUESTIONNAIRE

Name_____ Age _____ Date _____

Home Address

_____

Phone _____

SPORT: BODY BUILDING_____ POWER LIFTING_____ INACTIVE _____
OTHER _____ (explain)

AGE _____ HEIGHT _____ DATE OF BIRTH _____

Have you ever had any of the following conditions:
(Check those that apply)

_____ Diabetes or family history of diabetes
_____ Paget's Disease
_____ Thyroid surgery or take thyroid medication
_____ Fractures of the bones (specify)_____
_____
_____ Joint problems (arthritis)
_____ Heart problems
_____ Liver disorders
_____ Cancer disorders
_____ Stomach surgery
_____ Seizures, epilepsy
_____ Chronic diarrhea (within last 2 months)
_____ Paralysis of extremities legs, arms, hands
_____ Used steroids or growth hormone daily for more than 2 weeks
_____ Used more than 2ozs of hard liquor or 24ozs of beer per day
_____ Used birth control pills within the last ten years
_____ Ancestors of Northern European, Oriental or Hispanic origin
(Circle if appropriate)
_____ Do you smoke or have you in the past? Years quit?

Do you take any vitamin or nutritional supplements? Yes No

(Please list)_____

How many hours/week do you spend working out for your sport?_____

Do you follow any specific dietary regimen? Yes No
(If yes, please specify)_____
_____

Have you been on any weight loss programs in the past?
Yes No
(If yes, please specify and indicate amount of weight lost)_____

How would you rate the degree of pressure to maintain an ideal competitive body type? (circle)

LOW MODERATE HIGH

How old were you when your menstrual cycle began?_____

Have you had any irregular menstrual cycles in the past? (give a brief description)
_____
_____

Have you ever missed 3 periods in a row? Yes No
When?
_____

Do you currently have an irregular cycle?
(describe)_____
_____

Is your menstrual cycle regulated by oral contraceptives?
_____

If so, how long have you been using oral contraceptives?
_____

## Appendix C

Physical Activity History Questionnaire

for the Research Study

Bone Mineral Density in Resistance

Trained Premenopausal Females

Appendix C

PHYSICAL ACTIVITY HISTORY QUESTIONNAIRE

Name _____ Date _____ DOB _____ Age (yrs)____

Local Phone # _____ Local Address -_____

M / F (circle) Height (in.) ____ Weight (lbs.) ____ smoker Y / N

Which hand do you write with? R / L

Which foot do you kick with? R / L

Have you ever had a knee or shoulder injury? Y / N

If so, what type?_____When?_____to_____19____

Does it bother you at this time? Y / N If so, describe _____

Select the activity code that best describes your level of daily physical activity:

1. You have a sit-down job and no regular physical activity;

   OR

   three to four hours of walking or standing per day are usual. You have no regular organized physical activity during leisure time.

2. You lift weights four or more times per week and have been weight lifting for at least three years.

ACTIVITY CODE _____

ANSWER QUESTIONS TO THE NEAREST .1 YEARS (if years are requested)

---

If your Activity Code is 1, answer only this section:

How long (yrs.) have you maintained your current physical activity level? _____

Place a check next to any activities you have done in the past , the age(s) during which you did the activity, and the number of days per week you participated at that time:

| ACTIVITY | | AGE(S) | DAY/WK | ACTIVITY | | AGE(S) | DAY/WK |
|---|---|---|---|---|---|---|---|
| Free weights | ___ | ___ | _____ | Racquetball | ___ | ___ | _____ |
| Circuit weights | ___ | ___ | _____ | Squash | ___ | ___ | _____ |
| Walk/jog/run | ___ | ___ | _____ | Aerobics | ___ | ___ | _____ |
| Swimming | ___ | ___ | _____ | Downhill ski | ___ | ___ | _____ |
| Cycling | ___ | ___ | _____ | Basketball | ___ | ___ | _____ |

Comments:

_____

_____

_____

If your Activity Code is 2, answer only this section:

How long have you been training? (yrs)

_____

How frequently? (days/wk) _____

How long is a typical training session? _____

At what age did you start training 4 or more times/week for 10 or more months/year?

_____

Have you ever competed in a body building competition? Y / N

If so, # of competitions/year?

_____

If so, when was your last competition?

_____

Have you missed more than 3 consecutive mo. of training in the past 3 years? Y / N

If so, when?

_____

Do you participate in other types of physical activity? Y / N

If so, what type?_____

How long? (yrs.)_____

How frequently? (days/wk)_____

Comments:

## Appendix D

Individual Subject Data

TABLE D-1. BMD ($g \cdot cm^{-2}$) of the spine (L1-L4) for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 1.130 | 1.132 |
| 02 | 1.088 | 1.099 |
| 03 | 1.164 | 0.992 |
| 04 | 1.057 | 1.116 |
| 05 | 1.021 | 1.015 |
| 06 | 1.060 | 1.081 |
| 07 | 1.187 | 1.116 |
| 08 | 0.859 | 0.986 |
| 09 | 1.060 | 1.009 |
| 10 | 1.130 | 0.873 |
| 11 | 1.060 | 0.873 |
| 12 | 1.155 | 0.999 |
| 13 | 1.219 | 1.142 |

TABLE D-2. BMD ($g \cdot cm^{-2}$) of the total hip for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 1.078 | 0.920 |
| 02 | 0.825 | 0.884 |
| 03 | 1.076 | 0.809 |
| 04 | 0.976 | 1.076 |
| 05 | 1.175 | 0.823 |
| 06 | 1.023 | 1.087 |
| 07 | 1.265 | 1.069 |
| 08 | 0.908 | 0.871 |
| 09 | 1.008 | 0.908 |
| 10 | 0.996 | 0.983 |
| 11 | 0.911 | 0.851 |
| 12 | 0.920 | 0.783 |
| 13 | 1.265 | 1.215 |

TABLE D-3. BMD ($g \cdot cm^{-2}$) of Ward's triangle for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 0.879 | 0.805 |
| 02 | 0.673 | 0.797 |
| 03 | 0.868 | 0.942 |
| 04 | 0.723 | 0.931 |
| 05 | 1.064 | 0.624 |
| 06 | 0.868 | 0.849 |
| 07 | 0.966 | 0.839 |
| 08 | 0.677 | 0.777 |
| 09 | 0.795 | 0.726 |
| 10 | 0.785 | 0.771 |
| 11 | 0.713 | 0.650 |
| 12 | 0.676 | 0.742 |
| 13 | 0.942 | 1.105 |

TABLE D-4. BMD ($g \cdot cm^{-2}$) of the femoral neck for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 0.954 | 0.811 |
| 02 | 0.819 | 0.782 |
| 03 | 0.909 | 0.942 |
| 04 | 0.937 | 0.988 |
| 05 | 1.086 | 0.714 |
| 06 | 0.917 | 0.882 |
| 07 | 1.130 | 0.917 |
| 08 | 0.851 | 0.809 |
| 09 | 0.929 | 0.836 |
| 10 | 0.877 | 0.846 |
| 11 | 0.801 | 0.778 |
| 12 | 0.829 | 0.787 |
| 13 | 1.038 | 1.082 |

TABLE D-5. BMD ($g \cdot cm^{-2}$) of the trochanter for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 0.784 | 0.767 |
| 02 | 0.650 | 0.697 |
| 03 | 0.768 | 0.773 |
| 04 | 0.728 | 0.879 |
| 05 | 0.845 | 0.637 |
| 06 | 0.846 | 0.842 |
| 07 | 0.932 | 0.837 |
| 08 | 0.715 | 0.651 |
| 09 | 0.808 | 0.630 |
| 10 | 0.707 | 0.768 |
| 11 | 0.690 | 0.641 |
| 12 | 0.718 | 0.571 |
| 13 | 1.039 | 0.921 |

TABLE D-6. BMD ($g \cdot cm^{-2}$) of the ulna total for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 0.569 | 0.488 |
| 02 | 0.523 | 0.473 |
| 03 | 0.583 | 0.510 |
| 04 | 0.451 | 0.558 |
| 05 | 0.577 | 0.492 |
| 06 | 0.608 | 0.488 |
| 07 | 0.609 | 0.450 |
| 08 | 0.510 | 0.475 |
| 09 | 0.533 | 0.514 |
| 10 | 0.522 | 0.559 |
| 11 | 0.479 | 0.463 |
| 12 | 0.577 | 0.450 |
| 13 | 0.618 | 0.589 |

TABLE D-7. BMD ($g \cdot cm^{-2}$) of the radius total for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 0.595 | 0.557 |
| 02 | 0.575 | 0.538 |
| 03 | 0.583 | 0.563 |
| 04 | 0.521 | 0.572 |
| 05 | 0.654 | 0.544 |
| 06 | 0.629 | 0.544 |
| 07 | 0.653 | 0.491 |
| 08 | 0.557 | 0.511 |
| 09 | 0.551 | 0.534 |
| 10 | 0.552 | 0.606 |
| 11 | 0.544 | 0.496 |
| 12 | 0.607 | 0.537 |
| 13 | 0.655 | 0.642 |

TABLE D-8. BMD ($g \cdot cm^{-2}$) of the whole body for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 1.133 | 1.083 |
| 02 | 1.104 | 1.124 |
| 03 | 1.142 | 1.112 |
| 04 | 1.024 | 1.080 |
| 05 | 1.202 | 0.968 |
| 06 | 1.114 | 1.050 |
| 07 | 1.212 | 1.115 |
| 08 | 1.033 | 1.082 |
| 09 | 1.146 | 1.032 |
| 10 | 1.125 | 1.071 |
| 11 | 1.078 | 1.000 |
| 12 | 1.209 | 1.036 |
| 13 | 1.290 | 1.322 |

TABLE D-9. Bench press (kg) for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 54.5 | 39.2 |
| 02 | 43.8 | 50.1 |
| 03 | 56.4 | 42.7 |
| 04 | 44.3 | 40.2 |
| 05 | 36.4 | 20.0 |
| 06 | 37.4 | 41.8 |
| 07 | 52.8 | 33.6 |
| 08 | 50.0 | 33.6 |
| 09 | 57.4 | 25.5 |
| 10 | 48.3 | 39.2 |
| 11 | 55.5 | 36.4 |
| 12 | 45.5 | 20.7 |
| 13 | 45.5 | 34.6 |

TABLE D-10. Shoulder press (kg) for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 37.5 | 35.6 |
| 02 | 35.6 | 39.2 |
| 03 | 47.3 | 38.2 |
| 04 | 36.4 | 29.1 |
| 05 | 33.6 | 22.0 |
| 06 | 31.1 | 33.6 |
| 07 | 46.4 | 33.6 |
| 08 | 38.2 | 30.1 |
| 09 | 38.2 | 27.3 |
| 10 | 31.8 | 32.7 |
| 11 | 39.2 | 32.7 |
| 12 | 40.2 | 24.5 |
| 13 | 52.8 | 33.6 |

TABLE D-11. Leg press (kg) for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 168.2 | 150.0 |
| 02 | 154.5 | 195.5 |
| 03 | 245.5 | 163.6 |
| 04 | 154.5 | 136.4 |
| 05 | 150.0 | 100.0 |
| 06 | 163.6 | 150.0 |
| 07 | 209.1 | 150.0 |
| 08 | 190.9 | 150.0 |
| 09 | 195.5 | 136.4 |
| 10 | 154.5 | 154.5 |
| 11 | 181.8 | 95.5 |
| 12 | 186.4 | 136.4 |
| 13 | 250.0 | 136.4 |

TABLE D-12. Leg curl (kg) for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 15.5 | 10.0 |
| 02 | 17.8 | 14.5 |
| 03 | 14.5 | 12.3 |
| 04 | 14.5 | 5.5 |
| 05 | 12.3 | 5.5 |
| 06 | 10.0 | 7.7 |
| 07 | 21.4 | 7.7 |
| 08 | 16.8 | 10.0 |
| 09 | 16.8 | 7.7 |
| 10 | 14.5 | 14.5 |
| 11 | 12.3 | 5.5 |
| 12 | 21.4 | 10.0 |
| 13 | 21.4 | 7.7 |

TABLE D-13. Leg extension (kg) for R and S pairs.

| pair# | R | S |
|---|---|---|
| 01 | 88.6 | 61.4 |
| 02 | 88.6 | 68.2 |
| 03 | 95.5 | 75.0 |
| 04 | 102.3 | 68.2 |
| 05 | 88.6 | 40.9 |
| 06 | 81.8 | 61.4 |
| 07 | 125.0 | 47.7 |
| 08 | 95.5 | 68.2 |
| 09 | 88.6 | 68.2 |
| 10 | 88.6 | 68.2 |
| 11 | 95.5 | 40.9 |
| 12 | 109.1 | 68.2 |
| 13 | 102.3 | 40.9 |

TABLE D-14. Weight (kg) and height (cm) for matched R and S pairs.

| | R | | S | |
|---|---|---|---|---|
| pair# | WT | HT | WT | HT |
| 01 | 52.3 | 141.9 | 50.5 | 150.8 |
| 02 | 66.8 | 176.8 | 64.5 | 176.8 |
| 03 | 64.1 | 167.4 | 69.1 | 163.8 |
| 04 | 49.1 | 165.9 | 47.3 | 161.2 |
| 05 | 54.1 | 175.3 | 50.0 | 161.2 |
| 06 | 55.0 | 171.6 | 50.5 | 168.5 |
| 07 | 74.1 | 176.8 | 75.0 | 175.5 |
| 08 | 61.4 | 171.6 | 61.8 | 166.4 |
| 09 | 57.7 | 169.0 | 55.0 | 165.0 |
| 10 | 60.5 | 172.9 | 58.6 | 171.6 |
| 11 | 57.3 | 161.2 | 63.2 | 158.6 |
| 12 | 72.3 | 180.7 | 73.2 | 175.5 |
| 13 | 72.3 | 168.5 | 71.8 | 163.8 |

TABLE D-15. Percent fat and $VO_2max$ ($ml \cdot kg^{-1} \cdot min^{-1}$) for matched R and S pairs.

| | R | | S | |
|---|---|---|---|---|
| pair# | %Fat | $VO_2max$ | %Fat | $VO_2max$ |
| 01 | 16.02 | 43.27 | 23.28 | 43.32 |
| 02 | 18.30 | 46.26 | 20.62 | 39.07 |
| 03 | 16.40 | 33.24 | 32.61 | 40.80 |
| 04 | 14.87 | 50.05 | 12.60 | 37.85 |
| 05 | 9.96 | 59.53 | 24.42 | 32.91 |
| 06 | 19.01 | 56.23 | 19.64 | 42.94 |
| 07 | 18.92 | 48.48 | 34.76 | 34.43 |
| 08 | 20.29 | 45.60 | 29.38 | 35.16 |
| 09 | 20.77 | 47.91 | 22.69 | 38.15 |
| 10 | 26.00 | 47.53 | 22.21 | 34.26 |
| 11 | 25.26 | 43.67 | 42.85 | 31.43 |
| 12 | 15.49 | 53.74 | 37.50 | 31.56 |
| 13 | 13.40 | 50.99 | 34.21 | 23.71 |