FACTORS AFFECTING BONE DEVELOPMENT IN ADOLESCENT FEMALES

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TO MY MOTHER
FOR HER SUPPORT, LOVE AND ENCOURAGEMENT

TO GAYLE
I HOPE YOU FOUND WHAT YOU WERE LOOKING FOR

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Foreword

This thesis has been written in a format suitable for publication. The review of literature section entitled "Factors Affecting Bone Development in Adolescent Females" was written in the format of the journal "Sports Medicine". The second paper "Determinants and Correlates of Whole Body and Lumbar Spine Bone Mineral Content and Density in Healthy Adolescent Girls" was written in the format for the "Journal of Applied Physiology". The third paper entitled "Effect of 26 Weeks of Resistance Training on Whole Body and Lumbar Spine BMC and BMD in Healthy Adolescent Girls" was written for publication in the journal "Medicine and Science in Sports and Exercise". The reader will notice the different reference styles utilized in the different sections, reflecting the formats of the three publications.

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Introduction

Osteoporosis is a major cause of morbidity and mortality in our aging population. It is estimated that by the year 2000 the cost of hip fractures alone in the United States may well exceed $10 billion (Gleeson et al. 1990). The primary goal in the management of this problem is the prevention of bone loss and subsequent fractures. Current research (Matkovic et al. 1990) suggests that about 90% of adult peak bone mineral is accumulated in the years preceding, and during the adolescent growth spurt. Therefore it is important to identify those factors which contribute to the attainment of peak bone mass, and to determine their relative importance during the developmental years. Once these factors have been identified, intervention strategies may be employed during childhood and adolescence for the purpose of maximizing adult peak bone mass.

This chapter reviews the mechanisms underlying and examines the significance of a number of factors that currently are considered to affect childhood bone development; namely: ethnic and genetic influences, nutrition, physical activity, growth and maturation, and hormonal influences. It should be emphasized that although these factors will be considered separately, they probably act in an intergrated manner in the regulation of bone development during childhood.

Regulation of Bone

Bone structure and mineralization is governed largely by the mechanical demands imposed upon the bone tissue. The biological goal is the maintenance of a minimum adequate structure, in which the margin of safety between normal mechanical demands and fracture is balanced by the cost of excessive bone mass on mobility (Turner 1991). The "Mechanostat" theory proposed by Frost (1987) provides a paradigm for the study of bone regulation. He postulates that three separate biological mechanisms are responsible for the regulation of bone development: growth, modelling and remodelling. Each of these mechanisms responds in it's own way to mechanical, non-mechanical, local and circulating bone regulating influences.

Bone growth occurs at the growth plate to increase the length of bone. Growth adds new spongiosa to pre-existing spongiosa, and new length to the cortex (Parfitt 1984). Growth can only add trabecular and cortical bone to the bone bank, it cannot take away from bone which is already deposited (Frost 1991). The growth mechanism, therefore, functions to increase bone size by increasing the number of cells and the amount of intercellular material.

The bone modelling process involves drifts in both bone formation (new bone added) and resorption (bone removal) over broad regions of the skeleton (Raisz 1988). These drifts occur

primarily during longitudinal bone growth, begin to subside near skeletal maturity, and become almost non-existent by 25-30 years of age (Frost 1987). This mechanism increases the thickness of the outer shell and marrow cavity diameter as well as adding cortical bone along the entire length of bone (Raisz 1988). The effect of modelling is to provide a functionally and mechanically purposeful architecture.

Two types of modelling have been observed: micro-modelling and macro-modelling (Frost 1987). Micro-modelling differentiates the development of bone and collagen tissues into compact and trabecular bone. Macro-modelling determines whether, when and where new tissue is to be formed or old tissue is to be removed, and thus acts to promote or retard growth at various skeletal sites. Macro-modelling stops when skeletal growth ceases, while micro-modelling continues to form new tissue throughout life (Frost 1991).

Bone remodelling takes place through a cyclic process of erosion and repair of microscopic cavities on bone surfaces (Parfitt 1984). This process controls bone turnover, and functions in the maintenance, and replacement of bone and in bone micro-damage repair (Frost 1991).

Both modelling and remodelling are carried out by osteoblasts and osteoclasts; fully differentiated cells of finite life-span that require continuous renewal from separate stem cell populations (Lakkakorpi et al. 1991). The total rate

of bone remodelling is governed by the number of resorption/formation cycles that take place in a given time period, which in turn depends upon the rates of precursor cell proliferation and differentiation (Parfitt 1984). The total cycle of remodelling in adult humans is estimated to take approximately 120 days (Turner 1991). Remodelling consists of five successive stages: quiescence, activation, resorption, reversal, and formation (Parfitt 1984).

The first stage of the remodelling cycle is the state of inactivation or quiescence. In the young growing animal almost all bone surfaces free of articulation are either in the state of formation or resorption (Montoye 1987). By contrast in older animals, including adult men and women, about 80% of the trabecular bone surface and 95% of the intra-cortical bone surface is inactive with respect to bone remodelling (Parfitt 1984). During this period of quiescence, the outer bone surface is covered by a layer of extremely thin (0.1-1 um) flattened lining cells about 50um in diameter. The lining cells arise from terminal transformations of osteoblasts that have lost their ability to synthesize collagen, but which probably retain some form of hormonal receptors (Montoye 1987).

The second step in the process of remodelling is activation. Initiation of this cycle requires (1) recruitment of osteoclasts, (2) a means for osteoclasts to gain access to the

bone and (3) a mechanism for osteoclast attraction and attachment to the bone surface (Parfitt 1984). In a normal skeleton of average size, activation occurs somewhere about once every 10 seconds. The lining cells respond to bone-resorbing hormones by releasing proteolytic enzymes such as collagenase and plasminogen activator (Hamilton et al. 1985). These enzymes uncover bone surface and allow osteoclasts access to bone (Raisz 1988). Osteoclasts arise by fusion of precursor cells of the mononuclear phagocyte system (Lakkakorpi et al. 1991). These precursor cells are thought to originate in and travel through the bones' blood supply to reach mineralized bone surfaces, where their fusion into osteoclasts occurs (Melton et al. 1982). Osteoclast precursors display phagocytic recognition for bone mineral particles, but not for demineralized bone matrix. Thus, osteoclasts may initiate the resorption process by exposing the underlying bone to osteoclast precursors, by retracting bone cell linings (Raisz 1988).

The next phase, bone resorption, occurs when the newly formed osteoclasts begin to erode a cavity of characteristic shape and dimension on the bone surface. In trabecular bone this is known as a Howship's lacuna while in cortical bone it is referred to as a cutting zone (Lakkakorpi et al. 1991). The osteoclast has a ruffled border area with numerous cytoplasmic folds, surrounded by a clear zone where the cell is closely

attached to the bone surface (Bonucci 1981). The osteoclast requires firm attachment to the cell membrane of the bone surface to effectively isolate the resorption lacuna from the extracelluar fluid and to permit the maintenance of a cell generated pH gradient (Lakkakorpi et al. 1991).

Hydrogen ions secreted into the enclosed area dissolve the bone matrix, and lysosomal enzymes that are released degrade the collagen matrix at a low pH (Parfitt 1984). Osteoclasts are actively migrating cells that move from site to site. The ruffled sealing zone appears only during the resorption phases and disappears during the course of migration (Kanehisa et al. 1988). The osteoclast resorption front penetrates about 5-10 um/day perpendicular to the eroded surface and about 20-40 um/day parallel to and along the eroded surface (Parfitt 1984). The mean life-span of an osteoclast is 12.5 days, with a corresponding turnover of about 8% per day (Jaworski 1984). When the resorption cavity reaches a mean depth of 60 um from the surface of trabecular bone and 100 um in cortical bone, resorption at that particular site ceases (Carter 1984).

Once the osteoclasts have resorbed bone mineral and matrix the reversal phase begins. The reversal phase encompasses the interval between completion of resorption and commencement of bone formation at a particular location (Montoye 1987). In this process lining cells smooth over the ragged edges left by the resorptive process and deposit a thin layer of highly

mineralized, collagen poor bone matrix (known as cement substance) in preparation for bone formation (Tran van et al. 1982).

The final phase, formation, is characterized by the replacement of resorbed bone. The actual process of how osteoblasts deposit mineral in the collagen matrix is unknown. The osteoblasts are separated from a recently mineralized section of the resorption canal by a layer of non-mineralized matrix or osteoid (Raisz 1988). In both cortical and trabecular bone, the rate of matrix apposition is rapid (2-3 um/day) at the beginning when the osteoblasts are columnar and densely packed (Parfitt 1984). Following termination of the matrix synthesis, mineralization continues slowly until the osteoid seam disappears, and the cells remaining on the bone surface complete their morphologic and functional transformation to lining cells (Raisz 1988).

The bone surface has now returned to its original state of quiescence in adult bone. The process of remodelling is similar in young growing animals and occurs simultaneously with both the growth and modelling phases.

From infancy to adolescence the surface of bone is in a continual cyclic state of bone formation/resorption. In contrast 80-95% of adult bone rests in a state of quiescence. Bone mineral and architectural structure are influenced through the mechanical demands placed upon bone. A site

specific resorption/formation of bone occurs in response to altered mechanical demands in an attempt to enable bone to withstand the newly imposed force with a minimum adequate structure. If this newly imposed force produces stresses greater than those already present, bone formation exceeds resorption and bone mineralization increases. When the mechanical strain imposed on bone decreases, bone resorption exceeds formation in an attempt to provide adequate strength with the minimum required structure.

Ethnic and Genetic Determinants of Bone Mineralization:

Ethnic Determination of Bone Mineral

Trotter et al. (1960) was the first to examine the racial differences between bone density (BD) (BD; whole bone mineralized collagen in g/cm^{-3}) in blacks and whites. The results of this study showed that the bones of blacks were denser than those of whites, and this was especially significant when comparing black to white females. This ethnic difference in bone mineral density (BMD; local bone mineral in g/cm^{-2}) between whites and blacks is evident even before birth (Choi and Trotter 1970). An examination of the radial BMD in children between the ages 1-6 years showed that black children had higher bone mineral across all ages compared to whites (Li et al. 1989). Differences in spinal BMD between black and white children are detectable from the age of 5 years

(McCormick et al. 1991). This racial difference in BMC is evident in both weight bearing and non-weight bearing bones in children 7-12 years of age (Bell et al. 1988).

Regardless of ethnicity, spinal BMD shows a high degree of correlation with age, body weight, and height from childhood to adolescence (McCormick et al. 1991). Indirect evidence from BMD and total body water estimations indicate that black adults have approximately 10% more total bone mineral than do young white adults even after adjusting for stature (Schulte et al. 1984). In ossification centres of the hand and wrist, American black children are advanced compared to white children even though their socio-economic status is lower (Garn et al. 1972). Although black babies are generally smaller at birth, they quickly gain and then exceed white children in height and weight after the first 2 years (Pollitzer and Anderson 1989). Both black boys and girls grow more rapidly than whites through adolescence and are on average 2 cm taller at given ages (Garn and Clark 1975). However, even when matched for age, height, and weight, black children retain 5-6% more bone mineral than whites. Thus normal values of bone mineralization for whites are substantially lower than for blacks (Pollitzer and Anderson 1989).

Both past and present populations have been studied world wide to determine broader estimates of the ethnic variation in

bone mineral. The Chinese and Japanese have significantly less cortical bone than whites (Garn 1963). Even with lower bone mass the incidence of hip fractures in Japan is lower by 12.5 to 50% compared to western countries. Type I osteoporosis (naturally occuring osteoporosis) is so rare in Japan that many clinicians doubt that it even exists (Fujita 1992).

Whites of both sexes have about 5% larger metacarpal cortical bone area than Mexican-Americans from 1 to 17 years of age (Pollitzer and Anderson 1989). The Eskimo of the St-Lawrence Island have lower BMC than whites, and tend to lose bone at an earlier age, and faster rate than most populations. The female Eskimo apparently has a faster rate of bone loss than the Eskimo male (Harper et al. 1984). Alaskan Eskimo children have 5-10% lower BMC than white children and adults have a greater rate of loss of BMC with age. After age 40, Eskimo of both sexes have 10-15% lower BMC that whites (Mazes and Matthew 1974).

Generally, fractures of the proximal femur are common in temperate zones and uncommon in the tropics; this suggests that apparent ethnic differences may be confounded by other factors such as climate, and differences in diet, exercise, and socio-economic conditions (Pollitzer and Anderson 1989).

Genetic Determination of Bone Mineral

Proof of genetic causation cannot be inferred from the fact

that a particular condition runs in families. Rather it must be shown that established principles of Mendelian inheritance are followed or, that a characteristic is far more frequent in monozygotic (MZ) twins who have 100% of their genes in common than in dizygotic (DZ) twins who have 50% of common genes (Pollitzer and Anderson 1989).

In an examination of total and cortical width of three metacarpal bones, Moller et al. (1978) showed four to five times greater intra-pair variance among DZ than MZ twins. The authors concluded that there was a fairly high genetic contribution of about 80% to the determination of bone mineral. Smith et al. (1973) examined 71 juvenile twin pairs of both sexes and 80 adult male twin pairs. The within-pair variance for BMC among the young twins for both sexes was four times as great for DZ twins as for MZ twins. The larger variation among DZ twins was thought to indicate a strong genetic determination, but the increasing intra-pair difference with increasing age was considered to be due to genetic and environmental interactions. Slemenda et al. (1991) examined 171 pre- and post-menopausal female twins aged 25-80 years. At all skeletal sites MZ intra-class correlations exceeded DZ correlations for both pre- and post-menopausal women. However there was increasing within MZ pair variability in older women, which indicated that the genetic influence on bone mineral diminished with age.

A family history of osteoporosis has been cited as an important risk factor in the development of the disease, despite the existence of little evidence to substantiate this view (Seeman et al. 1989). Of 34 adult mother-daughter pairs and 29 adult female sibling pairs, Sowers et al. (1986) found no consistent evidence of a significant between pair resemblance for BMC of the distal radius. In contrast Seeman et al. (1989) found lower bone mass in the daughters of osteoporotic mothers than in the daughters of non-osteoporotic mothers.

Tylavsky et al. (1989) evaluated radial BMC and BMD in pre-menopausal mothers and their biological daughters. By the age of 18 the daughters accumulated 90-95% of the BMC of their mothers. Lutz and Tesar (1990) found significant positive correlations between mothers and daughters for BMD of the lumbar spine and femur, however the correlation weakened with increasing age of the mothers.

The nature of inheritance of bone mass in women may have at least two major components. One influences peak bone mass attained during growth. The second regulates post menopausal hormonal changes associated with bone loss (Seeman et al. 1989). These components may be regulated by different genetic factors which may not be equally transmittable from mothers to daughters.

The accumulation of peak bone mass is determined to a large

extent by ethnic/genetic variation. Studies comparing blacks to whites consistently find greater total bone mineral deposited in the skeleton of blacks. The intra-class variation in bone mineral is less for monozygotic than for dizygotic twin pairs. The most important finding, relevant to the optimization of adult peak bone mineral, is that by the age of 18, females have reached upwards of 90% of the bone mineral found in their pre-menopausal mothers. This suggests that intervention programmes designed at optimizing adult bone mineral should be initiated at an early age.

Nutrition and Bone Mineralization:

Calcium

Most of the body's calcium reserve resides as calcium phosphate salts within the skeleton. This reserve serves two important functions. First, with its incorporation into the skeleton, it provides protection to vital organs, and a framework for locomotion. Second, it acts as a reservoir for the body's calcium requirements. Because of the body's need to maintain plasma (ionic) calcium concentration for the preservation of ionic balance, calcium is resorbed from the skeleton whenever dietary calcium intake falls short of obligatory losses (Nordin and Morris 1988). These losses occur every day through urine, skin and the gastrointestinal tract (Charles et al. 1991a). Long term skeletal calcium homeostasis

is mainly based on the balance between the absorption of calcium from the intestine and the difference between bone mineralization and resorption rates.

Given the varying activity of the growth, modelling and remodelling processes at varying stages of development it has been considered appropriate to characterize or define the need for calcium in relation to the specific phases of bone development. This is reflected in the different Recommended Dietary Allowances (RDA'S) of calcium for children, adolescents and adults (Dawson-Hughes 1991a).

From birth to puberty, the skeletal mass increases by seven fold and by a further three fold during adolescence (Garn et al. 1972). Because 60% of the weight of mature bone is mineral, in the form of calcium phosphate, a continuous supply of dietary calcium is an essential requirement for healthy bone development during childhood and adolescence (Peacock 1991). Calcium is absorbed by the small intestine with little or no absorption by the colon. There are two components to calcium absorption, an active transport mechanism and a diffusion component. The major determinants of calcium absorption are the amount of bio-available calcium in the diet and the vitamin D status of the individual (Peacock 1991).

The skeleton of a newborn infant contains approximately 25 grams of calcium, comprising about 1% of the infant's weight (Chan 1991). During the first two years of life the calcium

content increases faster in relation to body size than during any other time of life (Tanner 1962). The degree of positive calcium balance in infancy necessary to achieve peak bone mass and density is unknown. Peak height velocity during infancy (birth - 1yr) reaches approximately 18 cm/yr . With adequate vitamin D status, infants retain about 44% of their calcium intake with very low calcium excretion in the urine (average 38 mg/day) (Matkovic 1991).

Growth rate begins to slow between 2-8 years of age (childhood). Peak height velocity declines from 9 cm/yr following infancy to 5.5 cm/yr for both boys and girls during this period. The body's retention of dietary calcium declines to approximately 25 % (Matkovic 1991). Required calcium retention during this period is estimated to be 100 mg/day (Chan 1991), and the recommended dietary intake is 1000 mg/day. The change in RDI between infancy and childhood reflects both the lower retention and greater urinary output in the older children.

Calcium needs are greater during pre-adolescence and puberty (ages 9-17 years) than in either childhood or adulthood (Char et al. 1987). This is a period characterized by accelerated skeletal development. During the adolescent growth spurt approximately 45% of the adult skeletal volume is formed, with BMC increasing at a rate of 8.5% per year (Matkovic et al. 1990). Girls reach their peak height velocity

of 8.5 cm/year by the age of twelve. Peak height velocity for boys is 9.5 cm/year and is achieved by age 14 (Tanner 1962). In general, adolescents retain more calcium than either children or young adults. Despite this higher calcium retention, required calcium intake is higher during this period than during childhood or adulthood due to their greater excretion of calcium in urine (Peacock 1991).

Adolescent females may be at risk for skeletal inadequacies because their ratio between calcium intake and optimal calcium retention is low (Chan 1991). An examination of peri-menarcheal females by Forbes (1992) demonstrated that urinary hydroxyproline excretion (a marker for collagen turnover) rose significantly approximately one year in advance of menarche then fell to levels below those of the late childhood years. This finding suggests that bone turnover and/or remodelling proceeds at a relatively rapid rate in the immediate pre-menarchal years in females.

There are few studies examining the current level of dietary calcium intake and bone mineralization in children and adolescents. Chan (1991) and Chan et al. (1987) demonstrated a positive correlation ($r=0.45$, $p<=0.01$) between dietary calcium intake and BMC of the radius in adolescent females. No correlation between current calcium intake and BMC of the proximal femur was found in women aged 23-75 yrs (Angus et al. 1988). These results were also confirmed by Kanders et al.

(1988). Dietary modification in the form of dairy products was shown to retard vertebral bone loss in women aged 30-42 yrs (Baran et al. 1989).

The effectiveness of calcium supplementation on bone growth has not been extensively studied. Most studies have concentrated on middle-aged women. A two year examination of the effectiveness of calcium supplementation in peri-menopausal women aged 46-55 yrs revealed a 3.5% loss of lumbar spine in the control group, while those supplemented with 1000 mg/day lost 1.3% and those supplemented with 2000 mg/day lost 0.7%. However, the effectiveness of calcium supplementation on bone loss was significant only during the first year (Elders et al. 1991). A controlled trial of the effect of calcium supplementation on bone density in post-menopausal women was performed by Dawson-Hughes et al.(1990). Calcium supplementation was ineffective in retarding lumbar spine bone loss in women within five years of menopause. Calcium supplementation was effective however, in retarding bone loss in those women six years and more, post-menopause. Sandler et al. (1985) reported that milk consumption during childhood and adolescence had a positive effect on post-menopausal bone density. Calcium supplementation (2000 mg/day) slowed the loss of cortical bone but not trabecular bone in a two year examination of post-menopausal women (Riis et al. 1987).

The effectiveness of calcium supplementation in regards to

optimizing bone formation appears to be inconclusive. There is little to no information concerning the efficacy of calcium supplementation on bone formation during the developmental years of infancy to adolescence. Calcium supplementation does appear to have a positive effect on bone loss in post-menopausal women, but only after the initial period of rapid bone loss associated with the cessation of menses.

In summary these studies indicate that percent net calcium absorption is relatively high during infancy (40%) and adolescence (30%) while lower in children (25%) and young adults (20%) (Matkovic 1991). Greater rates of calcium retention are reflective of the greater rate of bone formation associated with infancy and adolescence.

Adult calcium requirement is defined as the amount of calcium required to preserve calcium balance (Cumming 1990). If input is equal to output the calcium balance will equal zero. Young individuals need to be in positive calcium balance to meet the needs of skeletal growth and consolidation. This point is further emphasized by Matkovic et al. (1990) who estimated that 16 year old daughters have accumulated 90-97% of the bone mass of their pre-menopausal mothers. In light of this evidence the draft statement for the U.S. health goals for the year 2000 includes a calcium intake that amounts to an approximate doubling of the current RDA (Avioli 1991). If young females were to maximize their calcium absorption

potential it is estimated that their adult peak bone mass may be increased by as much as 10% (Peacock 1991).

Protein

Dietary proteins provide the necessary sources of amino acids and nitrogen for the synthesis of body proteins and nitrogen containing compounds. The predominant component of bone matrix is protein, which exists primarily in the form of collagen. Bone protein is responsible for bones intrinsic plastic stiffness properties (Boskey 1981).

Dietary protein exhibits both direct and indirect effects on calcium metabolism including: a dose-dependant inhibition of calcium absorption from the gut; a dose-dependant increase in urinary calcium excretion; and the resultant alteration in the body's calcium requirements (Widhe 1982).

Faridi et al. (1984) examined 100 children aged 6 months to 6 yrs suffering from protein calorie malnutrition. Radiographic examination revealed 75% of children to suffer from osteopenia, while 54% demonstrated retarded growth. Decreased osteoblastic activity was the cited cause.

A diet high in protein has been associated with increased urinary calcium excretion. In young adult females, a normal dietary protein intake had no effect on calcium absorption, but on a high intake (142 gm/day) increased urinary calcium excretion resulted in negative calcium balance (Simmons et al.

1990).

Low dietary protein results in decreased osteoblastic activity, osteopenia and growth retardation. Hypercalciuria on the other hand, and decreased calcium retention are caused by a high protein diet.

Lipids

Dietary lipids provide a major source of energy, containing more than twice the calories per gram than carbohydrates or protein. Recent research suggests that lipids may play an important role in the calcification process of bone, and are generally thought to concentrate at the boundary between calcified and non-calcified tissue (Simmons et al. 1990).

The extracelluar matrix of epiphyseal cartilage contains lipid rich vesicles that are believed to be the site of initial mineral deposition (Anderson and Toverud 1986). The important role of dietary lipids is to supply the body with adequate amounts of essential fatty acids in order to maintain normal membrane phospholipid composition.

Fiber

Because insoluble fibers increase the speed at which the gastrointestinal tract empties, a very high fiber intake may decrease absorption of dietary calcium. Natural fibers are also believed to bind to calcium in the intestinal tract.

Aloia (1989) suggests that high fiber diets decrease calcium absorption from the diet and should be avoided in osteoporotic women.

It is through our diet that we acquire the nutrients essential for proper bodily functions. The most important nutrient absorbed through the diet in regards to bone growth and formation is calcium. The amount of calcium retained in the body is a result of the amount of calcium available in the diet and the percentage absorbed through the intestine. Diets high in fiber and protein composition have been shown to decrease calcium absorption. Current research suggests that the RDA for calcium be increased predominantly during the growing years and continued up to menopause. Calcium supplementation in the years post-menopause does not appear to be effective in reducing bone mineral loss. The current RDI guidelines for fiber, protein and lipids are designed to promote global well-being of the human body. There is however, a lack of RDI guidelines for these secondary nutrients in regards to optimal bone growth and mineral development.

Physical Activity and Bone Mineral:

Mechanical strain

Although the mechanism(s) remains obscure, there is abundant evidence that bone mineralization is influenced by the mechanical stresses inherent to physical activity.

All forces imposed on bone produce strain of some magnitude. The biological control of bone's structure is accomplished largely through direct or secondary effects of mechanical strains on the osteoblasts or osteoblast precursors (Riggs et al. 1986). It has been proposed that changes in bone structure are brought about by a feedback system in which changes in peak mechanical strain drive bone cells to change bone structure (Turner 1991a). Lanyon (1984) hypothesizes that adaptive remodelling takes place to reduce strain when a bone's functional strain level has been exceeded. Carter (1984) states that remodelling is stimulated when strain histories cause fatigue micro-damage. Frost (1987) describes a "mechanostat" theory in which mechanical strains work through a feedback control system to maintain the desired bone mineralization. Using the postulates of Frost (1987), Turner (1991a) describes a feedback theory for the homeostatic control of bone structure. This hypothesis contains two basic premises: (1) a threshold level exists for mechanical strains, above or below which bone adaptation is initiated, and (2) the set point for normal bone structure can be modulated by hormones.

Bone structure is maintained such that ordinary mechanical strains do not exceed a minimum effective strain (MES). Frost (1987) speculates the MES to equal 1500-2500 micro-strain (1 micro-strain equals a bone deformation of 0.0001%). If

local strains surpass the MES, bone modelling will change bone structure to reduce the local strains to below the MES. Martin and Burr (1989) and Frost (1987) have suggested another effective strain level of 50-200 micro-strain. Strains below this level will cause bone resorption leading to a weakening in bone structure until local strains are increased. Therefore a physiological window exists (Figure 1) for bone between the MES and the lower effective strain. If the mechanical load imposed upon bone falls outside this physiological window an adaptation response occurs in which bone structure is altered through either modelling or resorption to maintain functional homeostasis. The control system for bone structure is outlined in Figure (2). The effector change in bone rigidity is the modelling/remodelling system of osteoblasts and osteoclasts. The variability of daily loading is reflected in the error signal. Through increasing or decreasing the strain on bone one alters the error signal. When the error signal is altered beyond the bounds of the physiological window in either a positive or negative direction modelling/remodelling is activated. This will increase or decrease bone rigidity in an attempt to return the error signal to within the confines of the physiological window.

Examining the relationship between stress variation and subsequent bone remodelling, Hert (1971) showed that static loads, regardless of magnitude, have no affect on bone

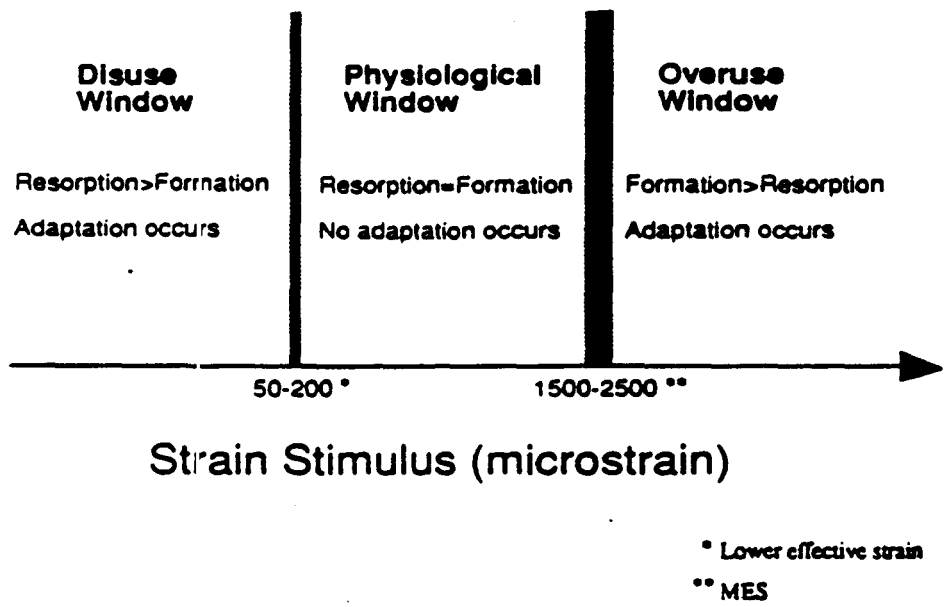

Figure 1: Depiction of the strain stimulus response

Taken from: C.H. Turner (1991b)

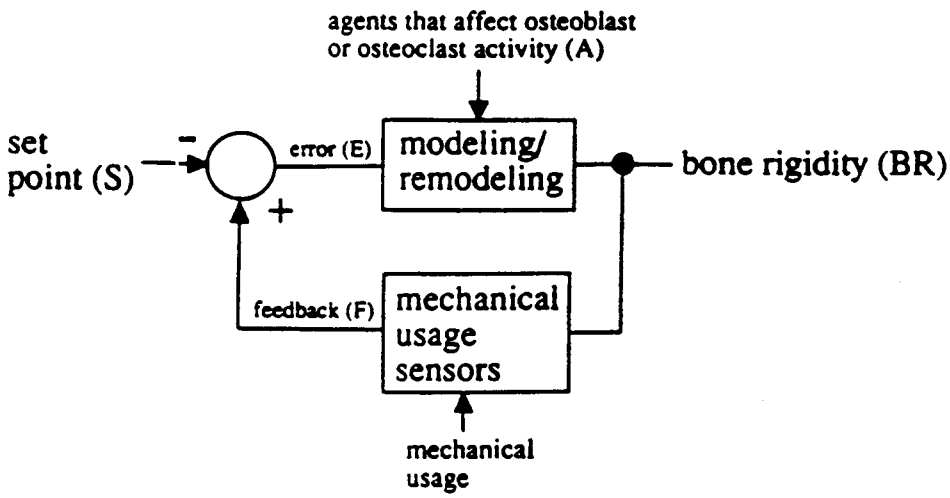

Figure 2: Feedback control system for bone structure

Taken from: C.H. Turner (1991b)

adaptation. Lanyon and Rubin (1984) demonstrated an increase in bone mineralization following dynamic loading, with the greatest increase activities associated with high loading and low repetitions (Martin and McCulloch 1987).

General Physical Activity:

Cross-sectional studies

Cross-sectional studies generally indicate a greater bone density in athletes and people with higher levels of physical activity than in the average population (Bailey and McCulloch 1990) An inherent problem in cross-sectional research is subject bias, since the underlying genotype for bone density preceding participation in physical activity is not known (Snow-Harter and Marcus 1991).

Viridakis et al. (1990) compared the non-dominant forearm of male junior competitive weight lifters with matched controls aged 15-20 yrs. Distal radius BMC in the exercise group was significantly above that for the control group. The effect of physical activity on monozygotic twins aged 5-14 yrs was examined by Slemenda et al. (1991). The results suggest a positive association between total time spent in weight-bearing activity and skeletal BMD. Physical activity resulted in 0.03-0.04 g/cm^2 per year increase in BMD, implying that children with activity levels one standard deviation (SD) above the mean of 2.7 h/day would have approximately 0.06

gm/cm^2 higher BMD than children with average activity levels. The implication is that active children may emerge from adolescence with 5-10% greater bone mass than sedentary children. In contrast, however, some investigators have found no correlation between physical activity and adolescent bone mineralization (Bachrach et al. 1990,1991).

The os calcis BMD of basketball and volleyball players, swimmers and controls, was recently studied by Risser et al. (1990). The swimmers demonstrated significantly lower BMD of the lumbar spine. Both the basketball and volleyball athletes had a greater mean os calcis BMD than the swimmers and controls. The BMD of the lumbar spine for the swimmers was observed to be lower than that of published data for amenorrheic runners. The lumbar spine BMD of female college swimmers was also demonstrated by Jacobson et al. (1984) to be similar to non-athletic age matched controls. Buchanan et al (1988) found no correlation between bone mineralization and physical activity levels in females aged 18-22 yrs. However in peri-menopausal women, level of physical activity has been shown to be significantly correlated (r=0.41) to BMD of the spine and (r=0.51) to total body calcium levels (Aloia et al. 1988). In general these studies suggest that weight-supported exercise such as swimming have little effect on optimizing spinal bone mineralization

Comparing adult male tennis players to age matched controls,

Huddelston et al. (1980) found the dominant radius of the athletes to have significantly higher bone mineral than the non-dominant radius, and to be higher than controls. Similar results were observed by Pirnay et al (1987); adult professional tennis players demonstrated 34% and 15% greater BMD of the dominant and non-dominant forearms compared to sedentary university students. This unilateral increase in BMD of the dominant arm may suggest a site specific adaptation of bone to locally imposed strain.

The physical activity level of peri-menopausal women has also been shown to be significantly correlated (r=0.41) to BMD of the spine and (r=0.51) to total body calcium levels (Aloia et al. 1988). Physical activity has also been suggested to slow the rate of bone loss associated with ageing. Jacobson et al. (1984) demonstrated a 0.7% per year loss of spinal BMD in sedentary women beyond the age of 50 yrs, which was not observed in a group of active age matched controls. These results are supported by Talmage et al. (1986) who also found an accelerated loss of lumbar spine BMD in sedentary women between the ages of 45-55 yrs, while an age matched group of athletic women demonstrated no accelerated loss of bone mineral. Likewise in a retrospective study of 108 women aged 50-60 yrs, Cheng et al. (1991) found women who participated in vigorous exercise four hours a week demonstrated greater BMD of the calcaneus than those who exercised less.

Generally, most studies indicate a positive association between level of habitual physical activity and bone mineralization during childhood, adolescence and even during adulthood. Therefore physical activity should be encouraged during the developmental years to promote optimal bone mineralization.

Endurance Training

Runners have also demonstrated increased bone mass. Brewer et al. (1983) compared BMD of the distal radius, hand and calcaneus in 30-40 yr old female marathon runners to age matched controls. The runners demonstrated significantly greater BMD of the mid-shaft radius and hand. Lane et al (1986) examined 41 male and female long distance runners 50-72 yrs. Bone mineralization of the first lumbar vertebrae was examined by computed tomography. Both male and female runners demonstrated on average, 40% greater BMC than sedentary controls. Similar results were also reported by Marcus et al. (1985) for lumbar spine BMD, however, no significant difference in distal radial BMD was demonstrated between exercise and control groups.

There are no studies examining the relationship between endurance training and bone mineralization in childhood and adolescence. Studies examining adults generally suggest a positive relationship between endurance training and bone

mineralization.

Endurance + Resistance Training

Menstruating resistance and endurance trained athletes aged 17-38 yrs were examined by Heinrich et al. (1990). Resistance trained athletes demonstrated greater BMC of the radius, lumbar spine, and hip compared to swimmers, collegiate runners, recreational runners and controls, while no significant difference in BMD was observed between these latter groups. Davee et al. (1990) found that college age women who supplemented their aerobic exercise training with resistance training, demonstrated higher lumbar spine bone densities than sedentary controls and those participating in aerobic exercise alone.

The effects of resistance training have not been examined in females during childhood and adolescence. In the adult population resistance training appears to be the preferential stimulus to optimize bone mineral over endurance training.

Anthropometric Variables

Cross-sectional studies have also been used to determine somatic correlates and predictors of bone mineralization, in children and adults

An examination of anorexic adolescent girls by Bachrach et al. (1991) demonstrated a significant correlation between

lumbar spine and whole body BMC with body mass index (BMI). Kroger et al. (1992) in an examination of 84 Finish children aged 6-19 yrs found BMC and BMD of the femur to be correlated with age, height, and weight ($0.74 \leq r \leq 0.92$). Miller et al. (1991) found height to be the best predictor of bone mass in children aged 5.3-14.4 yrs. These results were confirmed by Troverbach et al. (1991) who found BMC of the 1st metacarpal to be significantly correlated with height, weight, and age in children aged 6.8-10.7 yrs.

Examining young adult women, White et al. (1992) found BMD of the lumbar spine to be significantly correlated with body weight and fat-free mass in eumenorrheic and amenorrheic females age 15-21 yrs. Heinrich et al.(1990) demonstrated a significant correlation (r=0.38) between body weight and BMC of the distal radius in 17-38 yr old female athletes. Linnell et al. (1984) in an examination of amenorrheic vs eumenorrheic female runners found no difference in bone mineralization at the distal radius, however body fat was significantly correlated with BMC in the amenorrheic group only.

Body weight consistently appears to be one of the single most important predictors of bone mineral during childhood and adolescence. Maintenance of an adequate body weight seems essential for the optimal development of bone mineral during the formative growth years.

Physical Fitness

Snow-Harter et al. (1990) examining 59 females aged 18-31 yrs found BMD at the proximal femur to be correlated with muscle strength. Biceps strength proved to be the best predictor of proximal femur BMD and grip strength best predicted BMD of the lumbar spine and radius. Schoutens et al. (1989) found femoral bone mass of 19 yr old females to be significantly correlated (r=0.82) with quadriceps strength. Buchanan et al. (1988) found no correlation between bone mineralization and physical activity levels in females aged 18-22 yrs.

Pocock et al. (1986) observed physical fitness determined by the Astrand-Rhyming test as well as age and weight to be determinants of lumbar spine and femoral neck BMD in women aged 20-75 yrs. Pocock et al (1989) examining 78 women aged 23-75 yrs found biceps and quadriceps strength to be predictors of proximal femur and lumbar spine BMD. A positive correlation between trunk flexor/extensor strength and lumbar spine and proximal femur BMD in women aged 38-78 yrs was also observed by Halle et al. (1990)

An examination of post-menopausal women by Sinaki et al. (1989) found a weak, but nonetheless significant correlation (r=0.23) between BMD of the lumbar spine and back extensor strength. Bevier et al. (1989) demonstrated a positive correlation between grip strength and forearm BMC (r=0.37) and

spine (r=0.28) in women aged 61-84 yrs.

The correlation between muscular strength and bone mineral has not been examined thoroughly during the years of childhood and adolescence. The relationship between muscular strength and physical fitness to BMD/BMC is inconclusive in the adult population.

Generally the results from cross-sectional analysis suggest a positive association between level of habitual physical activity and peak bone mineralization. Body weight consistently appears as the best predictor of bone mineral in the younger age groups, while physical activity appears to slow the age associated loss of bone mineral post menopause.

Longitudinal Studies:

Longitudinal studies investigate the effect of an imposed exercise program on BMD over a given period of time. Problems associated with many of these trials include non-randomization, lack of adequate controls, poor subject compliance, and choice of measurement sites not stressed by the exercise protocol (Snow-Harter and Marcus 1991).

There is a paucity of intervention studies investigating the effect of exercise on bone mineralization during the formative growing years.

General Exercise Intervention

Bachrach et al. (1991) examined 15 anorexic females over a period of 12-15 months. An intervention program designed to increase body weight was demonstrated to improve lumbar spine BMD by as much as 30.3%. Leichter et al. (1989) reported an increase in tibial BMD (7.5%) in male army recruits aged 18-21 yrs following 14 weeks of strenuous physical training.

Resistance + Endurance Training

The effect of running verses weight-training on lumbar spine BMD of adolescent females with a mean age of 19 yrs was investigated by Snow-Harter and Marcus (1991). The exercise groups trained for eight months. During the final two months the runners averaged 10 miles per week, while the resistance group performed three sets of eight repetitions at 85% of one repetition maximum three times a week. The lumbar spine BMD of both the runners and the resistance trained groups increased significantly by 1.32% and 1.21% , respectively, compared to controls. The effect of weight-training (high intensity activity) and jogging (high repetition-low intensity) on whole body and lumbar spine BMD of 62 females, mean age 19.5 yrs, was examined by Lane et al. (1988). Bone density increased over a five month period in both exercise groups but was slightly higher in the weight-trained group. Peterson et al. (1991) subdivided women aged 36-67 yrs into aerobic dance,

aerobic dance + weight-training and control groups. An increase in muscle strength was observed with no parallel increase in BMD in the exercise groups. Chow et al. (1987) also randomized post-menopausal women age 50-62 yrs into aerobic and aerobic + resistance exercise groups. Bone mineralization determined by neutron activation increased significantly in both groups over controls, however no significant difference was observed between the two intervention groups. Rikli and McManis (1987) examined the effect of a 10 month exercise program on the distal radial BMC of post-menopausal women aged 57-83 yrs. The women were divided into three groups: general aerobics; aerobics + weight-training and control. There was a significant increase in BMC for both exercise groups of 1.3% while the control group demonstrated a decline of 2.5%. Similar results were observed for a 9 month weight-training program by Pruitt et al. (1992) and Chow et al. (1987).

There are no studies investigating the comparative effect of resistance training or resistance training supplemented with endurance training in females during childhood or adolescence.

Resistance Training

Pruitt et al. (1992) found that nine months of resistance training three times a week resulted in an increase of 1.6% of lumbar spine BMD of middle aged women while a decrease of 3.6%

was observed in the control group. No effect however was observed in regards to femoral neck or distal radius BMD.

A one year exercise program consisting of trunk resistive exercises did not increase lumbar spine or proximal femur BMD in 49 post-menopausal women (Smidt et al. 1992). Similar results were also found by Sinaki et al. (1989) who found no increase in spinal BMD following two years of an exercise program designed to increase back extensor strength. Simkin et al. (1987) examined the effects of dynamic loading exercises of the distal radius in 14 post-menopausal women diagnosed as osteoporotic. Following five months of exercise intervention BMD decreased by 1.9% in the control group and increased 3.8% in the exercise group. Dynamic loading of the distal radius in 14 women age 53-74 yrs for five months resulted in a 3.8% increase in distal radial BMD for the exercise group, while the control group decreased by 1.9% (Ayalon et al. 1987). The effect of squeezing a tennis ball 30 seconds a day for six weeks on the distal radius BMD of 99 elderly women was examined by Beverly et al. (1989). Grip strength increased by 14.5 % while BMC increased 3.4%. Six months post exercise BMC had reversed to baseline levels. The effectiveness of resistance training has not been examined in females during childhood or adolescence. Studies in adults generally support a positive effect of resistance exercise on bone mineralization.

Endurance Training

Dalsky et al. (1988) examined the effect of 22 months of walking and stair climbing on lumbar spine BMD of women aged 55-70 yrs. Lumbar spine BMD increased 5.2% in the exercise group while a decrease of 1.4% was observed in the control group. Sandler et al. (1985) examined the effect of a three year walking intervention trial on post-menopausal radial bone loss in 255 women. Walking alone did not increase bone mineral but when combined with adequate grip strength a positive effect on bone mineralization was observed.

The efficacy of 4 yrs of exercise intervention in deterring bone loss in middle age women was examined by Smith et al. (1989). Women with a mean age of 50 yrs exercised 3 times a week for 45 minutes a session. BMC of the radius, ulna and humerus were examined by single photon absorptiometry. Regardless of menopausal status, exercise subjects demonstrated lower bone loss rates than control subjects.

Generally endurance training tends to suppress the age related loss of bone in peri-menopausal women, however, the effectiveness of this type of training for optimizing bone mineral during the developmental years of bone growth is yet to be determined.

Exercise with Calcium or Estrogen Intervention

Exercise supplemented with calcium intervention has also been investigated in peri-menopausal women. Gleeson et al. (1990) examined the effect of a 12 month resistance training program on the lumbar spine and os calcis BMD of 68 pre-menopausal women. Both groups consumed 500 mg/day of supplemental calcium. Following the intervention period the exercise group demonstrated a non-significant increase of 0.8% in lumbar spine BMD while the control group decreased by 0.5%. Rockwell et al. (1990) examined the effect of weight-training on the lumbar spine of women with a mean age of 32.6 yrs. Bone density was measured by quantitative digital radiography. The intervention period lasted nine months during which time each subject ingested 500 mg/day of supplemental calcium. Even though strength increased by 57% in the exercise group lumbar spine BMD decreased by 3.96%. No change in BMD was observed in the control group.

Estrogen therapy alone and estrogen therapy plus resistance training was examined by Notelovitz (1991) in surgically induced menopausal women, with a mean age of 44 yrs. Variable resistance training increased BMD of the spine 8.3% as opposed to the increase of 1.5% observed in those women treated with estrogen alone.

Neither calcium nor estrogen intervention has been combined with exercise intervention in females during childhood or adolescence. In adult females the effectiveness of calcium

intervention when combined with exercise is inconclusive. Estrogen therapy, however, tends to enhance the effectiveness of resistance training in post-menopausal women.

The results of intervention trials tend to favour resistance training as the mode of exercise to optimize bone mineralization in the adult population. There are no long term intervention trials concerning the paediatric or adolescent female population. Longitudinal studies should be performed for at least one year to allow the bone to adapt to the newly imposed strain. The increase in BMD is transient and is lost if the imposed strain is removed.

The goals for exercise prevention and treatment of osteoporosis are different. The most important goal for non-osteoporotic women is to achieve optimal peak bone mass and to prevent rapid bone loss following menopause. If osteoporosis is present exercise should be initiated for reasons other than its affect on bone mass: to restore confidence, maintain joint mobility, improve posture, decrease pain and attain cardiorespiratory fitness (Aloia 1989).

The specific intensity, duration and frequency of exercise necessary to enhance bone mineralization have not yet been determined. The frequency by which a given area of bone surface undergoes remodelling is termed the activation frequency (Charles et al. 1991). This variable appears to be the primary determinant of bone turnover rate. If the level of

physical activity is too light, it cannot reach the threshold level required for changing bone metabolism. When the MES is exceeded, bone remodelling occurs in an attempt to increase bone strength and return the level of strain to within normal limits. Those forms of activity required to produce adequate strain appear to be weight bearing activities in which the muscular system has been overloaded for an extended period of time.

Hormones and Bone Mineral:

Estrogen

Estrogen has been described as an anti-resorptive agent that slows bone turnover by depressing resorption (Turner 1991a). Amenorrhea in young females has been associated with bone loss (Drinkwater et al. 1984), while the onset of female puberty with its associated increase in estrogen production increases adolescent bone mineral (Gilsanz et al. 1988). The effect of estrogen may be mediated through decreased responsiveness of bone to parathyroid hormone (PTH).

Estrogen may affect bone mass through changes in the set point for mechanical feedback as outlined by Turner (1991a). Osteoblasts have been shown to have estradiol binding sites suggesting that estrogen can act directly on bone to modulate remodelling (Benz et al. 1991). Based on evidence that decreased estrogen levels have been shown to: increase activation of remodelling, increase bone resorption and cause

a negative bone balance, Turner (1991b) hypothesized that a decrease in the mechanical set point of bone through estrogen deprivation would lead to greater bone resorption and thus a greater loss of bone mineral.

Georgiou et al. (1989) examined the effect of prior menstrual history on BMC of the forearm in 173 post-menopausal women. Results indicated that those women with the greatest number of regular menstrual cycles in the pre-menopausal years had the highest BMC. Genant et al. (1982) found that estrogen therapy following oophorectomy preserved bone mass. Studd et al. (1990) demonstrated that high dose estrogen replacement does increase lumbar spine BMD in post-menopausal women by 8.3%, but only after one year of therapy. A decreased rate of bone loss in adults with Turner's syndrome was observed following treatment with estrogen replacement. Nilas and Christiansen (1987) found that women of the same age but different menopausal status had significantly different bone masses, with pre-menopausal women having greater BMD, whereas a five year difference in age had no effect on bone mass in women with the same menopausal status. High dose estrogen therapy did not increase lumbar spine BMD of normal rat skeletons (Tobias et al. 1990), supporting the postulate of Frost that estrogen affects the set point for bone adaptation.

Estrogen deficiency leads to a significant bone loss in the remodelling skeleton, while estrogen replacement therapy

reverses this trend by reducing skeletal turnover. The anti-catabolic effect of estrogen appears to be through decreasing the responsiveness of bone mineral to parathyroid hormone. Anabolic effects occur through stimulation of matrix synthesis and cell proliferation. Lindsay (1987) suggests that estrogen treatment for post-menopausal women may reduce the incidence of hip fractures by 50% to equal the incidence of that found in men.

The onset of menses during early female adolescence appears to stimulate rapid bone formation. Amenorrhea during adolescence, whether primary or secondary, is associated with decreased bone formation. Therefore an adequate concentration of circulating estrogen is essential to the pubertal growth spurt and for the maintenance and optimization of bone mass during adolescence.

Progesterone

Progesterone is classified as a secondary female sex hormone. Current research of the effect of progesterone on bone mineralization is sparce; however, data suggests that progesterone has both direct and indirect effects on osteobalst cells (Prior 1990). Estrogen is proposed to act on the resorption side of the bone remodelling equation, while progesterone promotes bone formation. Total serum alkaline phosphatase and urine hydroxyproline excretion (markers of

bone resorption) have decreased during progesterone therapy (Abdalla et al. 1985). Progesterone production is high in the luteal phase of the ovulatory cycle and is low in the follicular phase (Prior 1990). Examining the effect of exercise on bone formation Prior (1992) demonstrated that inadequate production of progesterone (in cycles with short luteal phases and anovulatory cycles) is associated with accelerated bone loss, despite normal production of estradiol and the preservation of normal cycle intervals. These results suggest that the maintenance of peak bone density throughout adulthood requires normal ovarian production of both estrogen and progesterone.

Calcitonin

The primary role of calcitonin (CT) is to lower plasma calcium by inhibiting osteoclast mediated bone resorption (Chambers and Moore 1983). The administration of CT to animals caused osteoclasts to rapidly detach from bone surfaces and to lose their ruffled borders (Austin and Heath 1991). Besides its global inhibitory role, CT also diminishes osteoclast recruitment, where it is thought to work through the C-AMP second messenger system (MacIntyre 1992). This hypocalcemic hormone is secreted by the C-cells of the thyroid gland where its effect in slowing bone demineralization could protect the bone under extreme conditions of hypercalcemia (Manson and

Hirsh 1992). This was confirmed by Care (1992) who found a linear relationship between the circulating calcium concentration and the secretion of CT. A reduction in tubular resorption of calcium, and the direct stimulation of osteoblast and cartilage growth also appear to be secondary functions of CT (Wisneski 1992). In acute vertebral fractures CT has been found to prevent bone loss and possibly reduce healing time (MacIntyre 1992). Potts (1992) found eel or salmon CT to be more potent in man than CT from human's or other mammals.

As an agent inhibiting bone resorption, CT could possess therapeutic efficacy in those metabolic bone diseases associated with an increase in bone resorption. Without adequate levels of CT during childhood/adolescence normal bone formation would be hindered, as CT protects the skeleton during times of increased calcium need.

Parathyroid Hormone

Parathyroid hormone (PTH) is essential for the physiological maintenance of calcium homeostasis. The secretion of PTH is through the parathyroid gland; the principle stimulus for secretion is a decrease in plasma calcium ion concentration (Lui et al. 1991). In regulating calcium homeostasis, the hormone exerts important effects on bone and kidney, and indirectly affects the gastrointestinal tract. PTH increases

the release of the active form of vitamin D, calcitroil, by the kidney. The result is increased calcium absorption from the intestine and suppressed calcium excretion in the urine (Aloia 1989). The best documented effect of PTH is a catabolic effect on bone. The end result is the breakdown of bone mineral, manifested in an increased serum calcium concentration (Fatemi et al. 1991).

Osteoclasts bind PTH in a manner that displays the properties of receptor-dependant hormone binding (Agarwala and Gay 1992). Two phases of calcium mobilization have been described. An early phase occurs from 10 minutes to 3 hours after hormone exposure to bring about an increased activity of pre-existing osteoclasts (Lui et al. 1991). The second more sustained phase occurs over 24 hours and involves: an increase of osteoclast synthesis; an increase in ruffled border size and increased secretion of the lysosomal enzyme acid phosphatase. (Agarwala and Gay 1992).

Primary hyperparathyroidism is associated with a decrease in bone mass to about 10% below that of age matched controls (Fatemi et al. 1991). Marked excess or deficiency of PTH can cause severe or potentially fatal illness. Increased levels of PTH during the developmental growth spurt could lead to long term inadequacies in bone mineralization.

Glucocorticoids

The use of cortisol (glucocorticoid drugs) is associated with the rapid development of osteoporosis, as well as the inhibition of bone growth and fracture healing (Aloia 1989). Glucocorticoids have complex direct and indirect effects on skeletal tissue. The major indirect effect is the inhibition of calcium absorption from the intestine. This is thought to prompt secondary hyperparathyroidism (Gennari 1991). The major direct effect is a dose-related decrease in bone formation caused by a decrease in replication or differentiation of osteoblast precursors (Hahn et al. 1984). Cortisol can also have a positive effect on bone in physiological concentrations by increasing collagen synthesis and alkaline phosphatase levels (Gennari 1991). High levels of glucocorticoids have been found to magnify the cyclic-AMP response of bone cells to PTH, and may be one mechanism for the development of glucocorticoid-mediated osteopenia (Riggs and Melton 1986).

The main effect of glucocorticoids is the inhibition of calcium absorption from the intestine. Childhood and adolescence are times when optimal absorption of calcium from the diet is required to ensure normal bone growth. Therefore use of glucocorticoids in children should be avoided in order to prevent possible negative effects on bone growth.

Vitamin D

Technically, vitamin D is not a true vitamin because it can

be produced by the body. The most familiar property of vitamin D is its ability to cure rickets. The physiologically active form of vitamin D is $1,25(OH)_2D_3$ (Prince et al. 1988). Vitamin D is absorbed from the diet or synthesized from precursors in the skin in response to ultra-violet light (Tsai et al. 1987). Vitamin D must undergo two hydroxylation reactions, first in the liver, then in the kidney, before it can assume the role as a major calcitropic hormone (Tsai et al. 1984). The intestine and bone are the main target organs for vitamin D. Vitamin D stimulates the intestinal absorption of both calcium and phosphate (Prince et al. 1988). Under normal physiological concentrations vitamin D may promote bone mineralization directly, however under high concentrations it's actions may be to stimulate bone resorption (Maierhoffer et al. 1983).

Plasma levels of vitamin D decrease with age by more than 50%. The major contributors being decreased exposure to the sun and decreased renal production (Drezner and Nesbitt 1990). Increased intestinal calcium absorption can be detected within two hours after vitamin D administration . Parathyroid hormone appears to be the main hormonal stimulus for the increase in vitamin D production (Tsai et al. 1987). Calcitriol (vitamin D metabolite) has been suggested as a drug that may decrease bone resorption sufficiently to preserve trabecular bone structure in pre-menopausal women. Drezner and Nesbitt (1990) found that calcitriol abolished the loss of bone mineral

observed in oophorectomized beagles.

It is not clear whether the defect in age-related calcium absorption is secondary to the decrease in renal $1,25(OH)_2D_3$ production or whether it arises from an intrinsic age-related defect of intestinal calcium transport (Prince et al. 1988). In view of the fact that serum calcium levels rise post-menopausaly, PTH levels would be expected to fall. However PTH levels remain constant in this age group (Tsai et al. 1989). Therefore it is possible that in the absence of female sex steroids, the set point for calcium inhibition of PTH rises, resulting in higher calcium levels for the same PTH levels (Drezner and Nesbitt 1990).

In summary a decrease in vitamin D production causes a reduction in intestinal calcium absorption which in turn results in decreased serum calcium levels. The net result is onset of secondary hyperparathyroidism which leads to increased bone resorption.

Vitamin D is required to mobilize calcium and phosphorous from the intestine, therefore adequate levels are essential during childhood/adolescence when maximal calcium absorption is required via the intestine.

Prostaglandin E_2

Prostaglandin E_2 (PGE_2) has been shown to aid in both bone resorption and bone formation through alteration of the set

point of the bone structure control system (Turner 1991b). PGE_2 appears to be an "activating" agent - it activates bone remodelling, which begins with bone resorption, or bone modelling, which begins with either bone formation or resorption (Shin and Nordin 1986). Physical or mechanical stimuli to bone elicit an increase in the synthesis of PGE_2 which in turn increases cyclic AMP production and finally increased DNA synthesis (Binderman and Somjen 1984). Therefore PGE_2 acts as a first messenger in the transduction of mechanical usage to bone cells, and may have the ability to override or augment the effects of mechanical feedback signals, altering the set point of the bone structure control system.

The effect of PGE_2 on bone is biphasic, with its action depending on the concentration. Therefore inadequate levels during the childhood/adolescent growth spurt may effect the set point for bone formation leading to an inadequate bone structure.

Growth Hormone

Growth Hormone (GH) has been demonstrated to have anabolic effects on bone similar to PGE_2 by increasing bone formation (DiMartino-Nardi et al. 1991). GH also acts to increase the efficiency of calcium absorption and to decrease calcium excretion. GH in combination with calcitonin has been shown to

increase total body calcium (Aloia 1989). Anabolic effects of growth hormone on the skeleton are thought to be mediated by Insulin Like Growth Factor 1 (IGF-1). IGF-1 is present in skeletal tissue where its action is thought to stimulate proliferation of bone cells (Sklar et al. 1989).

Proper bone formation during the developmental growth spurt depends on the presence of GH where it is required to stimulate the proliferation of osteoblast-like cells.

The primary role of circulating hormones is the maintenance of plasma ionic calcium. Calcium is resorbed from bone when plasma calcium levels fall, or is deposited in bone when adequate plasma calcium is present. Estrogen is the single most important determinant of bone mineral in menarcheal and post-menopausal females. Bone mineral is rapidly accumulated during the years following the onset of menarche, and rapidly decreased in the years following menopause. Estrogen replacement therapy may be the most valuable weapon in the fight against osteoporosis.

Conclusion

Human growth and skeletal maturation are dynamic processes that start in utero and end somewhere during the third decade of life. This is the period when genetic and endogenous factors, interact with various environmental factors to determine skeletal peak bone mass.

Assuming that bone mass and its determinants could be measured with an error close to zero one could theoretically calculate the relative importance of the possible determining factors to the variability in bone mineral (Table 1)

The single most important variable in the determination of peak bone mass appears to be ones' genetic constitution. Body weight, height and parent's bone mineral status constantly appear as the most important other determinants of bone mass during the growing years.

When dietary calcium is low, plasma calcium level is regulated via bone resorption in response to hormonal stimulation. The amount of intestinal calcium absorption varies during the growing years, with highest values in infancy and adolescence. During these stages of development inadequate calcium intake may translate into inadequate calcium retention and consequently to a reduction of peak bone mass (Matkovic et al. 1990).

The fact that children are able to retain more calcium with an increase in calcium intake is possibly important. This

Factor	Attributable portion of population variance	
Heredity		0.25
Weight		0.10
Calcium deficiency		
Low intake	0.05	
Excess loss	0.05	0.15
Low absorption	0.05	
Exercise		0.15
Alcohol/smoking		0.10
Medications		0.05
Other		0.20
Total		1.00

Table 1: Proportion of variables determining adult bone

Taken from Heaney (1991)

could mean that a higher bone mass and density is possible but is not being achieved on current intake levels. Therefore a higher RDA for these age groups should be and is being considered (Avioli 1991).

The remodelling response of bone to mechanical strain during physical exercise may be site specific, requiring a dynamic rather than a static load. Bone tends to respond greater to mechanical strain associated with weight-bearing activity as opposed to non-weight-bearing activities. In most cases endurance training appears to have little affect on increasing bone formation, the greatest response of bone to exercise appears to be associated with resistance training. The adaptation of bone to the physical strain placed upon it appears to work through a tightly controlled physiological window. Strain is controlled by adjusting the mass and geometry of the bone while keeping the matrix composition constant. Once a functional level of bone mass has been achieved it is only maintained if the mechanically related stimulus continues.

The role of hormones is to regulate the extraction\formation of bone mineral . The feedback mechanism for this control is the level of circulating calcium. Estrogen, calcitonin, and growth hormone tend to promote bone formation leading to increased bone mineral, whereas parathyroid hormone and glucocorticoids tend to increase bone resorption leading to a

decrease in bone mineralization.

Because the developmental years are formative in the attainment of adult bone mineralization (which determines, in part, future fracture risk) it is important to identify and then to modify those factors which are determinants of bone mineral during this period. Although much is known about the physiological basis for the development and modification of bone, little is known about the factors which influence bone mass, their absolute or relative importance, or their responsiveness to intervention during childhood and adolescence.

This thesis is comprised of two related, yet separate studies. The first study is a cross-sectional investigation of the correlates and determinants of bone mass and density in healthy adolescent females. The second study is a prospective investigation of the effectiveness of a highly specialized type of physical activity, namely resistance training, in increasing bone mass and density in young girls during adolescence.

REFERENCES

Abdalla, H., Hart, D., Lindsay, R., Leggate, I., and Hooke , A., Prevention of bone mineral loss in postmenopausal women by norethisterone. Obstet. Gynecol. 66: 789-792, 1985.

Agarwala, N., and Gay, C., Specific binding of PTH to living cells. J. Bone Min. Res. 7: 531-539, 1992.

Aloia, J., **Osteoporosis: a guide to prevention and treatment.** Leisure Press, Champange Ill, 1989.

Aloia, J., Vaswani, A.,Yeh, J., and Cohn, S., Premenopausal bone mass is related to physical activity. Arch. Int. Med. 148:121-123, 1988.

Anderson, T., Toverud, J., Purification and characteriation of acid phosphatase from developing rat bone. Arch. Biochem. Biophys. 247: 131-139, 1986.

Angus, R., Sambrook, P., Pocock, N., and Eisman, J., Dietary intake and bone mineral density. Bone and Mineral. 4: 265-277, 1988.

Austin, K., Heath, H., Calcitonin: physiologhy and pathophysiology. N. Eng. J. Med. 304: 267-278, 1991.

Avioli, L.V. Calcium intake and bone health. Calcif. Tissue Int. 48: 221-223, 1991.

Ayalon, J., Simkin, A., Leichter, I., and Shalmoit, R., Dynamic bone loading exercises for postmenopausal women: effect on the density of the distal radius. Arch. Phys. Med. Rehabil. 68:280-283, 1987.

Bachrach, L.K., Katzman, G.D., Litt, I.F., Marcus, R. Decreased bone density in adolescent girls with anorexia nervosa. Pediatrics. 86:440-447, 1990.

Bachrach, L.K., Katzman D. K, Litt. I,F., Guido, D., Marcus, R.,Recovery from osteopenia in adolescent girls with anorexia nervosa. J. Clin. Endocrinol. Metab. 72: 602-606, 1991.

Bailey, D., and McCulloch, R., Bone tissue and physical activity. Can. J. Spt. Sci. 15: 229-239, 1990.

Baran, D., Sorenson, A., Grimes, J., Lew, R., Karellas, A., Johnson, B., and Roche, J., Dietary modification with dairy products for preventing vertebral bone loss in premenopausal women: a three year prospective study. .J Clin. Endocrinol. Metab. 70: 264-270, 1989.

Bell, N., Godsen, R., Henry, D., Sharp, J., ans Epstein, S., The effects of muscle building exercise on vitamin D and mineral metabolism. J Bone Min. Res. 3:369-373, 1988.

Benz, D., Haussler, M., Komm, B., Estrogen binding and estrogenic responses in normal human osteobalst-like cells. J Bone Min. Res. 6 (6): 531-541, 1991.

Bergstralh, E., Sinaki, M., Offord, K., Wahner, H., and Melton, J., Effect of season on physical activity score, back extensor muscle strength, and lumbar bone mineral density. J. Bone Min. Res. 5: 371-377, 1990.

Beverly, M., Rider, T., Evans, M., Smith, R., Local bone mineral resopose to brief exercise that stresses the skeleton. B. M. J. 229: 233-235, 1989.

Bevier, W., Wiswell, R., Pyka, G., Kozak, K., Newhall, K., and Marcus, R., Relationship of body composition, muscle strength, and aerobic capacity to bone mineral density in older men and women. J. Bone Min. Res. 4: 421-432, 1989.

Binderman, I., and Somjen, D., Biochemical pathways involved in the translation of physical stimulus into biochemical message. Calcif. Tissue Int. 36: S82-85, 1984.

Block, J., Friendlander, A., Brooks, G., Stieger, P., Stubbs, H.,and Genant, H., Determinants of bone density among athletes engaged in weight-bearing and non-weight-bearing activity. J. Appl. Physiol. 67: 1100-1105, 1989.

Bonucci, E., New knowlwdge on the origin, function and fate of osteoclasts. Clin. Orthop. 158: 252-269, 1981.

Boskey, A., Current concepts in the physiology and biochemistry of calcification. Clin. Orthop. 157: 225-257, 1981.

Brewer, V., Meyer, B., and Keele, M., Role of exercise in prevention of involutional bone loss. Med. Sci. Sport Exerc. 15: 445-449, 1983.

Buchanan, J., Myers, C., LLoyd, T., Leuenberger, P., and Demers,L., Determinants of peak trabecular bone density in women: The role of androgens, estrogen, and exercise. J. Bone Min. Res. 3: 673-680, 1988.

Buchanan, J., Myers, C., LLoyd, T., and Greer, R., Early vertebral bone loss in normal premenopausal women. J Bone Min Res. 3:583-587, 1988.

Cann, C., Martin, C., Genant, H., and Jaffe, R., Decreased spinal bone mineral content in amenorrheic women. J.A.M.A. 251: 626-629, 1984.

Care, A. The regulation of the secretion of calcatonin. Bone Mineral. 16: 182-185, 1992.

Carter, D., Mechanical loading histories and cortical bone remodelling. Calcif. Tissue Int. 36: S19-S24, 1984.

Chambers, T., Moore, A., The sensitivity of isolated osteoclasts to morphological transformation by calcitonin. J. Clin. Endocrinol. Metab. 57: 819-824, 1983.

Chan, G. Dietary calcium and bone mineral status of children and adolescents. A.J.D.C. 145: 631-634, 1991.

Chan, G., McMurray, M., Westover, K., and Thomas, R., Effects of increased dietary calcium intake upon the calcium and bone mineral status of lactating adolescent and adult women. Am. J. Clin. Nutr. 46:319-23, 1987.

Charles, P., Erikson, E., Hasling, C., Sondergard, K., and Moseklide, L., Dermal, intestinal, and renal obligatory losses of calcium: relation to skeletal calcium loss. Am. J. Clin. Nutr. 266S-735, 1991.

Cheng, S., Suomin, H., Rantanen, T., Parkatti, t., and Heikkinen,E., Bone mineral density and physical in 50-06 year old women. Bone and Mineral. 12: 123-32. 1991.

Choi, S., and Trotter, M., A statistical study of the multivariate structure and race-sex differences of american white and negro fetal skeletons. Am. J. Phys. Anthropol. 33: 307-312, 1970.

Chow, R., Harrison, J., and Notarius, C., Effect of two randomized exercise programmes on bone mass of healthy postmenopausal women. B. M. J. 295: 1441-1444, 1987.

Chow, R., Harrison, J., Brown, C., and Vlasta, h., Physical fitness effect on bone mass in postmenopausal women. Arch. Phys. Med. Rehabil. 67: 231-234, 1986.

Cooper, C., Barker, D., Wickham, C., Physical activity, muscle strength, and calcium intake in fracture of the proximal femur in britain. B. M. J. 297: 1443-1446, 1988.

Cumming, R. G., Calcium intake and bone mass: a quantitative review of the evidence. Calcif. Tissue. Int. 47: 194-201, 1990.

Currey, J., and Butler, G., The mechanical properties of bone tissue in children. J. Bone and Joint Surg. 57A: 810-817, 1975.

Dalsky, G., Stocke, K., Ehsani, A., Slatopolsky, E., Lee. W., and Birge, S., Weight-bearing exercise training and lumbar spine bone mineral content in premenopausal women. Ann. Int. Med. 108: 824-828, 1988.

Davee, A., Rosen, C., and Adler, R., Exercise patterns and trabecular bone density in college women. J. Bone Min. Res. 5:245-250, 1990.

Dawson-Hughes, B., Calcium supplementation and bone loss: a review of controlled clinical trials. Am. J. Clin. Nutr. 54: 274S-280S, 1991.

Dawson-Hughes, B., Dallan, G., Krall, E., Sadowski, L., Sahyoun,N., and Tannenbaum, S., A controlled trial of the effect of calcium supplementation on bone density in postmenopausal women. N. Eng. J. Med. 332: 878-883, 1990.

DiMartino-Nardi, J., Wu. R., Fishman, K., Saenger, P., The effect of long-acting analog of luteinizing hormone-releasing hormone on growth hormone secretory dynamics in children with precocious puberty. J. Clin. Endocrinol. Metab. 73: 902-906, 1991.

Drezner, M., and Nesbitt, T., Role of calcitroil in prevention of osteoporosis. Metabolism. 39: 18-23, 1990.

Drinkwater, B., Nilson, K., Chestnut, C., Bremmer, W., Sahinholtz,S., and Southworth, M., Bone mineral content of amenorrheic and eumenorrehic athletes. New Eng. J. Med. 311: 277-281, 1984.

Eastell, R., Yergey, A., Vieira, N., Cedel, S., Kumar, R., and Riggs, L., Inter-relationship among vitamin D metabolism, true alcium absorption, parathyroid function, and age in women: evidence of an age-related intestinal resistance to 1,25 dihydroxyvitamin D action. J. Bone. Min. Res. 2: 125-132, 1991.

Elders, P., Netelenbos, J., Lips, P., van Gilken, F., and van der Stelt, P., Calcium supplementation reduces vertebral bone loss in perimenopausal women: A controlled trial in 248 women between 46-55 years. J. Clin. Endocrinol. Metab. 73: 533-540, 1991.

Ernst, M., Heath, J., Schmid, C., Froesch, R., and Rodan, G., Evidence for the direct effect of estrogen on bone cells in vitro. J Steriod Biochem. 34: 279-284, 1989.

Everett, S., Reddan, W., Smith, P., Physical activity and calcium modalities for bone mineral increase in aged women. Med. Sci. Sports Exerc. 13: 60-64, 1981.

Faridi, M., Ansari, Z., and Bhagava, S., Imprints of protein energy malnutrition on the skeleton of children. J. Trop. Pediatr. 3: 150-153, 1984.

Fatemi, S., Ryzen, E., Flores, J., Enders, D., and Rude, R., Effect of experimental human magnesiun depletion on parathyriod hormone secretion and 1,25 dihydroxyvitamin D metabolism. J. Clin. Endocrinol. Metab. 71: 1067-1072, 1991.

Forbes, G., Body size and composition of perimenarchal girls. A. J. D. C. 146: 63-66, 1992.

Frost H. M., Bone mass and the "mechanostat": a proposal. The Anatomical Record. 219: 1-9, 1987.

Frost, H. M., Some ABC'S of skeletal pathophysiology. Calcif. Tissue. Int. 49:229-231, 1991.

Frost, H., Pathomechanics of osteoporosis. Clin. Orthop. and Related Res. 200: 1024-1027, 1989.

Fujita, T. Osteoporosis - east and west. Calcif. Tissue Int. 48:151-152, 1992.

Garn, S., Human biology and research in body composition. Ann NY Acad. Sci. 110: 429-426, 1963.

Garn, S., Sandusky, T., and Nagy, J., Advanced skeletal development in low income negro children. J. Pediatr. 80: 965-969, 1972.

Garn, S., and Clark, D., Nutrition, growth, development and maturation: findings from the ten-state survey 1968-1970. Pediatrics. 56: 306-319, 1975.

Genant, H., Cann, C., Ettinger, B., and Gordan, G., Quantitative computed tomography of vertebral spongiosa: a sensitive method for detecting bone loss after oophorectomy. Ann. Intern. Med. 97: 699-705, 1982.

Gennari, C., Glucocorticoids and bone. Bone Min. Res. 3: 213-231. 1991.

Georgiou, E., Galles, K., Pappeorgiou, A., Korkotsidis, A., Proukakis, C, Bone mineral loss related to menstrual history. Acta. Orthop. Scand. 2: 192-194, 1989.

Gilsanz, V., Gibbens, D., Carlson, M., Cann, C., and Schultz, E., Peak trabecular vertebral density: A comparison of adolescent and adult females. Calcif. Tissue Int. 43: 260-262, 1988.

Gleeson, P., Protas, E., LeBlanc, A., Schnieder, V., and Evans, H., Effects of weight lifting on bone mineral density in premenopausal women. J. Bone. Min. Res. 5: 264-269, 1990.

Granhed, H., Ragnar, J., and Hanson, T., The loads on the lumbar spine during extreme weight lifting. Spine. 2: 146-149, 1987.

Halle, J., Smidt, G., and O'Dwyer, K., Relationship between trunk muscle torque and bone mineral content of the lumbar spine and hip in healthy post menopausal women. Phys. Therapy. 70: 690-699, 1990.

Hamilton, J., Lingelbach, S., Partridge, N., and Martin, T., Regulation of plasminogen activator production by bone-resorbing hormones in normal and malignant osteobalsts. Endocrinol. 116: 2186-2191, 1985.

Harper, A., Laughlin, W., and Mazess, R., Bone mineral content in St. Lawrence island eskimos. Hum. Biol. 56: 63-78, 1984.

Hann, T., Westbrook, S., Halstead, R., Cortisol modulation fo osteoblast metabolic activity in cultured neonatal rat bone. Endocrinology. 114: 1864-1870, 1984.

Heanet, R., Calcium in the osteoporotic fracture context. Am. J. Clin. Nutr. 54: 242S-243, 1991.

Heaney, R., Calcium intake in the osteoporotic fracture context. Am. J. Clin. Nutr. 54: 242S-244S, 1991.

Heaney, R., Recker, R., and Savill, P., Menopausal changes in bone remodelling. J. Lab. and Clinical Med. 92: 964-970, 1978.

Heikkinen, J., Mattero, E., Kyllonen, E., Vuori, J., Takala, T., Moderate exercise does not enhance the positive effect of estrogen on bone mineral content in postmenopausal women. Calcif. Tissue Int. 49: S83-84, 1991.

Heinrich, C., Going, S., Pamenter, R., Perry, C., Boyden, T., and Lohman, T., Bone mineral content of cyclically menstrating female resistance and endurance trained athletes. Med. Sci. Sport Exerc. 22: 558-563, 1990.

Hert, J., Liskova, M., and Landgret, B., Reaction of bone to mechanical stimuli: continuous and intermittent loading of tibia in rabbit. Folia. Morph. 19: 290, 1971.

Huddelston, A., Rockwell, D., Kulund, D., and Harrison, B., Bone mass in lifetime tennis athletes. J.A.M.A. 244: 67-74, 1980.

Hurt, J., Liskova, M.m and Landrgot, B., Influence of long term continuous bending on bone. Fiola Morphilogica. 17: 389-399, 1969.

Jacobson, P., Beaver, W., Grubb, A., Taft, T., and Talmage, R., Bone density in women: college athletes and older athletic women. J. Orthop. Res. 2: 328-332, 1984.

Jaworski, Z., Lamellar bone turnover system and its effector organ. Calcif. Tissue Int. 36: S46-55, 1984.

Kanders, B., Dempsters, D., and Lindsay, R., Interaction of calcium nutrition and physical activity on bone mass in young women. J. Bone. Min. Res. 3: 145-149, 1988.

Kaneshisa, J., and Heersche, J., Osteoclastic bone resorption: invitro analysis of the rate of resorption and migration of individual osteoclasts. Bone. 9: 73-79, 1988.

Kelly, P., Pocock, N., Sambrook, P., and Eisman, J., Age and menopause-related changes in indicies of bone turnover. J. Clin. Endocrinol. Metab. 69: 1160-1165, 1989.

Kiiskinen, A., and Suominen, H., Blood circulation of long bone in trained growing rats and mice: biochemistry of long bones. J. Appl. Physiol. 44: 50-54, 1975.

Kroger, H., Kotaniemi, A., Vainio, P., and Alhaua, E., Bone densitometry of the lumbar spine and femur in children by dual-energy x-ray absorptiometry. Bone Min. Res. 17: 75-85, 1992.

Krolner, B., Toft, B., Nielsen, S., and Tonevold, E., Physical exercise as prophylaxis against involutional vertebral loss: a controlled trial. Clinical Science 64: 541-546, 1983.

Komn, B., Terpending, C., Benz, D., Graeme, K., Gallegos, A., Korc, M., and Haussler, M., Estrogen binding, receptor mRNA, and biologic response in osteoblast-like osteosarcoma cells. Science. 24: 81-84, 1988.

Laitinen, K., Valimaki, M., and Keto, P., Bone mineral density measured by dual-energy x-ray absorptiometry in healthy finish women. Calcif. Tissue Int. 48: 224-231, 1991.

Lakkakorpi, P., Vaananen, H. K., Kinematics of the osteoclast cytoskeleton during the resorption cycle in vitro. J. Bone Min.Res. 8: 817-826, 1991.

Lane, N., Bevier, W., Bouxsein, M., Carter, D., Marcus. R., Effect of exercise intensity on bone mineral. Med. Sci. Sport Exercise. 20: 198-207, 1988.

Lane, N., Bloch, D., Jones, H., Marshall, W., Fries, J., Long distance running, bone density and osteoarthritis. J.A.M.A. 255: 103-108, 1986.

Lanyon, L. E., Functional strain as a determinant for bone remodelling. Calcif. Tissue Int. 36: S56-61, 1984.

Lanyon, L., Rubin, C., Static vs dynamic loads as an influence on bone remodelling. Biomechanics 17: 897-905, 1984.

Leblanc, A., Schneider, V., Evans, H., Engelbretson, D., and Krebs, J., Bone mineral loss and recovery after 17 weeks of bed rest. J. Bone . Min. Research. 5: 843-850, 1990.

Leichter, I., Simkin, A., Marguilles, J., Biuas, A., Giladi, M., Milgrom, C., Gain in mass density of bone following streneous physical activity. J. Orthop. Research. 7: 86-90, 1989.

Leuenberger, P., Buchanan, J., Myers, C., Lloyd, T., and Demers,L., Determination of peak trabecular bone density: interplay of dietary fiber, carbohydrate, and androgens. Am. J. Clin. Nutr. 50: 955-61, 1984.

Li, J., Spencer, B., Ho, M., and Tsang, R., Bone mineral content in black and white children 1 to 6 years of age, early appearance of race and sex differences. Am. J. Dis. Child. 143: 1346-1349, 1989.

Lindsay, R., Estrogen and osteoporosis. Phys. and Sport Med. 11: 105-108, 1987.

Lindsay, R., Tohme, J., and Kanders, B., The effect of oral contraceptive use on vertebral bone mass in pre and post menopausal women. Contraception. 34: 333-340, 1986.

Linnell, S., Stager, J., Blue, P., Oyster, N., and Shaw, R., Bone mineral content and menstrual regularity in female runners. Med. Sci. Sport Exerc. 16: 343-348, 1984.

Liu, C., and Kalu, D., Human parathyroid hormone (1-34) prevents bone loss and augments bone formation in sexually mature ovarianectomized rats. J. Bone Min. Res. 5: 973-982, 1990.

Lohman, T., Going, S., Pamenter, R., Boyden, T., Houtkooper, L., Ritenbaugh, C., and Aickin, M., Effects of weight training on lumbar spine and femur bone mineral density in pre-menopausal females. Med. Sci. Sports Exerc. 24: S188, 1992.

Lui, C., Kalu, D., Salerno, E., Echon, R., Hollis, B., and Ray, M., Preexisting bone loss associated with ovarienectomy in rats is reversed by parathyroid hormone. J. bone Min. Res. 6: 1071-1080, 1991.

Lutz, J., Bone mineral, serum calcium, and dietary intakes fo mother/daughter pairs. Am. J. Clin. Nutr. 44: 99- 106, 1986.

Lutz, J., and Tesar, R., Mother-daughter pairs: spinal and femoral bone densities and dietary intakes. Am. J. Clin. Nutr. 52: 872-877, 1990.

MacIntyre, I., The calcitonin peptide family: relationship and mode of action. Bone Mineral. 16: 160-161, 1992.

Maierhoffer, W., Gray, R., Cheng, H., and Lemann, J., Bone resorption stimulated by elevated serum 1,25 (OH2) vitamin D concentrations in healthy women. Kideny Int. 24: 555-560, 1983.

Manson, T., and Hirsch, J., Physiology of calcatonin. Bone Min. 16: 162-165, 1992.

Marcus, R., Cann, P., and Madgiv, P., Menstrual function and bone mass in elite women distance runners. Ann. Int. Med. 102: 158-163, 1985.

Marcus, R., Kosek, J., Pfefferbaum, A., and Horning, S., Age related loss of trabecular bone in premenopausal women: A biopsy study. Calcif. Tissue Int. 35: 406-409, 1983.

Martin, M., and Burr, A., **Structure function and adaptation of compact bone.** New York. Raven Press, 1989.

Martin, A. D., McCulloch, R. G., Bone dynamics: stress, strain and fracture. J. Sports. Sci. 5:155-163. 1987.

Matkovic, V., Heanet, R., Calcium balalce during human growth: Evidence for threshold behaviour. Am. J. Clin. Nutr. 55: 992-996, 1992.

Matkovic, V. Calcium metablism and calcium requirements during skeletal modelling and consolidation of bone mass. Am. J. Clin. Nutr. 54: 254S-605, 1991.

Matkovic, V., Fontana, D., Tominac, C., Goel, P., Chestnut, C.,.Factors that influence peak bone mass formation: a study of calcium balance and the inheritance of bone mass in adolescent females. Am. J. Clin. Nutr. 52:878-88, 1990.

Mazes, R., and Matthew, W., Bone mineral content of North American alaskan eskimos. Am J Clin Nutr. 27: 916-925, 1974.

Mazes, R., and Cameron, J., Skeletal growth in school children: maturation and bone mass. Am. J. Phys. Anthropol. 35: 399-343, 1971.

McCormick, D., Ponder, S., Fawcett, H., and Palmer, J., Spinal bone mineral density in 335 normal and obese children and adolescents: evidence for ethnic and sex differences. J. Bone Min. Res. 6: 224-229, 1991.

McCulloch, R., Bailey, D., Houston, C., and Dobb, B., Effects of physical activity, dietary calcium intake and selected lifestyle factors on bone density in young women. C.M.A.J. 142: 221-227, 1990.

Melton, L., Ilstrup, D., Riggs, B., and Beckenbauch, R., five year trend in hip fracture incidence. Clin. Orthop. 162: 144-149, 1982.

Meuleman, J., Osteoporosis and the elderly. Geriatric Med. 73: 91-96, 1989.

Miller, J., Slemenda, C., Meaney, J., Reister, T., Hiu, S., and Johnston, C., The relationship of bone mineral density and anthropometric variables in healthy male and female children. Bone Min. Res. 14: 137-152, 1991.

Moller, M., Hursman, A., Harvald, B., Hauge, M., Henningson, K., and Nordin, B., Metacarpal morphology in monozygotic and dizygotic elderly twins. Calcif. Tissue Res. 25: 197-201, 1978.

Montoye, H. J. Better bone and biodynamics. Research. Q. 58: 334-348, 1987.

Nilas, C., Christiansen, C., Bone mass and its relationship to age and the menopause. J Clin. Endocrinol. Metab. 65: 679-702, 1987.

Nordin, C., Morris, H., The calcium deficiency model for osteoporosis. Nutrition Review. 47: 65-72, 1988.

Nordin, B., and Plooey, K., Metabolic consequences of the menopause. A cross-sectional, longitudinal and intervention study on 557 normal and post-menopausal women. Calcif. Tissue Int. 41: S1-59, 1987.

Notelovitz, M., Martin, D., Tesar, R., Kham, F., Probart, C., Fields, C., and McKienzie, L., Estrogen therapy and variable-resistance weight-training increases bone mineral in surgically menopausal women. J. Bone. Min. Res. 6: 583-590, 1991.

Parfitt, A. M. The cellular basis of bone remodelling: the quantum concept reexamined in light of recent advances in the cell biology. Calcif. Tissue. Int. 36: S37-S45, 1984.

Peacock, M. Calcium absorption efficience and calcium requirements in children and adolescents. Am. J. Clin. Nutr. 54: 261S-5, 1991.

Peterson, S., Peterson, M., Raymond, G., Gilligan, C., and Smith,E., Muscular strength and bone density with weight-training in middle aged women. Med. Sci. Sport Exerc. 23: 499-504, 1991.

Picard, D., ste Marie, L., Carrier, L., Lepage, R., and D'Amour,P., Premenopausal bone mineral content relates to height, weight and calcium intake during early adulthood. Bone and Mineral. 4: 229-309, 1988.

Pirnay, F., Bodeux, J., and Crielaard, M., Bone mineral content and physiacl activity. Int. J. Sport Med. 8: 331-335, 1987.

Pocock, N., Eisman, J., Gwinn, T., Sambrook, P., Freund, J., and Yeates, M., Muscle strength, physical fitness, and weight but not age predicted femoral neck bone mass. J. Bone . Min. Research. 4: 441-447, 1989.

Pocock, N., Eisman, J., Yates, M.m and Eberl, S., Physical fitness is a major determinant of femoral neck and lumbar spine bone mineral density. J. Clin. Invest. 78: 618-621, 1986.

Pollitzer, W., and Anderson, J., Ethnic and genetic differences in bone mass: a review with a heridetary vs environmental perspective. Am. J. Clin. Nutr. 50: 1244-1259, 1989.

Pollock, M., Garzarella, L., Graves, J., Carpenter, D., Tucci, J., and Manaquill, R., Effects of isolated lumbar extension resistance training on bone mineral density of the elderly. Med. Sci. Sports Exerc. 24: S66, 1992.

Potts, J., Chemistry of the calcitonins. Bone Mineral. 16: 169-173, 1992.

Prince, R., Dick, I., Boyd, K., and Garcia-Webb, P., The effects of dietary calcium deprivation on serum calcatroil levels in premenopausal and postmenopausal women. Metabolism. 37: 727-731, 1988.

Prior, J., Progesterone as a bone-tropic hormone. Endocr. Rev. 11: 386-398, 1990.

Prior, J., Vigna, Y., and Schiechter, M., Spinal bone loss and ovulatory disturbances. New Eng. J. Med. 323: 1221-1227, 1992.

Pruitt, L., Jackson, R., Bartels, R., Lehnard, H., Weight-training effects on bone mineral density in early postmenopausal women. J. Bone. Min. Research. 61: 179-185, 1992.

Raisz, L., Local and systemic factors in the pathogenesis of osteoporosis. New Eng. J. Med. 318: 818-828, 1988.

Raisz, L., and Kream, B., Regulation of bone formation. New Eng. J. Med. 309: 29-35, 1983.

Riis, B., Thomsen, K., and Christiansen, C., Does calcium supplementation prevent post-menopausal bone loss. N. Eng. J. Med. 316: 173-177, 1987.

Riggs, L., Wahner, H., Melton, M., Richelson, L., and Offord, K., Rates of bone loss in appendicular and axial skeletons of women. J. Clin. Invest. 77: 1487-1491, 1986.

Riggs, B., Wahner, H., Seeman, E., and Dunn, W., Changes in bone mineral density of the proximal femur and spine with aging. J. Clin. Invest. 70: 716-723, 1982.

Riggs, B., and Melton, L., Involutional osteoporosis. New Eng. J. Med. 314: 1676-1686, 1986.

Rikli, R., McManis, B., The effects of exercise on bone mineral content in postmenopausal women. Reasch.Quart. Exerc. and Sport. 61: 243-249, 1987.

Riuki, S., Cauley, J., Hom, D., Sashin, D., and Kriska, a., The effects of walking on the cross-sectional dimensions of the radius in post menopausal women. Calcif. Tissue Int. 41: 65-69, 1987.

Risser, W., Lee, E., LeBlanc, A., Poindexter, H., Risser, I., and Schneider, V., Bone density in eumenorrehiec female athletes. Med. Sci. Sports Exerc. 22: 570-574, 1990.

Rockwell, J., Sorensen, A., Baker, S., Leahey, D., Stock, I., and Baran, D., weight training decreases vertebral bone density in premenopausal women: A prospective study. J. Clin. Endcrinol. Metab. 71: 988-993, 1990.

Rodan, G., Mechanical loading, estrogen deficiency, and the coupling of bone formation to bone resorption. J. Bone Min. Res. 6: 527-530, 1991.

Rose, S., Municchi, G., Barnes, K., Kamp, G., Ross, J., Cassorla, F., and Cutler, G., Spontaneous growth hormone secretion increases during puberty in normal girls and boys. J. Clin. Endocrinol. Metab. 73: 428-435, 1991.

Rubin, C., Lanyon, L., Regulation of bone mass by mechanical strain magnitude. Calcif. Tissue Int. 37: 411-417, 1985.

Sandler, Riuka, Slemenda, C., LaPorte, R., Cauley, J., Schramm, M., Barresi, M., and Kriska, A., Post menopausal bone density and milk consumption in childhood and adolescence. Am. J. Clin. Nutr. 42: 270-274, 1985.

Schoutens, A., Laurent, E., and Poortmans. I., Effects of inactivity and exercise on bone. Sport Med. 7: 71-81, 1989.

Schulte, J., Townsend, E., and Hogg, J., Density of lean body mass is greater in blacks than in whites. J. Appl. Physiol. 56: 1647-1649, 1984.

Seeman, E., and Terri, A., Risk factors for osteoporosis. Aust NZ. J. Med. 19: 69-75, 1989.

Seeman, E., Hopper, J., Bach, L., Cooper, M., Parkinson, E., McKay, J., and Jerums, G., Reduced bone mass in daughters of women with osteoporosis. New Eng. J. Med. 320: 554-558, 1989.

Shaw, A., Wheeldon, J., Brockelbank, J., The tubular maximum for calcium reabsorption: A normal range for children. Clin. Endocrinol. 36: 193-195, 1992.

Shin, M., and Norrdin, R., Effects of prostaglandins on regional remodelling changes during tibial healing in beagles: a histomorphic study. Calcif Tissue Int. 39: 191-197, 1986.

Simkin, A., Aylon, J., Leichter, I., Increased trabecular bone density due to bone-loading exercises in post menopausal osteoporotic womwn. Calcif. Tissue Int. 40: 59-63, 1987.

Simmoms, A., Kinder, L., and Thomas, M., Effect of cortisone on cells at the bone-marrow interface. Calcif. Tissue Int. 46: 327-332, 1990.

Sinaki, M., Wahner, H., Offord, K., Hodgson, S., Efficacy of non-loading exercises in prevention of vertebral bone loss in postmenopausal women. Mayo Clin. Proc. 64:762-769, 1989.

Sinaki, M., Offord, K., Physical activity in post-menopausal women: effect on back muscle strength and bone mineral density of the spine. Arch. Phys. Med. Rehab. 69: 126-130, 1988.

Sklar, C.,Rothenberg, D., Blumberg, D., Oberfield, S., Levine, L. Suppression of the pituitary-gonadal axis in children with central precocious puberty: effects on growth, growth hormone, insulin-like growth factor-1, and prolactin secretion. J. Clin. Endocrinol. Metab. 73: 734-738. 1989.

Slemenda, W., Miller, J., Hiu, S., Reister, T. Role of physical activity in the development of skeletal mass in children. J. Bone Miner. Res. 6: 1227-1233, 1991.

Slemenda, C., Christian, J., Williams, C., Norton, J., and Johnston, C., Genetic determinants of bone mass in adult women: a reevaluation of the twin model and the potential importance of gene interaction on heritability estimates. J. Bone Min. Res. 6: 561-567, 1991.

Smidt, G., Lin, S., O'Dwyer, K., and Blanpied, P., The effect of high-intensity trunk exercise on bone mineral density in postmenopausal women. Spine 3: 265-270, 1992.

Smith, E., Gilligan, C., McAdam, M., and Smith, P., detering bone loss by exercise intervention in premenopausal and postmenopausal women. Calcif. Tissue Int. 44: 312-321, 1989.

Smith, E., and Gilligan, C., Effects of inactivity and exercise on bone. Phys. Sports Med. 15: 109-112, 1987.

Smith, D., Nance, W., Wonkang, K., Christian, J., and Jonhston, C., Genetic factors in determining bone mass. J. Clin. Invest. 52: 2800-2808, 1973.

Snow-Harter, C., and Marcus, R., Exercise, bone mineral density and osteoporosis. Exerc. Sport Sci. Rev. 19: 351-389, 1991.

Snow-Harter, C., Bouxsein, M., Lewis, B., Charette, S., Weinstein,P., Marcus, R., Muscle Strength as a predictor of bone mineral density in young women. J. Bone Min. Res. 5: 589-95, 1990.

Sowers, M., Burns, T., and Wallace, R., Familial resemblance of bone mass in adult women. Genet Epidemiol. 3: 85-93, 1986.

Studd, J., Sauvas, M., Watson, N., Garrett, T., Fogelman, I., and Cooper, D., The relationship between plasma estradiol and the increase in bone density in postmenopausal women after with subcutaneous hormone implants. Am. J. Obstet. Gynecol. 163: 1474-1479, 1990.

Swissa-Sivan, A., Simkin, A., Leichter, I., Nyska, A, Effect of swimming on bone growth and development in young rats. Bone and Mineral. 7: 91-105, 1989.

Talmage, R., Stinnett, J., Landwher, L., Vincent, L., and McCartney, W., Age-related loss of bone mineral density in non-athletic and athletic women. Bone Mineral. 1: 115-125, 1986.

Tanner, J., **Growth and adolescence** (2nd ed.). Oxford: Blackwell. 1962.

Tobias, J., Maxwell, J., and Chambers, I., Hormone replacement for osteoporosis. Lancet. 335: 1471, 1990.

Tran van, I., Vingery, A., and Baron, C., Cellular kinetics of the bone remodelling sequence in the rat. Anat. Rec. 202: 445-451, 1982.

Trevisan, C., Ortolani, S., Bianchi, M., Caraceni, M., Vlivier, F., and Polli, E., Age, Time since menopause, and body paramaters as determinants of female spinal bone mass: A mathematical model. Calcif. Tissue Int. 49: 1-5, 1991.

Trotter,M., Broman, G., and Peterson, R., Densities of bones of white and negro skeletons. J. Bone Joint Surgery. 42-A:58, 1960

Troverbach, W., de Mann, S., Gommers, D., and Grobbee, D., Determinants of bone mineral content in children. Bone Min. Res. 13: 55-67, 1991.

Turner, C. Do estrogens increase bone formation?. Bone. 12: 305-306, 1991a.

Turner, C. Homeostatic control of bone structure: an application of feedback theory. Bone. 12:203-217, 1991b.

Tsai, K., Ebeling, P., and Riggs, L., Bone responsiviness to parathyroid hormone in normal and osteoporotic women. J. Clin. Endocrinol. Metab. 69: 1024-1027, 1989.

Tsai, K., Wahner, H., Offord, K., Melton, J., Kumar, R., and Riggs, B., The effect of ageing on vitamin D stores and bone density in women. Calcif. Tissue Int. 40: 241-243, 1987.

Tsai, K., Heath, H., Kumar, R., and Riggs, B., Impaired vitamin D metabolism with ageing in women: Possible role in pathogenesis of osteoporosis. J. Clin. Invest. 73: 1668-1672, 1984.

Tylavsky, F., Bortz, R., Hancock, R., Anderson, J., Familial resemblences of radial bone mass between premenopausal mothers and their college age daughters. Calcif. Tissue Int. 45: 265-272, 1989.

Virvididakis, K., Georgiou, E., Korkotsidis, A., Ntalles, K., and Proukakis, C., Bone mineral content of junior competitive weightlifters. Int. J. Sports Med. 11: 244-246, 1990.

Wall, J., Chatterji, S., and Jeffery, J., Age related changes in the density and tensile strength of human femoral cortical bone. Calcif. Tissue Int. 27: 105-108, 1979.

White, C., Hergenroeder, A., and Klish, W., Bone mineral density in 15 to 21 year old eumenorrheic and amenorrheic subjects. A. J. D. C. 146: 31-35, 1992.

Widhe, J., Calcium metabolism in young undernourished rats fed diets differing in protein and calcium content. Clin. Orthop. 162: 304-309, 1982.

Wisneski, L., Review of calcatonin: future perspectives and new opportunities in therapy. Bone Mineral. 16: 169-173, 1992.

Wolman, R., Clark, P., McNally, E., Harries, M., and Reeve, J., Dietary calcium as a statistical determinant of spinal trabecular bone density in amenorrheic and estrogen-relpete athletes. Bone and Min. 17: 415-423, 1992.

Correlates and Determinants of Bone Mineral Content and Density In Healthy Adolescent Girls

by

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Abstract

The relationships between whole body (WB) and lumbar spine (LS) bone mineral content (BMC) and density (BMD), and measures of chronologic age, body composition, physical activity, cardiorespiratory and strength fitness, gynaecologic, sexual maturity, endocrine, and nutrition status were studied in 36 healthy menarcheal girls (14 - 18 y). Body mass ($0.464 < r < 0.704$), growth hormone ($-0.34 < r < -0.42$), and one repetition maximum double leg press strength ($0.343 < r < 0.467$) were significantly ($P < 0.05$), and moderately correlated with each of the 4 bone mineral measures. Average daily caloric intake was the most consistent moderately correlated dietary variable, but was significantly correlated with only LSBMC ($r = 0.478$) and LSBMD ($r = 0.382$). Multiple regression analysis indicated that body mass accounted for the largest significant proportion of the explained variance (27.3 - 55.1%) in each of the 4 bone mineral measures. Age at 1st menses accounted for a smaller but still significant proportion of the variance in WBBMD (9.3%), LSBMC (16.5%), and LSBMD (18.4%), and past yearly level of physical activity explained between 6.7% and 15% of the variance in WBBMC, WBBMD, and LSBMC. Average daily caloric intake accounted for a significant proportion of the variance in LSBMC (11.3%), and although just failing to reach significance, growth hormone

entered the regression model as an important predictor of LSBMD, accounting for 7.3% of the explained variance in this variable. Neither age, cardiorespiratory fitness, or strength contributed significantly to the explained variance in any of the bone mineral measures. Body mass appears to be an important determinant of bone mineral, and level of physical activity, although of relatively less importance, also seems to have a positive modulating effect on bone mineralization in girls during adolescence. Other nutritional, endocrine, and gynaecological factors also appear to contribute to the variability in bone mineral among females during this developmental period.

Index Terms

Bone Density; Adolescent Girls; Body Composition; Physical Fitness; Gynaecologic, Endocrine, and Nutritional Factors

Introduction

Osteoporosis is a major health problem in North America (34, 35). Concerns about personal physical disability and socio-economic costs associated with this condition have sparked considerable research into the topic of bone health in humans. Most of the research in this area has focused on adults, and peri- and post-menopausal women in particular, and numerous factors have been identified as important determinants of bone mass and predictors of risk for osteoporosis in these populations (11, 17, 23, 40).

In contrast, little is known about the determinants of bone mass, and the factors which influence bone mineralization in adolescent females. This is surprising since the amount of bone mass at any point in time during adulthood, and thus the risk of osteoporosis is partly a function of the bone mass achieved at maturity (35). Adolescence may be a critical period since 45-60 % of peak adult bone mass is deposited between puberty and adolescence (5, 40), and although not yet unequivocally established, this may also be the period when peak bone mass and density, at least for predominantly trabecular bone, achieve maturity (5, 15, 40).

It has been suggested that osteoporosis might be prevented or delayed by maximizing bone mass during the formative growth years (11, 23, 35). The effectiveness of this

approach depends first on identifying those factors which influence bone mass development, and second, on determining their relative importance to the bone mineralization process. The purposes of this study were to : 1) describe the correlational relationships between whole body (WB) and lumbar spine (LS) bone mineral content (BMC) and density (BMD), and selected lifestyle, fitness, gynaecologic, endocrine, and body composition variables and , 2) to identify the key predictors of predominantly cortical (whole body) and trabecular (lumbar spine) BMC and BMD from among these different classes of independent variables in healthy adolescent females.

METHODS

Subjects. Subjects were 36 girls recruited from a local high school. All subjects were given a medical and physical examination to rule out metabolic bone disorders and any other disorder which would preclude participation in physical activity or exercise. The study was approved by both internal (Chedoke-McMaster Hospitals) and external (St. Joseph's Hospital) human ethics medical review committees, and subjects and their parents signed consent forms approved by these committees.

Anthropometric and Cardiorespiratory Fitness Testing. Height

was measured with subjects standing in their stockings, and body mass was determined on a balance scale with subjects wearing only shorts and a T-shirt. Body mass index (weight/height^2) was calculated from these measurements. Skinfold thickness was measured at two sites (triceps and subscapular) using a Harpenden skinfold calliper. Percent body fat was predicted from the sum of skinfolds using an age and sex appropriate equation (36). Fat mass and fat free mass were calculated from measures of percent body fat and body mass. Peak oxygen uptake ($\dot{V}O_2$ peak) was obtained using an open circuit system on a Jaeger electronically braked cycle ergometer. The test protocol consisted of progressive increments (15 Watts) in work load every minute commencing at 15 Watts, until the subject could no longer maintain a cadence of 50-55 rpm. Oxygen uptake and other select cardiorespiratory variables were measured at 20 sec intervals throughout the test. Peak $\dot{V}O_2$ was determined as the highest value during the last minute of the test. Subjects remained seated during the test and were given verbal encouragement to continue until exhausted.

Questionnaires. Questionnaires were completed to determine past medical and gynaecological history. The age at onset of menses (determined to the nearest month) and the frequency of periods within the past year were determined from the

gynaecological history. Gynaecologic age was determined as the difference between age at onset of menses and current age. Sexual maturity status based on pubic hair development was determined by a nurse using the criteria of Tanner (43).

A modified questionnaire was used to determine past yearly and monthly energy expenditure in leisure activities (41). From a list of physical activities, subjects chose those in which they participated during the previous year or month and indicated the average intensity, frequency and duration of participation in each activity. With the exception of cycling and swimming, the other activities on the list were all weight bearing in nature. Subjects were also encouraged to complete an open-ended question about participation in activities not included in the list. Non-weight bearing activities were those in which the body mass was supported, for example, cycling, sailing, canoeing, swimming, and by exclusion, all other activities were classified as weight bearing. The Met activity scores for the past year and month were determined separately for total, weight bearing, and non-weight bearing activity as the summed product of the frequency, intensity and duration of selected activities during the specified time period. Current level of physical activity averaged over 2 weekdays and 1 day of the weekend was also assessed using the physical activity questionnaire developed by Bouchard et al. (6). Each 24 h period was subdivided into 15 min intervals and subjects

recorded the average intensity of physical activity (ranging from 1-9 Mets) for each interval. The scores were averaged over the 72 h period to provide a mean 15 min Met activity score. Subjects were also classified according to level of participation in organized sports. Girls involved in competitive sports (individual or team) were classified as highly active. Girls who participated in organized sports for leisure or recreational purposes were classified as active and those not involved in any form of organized sport were classified as inactive.

Strength Measurements. One repetition maximum lifts (1 RM) were determined on a Global Gym multi-station for double leg press and bench press using previously described procedures (33). In brief, subjects were given technical instruction in the proper execution of the lifts, and one practice session at low resistance to become familiar with the techniques. On a separate occasion, and following a specific warm-up of 6-8 repetitions at minimal resistance, subjects were given a series of trials of increasing resistance until the maximal load that could be lifted only once (the 1 RM) was established. The 1 RM load was determined to within 0.5 kg by the addition of small weight plates, and the maximum load was usually achieved after 6-8 trials. The order of testing for these lifts was randomized. Maximal voluntary quasi-isokinetic

strength was also determined for double elbow flexion, double knee extension, and the squat thrust using the HFMAXX hydraulic resistance machine (Hydra-Fitness Ind., Belton, Texas). Subjects were given instruction in the proper execution of these exercises, and 2 practice sessions at low and intermediate resistance to become familiar with the techniques. A load cell was mounted to each station and an electrical output proportional to the applied force was recorded on a strip chart recorder. The output from the load cell for each station was compared to standard force-velocity curves derived during maximal effort at various valve settings using a pre-calibrated strain gauge attached to the lever arms of the device. Three trials were performed for each exercise at valve settings corresponding to low (valve 1), intermediate (valve 3), and high (valve 6) resistance. The order of valve selection was randomized, and the load cell output from the two highest trials was averaged, converted to force in kg, and taken as the criterion measure. The order of testing for elbow flexion and knee extension was also randomized, and the squat thrust was always tested last. Maximal voluntary isometric strength was also determined for the right elbow flexors and right knee extensors using previously described procedures (3, 4). Measurements were made at a joint angle of 90 degrees (180 degrees is full extension). Isometric strength testing preceded the 1 RM testing, and both were done during a 2 hour

visit to the laboratory. Quasi-isokinetic strength testing (on the HFMAXX machine) was done on a separate occasion, and all strength tests were completed within a 2-3 week period.

Dietary Analysis. Current dietary intake was assessed using a 3 day food diary, consisting of two weekdays and one weekend day. Subjects were given written and verbal instruction regarding the recording procedure and were encouraged to be as accurate as possible in identifying the type and quantity of food consumed. The Nutritionist III food analysis program (N-Squared Computing Company, Silverton, Oregon) was used to determine total calorie (absolute and per kg mass), calcium and vitamin D intakes.

Blood Sampling. Subjects reported in a non-fasted state to a nurse between 3:15 and 9:30 am, 14 to 18 days following the last day of their most recent period (predetermined by interview). Venous blood was drawn, allowed to clot and then centrifuged. The serum was separated into aliquots and stored at -20° C until analyzed in batch for estradiol, progesterone, testosterone, cortisol, and growth hormone. Bloods were taken within 1-3 weeks of all other measurements.

Hormone Assays. Estradiol, progesterone, testosterone, cortisol, and growth hormone were measured by

radioimmunioassay (reagents from Diagnostics Products Corporation, Calbiochem, and Intermedico). Assays were performed by the Departments of Clinical Chemistry at Chedoke-McMaster Hospitals, St. Joseph's Hospital, and The General Hospital in Hamilton.

Bone Mineral Measurements. BMC and BMD for the whole body and for L2-L4 lumbar vertebrae were determined by dual photon absorptiometry (Norland 2600 dichromatic densitometer), using a ^{153}Gd radiation source. Separate scans were taken for the whole body and lumbar spine with subjects in the supine position. The densitometer was calibrated daily using a phantom supplied by the manufacturer. The accuracy of this technique in our laboratory has been established to be within 3.0 % of true values based on ash weight of a cadaver spine, and the precision, calculated as the ratio of standard deviation to the mean of repeated measurements of tissue equivalent phantom spine, to be 1.6 % (14, 45).

Statistical Analysis. Pearson product-moment correlation coefficients were computed between the 4 measures of bone mineral (WBBMC, WBBMD, LSBMC, LSBMD) and measures from the following classes of variables: anthropometric, physical activity, cardiorespiratory and strength fitness, sexual maturity and gynaecologic, dietary, and endocrine. Some

classes included multiple measures. For example, the anthropometric class variables included measures of body mass, body mass index, fat mass, fat free mass, percent body fat, and height; all measures described under each class variable heading in Table 1 were included in this analysis. A series of stepwise multiple regression analyses (MINITAB, Minitab Inc., Valley Forge, PA) with each of the four measures of bone mineral as the dependent variable was then computed. In addition to the general variable age, the single most consistent (across all bone mineral measures) and best correlated measure from each category of class variables was selected as a predictor variable in the regression analysis. Relationships were considered significant if $P < 0.05$.

RESULTS

The means and standard deviations for the bone mineral, anthropometric, physical activity, sexual maturity, gynaecologic, cardiorespiratory fitness, strength fitness, dietary, and endocrine measures are summarized in Table 1.

A summary of the correlational (univariate) relationships between the measures of bone mineral and the most theoretically important and highly correlated independent variables is given in Table 2. Chronologic age was not

Table 1. Descriptive characteristics of subjects

Bone Mineral Measures

WB-BMC(g)	2269.8 ± 350.4	LS-BMC(g)	4.309 ± 0.573
WB-BMD($g \cdot cm^{-2}$)	0.957 ± 0.070	LS-BMD($g \cdot cm^{-2}$)	1.054 ± 0.112

Anthropometric Variables

Age (y)	1.61 ± 1.0	Σ2Skf (mm)	33.2 ± 9.5
Height (cm)	164.3 ± 7.0	+Body Fat (%)	27.4 ± 5.6
Body Mass (kg)	59.1 ± 10.2	Fat Free Mass (kg)	42.4 ± 5.1
Body Mass Index ($kg \cdot m^{-2}$)	21.7 ± 3.2	Fat Mass (kg)	16.6 ± 6.3

Sexual Maturity and Gynecologic Variables

Pubic Hair (1-5 Stages)	4.2 ± 0.7	Time Since 1st Menses (y)	3.2 ± 1.3
Age at 1st Menses (y)	12.9 ± 1.0		
		Frequency of Menses Past Year	9-12/y n=31 <9/y n=2 none past 6 mths n=1 missing n=2

Physical Activity and Cardiorespiratory Fitness Variables

Total Past Year Act. ($Mets \cdot y^{-1}$)	110625 ±131279	Past Mth. Act. ($Mets \cdot y^{-1}$)	9258 ±13310
Past Year Weight Bearing Act. ($Mets \cdot y^{-1}$)	90714 ±120990	Current Act. ($Mets \cdot 15min^{-1}$)	2.1 ±0.2
Past Year Non-Weight Bearing Act. ($Mets \cdot y^{-1}$)	23676 ±33876	Peak $\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	38.9 ±5.4

Strength Variables

1 RM Bench Press (kg)	26.6 ± 6.3	Isokinetic Squat Thrust (kg)	131.1 ± 28.5
1 RM Double Leg Press (kg)	127.9 ± 19.2	Isometric Elbow Flexion (Nm)	34.5 ± 6.1
Isokinetic Elbow Flexion (kg)	18.2 ± 3.8	Isometric Knee Extension (Nm)	166.1 ± 31.2
Isokinetic Leg Extension (kg)	59.2 ± 11.2		

Dietary Variables

Daily Caloric Intake ($Kcal \cdot d^{-1}$)	1652 ± 532	Daily Calcium Intake ($mg \cdot d^{-1}$)	889.7 ± 474.7
Daily Caloric Intake ($Kcal \cdot d^{-1} \cdot kg^{-1}$)	28.7 ± 8.6	Daily Vit. D Intake ($IU \cdot d^{-1}$)	168.4 ± 131.5

Endocrine Variables

Estradiol ($pmol \cdot L^{-1}$)	228.5 ± 140.8	Cortisol ($nmol \cdot L^{-1}$)	351.9 ± 137.4
++Progesterone ($nmol \cdot L^{-1}$)	7.9 ± 9.7	Growth Hormone ($ug \cdot L^{-1}$)	3.8 ± 6.0
Testosterone ($nmol \cdot L^{-1}$)	1.4 ± 0.7		

Values are means ± S.D.
+Based on the age and sex specific equation of Slaughter et al. (36).
++Progesterone assay had a lower sensitivity of 2.0 $nmol \cdot L^{-1}$. Samples below this level (n=18) were ascribed the minimum value of 2.0 ($nmol \cdot L^{-1}$).

Table 2 : Pearson product-moment correlation coefficients for selected variables

CLASS VARIABLE CATEGORIES	WBBMC	WBBMD	LSBMC	LSBMD
General				
Age	-.064	-.006	-.044	-.034
Anthropometric Variables				
Height	.596*	.389*	.451*	.295
Body Mass	.704*	.464*	.588*	.511*
% Body Fat	.478*	.343*	.449*	.440*
Fat Free Mass	.668*	.429*	.523*	.420*
Fat Mass	.610*	.409*	.540*	.489*
Sexual Maturity & Gynacologic Variables				
Age at first Menses	-.048	-.164	-.283	-.215
Physical Activity & Cardiorespiratory Fitness Variables				
Total Past Year Act.	.135	.189	.320	.257
Past Year Act. (Non-Weight Bearing)	.109	.028	.077	.107
Past Year Act. (Weight Bearing)	.069	.181	.096	.093
Peak VO_2	-.130	-.063	-.008	-.039
Strength Variables				
IRM Double Leg Press	.467*	.343*	.409*	.405*
Isok. Leg Extension	.434*	.350*	.315	.302
Isom. Elbow Flexion	.396*	.233	.315	.171
Dietary Variables				
Daily Caloric Intake ($Kcal.d^{-1}$)	.368	.251	.478*	.382*
Daily Calcium Intake	.165	.049	.305	.293
Daily Vit. D. Intake	.233	.188	.385*	.372
Endocrine Variables				
Estradiol	.043	-.056	.052	.084
Growth Hormone	-.418*	-.420*	-.340*	-.389*

N = 36, except for the dietary variables where n = 27

* Coefficients are significant at $P \leq 0.05$ for n = 27 and n = 36 when $r \geq .374$ and $r \geq .325$, respectively.

significantly correlated with any of the bone mineral measures. With the exception of the relationship between height and LSBMD, all anthropometric variables were significantly ($P < 0.05$) and moderately correlated with the four bone mineral measures. Body mass was the most highly correlated anthropometric variable, and also displayed the highest of all correlations from the various class variables with the bone mineral measures. Correlations were higher between body mass and the measures of bone mineral content, than between body mass and bone mineral density.

None of the sexual maturity and gynaecologic variables correlated significantly ($P > 0.05$) with any of the bone mineral measures. Within this class however, age at first menses had the strongest and most consistent correlations with the four bone mineral measures.

There were no significant correlations between the measures of physical activity and cardiorespiratory fitness, and any of the measures of bone mineral. Total past yearly level of physical activity had the strongest and most consistent correlations with the four bone mineral measures within this class of variables, and just failed to reach the threshold level of significance for its relationship with LSBMC. There were no consistent or substantial differences in the strength of the correlations between weight bearing and non-weight bearing activity.

One RM double leg press strength was significantly positively correlated with all bone mineral measures. Quasi-isokinetic double leg extension strength was significantly positively correlated with only the whole body bone mineral measures, and isometric elbow flexion strength was significantly positively correlated with only WBBMC. None of the other strength variables correlated significantly with any of the bone mineral measures.

Daily caloric intake had the strongest and most consistent correlations of all dietary variables with the bone mineral measures, but was significantly correlated with only LSBMC and LSBMD. Daily vitamin D intake was significantly correlated with LSBMC, and just failed to reach significance for LSBMD. None of the remaining dietary variables correlated significantly with any of the bone mineral measures.

Growth hormone was significantly negatively correlated with each of the bone mineral measures. Neither estradiol, nor any of the other endocrine variables correlated significantly with any of the measures of bone mineral.

The results of the regression analyses are summarized in Table 3. Body weight accounted for the largest significant proportion (27.3 to 55.1%) of the explained variance for the four measures of bone mineral. Age at 1st menses accounted for a substantially smaller, but nonetheless, significant proportion of the variance in WBBMD (9.3%), LSBMC (16.5%), and

Table 3. **Regression models for the bone mineral variables**

Bone Mineral Variable	Constant ± SE	bX_1 ± SE Units t-Ratio R^2	bX_2 ± SE Units t-Ratio R^2	bX_3 ± SE Units t-Ratio R^2	bX_4 ± SE Units t-Ratio R^2	Cumulative R^2
Lumbar Spine (L_2-L_4)						
Content (g)	7.241 ±.206	.0341 Body Mass ±.0002 kg 4.56 42.2%	-.321 Age 1st Menses ±.0023 y -4.01 16.5%	.0004 Cal. Intake ±.0000052 Kcal 3.01 11.3%	.0000 Past Year Act. ±.0000 Mets·y^{-1} 2.32 6.7%	76.7%
Density ($g\,cm^{-2}$)	1.6823 ±.0103	.006 Body Mass ±.00005 kg 3.24 33.1%	-.060 Age 1st Menses ±.00056 y -3.37 18.4%	(-.0049 Growth Horm.) ±.00007 ug·L^{-1} -1.77 7.3%		58.8%
Whole Body						
Content (g)	2894 ±30.86	23.719 Body Mass ±.1502 kg 4.51 55.1%	.0012 Past Year Act. ±.0000 Mets·y^{-1} 1.77 6.7			61.8%
Density ($g\,cm^{-2}$)	1.1938 ±.0074	.0040 Body Mass ±.00023 kg 2.73 27.3%	.0000 Past Year Act. ±.0000 Mets·y^{-1} 2.29 15.0%	-.027 Age 1st Menses ±.0617 y -2.16 9.3%		51.6%

The following variables were included in each regression model, and represent the best univariate correlate from the different classes of variables with the bone mineral measures: age, body mass, age at 1st menses, past year activity, 1 RM double leg press strength, average daily caloric intake, and growth hormone level.

b = regression coefficients for the significant ($P<0.05$) predictor variables X_1 - X_n and R^2 indicates the percent of explained variation with the stepwise inclusion of independent variables. Cumulative R^2 is the total percent of explained variation attributed to the included predictor variables. Predictor variables in brackets are those which entered into the regression model, but which just failed to reach significance.

LSBMD (18.4%). Likewise, past yearly level of physical activity accounted for a small, but still significant proportion of the variance in WBBMC (6.7%), WBBMD (15.0%), and LSBMC (6.7%). A significant proportion of the variance in LSBMC was also attributed to average daily calorie intake (11.3%). Although just failing to reach significance (P = 0.084), serum growth hormone level did enter the regression model as an important predictor of LSBMD, accounting for 7.3% of the explained variance in this variable. Neither chronologic age, strength fitness (1 RM double leg press strength) nor cardiorespiratory fitness (peak VO_2) entered into the regression model for any of the dependent variables. Together and in various combinations, these variables explained between 51.6 and 76.7% of the variance in the four bone mineral measures.

DISCUSSION

There have been a number of recent investigations of the development of bone mass and density, and the determinants of bone mineralization in children and adolescents (1, 5, 15, 16, 18, 21, 28, 32, 38, 44). Most of these studies have spanned developmental periods characterized by tremendous changes in somatic growth (5, 15, 16, 21, 32, 38). Fewer studies have investigated the correlates and determinants of bone

mineralization during adolescence, a period during which somatic growth, especially for females is nearly complete (1, 18, 44). These studies have provided important information mostly about the univariate, and to a lesser extent, the multivariate relationships of a rather limited set of independent variables with various measures of bone mineral. With a multivariate approach, and by including a greater variety of theoretically important independent variables than has been used previously, we have tried to describe and quantify the relationships between these variables and bone mineral in a population of healthy adolescent females.

The primary finding of this study is that body mass was most highly correlated with and accounted for the largest proportion of the explained variance in the four bone mineral measures. These results are consistent with findings in healthy non-athletic children and adolescents (1, 18, 21) and adolescent male weightlifters (44). The results from the correlational analyses also indicated that there were positive associations between the bone mineral measures and virtually all other anthropometric variables. No physiological significance is attached to these findings however, since body mass was the only anthropometric variable to contribute significantly to the variance of the bone mineral measures in a separate regression analysis incorporating only the anthropometric measures. The positive associations between

body mass and bone mineral may reflect an adaptive response to the mechanical stress imposed by this mass (11).

Body mass was more highly correlated with, and accounted for a larger proportion of the variance in the bone mineral content than in the bone mineral density measures. Similar findings have been reported for healthy adolescent females (18). The higher correlations with bone mineral content probably reflect the effect of growth related bone expansion on mineral status during this period. Recent studies have shown that strong positive correlations between measures of bone mineral content and density and anthropometric variables diminish or disappear when bone mineral measures are corrected for bone size (18, 21). Various anthropometric correction factors based on geometric assumptions about the shapes of bones have been utilized to obtain bone size unrelated density measures (18, 21). To date, there is no generally accepted normalization procedure to account for the effects of bone expansion with growth on volumetric bone density, and no attempt was made in the present study to account for this effect. Since the bone mineral measures in the present study were uncorrected for bone size, it is difficult to predict with certainty, either the absolute or relative importance of the anthropometric or other variables to the development of true volumetric bone density during adolescence. Elaboration of these relationships await further development of a valid

and generally accepted normalizing procedure to account for the effects of bone expansion on bone mineral status during periods of growth.

Chronologic age was not correlated with, and did not contribute significantly to the variance in any of the bone mineral measures in this study. Age has been shown to be significantly correlated with (5, 21), and to be a significant predictor of bone mineral (38) in studies involving children of a wider age range than in the present study. The relative unimportance of age in this study probably reflects the narrow age range of subjects (14-18 y), and the failure of other studies to control for the covariance of age with other potentially more important predictor variables of bone mineral.

Height has also been shown to be significantly correlated with (5, 21), and to be a significant predictor of bone mineral (28) in children and adolescents. Height was moderately correlated with 3 of the 4 bone mineral measures in the present study, but failed to enter as a significant predictor of bone mineral after controlling for its covariance with body mass. The relative unimportance of height as a predictor of bone mineral in this study is probably due to reduced inter-subject variability in height since growth in stature is nearly complete in females at this stage of development. These findings are consistent with those of

Bonjour et al. (5) which indicated a fairly strong association between height and lumbar spine bone mineral in girls between 9 and 13 years of age, but a diminished correlation beyond this age range.

Body fatness, as reflected by both % body fat and fat mass was moderately positively correlated with all bone mineral measures in this study. These findings are consistent with previously reported positive correlations between body fatness and bone mineral in pre-pubescent children (32) and post-menopausal women (37), but are in contrast to the negative correlations recently reported by Miller et al. (28) in mostly pre-pubertal girls. Considerable changes occur in the development and distribution of fat throughout childhood, especially in females during the transition from childhood to adolescence (24). Developmental differences may account for the apparent discrepant results between the findings of Miller et al. (28), and our findings and those of others (32, 37).

Menstrual history has been identified as an important correlate of bone mass and density in adolescent girls (1, 5). None of the measures of sexual maturity or gynaecological status correlated significantly with any of the measures of bone mineral in the present study. Age at first menses, however, an index of relative reproductive maturity, did account for a significant proportion of the variance in three of the four bone mineral measures. Since age at first menses

was negatively correlated with the bone mineral measures, this suggests that the earlier the age at onset of menses, the higher the bone mineral content and density in these adolescent girls. This relationship may be explained by a greater total exposure of osteoblasts to estrogen during the longer interval since onset of menses (17, 38). Alternatively, some independent factor, perhaps some form of genetic regulation, may cause covariation in both age at onset of menses and bone development during the circumpubertal period. Both age at onset of menses and bone growth are believed to be highly genetically regulated (24). A common genetic influence appears to be the most likely explanation for this association since the interval between age at first menses and current age was only weakly correlated with the bone mineral measures.

Pubertal status was not significantly correlated with, and failed to account for a significant proportion of the variance in bone mineral in the present study. These results seem to be in contrast with those from a number of recent studies (5, 16, 18) which have demonstrated a positive association between bone mineral and pubertal status in samples of children spanning different stages of puberty. Most of the subjects in the present study, however, had already reached pubertal stage 5, and given their advanced level of sexual maturity it is not surprising that pubertal status was

not a significant predictor of bone mineral in these girls.

The various measures of physical activity and sports participation were only weakly correlated with the measures of bone mineral, and there was no apparent positive differential effect of weight bearing compared to non-weight bearing activity on bone mineral in this study. Despite these rather weak correlational relationships, past yearly level of physical activity did contribute modestly, but nevertheless significantly, to the explained variance in three of the four bone mineral measures in the regression models. The significance of physical activity in the multivariate, but not in the univariate analysis results from differences in the properties of the correlation and regression coefficients. The correlation coefficient measures the size of the relationship between the various measures of bone mineral and physical activity, without consideration of the covariance effects of other independent variables. The regression coefficient measures the effect of a change in physical activity on the unexplained portion of the variance in the bone mineral measures after controlling for the effects of other predictor variables and covariates (9). Whereas physical activity appeared to be an insignificant predictor of the total variation in bone mineral, it did significantly reduce the residual variation after accounting for the effects of other independent variables, most notably body mass.

Levels of physical activity and exercise during childhood have been positively associated with increased bone mass in females during adulthood (27, 42). Increased physical activity has also been shown to be positively correlated with bone density during childhood (38), and to be associated with increased bone mass both during childhood (38) and during the period spanning childhood and adolescence (21). Our findings are consistent with the aforementioned studies, and to our knowledge this is the first report of a positive association between current levels of physical activity and bone density in adolescent females.

Only two other studies (1, 18) have investigated the relationship between current levels of physical activity and bone mass and density in healthy females during adolescence. In both studies, there were no significant correlations between exercise habits and measures of bone mineral density. These findings are consistent with results from our correlational analyses, but not with the results of our regression analyses. The apparent discrepancy between studies may be explained by differences in sample characteristics, methods used to assess physical activity and statistical procedures used to determine the extent of relationship between physical activity and the various bone mineral measures.

Cardiorespiratory fitness was weakly correlated with, and

did not contribute significantly to the variance in any of the bone mineral measures in this study. These results suggest that cardiorespiratory fitness as measured by peak oxygen uptake is, as it also appears to be in adults (40), an unreliable and unimportant independent predictor of bone density in adolescent females. There are no other studies to our knowledge of the relationship between cardiorespiratory fitness and bone density in healthy adolescent females.

The relationship between muscle strength and bone mass and density has not been studied previously in adolescent females. In the present study, significant positive correlations were observed between 1 RM double leg press strength and all measures of bone mineral, and between quasi-isokinetic knee extensor strength and measures of whole body bone mineral. The association between 1 RM leg press strength and lumbar spine bone mineral, in the absence of a significant relationship with knee extensor strength, probably reflects the greater involvement of muscles of the lower trunk and hip in the leg press maneuver, and may represent a site-specific relationship with lumbar bone mineral. Despite these correlational relationships, however, strength fitness itself appeared not to be a significant independent predictor of variance in any of the bone mineral measures. The correlational relationships probably reflect the covariance of strength with some other independent variable (perhaps body

mass) which is a more important determinant of bone mineral than strength fitness itself.

Nutritional factors have been implicated as important determinants of bone health for adolescent females (17, 23). Calcium intake has been identified as one of the potentially most important nutritional factors in the development of bone mass in females during adolescence (25, 35). Dietary calcium intake was below the Canadian recommended level (13) for 13 girls, and the group's average daily calcium intake was below recommended levels according to the American allowance.

Despite its marginal adequacy, calcium intake was not significantly correlated with, and did not contribute significantly to the variance in any of the measures of bone mineral in the present study. These results are consistent with the findings of Bachrach et al. (1) and Katzman et al. (18) for circumpubertal and adolescent females, but are at variance with the findings of Sentipal et al. (35) for healthy preadolescent and adolescent girls. The discrepancy between studies is most likely explained by differences in sample characteristics and to a lesser extent, differences in approaches used to assess dietary intake between studies.

The positive correlations between daily calorie intake and lumbar spine bone mineral measures, and the significant contribution of daily calorie intake to the variance in LSBMC were two unexpected findings in this study. These findings

suggest that energy intake, perhaps by influencing trace elements such as manganese, phosphorous, copper and zinc which are required for normal bone development (17), may be an additional important independent dietary determinant of lumbar spine mineralization in adolescent girls. These results are consistent with findings in young amenorrheic runners, where low bone mass was associated with a reduced energy intake (30). In terms of maximizing bone development during adolescence, our results emphasize the importance of maintaining a diet that satisfies not only the recommended daily intake of specific macro-nutrients, but also an adequate energy intake.

Endocrine status has been implicated as an important regulator of bone mineralization in both adolescent and young adult females. Reduced serum estrogen related to athletic amenorrhea (11, 12, 30) and anorexia nervosa (1) has been associated with reduced bone mineral. Serum estrogen levels were only weakly correlated with, and did not contribute significantly to the variance in any of the bone mineral measures in the present study. These results are consistent with the findings of Drinkwater et al. (12) and suggest that current estrogen levels as assessed by a single blood sample may not be a good predictor of bone mineral in adolescent girls. Estrogen should not, however, be dismissed as an important regulator of bone mineral in this age group. The

cumulative influence on osteoblast activity and calcium metabolism of prolonged exposure to estrogen during the interval since onset of menses might prove to be a more important determinant of bone mineralization than current circulating estrogen levels (30).

The significant negative correlations between serum growth hormone and all measures of bone mineral, and the near significant contribution of growth hormone to the variance in LSBMD were unexpected findings. Mean serum growth hormone levels increase dramatically, and are associated with increased growth rate during puberty (24, 26). After reaching peak levels during late puberty, however, growth hormone levels decline progressively during adolescence toward adult values. Bone mass and density continue to increase toward adult peak levels (5, 18, 20) during this period of deceleration in both growth rate and growth hormone secretion. The negative associations between growth hormone and bone mineral in the present study probably reflects the divergent developmental patterns of these physiological processes. The results from this study suggest that the older more mature girls who have attained full or near full growth, have lower growth hormone and higher bone mineral levels than the younger girls whose growth is incomplete.

Despite the significant correlations, growth hormone failed to account for a significant proportion of the variance

in any of the bone mineral measures. The correlational relationships probably reflect the covariance of growth hormone with some other independent variable which may be a more important determinant of bone mineral than growth hormone itself. To establish the importance of growth hormone as an independent determinant of bone mineral during adolescence would require a more controlled longitudinal study, with serial fasting measures of either growth hormone or its related growth factors, and repeated measures of bone mineral spanning the period from late puberty to adulthood.

Although the contribution was not statistically significant, growth hormone level did enter the regression model as an important predictor of LSBMD in this study. This may reflect a lag or delay in the mineralization of the vertebral bodies which have a delayed growth spurt compared to other skeletal dimensions, and which continue to increase in length even into adolescence (10, 24). Younger less mature girls experiencing the largest increase in vertebral growth in response to relatively high levels of growth hormone would be expected to have the greatest lag in bone mineralization and consequently, lower lumbar bone mineral density. Other investigators have postulated a temporary dissociation between bone growth and mineralization during the pubertal growth spurt (2, 20), but this phenomenon remains to be confirmed and correlated with hormonal changes.

Neither serum cortisol, progesterone, nor testosterone correlated significantly with, or were significant predictors of the measures of bone mineral in this study. These results suggest that for these hormones, normal fluctuations within the physiological range have little influence on bone mineralization in healthy adolescent girls. There are no other studies of the relationship between these hormones and bone density in adolescent females. Our results are consistent however, with the findings of Drinkwater et al. (12) regarding the lack of association between progesterone and testosterone and bone mineral in young athletic women.

In various combinations, the independent variables accounted for between 51.6 % and 76.7 % of the variance in the bone mineral measures in this study. Much of the remaining variance may be attributed to genetic factors. Numerous studies have demonstrated a significant heritability of various bone mineral measures at commonly measured skeletal sites (8, 29, 31, 39). It also appears that the relative strength of this genetic effect may vary with age (8, 31, 39). It was not possible with the design of this study to determine the influence of genetics on the development of bone mineral in healthy girls during adolescence. In one study (39), there was evidence of a stronger genetic influence on bone mineral in juvenile (6-18 year olds) than in adult twins, but there was no consideration of the genetic effect during adolescence,

separately. The magnitude and significance of the genetic effect on bone mineral development in females during adolescence, and the relative strength of this effect compared to other stages of development and ageing remain to be determined.

CONCLUSION

The results from this study indicate that body mass is an important predictor of bone mineral content and density in healthy adolescent girls. Daily energy intake and past yearly level of physical activity were also identified as minor, but nonetheless significant predictors of selected measures of bone mineral in this study. Each of these variables is to some extent self-controlled, and can be manipulated by the individual. The importance of weight, diet and exercise management during adolescence both for optimal bone development during the growth years, and for the maximization of eventual adult peak bone mass should not be underestimated. Besides those variables which are predominantly self-controlled, other complex and biologically regulated factors which influence reproductive maturity and endocrine status also seem to play an important role in the development of bone mineral in young females during adolescence.

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REFERENCES

1. BACHRACH, L.K., D. GUIDO, D. KATZMAN, I.F. LITT, AND R.MARCUS. Decreased bone density in adolescent girls with Anorexia Nervosa. Pediatr. 86: 440-447, 1990.

2. BAILEY, D.A., J.H. WEDGE, R.G. McCULLOCH, A.D. MARTIN, AND S.C. BERNHARDSON. Epidemiology of fractures of the distal end of the radius in children as associated with growth. J. Bone Joint Surg. 71-A: 1225-1230, 1989.

3. BLIMKIE, C.J.R., B. EBBESEN, D. MacDOUGALL, O. BAR-OR, AND D. SALE. Voluntary and electrically evoked strength characteristics of obese and non-obese preadolescent boys. Hum. Biol. 61: 515-532, 1989.

4. BLIMKIE, C.J.R., D. SALE, AND O. BAR-OR. Voluntary strength, evoked twitch contractile properties and motor unit activation of the knee extensors in obese and non-obese adolescent males. Eur. J. Appl. Physiol. 61: 313-318,199

5. BONJOUR, J-P, G. THEINTZ, B. BUCHS, D. SLOSMAN, AND R. RIZZOLI. Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. J. Clin. Endocrinol. Metab. 73: 555-563, 1991.

6. BOUCHARD, C., A. TREMBLAY, C. LEBLANC, G. LORTIE, R. SAVARD, AND G. THERIAULT. A method to assess energy expenditure in children and adults. Am. J. Clin. Nutr. 37: 461-467, 1983.

7. CANADA FITNESS SURVEY. **Canadian Youth and Physical Activity**. Ottawa, Canada: Government of Canada, Fitness and Amateur Sport, 1983.

8. CHRISTIAN, J.C., P.-L. YU, C.W. SLEMENDA, AND C.C. JOHNSTON JR. Heritability of bone mass: a longitudinal study in aging male twins. Am. J. Hum. Genet. 44: 429-433, 1989.

9. DARLINGTON, R.B. Regression and linear models. New York: McGraw-Hill Publishing Company, 1990.

10. DESCHEPPER, J., M.P. DERDE, M. VAN DER BROECK, A. PIEPSZ, AND M.H. JONEKHEER. Normative data for lumbar spine bone mineral content in children: influence of age, height, weight, and pubertal stage. J. Nucl. Med. 32: 216-220, 1991.

11. DRINKWATER, B.L., B. BRUEMNER, AND C.H. CHESTNUT III. Menstrual history as a determinant of current bone density in young athletes. J.A.M.A. 263: 545-548, 1990.

12. DRINKWATER, B.L., K. NILSON, C.H. CHESTNUT III, W.J. BREMNER, S. SHAINHOLTZ, AND M.B SOUTHWORTH. Bone mineral content of amenorrheic and eumenorrheic athletes. N. Engl. J. Med. 311: 277-281, 1984.

13. FREMES, R., AND Z. SABRY. **Nutri Score**. Toronto: Methuen. 1976.

14. GALEA, V., S. ORMEROD, N. WHITE, J.D. MACDOUGALL, AND C.E. WEBBER. Body composition by photon absorptiometry. Can. J. Sport Sci. 15: 143-148, 1990.

15. GILSANZ V., D.T. GIBBENS, T.F. ROE, M. CARLSON, M.O. SENAC, M.I. BOECHAT, H.K. HUANG, E.E. SCHULZ, C.R. LIBANTI, AND C. CANN. Vertebral bone density in children: Effect of puberty. Radiol. 166: 847-850, 1988.

16. GLASTRE, C., P. BRAILLON, L. DAVID, P. COCHAT, P.J. MEUNIER, AND P.D. DELMAS. Measurement of bone mineral content of the lumbar spine by dual energy x ray absorptiometry in normal children: correlations with growth parameters. J. Clin. Endocrinol. Metab. 70: 1330-1333, 1990.

17. HEANEY, R.P. Nutritional factors in bone health. In: **Osteoporosis: Etiology, Diagnosis, and Management,** edited by B.L. Riggs, and L.J. Melton III. New York: Raven Press, 1938; p. 359-372.

18. KATZMAN, D.K., L.K. BACHRACH, D.R. CARTER, AND R. MARCUS. Clinical and anthropometric correlates of bone mineral acquisition in healthy adolescent girls. J. Clin. Endocrinol. Metab. 73: 1332-1339, 1991.

19. KAUFMAN, J.M., P. TAELMAN, A. VERMEULEN, AND M. VANDEWEGHE. Bone mineral status in growth hormone-deficient males with isolated and multiple pituitary deficiencies of childhood onset. J. Clin. Endocrinol. Metab. 74: 118-123, 1992.

20. KRABBE, S., C. CHRISTIANSEN, P. RODBROW, AND I. TRANSBOL. Effects of puberty on rates of bone growth and mineralization: with observations in male delayed puberty. Arch. Dis. Child. 54: 950-953, 1979.

21. KROGER, H., A. KOTANIEMI, P. VAINIO, AND E. ALHAVA. Bone densitometry of the spine and femur in children by dual-energy x-ray absorptiometry. Bone and Mineral 17: 75-85, 1992.

22. LOHMAN, T.G. Measurement of body composition. J.O.P.E.R.D. 53: 67-70, 1982.

23. LOUCKS, A. Osteoporosis prevention begins in childhood. In: **Competitive Sports For Children and Youth**, edited by E.W. Brown, and C.F. Branta. Champaign IL.: Human Kinetics Publishers Inc., 1988; p. 213-223.

24. MALINA, R.M., AND C. BOUCHARD. **Growth, Maturation, and Physical Activity**. Champaign, IL.: Human Kinetics Publishers Inc., 1991.

25. MATKOVIC, V., D. FONTANA, C. TOMINAC, P. GOEL, AND C.H. CHESTNUT III. Factors that influence peak bone mass formation: a study of calcium balance and the inheritance of bone mass in adolescent females. Am. J. Clin. Nutr. 52: 878-888, 1990.

26. MAURAS, N., R.M. BLIZZARD, K. LINK, M.L. JOHNSON, A.D. ROGOL, AND J.D. VELDHUYS. Augmentation of growth hormone secretion during puberty: evidence for a pulse amplitude-modulated phenomenon. J. Clin. Endocrinol. Metab. 64: 596-601, 1987.

27. McCULLOCH, R.G., D.A. BAILEY, S. HOUSTON, AND B.L. DODD. Effect of physical activity, dietary calcium intake and selected lifestyle factors on bone density in young women. Can. Med. Assoc. J. 142: 221-227, 1990.

28. MILLER, J.Z., C.W. SLEMENDA, F.J. MEANEY, T.K. REISTER, S. HUI, AND C.C. JOHNSTON. The relationship of bone mineral density and anthropometric variables in healthy male and female children. Bone and Mineral 14: 137-152, 1991.

29. MOLLER, M., A. HORSMAN, B. HARVALD, M. HAUGE, K. HENNINGSEN, AND B.E.C. NORDIN. Metacarpal morphometry in monozygotic and dizygotic elderly twins. Calcif. Tiss. Res. 25: 197-201, 1978.

30. NELSON, M.E., E.C. FISHER, P.D. CATSOS, C.N. MEREDITH, R.N. TURKSOY, AND W.J. EVANS. Diet and bone status in amenorrheic runners. Am. J. Clin. Nutr. 43: 910-916, 1986.

31. POLLITZER, W.S., AND J.J.B. ANDERSON. Ethnic and genetic differences in bone mass: a review with a hereditary vs environmental perspective. Am. J. Clin. Nutr. 50: 1244-1259, 1989.

32. PONDER, S.W., D.P. MCCORMICK, H.D. FAWCETT, J.L. PALMER, M.G. MCKERNAN, AND B.H. BROUCHARD. Spinal bone mineral density in children aged 5.00 through 11.99 years. A.J.D.C. 144: 1346-1348, 1990.

33. RAMSAY, J.A., C.J.R. BLIMKIE, K. SMITH, S. GARNER, J.D. MacDOUGALL, AND D. SALE. Strength training effects in prepubescent boys. Med. Sci. Sports Exerc. 22: 605-614, 1990.

34. RIGGS, B.L., AND L.J. MELTON. Involutional osteoporosis. N. Engl. J. Med. 314: 1676-1686, 1986.

35. SENTIPAL, J.M., G.M. WARDLAW, J. MAHAN, AND V. MATKOVIC. Influence of calcium intake and growth indexes on vertebral bone mineral density in young females. Am. J. Clin. Nutr. 54: 425-428, 1991.

36. SLAUGHTER, M.H., T.G. LOHMAN, R.A. BOILEAU, C.A. HORSWILL, R.J. STILLMAN, M.O. VANLOAN, AND D.A. BEMBER. Skinfold equations for estimation of body fatness in children and youth. Hum. Biol. 60: 709-723, 1988.

37. SLEMENDA, C.W., S.L. HUI, C.C. JOHNSTON JR., J.C. CHRISTIAN, C.J. WILLIAMS, AND F.J. MEANEY. Bone mass and anthropometric measurements in adult females. Bone Mineral 11: 101-109, 1990.

38. SLEMENDA, C.W., J.Z. MILLER, S.L. HUI, T.K. REISTER, AND C.C. JOHNSTON JR. Role of physical activity in the development of skeletal mass in children. J. Bone Min. Res. 6: 1227-1233, 1991.

39. SMITH, D.M., W.E. NANCE, K.W. KANG, J.C. CHRISTIAN, AND C.C. JOHNSTON JR. Genetic factors in determining bone mass. J. Clin. Invest. 52: 2800-2808, 1973.

40. SNOW-HARTER, C., AND R. MARCUS. Exercise, bone mineral density, and osteoporosis. In: **Exercise and Sport Sciences Reviews**, edited by J. Holloszy. Philadelphia: Williams and Wilkins, 1991; p. 351-388.

41. STEPHENS, T., AND C.L. CRAIG. **The Well-Being of Canadians: Highlights of the 1988 Campbell's Survey.** Ottawa, Canada: Canadian Fitness and Lifestyle Research Institute, 1990.

42. TALMAGE, R.V., AND J.J.B. ANDERSON. Bone density loss in women: effects of childhood activity, exercise, calcium intake and estrogen therapy. Calcif. Tissue Int. 36 (Suppl. 2): S52, Abstract, 1984.

43. TANNER, J.M. **Growth at Adolescence (2nd ed).** Oxford: Blackwell. 1962.

44. VIRVIDAKIS, K., E. GEORGIOU, A. KORKOTSIDIS, K. NTALLES, AND C. PROUKAKIS. Bone mineral content of junior competitive weightlifters. Int. J. Sports Med. 11: 244-246, 1990.

45. WEBBER, C.E. Some factors which influence the evaluation of a dual photon measurement of lumbar spine bone mineral mass. J. Can. Assoc. Radiol. 40: 87-91, 1989.

Effect of 26 Weeks of Resistance Training on Bone Mineral Content and Density in Healthy Adolescent Females

by

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Abstract

The purpose of this study was to: 1) determine the effect of 26 weeks of progressive resistance training (RT) on whole body and lumbar spine, bone mineral content (BMC), and bone mineral density (BMD) in healthy adolescent females and, 2) investigate the possible mechanism(s) underlying observed changes in bone mineral. Thirty five girls (mean age 16 yrs), matched for age, weight and sport participation, were randomized into either a training (N=17) or control group (N=18). Bone mineral density was determined by dual photon absorptiometry. Two separate scans were taken; one for the whole body and a second scan for the L2-L4 lumbar spine. Bone scans were taken at pre- and post-training for the whole body, and at pre-, mid- and post-testing for the lumbar spine. Progressive resistance training was performed on the HFMAXX and squat thrust hydraulic resistance machines. Training was comprised of 13 exercises at 7 stations, performed three times weekly for 26 weeks. One repetition maximum (1RM) strength was determined on a global gym for bench press and double leg press. Quasi-isokinetic strength was determined on the HFMAXX hydraulic resistance machines for double elbow flexion/extension, double knee flexion/extension and squat thrust. Isometric maximal voluntary strength was also determined for single elbow flexion and knee extension, both at a joint angle of 90 degrees. Strength measures were taken

at pre-, mid- and post-testing after 2-3 days recovery from the last training session. A three day dietary record was used to determine average daily calcium, vitamin-D and total caloric intake at the mid-point of the study. Venous blood samples were drawn at pre- and post-training, and analyzed for serum estrogen, creatine phosphokinase (CPK), acid-phosphatase and alkaline phosphatase. Training resulted in a significant increase of 4.6% ($p=0.001$) in lumbar spine BMD from pre- to mid-testing. No effect of training was observed for either whole body BMC and BMD or lumbar spine BMD from mid- or pre- to post-test measurements. Significant increases in strength were observed for the 1RM Bench Press measured on the global gym , and all strength measures on the HFMAXX resistance machine. No differential effect of training was observed for plasma estradiol, alkaline or acid phosphatase and creatine phosphokinase (CPK). Additionally, there was also no significant difference in dietary intake of calcium, vitamin D or total calories between groups. In conclusion, despite significant RT induced increases in voluntary strength, training had only a transient and relatively small positive effect on lumbar spine BMC at mid-point testing, while no significant effect of training was observed over the longer period of 26 weeks for any measure of bone mineral.

Introduction

One key to the prevention of problems associated with osteoporosis, may be the amount of bone present at skeletal maturity (Gleeson et al. 1990). Adolescence is a critical period in the attainment of peak bone mass, since 45-60% of adult peak bone mass is deposited during puberty and adolescence (Bonjour et al. 1991, Snow-Harter and Marcus 1991). Additionally, adolescence may be the period when adult peak bone mass and density achieve maturity (Bonjour et al. 1991, Gilsanz et al. 1988, Snow-Harter and Marcus 1991).

Relatively few studies have investigated the development and determinants of bone mass and density during childhood and adolescence (Bachrach et al. 1990, Bonjour et al. 1991, Gilsanz et al. 1988, Katzman et al. 1991, Virvidakis et al. 1990). Factors which have been identified as important determinants of bone mass and density during female adolescence and early adulthood include: body mass (Bachrach et al. 1991, Katzman et al. 1991, Kroger et al. 1992), physical activity (McCulloch et al. 1990, Slemenda et al. 1991), endocrine status (Bachrach et al. 1990, Drinkwater et al. 1990), muscular strength (Schoutens et al. 1989, Snow-Harter and Marcus 1991), nutritional factors (Heaney 1988, Loucks 1988) and menstrual history (Bachrach et al. 1990, Bonjour et al. 1991).

Level of habitual physical activity during childhood and

adolescence has been positively associated with increased bone mass in females during adulthood (McCulloch et al. 1990, Talmage and Anderson 1984). Recently, current levels of physical activity have been associated with increased bone mass in pre-pubescent male and female children (Slemenda et al 1991). Past yearly level of habitual physical activity has also recently been identified as a statistically significant determinant of variation in bone mineral mass and density in healthy adolescent females (Rice et al. in press)

Muscular strength has been positively correlated with bone mass and density in adult females (Halle et al. 1990, Schoutens et al. 1989, Sinaki et al. 1989, Snow-Harter and Marcus 1991), and the results from a recent study (Rice et al. in press) also indicate a moderate, yet significant positive correlation between 1RM double leg press strength and whole body and lumbar spine BMC and BMD in healthy adolescent girls. Results from these studies suggest that increased general physical activity, and activities that have a positive effect on skeletal muscle strength may have a positive influence on the development of bone mass and density in females from childhood to adulthood.

Resistance training (RT) is known to result in significant strength gains in both men and women (Kraemer et al. 1988). Resistance trained male athletes consistently demonstrate greater bone mass and density than endurance trained athletes

or controls (Virvidakis et al. 1990). Whether a similar positive association is evident in resistance trained female athletes remains to be determined. These results, however, suggest that RT may be the most effective form of physical activity or exercise for increasing bone mass and density in adults and perhaps even in children.

The effect of resistance training on bone mass and density has recently been investigated prospectively in adult females. Resistance training has been shown to slightly increase lumbar spine bone mineral density in pre-menopausal (Gleeson et al. 1990, Lane et al. 1986, Snow-Harter and Marcus 1991) and peri-menopausal women (Chow et al. 1987, Rikli and McManis 1987), but failed to significantly increase lumbar spine BMD in post-menopausal women (Sinaki et al. 1989, Smidt et al. 1992). In the latter two studies however, RT was successful in preventing the normal age related bone loss in these post-menopausal women.

To our knowledge there is no information regarding the responsiveness of either lumbar spine or whole body BMC or BMD to resistance training during the formative growth years in adolescent females. Therefore, the purposes of this study were to: 1) determine the effect of 26 weeks of progressive resistance training on whole body and lumbar spine BMC and BMD, and, 2) to investigate the mechanisms underlying resistance training induced changes in bone mineral content

and density in healthy menarcheal adolescent girls.

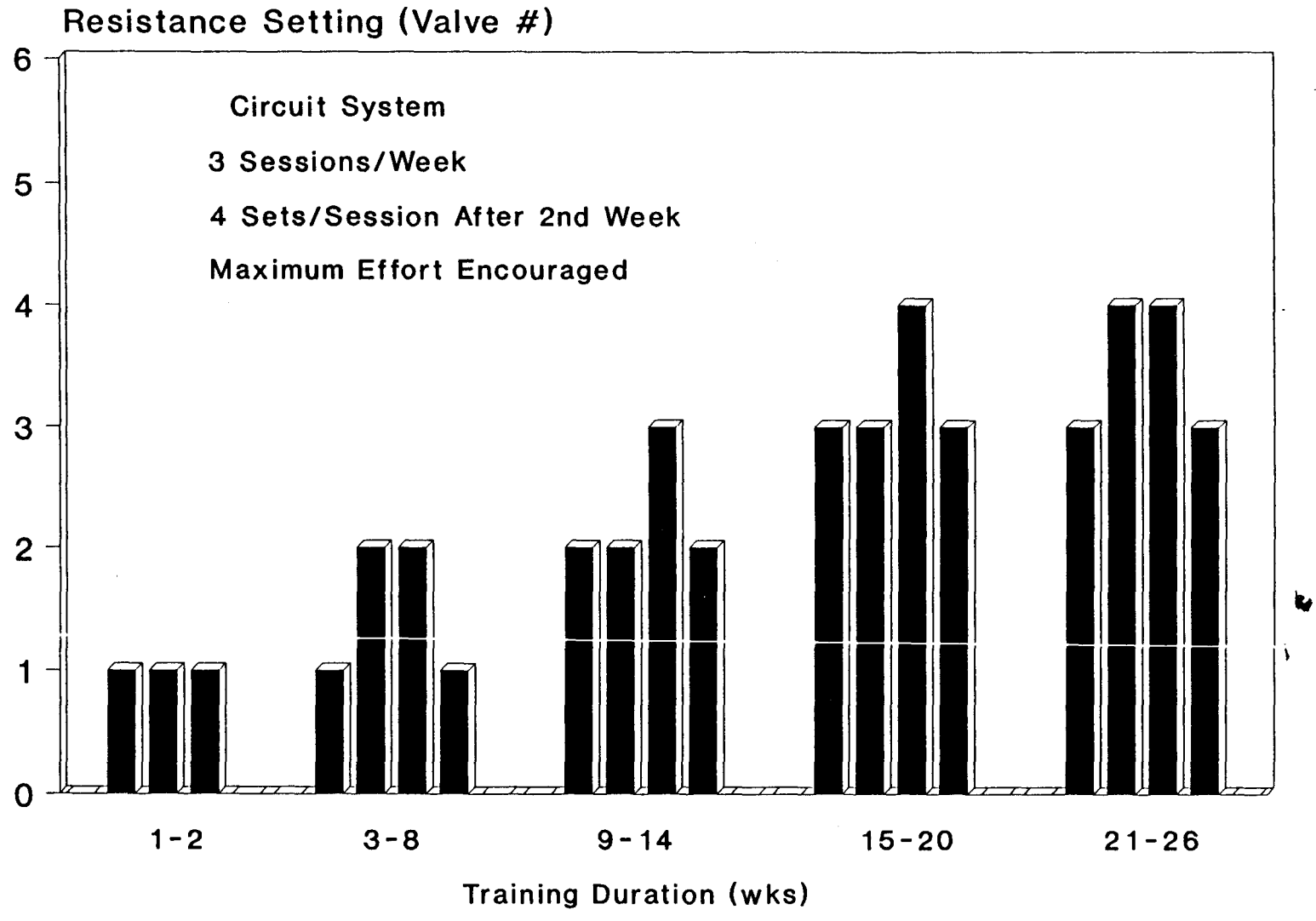
Methods

Subjects. Subjects were 35 girls (14-18 years of age) recruited from a local high school. Subjects were matched for age, body mass and sport participation and then randomized into an exercise (N=17) or control group (N=18). All subjects were given a medical and physical examination to rule out metabolic bone disorders and any other disorder which may have precluded participation in physical activity or exercise. The study was approved by both internal (Chedoke-McMaster Hospitals) and external (St. Joseph's Hospital) human ethics medical review committees. Both subjects and parents signed consent forms approved by these committees.

Training Regimen. Prior to pre-testing, both the exercise and control groups attended a lifting techniques training session. All subjects completed 2-3 practice sessions to become familiar with the proper execution of the lifting exercises selected for use in this study. Pre-test measurements were made on a separate day following the practice sessions. The experimental group trained three times per week for 26 weeks under supervision on the Hydra-Fitness HFMAXX and squat thrust hydraulic resistance training machines (Hydra-Fitness Ind., Beylon Texas). The first two weeks served as a break-in

period, during which training consisted of 3 sessions per week, of 2-3 sets of 10 repetitions (reps) of 13 different exercises with a light resistance (valve setting 1). The remaining 24 weeks training consisted of 3 sessions per week of 4 sets of 10-12 reps at varying resistance (Figure 1). A circuit training approach was used which consisted of 7 exercise stations. Training incorporated the following exercises: bicep curl / tricep press; knee extension / knee flexion; military press / lat pull down; squat press; abdominal crunch / trunk extension; seated incline press / row; and seated tricep dip / bicep pull. For the first 6 weeks immediately following the break-in period, training consisted of light to moderate resistance. The first and last sets were performed at a valve setting of 1, and the second and third sets at valve setting 2 (valve 1=minimal resistance, valve 6=maximal resistance). The resistance was increased at approximately 6 week intervals, as indicated by the valve settings in Figure 1. At the end of 26 weeks of training all subjects were training at a moderate to heavy resistance (the first and last sets were performed at valve setting 3, and the middle two sets at valve 4). Subjects were encouraged to give full effort during each repetition and to adequately rest between work stations. All exercises were performed reciprocally and concentrically with the exception of the squat lift. Training was interrupted for two weeks during

Training Program



Christmas and one week during the Easter holidays.

Strength Measurements. One repetition maximum lifts (1RM) were determined on a global gym multi-station for double leg press and bench press using previously described procedures (Ramsay et al. 1990). Subjects were given one practice session prior to testing to become familiar with the equipment and the proper execution of the exercises: the practice session involved sub-maximal lifting efforts only. The order of testing of these lifts was randomized. Maximal voluntary quasi-isokinetic strength was also determined for double elbow flexion/extension, double knee flexion/extension, and squat thrust using the HFMAXX and squat hydraulic resistance machines. A load cell was mounted to each station and an electrical output proportional to the applied force was recorded on a strip chart recorder. The output from the load cell for each station was compared to force velocity curves derived during maximal effort at various valve settings using a pre-calibrated strain gauge attached to the lever arm of the device. Three trials were performed at each valve setting corresponding to low (valve 1), intermediate (valve 3), and high (valve 6) resistance. The order of valve selection was randomized and the mean of the two highest trials was taken as the criterion measure. The order of testing for elbow flexion/extension and knee flexion/extension were randomized,

and the squat thrust was always tested last. Maximal voluntary isometric strength was also determined for the right elbow flexors and right knee extensors using previously described techniques (Blimkie et al 1990). Measurements were made at a joint angle of 90 degrees.Strength measurements were made at the beginning, mid-point (13th week) and end (26th week) of the study. All strength tests were completed within a 2-3 week period of the bone scan measurements, and for mid- and post-testing, a three day rest period was provided before the strength measurements, to control for residual training fatigue.

Anthropometry. Height was measured with the subjects standing in their stockings, and weight was determined on a balance scale with the subjects wearing only shorts and a T-shirt. Body mass index (weight/height^2) was calculated from these measurements. Skinfold thickness was measured at two sites (triceps and subscapular) using a Harpenden skinfold caliper. Percent body fat was predicted from the sum of skinfolds using an age and sex appropriate equation (Slaughter et al. 1988). Anthropometric testing was completed within 1-2 weeks prior to training, at mid-point, and within 1-2 weeks of post-training.

Blood Sampling. Subjects reported in a non-fasting state to a nurse between 8:15 and 9:15 am, 14 to 18 days following the

last day of their most recent period. Venous blood was drawn, allowed to clot and then centrifuged. The serum was separated into aliquots and stored at -20^0 C until analyzed in batch for estradiol, acid phosphatase, alkaline phosphatase and CPK. Estradiol was measured by radioimmunoassay (reagents from Diagnostics Products Corporation, Calbiochem, and Intermedico). CPK, acid and alkaline phosphatase were analyzed using a colorimetric enzymatic assay technique. Assays were performed by the departments of Clinical Chemistry at Chedoke-McMaster Hospitals, St. Joseph's Hospital, and the General Hospital in Hamilton. Blood samples were taken within 1-3 weeks of all other measurements prior to and following training.

Bone Mineral Measurements. BMC and BMD for the whole body and L2-L4 lumbar vertebrae were determined by dual photon absorptiometry (Norland 2600 dichromatic densitometer), using a ^{153}Gd radiation source with energies of 44 and 100 KeV. Bone mineral content represents the total amount of calcium phosphate crystal in a given scan, and bone mineral density is derived by dividing the BMC by the cross-sectional area of the skeleton for the whole body, and by the projected area of the scan between L2 and L4 vertebrae for the lumbar spine. Separate scans were taken for the whole body and lumbar spine with the subjects in the supine position. The densitometer was

calibrated daily using a phantom supplied by the manufacturer. The accuracy of this technique in our laboratory has been established to be within 3.0% of true values based on ash weight of a cadaver spine and the precision, calculated as the ratio of standard deviation to the mean of repeated measurements of a tissue equivalent phantom spine, to be 1.6% (Galea et al. 1990, Webber 1989). Whole body scans were taken pre- and post-training, whereas lumbar spine scans were taken at pre-, mid-and post-training.

Dietary Analysis. Current dietary intake was assessed using a 3 day food diary, consisting of two weekdays and one weekend day. Subjects were given verbal and written instructions regarding the recording procedure and were encouraged to be as accurate as possible in identifying the type and quantity of food consumed. The Nutritionist III food analysis program (N-Squared Computing Company, Silverton, Oregon) was used to determine total caloric, calcium and vitamin D intakes. Dietary analysis was determined for a total of 27 subjects (N=15, exercise group; N=12, control group). Dietary assessment was performed only once, mid-way through the study.

A summary of the time course for the assessment of the key dependant variables is presented in Figure 2.

Statistics. Two-way analyses of variance (ANOVA) with one

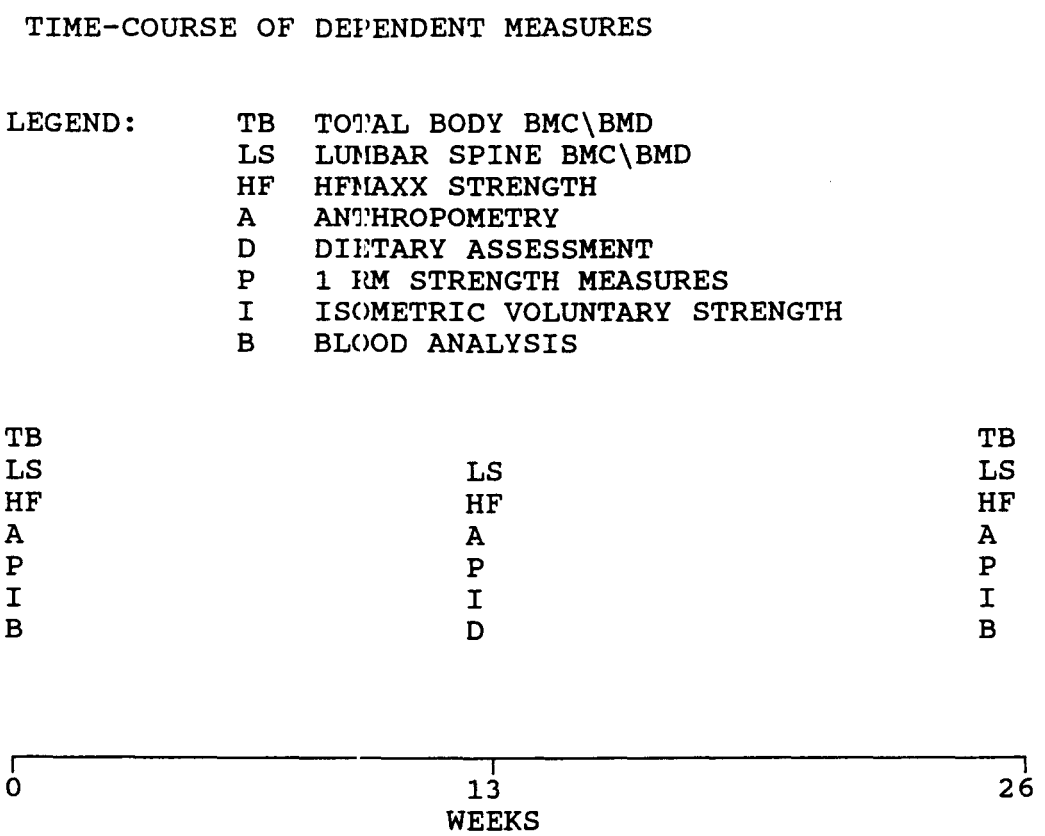


Figure 2. Time course for testing the dependent measures, including total body (TB) and lumbar spine (LS) BMC and BMD, HFMAXX and squat thrust strength (HF) anthropometry (A), 1 RM performance strength measures (P), isometric voluntary strength (I) and blood analysis (B). With the exception of dietary assessment, all other measurements were taken prior to (0) and after 13 and 26 wk of training.

variable repeated measures, were used to determine the significance of the training effect on the dependant variables. Analyses of covariance (ANCOVA) were performed when a significant difference in pre-test measures were found between the exercise and control group. The covariate used was the pre-test score for both the exercise and control group. A Tukey post-hoc test was used to identify differences among means when significance for main effects or interactions were found. A Students' two-tailed T-test was used to determine the statistical significance of differences between groups for the dietary analysis. Pearson product-moment correlation coefficients were computed between the largest percent changes in strength for leg flexion and squat thrust, at valves 3 and 6 respectively, and the percent change in whole body and lumbar spine BMC and BMD for the exercise group

Analyses were performed using the CSS statistical software package, with the level of significance set at $P<0.05$.

A summary of the statistical analyses used for the various dependent measures is provided in Table 1.

Results:

Descriptive Characteristics. A summary of the descriptive characteristics of the subjects is provided in Table 2. There were no significant differential effects of training on any of

Table 1: Summary of Statistical Analyses Used to Determine Significance for Dependent Variables

BONE				GLOBAL GYM		QUASI-ISOKINETIC STRENGTH															BLOOD ANALYSIS	DIET ANALYSIS	DESCRIPTIVE CHARACTERISTICS
						ARM FLEXION			ARM EXTENSION			LEG FLEXION			LEG EXTENSION			SQUAT					
TBBMD	TBBMC	LSBMD	LSBMC	BP	DLP	1	3	6	1	3	6	1	3	6	1	3	6	1	3	6			
A	C	A	A	C	A	A	A	A	A	A	A	A	A	C	A	A	A	A+C	C	C	A	T	A

A = ANALYSIS OF VARIANCE
C = ANALYSIS OF COVARIANCE
T = T-TEST
NUMBERS (1 3 6) REFER TO RESISTANCE SETTINGS
TBBMD = TOTAL BODY BONE MINERAL DENSITY
TBBMC = TOTAL BODY BONE MINERAL CONTENT
LSBMD = LUMBAR SPINE BONE MINERAL DENSITY
LSBMC = LUMBAR SPINE BONE MINERAL CONTENT
BP = BENCH PRESS
DLP = DOUBLE LEG PRESS

Table 2: Descriptive Characteristics of Subjects

	AGE (YR)	HEIGHT (CM)		WEIGHT (KG)		* BODY FAT (%)		BMI (kg/m2)	
	pre	pre	post	pre	post	pre	post	pre	post
Exercise	16.3 0.3	163.3 1.9	163.9 1.8	57.8 2.7	58.1 2.7	30.4 1.2	29.8 1.2	21.7 0.9	21.5 0.7
Control	16.1 0.2	165.2 1.6	165.3 1.6	59.8 2.3	60.5 2.3	30.9 0.7	29.9 0.8	21.7 0.6	22.1 0.6

* Based on the age and sex specific equation of Slaughter et al., Hum. Biol. 60: 709-723, 1988.
Results are reported as mean +/- SEM

the anthropometric or body composition variables.

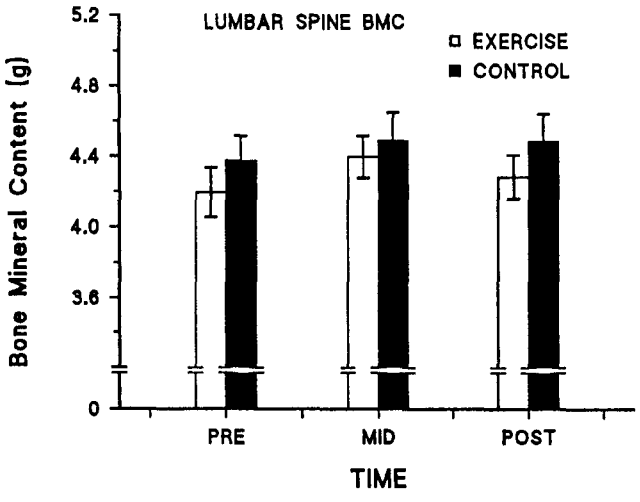
Bone Mineralization. The results for bone mineral are depicted in Figure 3. No difference between groups was observed in pre-test values for either total body or lumbar spine BMD. A difference was observed between exercise and control groups for total body BMC (2220 ± 339 g vs 2311 ± 384 g; P=0.00017), and lumbar spine BMC (4.2 ± 0.6 g/cm vs 4.4 ± 0.6 g/cm; p=0.005) respectively. ANCOVA revealed no effect of training on lumbar spine or total body BMC. No effect of training was observed for whole body BMD using ANOVA; however, a significant main effect for time (p=0.002) and a time by group interaction (p=0.02) was observed for lumbar spine BMD. Post-hoc analysis revealed a significant (p=0.001) increase of 4.6% (4.2 ± 0.6 to 4.4 ± 0.5 g/cm^2) for lumbar spine BMD in the exercise group from pre- to mid-testing; however, there was no difference between pre- to post-test values in the training group. There were no significant changes in lumbar spine BMD in the control group during the study period.

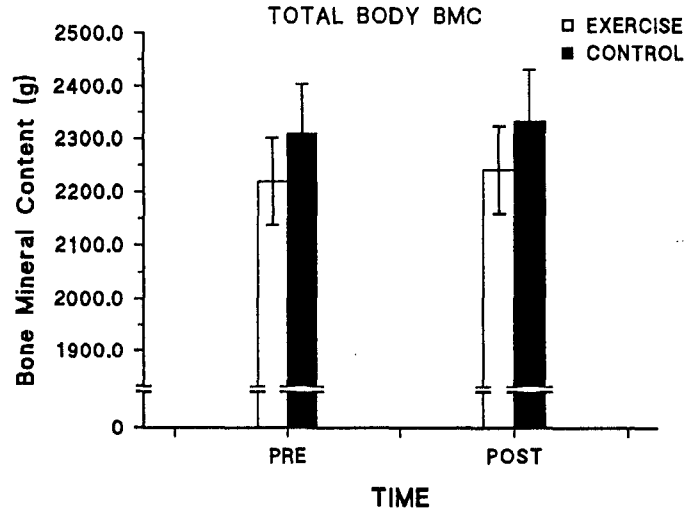
1 RM Performance Strength.

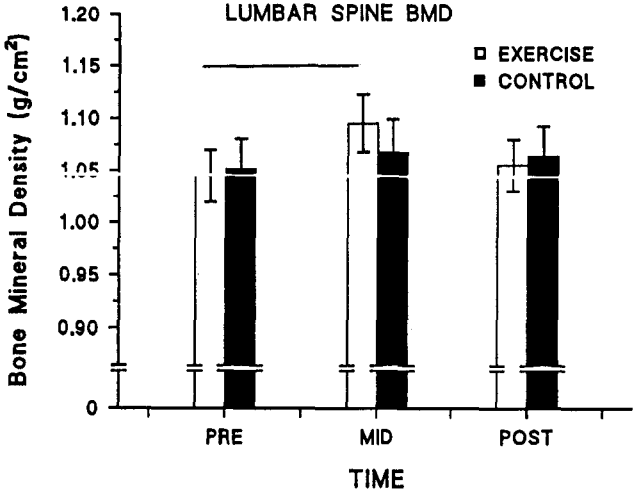
Global Gym. The results for the 1RM performance strength measures are provided in Tables 3 and 4. After adjusting for pre-test differences, ANCOVA revealed significantly (p=0.003) higher 1RM bench press strength in the trained versus the

Figure 3. Results for whole body and lumbar spine BMC and BMD for pre-, mid- and post testing.

Bar with the line across the top, indicates a significant difference bewteen pre- to mid-testing for lumbar spine BMD.


LUMBAR SPINE BMC
EXERCISE
CONTROL
Bone Mineral Content (g)
5.2
4.8
4.4
4.0
3.6
0
PRE
MID
POST
TIME


TOTAL BODY BMC
EXERCISE
CONTROL
Bone Mineral Content (g)
2500.0
2400.0
2300.0
2200.0
2100.0
2000.0
1900.0
0
PRE
POST
TIME


LUMBAR SPINE BMD
EXERCISE
CONTROL
Bone Mineral Density (g/cm²)
1.20
1.15
1.10
1.05
1.00
0.95
0.90
0
PRE
MID
POST
TIME

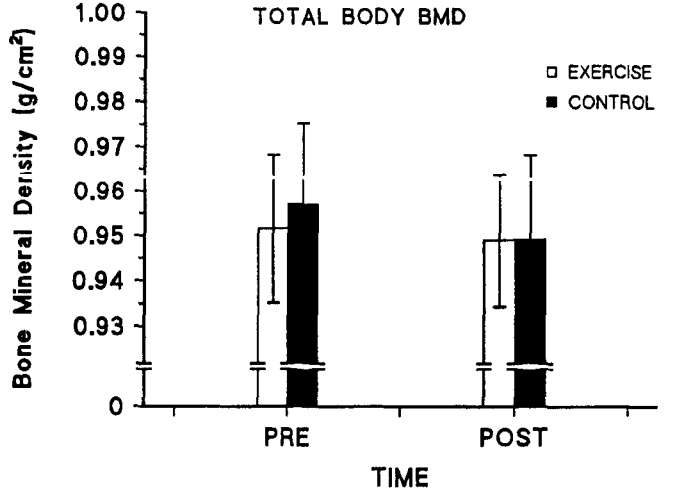

TOTAL BODY BMD
EXERCISE
CONTROL
Bone Mineral Density (g/cm²)
1.00
0.99
0.98
0.97
0.96
0.95
0.94
0.93
0
PRE
POST
TIME

Table 3: Summary of Strength Measures

	1 RM STRENGTH GLOBAL GYM (kg)				QUASI-ISOKENETIC STRENGTH HFMAXX (kg)										ISOMETRIC MAXIMAL VOLUNTARY STRENGTH (Nm)			
	BENCH PRESS		DOUBLE LEG PRESS		DOUBLE ELBOW FLEXION		DOUBLE ELBOW EXTENSION		DOUBLE LEG EXTENSION		DOUBLE LEG FLEXION		SQUAT THRUST		SINGLE ELBOW FLEXION		SINGLE KNEE EXTENSION	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST
EXERCISE	27.38 (1.41)	31.22 (1.44)	129.41 (4.90)	139.84 (4.52)	21.21 (1.61)	24.01 (1.69)	17.57 (0.76)	21.54 (1.14)	60.93 (2.13)	71.63 (2.75)	27.87 (2.61)	33.73 (2.09)	135.18 (7.92)	153.64 (6.98)	34.10 (1.81)	35.38 (1.74)	164.84 (8.63)	177.1 (11.9)
CONTROL	25.18 (1.48)	26.15 (1.52)	124.03 (4.11)	130.72 (3.73)	21.63 (1.08)	19.13 (1.12)	17.76 (0.63)	17.42 (0.74)	57.97 (3.13)	58.91 (3.16)	24.45 (1.27)	26.19 (1.52)	125.13 (5.42)	123.10 (4.80)	34.84 (1.15)	33.64 (1.41)	166.37 (6.69)	159.11 (8.38)

Values are mean +/- SEM.
Data represent measures obtained at maximal resistance setting on HFMAXX (valve setting 6).
Isometric measures were taken at a joint angle of 90 degrees.

Table 4: Percent Change in Muscular Strength

	1RM STRENGTH GLOBAL GYM (KG)		QUASI-ISOKENETIC STRENGTH HFMAXX (KG)					ISOMETRIC MAXIMAL VOLUNTARY STRENGTH (Nm)	
	BENCH PRESS	DOUBLE LEG PRESS	DOUBLE ELBOW FLEXION	DOUBLE ELBOW EXTENSION	DOUBLE LEG EXTENSION	DOUBLE LEG FLEXION	SQUAT THRUST	SINGLE ELBOW FLEXION	SINGLE KNEE EXTENSION
	% CHANGE	% CHANGE	% CHANGE	% CHANGE	% CHANGE	% CHANGE	% CHANGE	% CHANGE	% CHANGE
EXERCISE	19.0	8.1	13.2	22.6	17.6	21.0	13.7	3.8	7.4
CONTROL	3.85	5.4	-13.1	-2.0	0.4	7.1	-1.6	-3.6	-4.6

HFMAXX measures obtained at valve setting 6.
Isometric measures obtained at a joint angle of 90 degrees.

control group across time (main effect for group), and a significant ($p=0.007$) increase in strength in both groups from pre- to post-testing (main effect for time). Bench press strength increased by 14% in the exercise group, and 3.9% in the control group, with the largest increase in strength occurring between pre- to mid-testing. There were no significant interactions or group effects for double leg press (no differential effect of training), but strength did increase significantly ($p=0.00001$) over time in both groups (main effect for time).

HFMAXX (Quasi-Isokinetic Strength).

Double arm elbow flexion/extension. Training resulted in significant increases in both elbow flexion ($p<0.00005$), and extension ($p<0.005$) strength at all resistance settings at mid- and post-testing. Arm flexion strength increased by 12.3%, 20% and 13.2%, and arm extension strength by 19%, 20.3% and 22.6%, respectively in the training group at resistance settings of 1, 3 and 6 from pre- to post-testing. The largest increases occurred from pre- to mid-testing for both measures at all resistance settings. Arm flexion strength decreased substantially from pre- to post-testing in the control group at all valve settings (18.7%, 14.7%, 13.1%), whereas arm extension strength decreased to a lesser extent (1.1%, 0.5%, 2.0%).

Double leg flexion/extension. Training resulted in significant increases in double leg extension and flexion strength at valve settings 1, and 3 at mid- and post-testing (main group by time interaction effect). Double leg extension and flexion strength was also significantly greater in the trained compared to the control group (main effect for group) at all valve settings over the course of the study. Double leg extension strength increased by 6%, 23.7%, and 19.3% at low, moderate and high resistances, and leg flexion strength by 54%, 49.5%, and 23%. For the trained group, the largest percent improvements occurred between pre- to mid-testing for all resistance settings. Double leg extension strength did not change significantly at any resistance setting from pre- to post-testing for the control group; however, there was a significant improvement in leg flexion strength (average 12.6% increase across all 3 resistance settings) in the control group from pre- to post-testing (main effect for time).

Squat Thrust. After adjusting for initial strength differences, both ANOVA and ANCOVA revealed significantly greater squat thrust strength in the trained compared to the control group at each valve setting (main effect for group) over the course of the study. Squat thrust increased by 12.7%, 20.6%, and 13.7% at low, moderate, and high resistance

settings in the trained group from pre- to post-testing, with the largest increases occurring from pre- to mid-testing at all valve settings. Squat thrust strength decreased slightly (average of -1% across all 3 valve settings) in the control group from pre- to post-testing.

Isometric Strength. There were no significant interactions or group effects for either maximal voluntary isometric elbow flexor or knee extensor strength. There was a trend, however, for increased elbow flexor ($p=0.09$) and knee extensor ($p=0.08$) strength in the trained group (group by time interaction effect) from pre- to post-testing. This represented 3.7% and 7.4% increases in elbow flexor and knee extensor strength respectively. Elbow flexor and knee extensor strength decreased in the control group by -3.6% and -4.6%, respectively from pre- to post-testing.

Relationship between strength and bone mineral changes. The results of the Pearson product-moment correlational analyses are presented in table 5. No significant relationship was observed between changes in muscular strength and whole body or lumbar spine BMC and BMD. There was however, a significant negative ($r= -.655$) relationship between the percent change in leg flexion strength and lumbar spine BMD between pre- to post-testing.

Table 5: Results of correlational analyses between percent changes in strength and bone.

		TOTAL BODY BMC	TOTAL BODY BMD	LUMBAR SPINE BMC			LUMBAR SPINE BMD		
		P-P	P-P	P-M	M-P	P-P	P-M	M-P	P-P
LEG FLEXION	P-M			.023			-.249		
	M-P				.428			.194	
	P-P	.352	.091			.070			-.655
SQUAT THRUST	P-M			-.035			.016		
	M-P				.276			.068	
	P-P	.182	.144			.297			-.199

Correlations are significant at r=0.482
P-M= pre to mid testing
M-P= mid to post testing
P-P= pre to post testing

Blood Analysis. The results for the blood analysis are presented in Table 6. There were no significant differential effects of training on any of the blood variables (group by time interaction, or main effect for group), and with the exception of serum alkaline phosphatase, there was no change in these variables from pre- to post-testing. Serum alkaline phosphatase however, decreased significantly in both the trained (10.3%) and control (12.5%) groups from pre- to post-testing.

Dietary Analysis. The results of the dietary analysis are presented in Table 7. There were no significant differences between groups for either total caloric, vitamin D or calcium intake.

Discussion

The effect of resistance training on whole body or lumbar spine BMC or BMD has not been previously examined in females during adolescence. Snow-Harter and Marcus (1991) demonstrated a 1.2% increase in lumbar spine BMD following eight months of resistance training in females with a mean age of 19 yrs. Small increases, on the order of 1-2%, have been demonstrated for lumbar spine BMD in pre-menopausal women following 12 (Gleeson et al. 1990) and 18 (Lohman et al. 1992) months of resistance training. The effects of resistance training on

Table 6: Blood Serum Characteristics

	ESTRADIOL pmol/L		CPK IU/L		ALKALINE PHOSPHATASE IU/L		ACID PHOSPHATASE U/L	
	pre	post	pre	post	pre	post	pre	post
EXERCISE	227.82 (36.30)	211.18 (30.06)	89.41 (8.78)	84.29 (13.80)	86.0 (5.13)	77.94 (4.44)	4.02 (0.12)	3.93 (0.16)
CONTROL	237.11 (33.05)	363.28 (66.10)	92.06 16.65	76.00 (9.43)	88.44 (6.77)	78.67 (4.95)	4.02 (0.14)	3.75 (0.13)

Results are reported as mean +/- SEM.

Table 7: Dietary Analysis

KCAL		VITAMIN D (IU)		CALCIUM (mg)	
EXERCISE	CONTROL	EXERCISE	CONTROL	EXERCISE	CONTROL
1786.0 (150)	1524.0 (125)	181.0 (34.6)	153.8 (38.3)	996.0 (135)	747.0 (117)

Results are reported as mean+/- SEM.

Dietary analysis was performed on a total of 27 subjects: exercise (N=15), control (N=12).

bone mineral density in post-menopausal women have been equivocal; one study has reported a small reduction (4%) in lumbar spine BMD following 11 months of training (Rockwell et al. 1990), whereas Pruitt et al. (1992) reported rather small and insignificant increases in lumbar spine and greater trochanter BMD following 18 months of training.

The primary finding in this study was that resistance training induced a small, yet significant, transitory increase in lumbar spine BMD from pre- to mid-testing. Despite significant increases in strength there was a general lack of responsiveness of bone mineral at all other sites.

These results are generally in agreement with those studies which have examined the effect of resistance training on lumbar spine bone mineral in pre-menopausal, but not post-menopausal women. Collectively, they suggest that lumbar spine bone mass and density respond weakly to resistance training during both adolescence and young adulthood. The general lack of responsiveness of bone mineral at other sites such as the whole body, suggests a site specific effect of high resistance exercise, localized at the level of the spine. The localization of increased bone density at the spine may be related to the greater responsiveness and higher turnover of trabecular bone in this region, compared to the whole body, which is comprised predominantly of cortical bone (Lanyon 1992).

Bone remodelling is governed by the number of resorption-formation cycles that take place in a given time period (Parfitt 1984). During adolescence, most bone surfaces free of articulation, are either in a state of bone formation or resorption (Montoye 1987). Bone formation proceeds at a greater rate than resorption, and about 45-60% of adult peak bone mass is deposited between puberty and the end of adolescence (Bonjour et al. 1991, Snow-Harter and Marcus 1991). If a large percentage of bone surface is undergoing new bone formation during adolescence, bone resorption and remodelling in response to the extra strain imposed by resistance exercise may be relatively suppressed compared to periods when there is little new bone growth. A relative suppression of bone remodelling, may explain the rather small changes in bone mineral at the spine, as well as at the whole body level in response to resistance training in this study.

Given the dynamics (i.e. half-life) of bone turnover and formation, Dalsky et al. (1988) suggest that a period of exercise intervention be extended for a minimum of one year to allow bone to adapt to the newly imposed strain of exercise. The present study lasted 26 weeks; nevertheless, despite the short intervention period a positive effect on lumbar spine BMD was observed. This finding suggests that the minimum duration of exercise intervention required to positively impact on bone mineral may be shorter during adolescence than

during adulthood. The differences in intervention interval may be related to the higher turnover of bone mineral during adolescence, particularly in the spinal region (Lanyon 1992).

Cross-sectional studies generally indicate a greater bone density in athletes and individuals with higher levels of physical activity than in the average population (Bailey and McCulloch 1990). Mixed results have been reported in the childhood and adolescent periods. A positive association between physical activity, athletic training and bone mineral has been observed in some (Rice et al., in press, Slemenda et al. 1991, Virvidakis et al. 1990), but not all studies (Bachrach et al 1990, 1991).

Turner (1991) has described a feedback theory for the homeostatic control of bone structure. This hypothesis proposes that a threshold level exists for mechanical strains, above or below which bone adaptation is initiated. The majority of subjects in the present study may have been physically active enough to surpass the minimum strain threshold required to optimize bone formation. The extra mechanical strain imposed by resistance training may not have been adequate to augment cortical bone formation, but appears to have been sufficient for enhanced trabecular bone density at the spine. These results suggest that there may be different mechanical strain thresholds for optimal development of predominantly cortical and trabecular bone density during

adolescence. This remains a possible, but yet untested hypothesis.

The exercises performed during the resistance training program were specifically designed to load the lumbar vertebrae in a compressive/dynamic fashion. Compressive forces were directly applied to the lumbar vertebrae during the squat thrust and military press exercises, while flexion/extension dynamic loads were applied during the abdominal crunch/ back extension exercises. These exercises were effective in increasing both strength, and lumbar spine bone mineral density in the present study from pre- to mid-testing. In this sense, these results are consistent with the positive relationship which has been reported between muscular strength and bone mineral in females during early adulthood (Schoutens et al. 1989, Snow-Harter and Marcus 1991) and middle age (Halle et al. 1990, Pocock et al. 1989, Sinaki et al. 1989).

The increase in muscular strength demonstrated in this study was similar to that observed in other studies examining the effects of resistance training in young women (Cureton et al. 1988, Jette et al. 1988, Westcott 1979). However, the correlation (Table 5) between the largest percent increase in muscular strength (leg flexion - medium resistance) and percent change in lumbar spine BMD from pre- to mid-testing was non-significant and negative (r=-0.249). The lack of a significant correlation between changes in muscular strength

and bone mineral suggest that factors other than increased muscular strength contributed to the transitory increase in lumbar spine BMD observed in this study.

The mechanical strains imposed on bone by exercise appear to have greater osteogenic potential when the loads are dynamic rather than static, of high rather than low magnitude, and are applied infrequently instead of repetitively over an extended period of time (Lanyon 1992). According to Turners' (1991) hypothesis, the newly imposed loads by resistance training would place increased strain on the bone, beyond its normal physiological strain window. The consequence would be increased bone formation and establishment of a new physiological strain window, but at a higher strain threshold. Additionally, Lanyon (1992) suggests that mechanical strains must be perceived as being above this newly established physiological window, both to maintain prior gains in bone mass and to promote further new bone formation. It appears therefore, that to sustain or promote bone mass accretion, the strain input must consist of varied strain distribution patterns of high magnitude and short duration that are above the newly established strain window (Lanyon, 1992).

The mechanical aspects of compressive/dynamic loading of the lumbar vertebrae during training, rather than changes in muscular strength itself, may be the primary determinant of the transitory increase in lumbar spine BMD in this study.

The mechanical strains associated with resistance training from pre- to mid-testing may have been perceived as beyond the normal physiclogical strain window, and of sufficient magnitude to induce an osteogenic response, as evidenced by the increase in lumbar spine BMD. Strength gains from mid- to post-testing were small, compared to changes during the initial period of training, suggesting perhaps that there was an accommodation to the mechanical strains and resistive loads during this phase of the training program. Perhaps the mechanical strains were no longer of sufficient magnitude, or perceived as being sufficiently different from background strain levels to stimulate new bone formation, or even to sustain prior gains in bone density. An accommodation to strain loads during the latter phase of training could explain the small reduction in bone mass and density from mid- to post-testing, and the failure of training to significantly increase bone mass and density from pre- to post-testing.

Resistance training often (Wilmore 1978, Mayhew and Gross 1974), but not always (Hunter 1985), results in increased lean body mass in women. Lean body mass has been identified as an important correlate of bone mass and density in young adult (White et al. 1992) and adolescent females (Rice et al. in press). Resistance training did not result in any change in body composition in the present study. Perhaps the relationship between muscular strength and bone mineral is

dependant on increased lean body mass. The general lack of responsiveness of bone mineral despite significant strength gains in the present study may be attributed in part to the failure of the training program to elicit an increase in lean body mass.

Nutritional factors have been implicated as important determinants of bone mineral development in adolescent females during adolescence (Katzman et al. 1991, Malina and Bouchard 1991, Rice et al. in press). Calcium intake has been identified as potentially the most important nutritional factor in the development of bone mass in females (Matkovic et al. 1990, Sentipal et al. 1991). Vitamin D which stimulates the intestinal absorption of both calcium and phosphate (Prince et al. 1988), is also considered an important nutrient for bone development during childhood and adolescence.

Growing individuals need to be in positive calcium and vitamin D balance to meet the needs of skeletal growth and mineralization. Both the exercise, and control groups in the present study were below the RDA of 1000 mg/day for calcium and 400 IU for vitamin D (Aloia 1989). Perhaps there was an insufficient calcium phosphate pool to support the processes of growth-related new bone formation and increased mineralization in response to the remodelling process evoked by resistance training. The limited availability of calcium may partly account for the generally small changes in bone

mineral in response to resistance training in this study. Whether the effects of resistance training on bone density in adolescent females is influenced by state of calcium balance remains to be determined.

Endocrine status has also been implicated as an important determinant of bone mineralization in both adolescent and young adult females. Reduced serum estrogen levels associated with anorexia nervosa (Bachrach et al. 1990) and athletic amenorrhea (Drinkwater et al. 1984, 1990) have been associated with reduced bone mass and density. Resistance training had no differential effect on serum estrogen levels and these levels were within the normal physiological range in both groups at the beginning and end of the present study. It seems unlikely therefore, that the general unresponsiveness of bone mineral to resistance training in this study can be attributed to altered estrogen status.

Serum creatine phosphokinase (CPK) is generally considered an indicator of muscle damage, and may be used as an indirect indicator of the intensity of exercise training. There were no observable changes in CPK levels in either group between pre- to post-test measurements. The lack of change in CPK levels from pre- to post-testing in the trained group suggests that the training intensity may not have been sufficiently high to induce muscle adaptation, especially during the latter part (mid- to post-testing) of the study. Blood samples were not

taken at mid-point, so we can only speculate about CPK changes during the initial phase of training. Given that the largest increases in strength occurred from pre- to mid-testing, it is likely that training intensity during this period would have been sufficient to increase CPK levels. The lack of change in CPK levels between pre- and post-testing suggests, as do the comparatively small changes in strength from mid- to post-testing, that training intensity was not sustained at a high level during the latter part of the study.

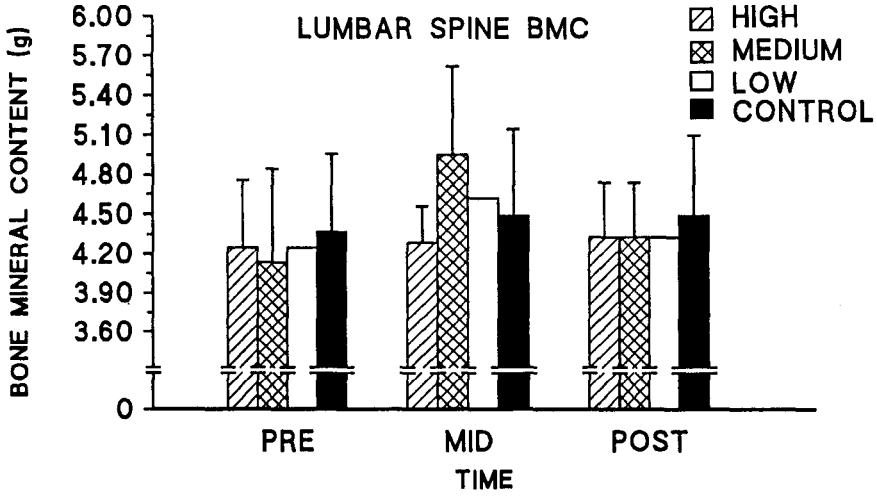
It was difficult with the training devices used in this study to monitor training effort. Training intensity is determined by the resistance setting on the device, but training effort is a product of both the resistance setting and level of subject motivation. Although subjects were encouraged to provide maximal effort at all times, it was evident during the latter part of the study the girls were loosing enthusiasm for training. Reduced motivation and enthusiasm probably accounts not only for the lack of change in CPK levels, but also for the unresponsiveness of strength and bone to resistance training from mid- to post-testing in this study.

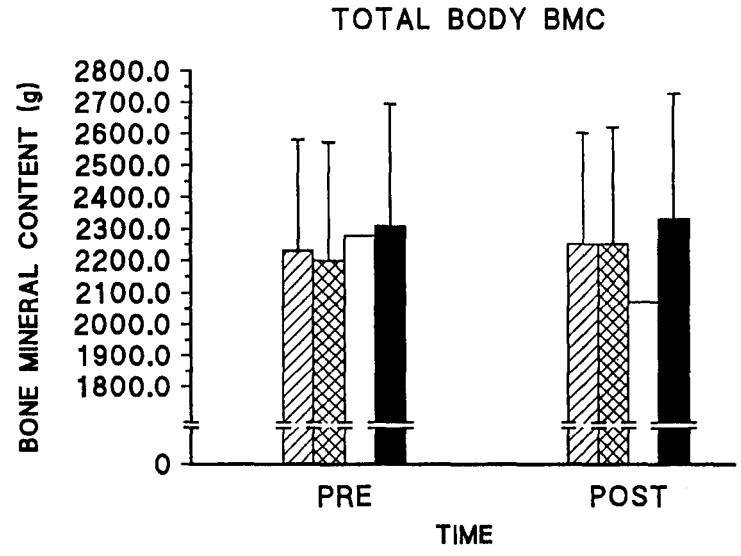
Serum acid phosphatase is considered a general marker of bone resorption, while alkaline phosphatase is considered a marker of bone formation (Aloia 1989). Acid phosphatase levels were similar between groups and remained unchanged throughout

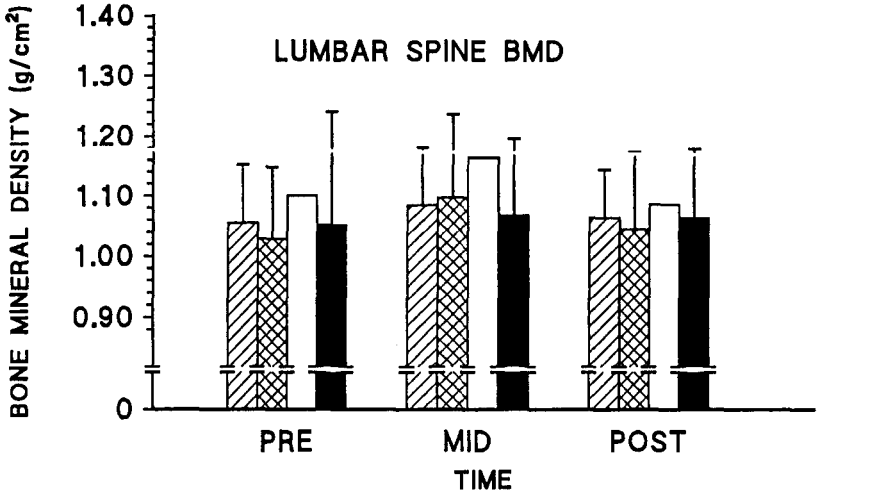
the study, implying that training had no effect on rate of bone resorption. Alternatively, perhaps acid phosphatase was not a sufficiently sensitive marker to detect subtle training induced changes in bone resorption in this study. Alkaline phosphatase levels declined proportionally in both groups from pre- to post-testing, implying an age or growth, but not training-related reduction in bone formation. Puberty and adolescence have been identified as periods of very rapid bone formation (Bonjour et al. 1991, Snow-Harter an Marcus 1991). The majority of the girls at the beginning of this study were in the latter stages of puberty and probably in the midst of the peak bone formation phase, as reflected by relatively high levels of alkaline phosphatase. The reduction in alkaline phosphatase from pre- to post-testing probably reflects a reduction in bone formation rates toward adult levels, as a consequence of continued maturation during the course of the study.

In addition to the factors discussed above, the results of this study may also have been affected by subject compliance to training. The effect of subject compliance on measures of bone mineral, and 1RM performance strength on the HFMAXX resistance machine are depicted in Figures 4 and 5. Despite on-site training and flexible training times, only 8 of the 17 girls in the exercise group demonstrated a compliance rate of greater than 80%. Eight subjects had compliance rates between

Figure 4. Effect of subject compliance on whole body and lumbar spine BMC and BMD.


LUMBAR SPINE BMC
HIGH
MEDIUM
LOW
CONTROL
BONE MINERAL CONTENT (g)
6.00
5.70
5.40
5.10
4.80
4.50
4.20
3.90
3.60
0
PRE
MID
POST
TIME


TOTAL BODY BMC
BONE MINERAL CONTENT (g)
2800.0
2700.0
2600.0
2500.0
2400.0
2300.0
2200.0
2100.0
2000.0
1900.0
1800.0
0
PRE
POST
TIME


LUMBAR SPINE BMD
BONE MINERAL DENSITY (g/cm²)
1.40
1.30
1.20
1.10
1.00
0.90
0
PRE
MID
POST
TIME

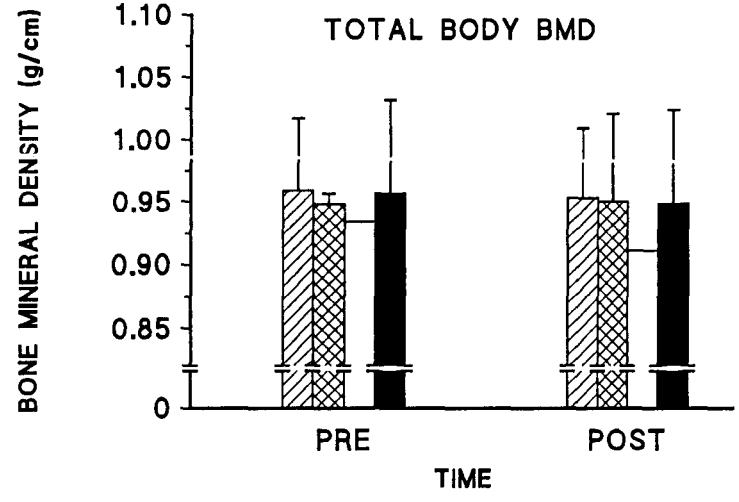

TOTAL BODY BMD
BONE MINERAL DENSITY (g/cm)
1.10
1.05
1.00
0.95
0.90
0.85
0
PRE
POST
TIME

Figure 5. Effect of subject compliance on leg flexion and squat thrust strength measures for pre-, mid- and post-testing.

50-80%, and one subject had a compliance rate of less than 50%. This poor compliance rate may have dampened the effect of training on strength and body composition outcomes, and ultimately bone mineral formation. Compliance appeared to have little effect on measures of bone mineral (Figure 4), however, there was a trend for greater strength increases in the high compliance group compared to the other two groups (Figure 5). As is evident, the increases in strength do not appear to correlate well with the observed changes in either whole body or lumbar spine BMC or BMD. It appears therefore, that some as yet unidentified factor or factors, besides compliance and strength gains per se, may be relatively more important determinants of the responsiveness of bone to resistance training in adolescent girls.

At 26 weeks, this is the longest resistance training study to date involving adolescent females. Despite significant increases in muscular strength, the training program failed to provide an adequate stimulus for sustaining of mid-point changes in lumbar spine bone density, or for enhanced whole body bone mineral density and content in this study. These results suggest that resistance training might not be as effective at increasing bone mass and density in adolescent girls, as many have suggested it might be (Loucks 1988, Lanyon 1992).

REFERENCES

Aloia, J., **Osteoporosis: A guide to prevention and treatment.** Leisure Press, Champange Ill, 1989.

Bachrach, L., Katzman, G., Litt, I., and Marcus, R., Decreased bone density in adolescent girls with anorexia nervosa. Pediatrics. 86:440-447, 1990.

Bachrach, L., Katzman, D., Litt, I., Guido, D., and Marcus, R., Recovery from osteopenia in adolescent girls with anorexia nervosa. J. Clin. Endocrinol. Metab. 72:602-606, 1991.

Bailey, D., and McCulloch, R., Bone tissue and physical activity. Can. J. Spt. Sci. 15: 229-239, 1990.

Blimkie, C., Sale, D., and Bar-Or, O., Voluntary strength, evoked twitch contractile properties and motor unit activation of the knee extensors in obese and non-obese adolescent males. Eur. J. Appl. Physiol. 61: 313-318, 1990.

Bonjour, J., Theintz, B., Buchs, B., Slosman, D., and Rizzoli, R. Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. J. Clin. Endocrinol. Metab. 73: 555-563, 1991.

Bevier, W., Wiswell, R., Pyka, G., Kozak, K., Newhall, K., and Marcus, R. Relationship of body composition, muscle strength, and aerobic capacity to bone mineral density in older men and women. J. Bone Min. Res. 4: 421-432, 1989.

Chow, R., Harrison, J., and Notarius, C., Effect of two randomized exercise programmes on bone mass of healthy postmenopausal women. B. M. J. 295: 1441-1444, 1987.

Cureton, K., Collins, M., Hill, D., and McElhannon, M., Muscle hypertrophy in men and women. Med. Sci. Sports Exerc. 4: 338-344, 1988.

Dalsky, G., Stocke, K., Ehsani, A., Slatopolsky, E., Lee, W., and Bridge, S. Weight-bearing exercise training and lumbar spine bone mineral content in premenopausal women. Ann. Int. Med. 108. 824-828, 1988.

Davee, A., Rosen, C., and Adler, R. Exercise patterns and trabecular bone density in college women. J. Bone Min. Res. 5: 245-250, 1990.

Drinkwater, B., Nilson, K., Chestnut, C., Bremmer, W., Sahinholtz, S., and Southworth, M. Bone mineral content of amenorrheic and eumenorrehic athletes. New Eng. J. Med. 311: 277-281, 1984.

Drinkwater, B., Bruemner, B., and Chestnut, C., Menstrual history as a determinant of current bone density in young athletes. J.A.M.A. 263: 545-548, 1990.

Galea, V., Ormerod, S., White, N., Macdougal, J., and Webber, C., Body composition by photon absorptiometry. Can. J. Sport Sci. 15: 143-148, 1990.

Gilsanz, V., Gibbens, D., Carlson, M., Cann, C., and Schultz, E. Peak trabecular vertebral density: A comparison of adolescent and adult females. Calcif. Tissue Int. 43: 260-262, 1988.

Gleeson, P., Protas, E., LeBlanc, A., Schnieder, V., and Evans, H. Effects of weight lifting on bone mineral density in premenopausal women. J. Bone Min. Res. 5: 264-269, 1990.

Halle, J., Smidt, G., and O'Dwyer, K., Relationship between trunk muscle torque and bone mineral content of the lumbar spine and hip in healthy post menopausal women. Phys. Therapy. 70: 690-699, 1990.

Heaney, R., Nutritional factors in bone health. In: **Osteoporosis: Etiology, Diagnosis, and Management,** edited by B.L. Riggs, and L.J. Melton. New York: Raven Press, 1988; p. 359-372.

Heinrich, C., Going, S., Pamenter, R., Perry, C., Boyden, T., and Lohman, T. Bone mineral content of cyclically menstruating female resistance and endurance trained athletes. Med. Sci. Sport Exerc. 22: 558-563, 1990.

Hunter, G. Changes in body composition, body build and performance associated with different weight training frequencies in males and females. Nat. Strength Conditioning J. 7: 26-28, 1985.

Jette, M., Sidney, K., Regimbal, M., Barsalou, J., and Montelpare, W. Effects of three heavy-resistance weight training programs on the upper body strength of young women. Can. J. Sport Sci. 12: 71-77, 1988.

Katzman, D., Bachrach, L., Carter, D., and Marcus, R. Clinical and anthropometric correlates of bone mineral acquisition in healthy adolescent girls. J. Clin. Endocrinol. Metab. 73: 1332-1339, 1991.

Kramer, W., Deschemes, M., Fleck, S. Physiological adaptations to resistance exercise. Implications for athletic conditioning. Sports Med. 6: 246-256, 1988.

Kroger, H., Kotaniemi, A., Vainio, P., and Alhaua, E., Bone densitometry of the lumbar spine and femur in children by dual-energy X-ray absorptiometry. Bone Min. Res. 17: 75-85, 1992.

Lane, N., Bloch, D., Jones, H., Marshall, W., Fries, J. Long distance running, bone density and osteoarthritis. J.A.M.A. 255: 103-108, 1986.

Lanyon, L. Functional Strain as a determinant for bone remodelling. Calcif. Tissue Int. 36: S56-S61, 1984.

Lanyon, L., The success and failure of the adaptive response to functional load-bearing in averting bone fracture. Bone. 13: S17-S21, 1992.

Loucks, A. Osteoporosis prevention begins in childhood. In: **Competitive Sports For Children and Youth**, edited by E.W. Brown, and C.F. Branta. Champaign Il. : Human Kinetics Publishers Inc., 1988; p. 213-223.

Lohman, T., Going, S., Pamenter, R., Boyden, T., Houtkooper, L., Ritenbaugh, C., and Aickin, A. Effects of weight training on lumbar spine and femur bone mineral density in pre-menopausal women. Med. Sci. Sports Exc. 24: S188, 1992.

Malina, R., and Bouchard, C., **Growth, Maturation, and Physical Activity**. Champaign, IL.: Human Kinetics Publishers, 1991.

Matkovic, V., Fontana, D., Tominac, C., Goel, P., and Chestnut, C., Factors that influence peak bone mass formation: a study of calcium balance and the inheritance of bone mass in adolescent females. Am. J. Clin. Nutr. 52: 878-888, 1990.

Mayhew, L., Gross, P., Body composition changes in young women with high intensity weight training. Res. Quart. 45: 433-440, 1974.

McCulloch, R., Bailey, D., Houston, S., and Dodd, B., Effect of physical activity, dietary calcium intake and selected lifestyle factors on bone density in young women. Can. Med. Assoc. J. 142: 221-227, 1990.

Moller, M., Horsman, B., Harvald, M., Hauge, K., Henningsen, K., and Nordin, B. Metacarpal morphometry in monozygotic and dizygotic elderly twins. Calcif. Tiss. Res. 25: 197-210, 1978.

Montoye, H. Better bone and biodynamics. Research Quart. 58: 679-702, 1987.

Parfitt, A., The cellular basis of bone remodelling: the quantum concept reexamined in light of recent advances in the cell biology. Calcif. Tissue Int. 36: S37-45, 1984.

Pocock, N., Eisman, J., Gwinn, T., Sambrook, P., Freund, J., and Yeates, M., Muscle strength, physical fitness, and weight but not age predicted femoral neck bone mass. J. Bone Min. Res. 4: 441-447, 1989.

Pollitzer, W., and Anderson, J., Ethnic and genetic differences in bone mass: a review with a hereditary vs environmental perspective. Am. J. Clin. Nutr. 50: 1244-1259, 1989.

Prince, R., Dick, I., Boyd, K., and Garcia-Webb, P. The effects of dietary calcium deprivation on serum calcatroil levels in premenopausal and postmenopausal women. Metabolism. 37: 727-731, 1988.

Pruitt, L., Jackson, R., Bartels, R., Lenhard, H. Weight-training effects on bone mineral density in early postmenopausal women. J. Bone Min. Res. 61: 179-185, 1992.

Ramsay, J., Blimkie, C., Smith, K., Garner, J., MacDougall, J., and Sale, D., Strength training effects in prepubescent boys. Med. Sci. Sports Exerc. 22: 605-614, 1990.

Rice, S., C.J.R, Blimkie., C, Webber., D. Levy, J. Martin, C, Gordan., and D, Parker. Correlates and determinates of bone mineral content and density in healthy adolescent girls. J. Appl. Physiol. in press.

Rikli, R., McManis, B. The effects of exercise on bone mineral content in post menopausal women. Reasch. Quart. Exerc. and Sport. 61: 243-249, 1987.

Rockwell, J., Sorensen, A., Baker, S., Leahey, D., Stock, I., and Baran, D. weight training decreases vertebral bone density in premenopausal women: A prospective study. J. Clin. Endocrinol. Metab. 71: 988-993, 1990.

Schoutens, A., Laurent, E., and Poortmans, I., Effects of inactivity and exercise on bone. Sport Med. 7: 71-81, 1989.

Sentipal, J., Wardlaw, G., Mahan, J., and Matkovic, V., Influence of calcium intake and growth indexes on vertebral bone mineral density in young females. Am. J. Clin. Nutr. 54: 425-428, 1991.

Sinaki, M., Wahner, H., Offord, K., Hodgson, S. Efficacy of non-loading exercises in prevention of vertebral bone loss in postmenopausal women. Mayo Clin. Proc. 64: 762-769, 1989.

Slaughter, M., Loham, T., Boileau, R., Horswill, C., Stillman, R., Vanloan, M., and Bember, D., Skinfold equations for estimation of body fatness in children and youth. Hum. Biol. 60: 709-723, 1988.

Slemenda, C., Miller, J., Hui, S., Reister, T., and Johnston, C. Role of physical activity in the development of skeletal mass in children. J. Bone Min. Res. 6: 1227-1233, 1991.

Smidt, G., Lin, S., O'Dwyer, K., and Blanpied, P. The effect of high-intensity trunk exercise on bone mineral density in postmenopausal women. Spine. 3: 265-270, 1992.

Snow-Harter, and Marcus R. Exercise, bone mineral density, and osteoporosis. In: **Exercise and Sport Sciences Reviews,** edited by J. Hollosyz. Philadelphia: Williams and Wilkins, 1991.

Talmage, R., and Anderson, J. Bone density loss in women: effects of childhood activity, exercise, calcium intake and estrogen therapy. Calcif. Tiss. Int. 36: (suppl 2) S52, 1984.

Thompson, W., and Smith, I. Effects of oestrogens on erythrocyte enzyme efflux in normal men and women. Clin. Chim. Acta. 103: 203-208, 1980.

Turner, C., Homeostatic control of bone structure: an application of feedback theory. Bone. 12: 203-217, 1991.

Tylavsky, F., Bortz, R., Hancock, R., and Anderson, J. Familial resemblenses of radial bone mass between premenopausal mothers and their college daughters. Calcif. Tiss. Int. 45: 265-272, 1989.

Virvidakis, K., Georgiou, E., Korkotsidis, A., Ntalles, K., and Proukakis, C., Bone mineral content of junior competitive weightlifters. Int. J. Sports Med. 11: 244-246, 1990.

Webber, C., Some factors which influence the elevation of a dual photon measurement of lumbar spine bone mineral mass. J. Can. Assoc. Radiol. 40: 87-91, 1989.

Westcott, W. Female response to weight training. J Phys. Educ. 77: 31-33, 1979.

White, C., Hergenroeder, A., and Klish, W., Bone mineral density in 15 to 20 yr old eumenorrheic and amenorreheic subjects. A.J.D.C. 146: 31-35, 1992.

Wilmore, J. Alterations in strength, body composition, and anthropometric measurements consequent to a 10 week weight training program. Med. Sci. Sport Exerec. 10: 79-84, 1978.

Summary

Osteoporosis is a major cause of morbidity and mortality in our aging population. The primary goal in the management of this problem is to prevent or delay bone loss and consequently, to curtail the incidence of fractures associated with low bone mass and density.

Given the relative ineffectiveness of most intervention studies in adult females to counter the age associated loss of bone leading to osteoporosis, one strategy for impacting on this disease is to strive for attainment of enhanced BMC/BMD during the childhood and adolescent years. Increased bone mass during the formative growth years theoretically would prolong the time before the critical bone mineral fracture threshold would be reached.

Human growth and skeletal maturation are dynamic processes that start in utero and end somewhere during the third decade of life. During this period, genetic and endogenous factors interact with the environment to determine skeletal peak bone mass. Matkovic et al. (1990) suggests that approximately 90% of adult peak bone mineral is accumulated in the years preceding, and during the adolescent growth spurt. Therefore it is important to identify those factors which contribute to the attainment of peak bone mass, and to determine their relative importance during the developmental years. Once these factors have been identified, intervention strategies may be

employed during childhood and adolescence for the purpose of maximizing adult peak bone mass.

Two separate studies were undertaken in this thesis; the first, was a study of the correlates and determinants of bone mass and density in adolescent females, and the second was a study of the effectiveness of resistance training to increase bone mineral density in young healthy adolescent girls.

The specific purposes of the first study were: 1) to describe the correlational relationships between whole body and lumbar spine BMC and BMD and selected lifestyle, fitness, gynaecologic-endocrine, nutritional, and body composition variables and 2) to identify the key predictors of cortical and trabecular BMC and BMD from amongst various classes of independent variables. The purposes of the second study were: 1) to determine the effectiveness of 26 weeks of progressive resistance training to increase whole body and lumbar spine bone mineral mass and density in young females during the formative growth years of adolescence, and 2) to investigate selected endocrine adaptations to this type of training which might impact on bone mineral development.

The primary finding of the first study was that of all the selected independent variables, body mass was most highly correlated with, and accounted for the largest proportion of the explained variance in the four bone mineral measures. These results are consistent with previous findings in adult

females (Dawson-Hughes et al 1990, Drinkwater et al. 1990) as well as in children (Kroger et al. 1992, Troverbach et al. 1992, and White et al. 1992). Caloric intake, level of habitual physical activity and time since the onset of menses were additional significant but relatively less important factors in determining female adolescent bone mass. The importance of these findings is that body weight, daily caloric intake, and level of physical activity are to some extent self-controlled variables. Therefore, girls (and their parents) in this age range should be counselled about the importance of maintaining both a healthy diet and body weight, and participation in regular habitual physical activity for optimal development of bone mass and density during adolescence.

The major limitation of this study was the limited sample size. A small sample of 36 subjects provides limited statistical power, when using multiple linear regression analysis. Given the small sample size, caution must be used in generalizing these results to the female adolescent population at large. There were also limitations associated with the methods used to determine level of habitual physical activity and endocrine status. Physical activity was assessed by recall questionnaire and by a motion detection device (Caltrac). The major limitation of recall questionnaires, is the degree of accuracy of recalled information over extended periods. The

motion detector only indicates motion in a single plane when the body's centre of gravity is moved outside a pre-defined range. Although providing an objective measure of physical activity, many activities such as cycling, or activities involving arms only are not registered on this device. Blood analysis was only sampled at one point in time, and therefore may not be reflective of the normal or integrated hormonal profile of the subjects.

In the second study, resistance training resulted in significant strength gains, and a transitory increase in lumbar spine BMD from pre- to mid-testing. The training program, however, had no significant effect on whole body BMC or BMD and lumbar spine BMC. To the authors' knowledge, this was the first study to examine the effects of resistance training on bone mineral in the adolescent female population. To date, the majority of studies examining the effectiveness of resistance training in maintaining or increasing present bone mineral levels have concentrated on the peri-menopausal age range. The majority of these studies (Gleeson et al. 1990, Pruitt et al. 1992, Sinaki et al.1989) support a weak, but nevertheless positive effect of resistance exercise on bone mineralization in the adult female population. The results from the present study suggest that resistance training may also have a weak positive effect on lumbar spine bone density in adolescent girls, but that this response is not permanent,

if not engendered by continued high intensity training.

There were three major limitations to this training study: subject compliance, training intensity, and possibly training duration.

Despite on site training and flexible training times, only 8 of the 17 girls in the training group had a compliance rate of greater than 80%. Eight subjects had compliance rates, between 50-80%, and one subject had a compliance rate of less than 50%. The second limitation was the training effort or intensity put forth by the subjects during the exercise program. Enthusiasm and motivation for training waned, especially during the latter phase of training despite numerous incentives to encourage participation.

The third limitation may have been the length of the training program itself. At 26 weeks this represented the longest resistance training program to date involving adolescent girls. While this was obviously of sufficient duration to induce significant strength gains, it was not of sufficient length to induce substantial changes in bone mineral status. Bone appears to be a slowly adapting tissue and its response to imposed mechanical strain may be slow compared to skeletal muscle. Some (Dalsky et al. 1988, Drinkwater et al. 1990) suggest training programs of a minimum duration of 1 year for realization of enhanced bone mineral. Additionally, the biological adaptive capacity of bone may be

fully taxed during adolescence in meeting the demands imposed by growth. Thus, there may be no reserve capacity for enhanced bone mineralization in response to the increased mechanical strains of physical activity during this period.

Future studies should examine the issue of improving subject compliance in this age range, ensuring progressive training intensity, as well as extending the training period to encompass at least one year. Adolescence is a period of optimal intestinal calcium retention. Future studies should also examine the effects of combining resistance training with calcium intervention on development of peak bone mineral content and density in adolescent females.

References

Dalsky, G., Stocke, K., Enhansi, A., Slatopolsky, E., Lee, W., and Birdge, S., Wieght-bearing exercise training and lumbar spine bone mineral content in pre-menopausal women. Ann. Int. Med. 108: 824-828, 1988.

Dawson-Hughes, B., Dallen, G., Krall, E., Sadowski, L., Sahyoun, N., and Tannenbaum, S., A controlled trial of the effect of calcium supplementation on bone density in postmenopausal women. N. Eng. J. Med. 332: 878-883, 1990.

Drinkwater, B., Chestnut, C., Bremmer, W., Menstrual history as a determinant of current bone density in young athletes. J.A.M.A. 236: 545-548, 1990.

Gleeson, P., Protas, E., LeBlanc, A., Schneider, V., and Evans, H., Effects of weight lifting on bone mineral density in premenopausal women. J. Bone Min. Res. 5: 264-269, 1990.

Kroger, H., Kotaniimen, A., Vaino, P., and Alhaua, E., Bone densitometry of the lumbar spine and femur in children by dual-energy x-ray absorptiometry. Bone Min. Res. 17: 75-85, 1992.

Matkovic, V., Fontana, D., Tominac, C., Goel, P., and Chestnut, C., Factors that influence peak bone mass formation: a study of calcium balance and the inhertiance of bone mass in adolescent females. Am. J. Clin. Nutr. 52: 878-888, 1990.

Pruitt, L., Jackson, R., Bartels, R., and Lehnard, H., Weight training effects on bone mineral density in early postmenopausal women. J. Bone. Min. Res. 61: 179-185, 1992.

Sinaki, M., Wahner, H., Offord, K., and Hodgson, S., Efficacy of non-loading exercises in prevention of vertabral bone loss in postmenopausal women. Mayo Clin. Proc. 64: 762-769, 1989.

Troverbach, W., de Mann, S., Gommers, D., and Grobbee, D., Determinants of bone mineral content in children. Bone Min. Res. 13: 55-67, 1992.

White, C., Hergenroeder, A., and Klish, W., Bone mineral density in 15 to 20 yr old amenorrheic and eumenorrheic subjects. A.J.D.C. 146: 31-35, 1992.

APPENDIX

ANOVA/ANCOVA TABLES

ANOVA SUMMARY TABLES

ESTROGEN

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	113860	33	32792.7	3.47211	0.07133
Time (T)	1	52433.2	33	33216.4	1.57854	0.21779
G x T	1	89158.7	33	33216.4	2.68418	0.11085

CPK

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	139.563	33	3880.52	0.03597	0.85075
Time (T)	1	1959.73	33	1403.37	1.39644	0.24577
G x T	1	522.988	33	1403.37	0.37267	0.54574

ACID PHOSPHATASE

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	0.15059	32	0.45158	0.33347	0.56766
Time (T)	1	0.56532	32	0.20195	2.79933	0.10405
G x T	1	0.13235	32	0.20195	0.65538	0.42417

ALKALINE PHOSPHATASE

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	43.926	33	893.223	0.04918	0.82587
Time (T)	1	1390.74	33	80.0917	17.3643	0.00021
G x T	1	12.917	33	80.0917	0.16127	0.69058

ANOVA SUMMARY TABLES

ISOMETRIC ARM MVC

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	4.11493	33	107.258	0.03836	0.84593
Time (T)	2	1.73307	66	5.6296	0.30785	0.73607
G x T	2	14.0217	66	5.6296	2.49116	0.09053

ISOMETRIC LEG MVC

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	1476.15	32	3701.85	0.39876	0.53222
Time (T)	2	611.976	64	291.967	2.26729	0.11189
G x T	2	745.255	64	291.967	2.55253	0.08579

ANCOVA SUMMARY TABLE

BENCH PRESS

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	161.323	32	10.0954	15.9798	0.00035
Time (T)	1	30.5542	32	3.71386	8.22707	0.00725
G x T	1	0.0047	32	3.71386	0.00126	0.97195

ANOVA SUMMARY TABLE

DOUBLE LEG PRESS

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	1693.87	33	918.127	1.84491	0.18359
Time (T)	2	642.56	66	47.8174	13.4378	1.3E-05
G x T	2	46.656	66	47.8174	0.97571	0.38229

ANOVA SUMMARY TABLES

ARM EXTENSION - VALVE 1

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	70.7085	33	10.5961	6.67308	0.01441
Time (T)	2	16.0994	66	1.57391	10.2289	0.00014
G x T	2	13.9666	66	1.57391	8.87379	0.00039

ARM EXTENSION - VALVE 3

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	100.556	33	15.3482	6.55164	0.01525
Time (T)	2	32.1661	66	1.44009	22.3361	1E-06
G x T	2	17.6879	66	1.44009	12.2825	2.9E-05

ARM EXTENSION - VALVE 6

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	134.191	33	34.61	3.87724	0.05739
Time (T)	2	49.4816	66	3.14129	15.752	3E-06
G x T	2	42.9122	66	3.14129	13.6607	1.1E-05

ANOVA SUMMARY TABLES

ARM FLEXION - VALVE 1

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	131.537	33	18.93	6.94858	0.01269
Time (T)	2	0.2195	66	1.57337	0.13949	0.87006
G x T	2	18.4479	66	1.57337	11.7251	4.4E-05

ARM FLEXION - VALVE 3

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	200.228	33	33.5547	5.97621	0.02009
Time (T)	2	2.428	66	1.8497	1.31262	0.27605
G x T	2	43.5051	66	1.8497	23.5201	1E-06

ARM FLEXION - VALVE 6

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	169.135	33	91.226	1.85402	0.18254
Time (T)	2	0.3412	66	2.98354	0.11434	0.89213
G x T	2	63.8829	66	2.98354	21.4117	1E-06

ANOVA SUMMARY TABLES

SQUAT THRUST - VALVE 1

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	3156.64	32	423.387	7.45568	0.0102
Time (T)	2	301.371	64	240.775	1.4717	0.23719
G x T	2	302.325	64	204.775	1.47637	0.23614

ANCOVA SUMMARY TABLES

SQUAT THRUST - VALVE 3

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	7208.31	31	266.189	27.0797	1.2E-05
Time (T)	1	12.781	31	13.9234	0.91796	0.34524
G x T	1	25.67	31	13.9234	1.84367	0.18432

SQUAT THRUST - VALVE 6

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	9078.42	31	236.172	38.4399	1E-06
Time (T)	1	6.918	31	47.3134	0.14621	0.70479
G x T	1	1.152	31	47.3134	0.02435	0.87702

ANOVA SUMMARY TABLES

LEG FLEXION - VALVE 1

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	241.602	33	39.3966	6.13256	0.01857
Time (T)	2	419.94	66	6.2132	67.5884	1E-06
G x T	2	84.6167	66	6.2132	13.6189	1.1E-05

LEG FLEXION - VALVE 3

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	482.095	33	57.1522	8.43529	0.00652
Time (T)	2	456.603	66	8.36946	54.5559	1E-06
G x T	2	132.6	66	8.36946	15.8433	2E-06

ANCOVA SUMMARY TABLE

LEG FLEXION - VALVE 6

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	461.52	32	29.18	15.8163	0.00037
Time (T)	1	4.0159	32	2.08015	1.93059	0.17429
G x T	1	4.9758	32	2.08015	2.39204	0.13179

ANOVA SUMMARY TABLES

LEG EXTENSION - VALVE 1

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	639.835	33	141.485	4.52229	0.04102
Time (T)	2	315.869	66	6.4488	48.9808	1E-05
G x T	2	188.798	66	6.4488	29.1838	1E-05

LEG EXTENSION - VALVE 3

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	1428.59	33	151.08	9.45585	0.00421
Time (T)	2	344.718	66	8.4146	40.9668	1E-06
G x T	2	207.204	66	8.4146	24.6245	1E-06

LEG EXTENSION - VALVE 6

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	2845.87	33	396.564	7.17632	0.01143
Time (T)	2	585.342	66	13.839	42.2966	1E-06
G x T	2	365.836	66	13.839	26.4352	1E-06

ANOVA SUMMARY TABLES

LUMBAR SPINE BONE MINERAL CONTENT

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	.654882	32	.937310	.69868	0.40943
Time (T)	2	.217764	64	11.12304	11.12304	7E-05
G x T	2	.030633	64	1.56466	0.217053	0.21705

LUMBAR SPINE BONE MINERAL DENSITY

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	0.00061	32	0.036962	0.01645	0.89876
Time (T)	2	0.00998	64	0.000791	12.62032	2.4E-05
G x T	2	0.00315	64	0.000791	3.97789	0.02353

TOTAL BODY BONE MINERAL DENSITY

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	0.00035	32	0.00915	0.038116	0.84644
Time (T)	1	0.00024	32	0.00053	0.385666	0.53899
G x T	1	1.2E-05	32	0.00053	0.021769	0.88363

ANCOVA SUMMARY TABLE

TOTAL BODY BONE MINERAL CONTENT

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	1774.86	31	7560.566	0.234752	0.63143

CONSENT FORM (CHILD)

McMASTER UNIVERSITY
School of Physical Education and Athletics
1280 Main Street West, Hamilton, Ontario L8S 4K1
Telephone: 525-9140 Ext. 3400

CONSENT FORM
(Child)

STRENGTH TRAINING AND BONE DEVELOPMENT IN ADOLESCENT GIRLS

I _____ consent to take part in a study which will
(Name - Print)
assess the effects of strength training on bone and strength development in adolescent girls.

I understand that I will be assigned to either a group that strength trains, 3 times per week, or a control group which will not be involved in any special training.

I also understand that I will be required to attend several testing sessions both at McMaster's Physical Education Complex and at the McMaster Medical Centre to determine my level of physical activity, cardiovascular and strength fitness, dietary intake, endocrine status (blood hormones), body composition, and the size and density of my muscles and bones.

I understand that I will receive a written summary of my results on these tests, that my results will be kept strictly confidential, and that I may withdraw from the study at any time, even after signing this consent form.

If you are willing to take part, please so indicate by signing below.

I understand the purpose and procedures of this study as described above, and agree to participate.

_____	_____	_____
(Name - Print)	Signature	Date

Please return this form to Mr. Fox, Principal, St. Mary's School by September 7th, 1990. You will be contacted by telephone, by the study coordinator, once the consent forms have been returned.

If you or your parents have any questions please do not hesitate to contact Dr. Cameron Blimkie at 525-9140, Ext. 4465, the chief investigator in this study.

CONSENT FORM (PARENT)

McMASTER UNIVERSITY
School of Physical Education and Athletics
1280 Main Street West, Hamilton, Ontario L8S 4K1
Telephone: 525-9140 Ext. 3400

CONSENT FORM
(Parents)

STRENGTH TRAINING AND BONE DEVELOPMENT IN ADOLESCENT GIRLS

I _____, consent to allow _____ to take
(Name) (Participants Name)
part in a study which will assess the effects of strength training on bone and strength development in adolescent girls.

I understand that my daughter will be assigned by the investigators into either the training group, which will take part in a 5 month strength training program, or a control group which will not train, but which will undergo all of the same tests and measurements (outlined below) as the training group.

I understand that the tests will involve a complete medical/physical examination by a physician, a series of maximal voluntary and electrically stimulated muscle strength measurements using standard laboratory techniques, assessment of body composition (weight and body fat), physical activity (questionnaire and activity monitors) and dietary intake (food diary). Cardiorespiratory fitness will be assessed during an 8-13 minute cycling test and muscle and bone cross-sectional area of the right leg, and whole body and spine mineral content and density will be determined using special radiological techniques. The radiation techniques are relatively safe, and the combined radiation exposure for the study is equal to the average annual whole body dose received by Ontarians as a consequence of exposure to natural environmental radiation.

I understand that my daughter will receive a written summary of her performance on the various tests upon completion of the study, that her individual results will be kept strictly confidential, and that she may withdraw from the study at any time, even after having signed the consent form.

If you and your daughter agree to take part, please so indicate by signing below.

.....2

I understand the purpose and procedures of this study as described above, and agree to allow my daughter to participate.

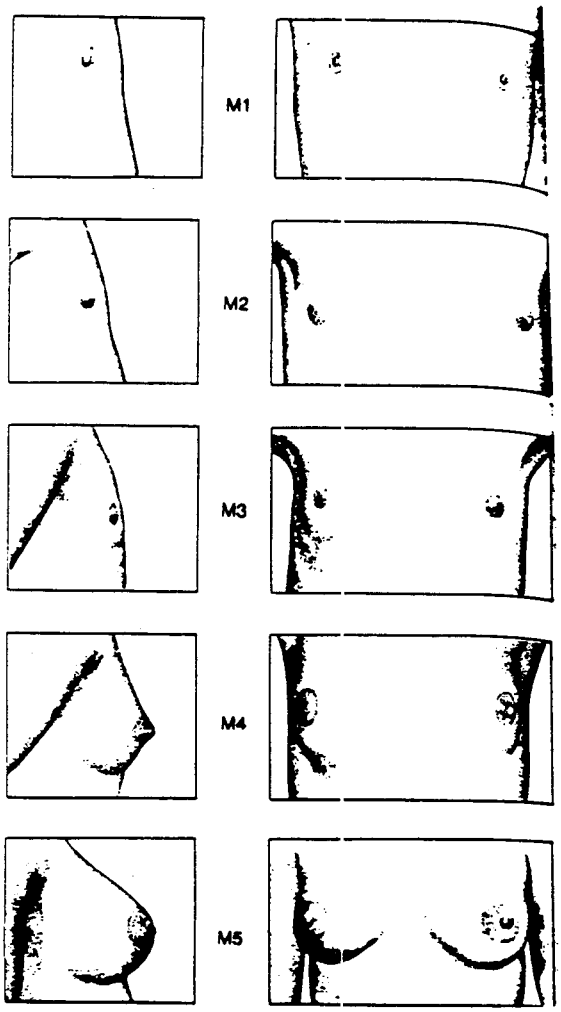
(Name - Print)	Signature	Date

Please return this form to Mr. Fox, Principal, St. Mary's School by September 7th, 1990. You will be contacted by telephone, by the study coordinator once the consent forms have been returned.

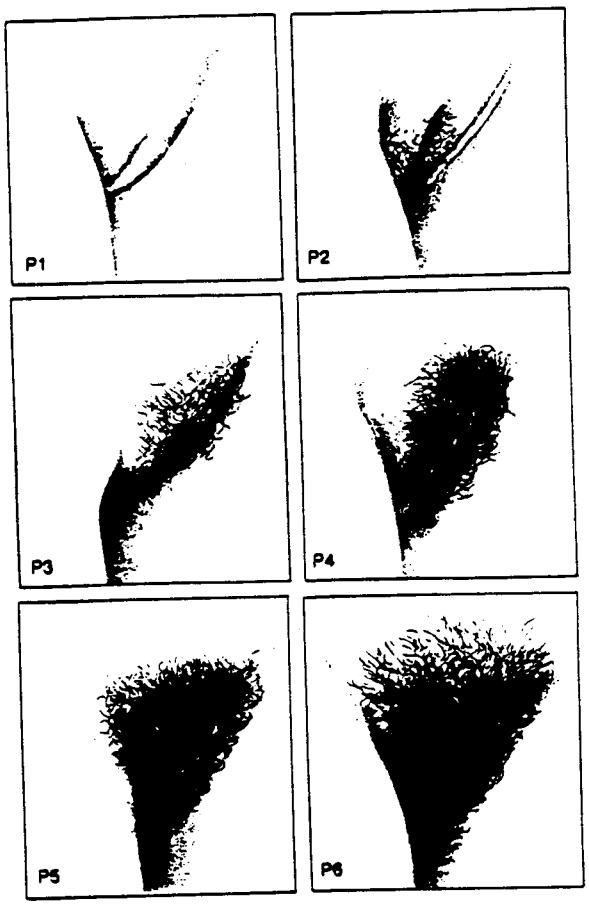
If you have any questions, please don't hesitate to contact Dr. Cameron Blimkie at 525-9140, Ext. 4465, the chief investigator in this study.

TANNER STAGING

Breast Development



Pubic Hair Development



PLEASE CIRCLE THE LETTERS WHICH BEST REFLECT YOUR CURRENT STAGE OF BREAST AND PUBIC HAIR DEVELOPMENT.

MENSTRUAL HISTORY QUESTIONAIRRE

ADOLESCENT GIRLS STRENGTH TRAINING STUDY

MENSTRUAL HISTORY QUESTIONNAIRE

Numerous studies have indicated that intensive training may alter menstrual function, and that changes in menstrual function might affect bone development. Please take a few minutes to complete this questionnaire about your menstrual history. Disregard any question that may be offensive. Remember that this information is strictly confidential and will not be shared with anyone other than the key investigators in this study. Thank you for your honesty and help.

Name: _____ Age: _____

Grade: _____

Have you ever weight-trained before? yes ☐ no ☐

If yes, for how long? _____

If yes, what type of training? For strength ☐

Powerlifting ☐

Olympic Lifting ☐

. Have you had your 1st period yet? yes ☐ no ☐

. At what age did you have your first menstrual period? State your age in years and months. For example, 12 years, 3 months.
years_____ months_____

. Is your current cycle regular? yes ☐ no ☐

If yes above, state the interval (# of days) between periods. Indicate the number of days between day 1 of your period (onset of flow) and day 1 of the following period. # of days _____.

. Has your menstrual cycle been regular from its onset?
yes ☐ no ☐

. How many menstrual periods have you had in the last year? Check one area only. 9-12 ☐, less than 9 ☐, none in the last 6 months ☐, none in the last 12 months ☐

. Have you ever used the birth control pill? yes ☐ no ☐

- if yes above, how long had you used it? Indicate the number of years and months. years _____, months _____.

- if yes above, at what age did you begin taking the pill? age _____.

. Are you currently using the birth control pill? yes ☐ no ☐

- if yes above, how long have you currently been on it?
  years _____, months _____.

. Please list all the sports activities you have been involved in during the past 12 months and provide information about your level of participation e.g. recreational, houseleague, competitive, a description of the amount of training involved with the sport, the season in which you participated, and the level of intensity of training and participation (1 being not at all intense, 10 being most intense).

Sport History

Sport	Level of Competition Rec.	House Lg.	Compet.	Training History hrs/day	days/wk.	Season	Train/Partic Int. (1-10)

Thank you for your cooperation.

ACTIVITY QUESTIONNAIRES

Directions For Activity Questionnaire Completion

A. Activities In The Past Year

- Mention importance of accuracy of detail.
- Record activities which subject did <u>besides</u> normal activity, as a form of leisure, recreation or sport.

  e.g. - walking to school not considered appropriate
  - bicycling to school not considered appropriate
  - walking exercise program is appropriate
  - stationary or outdoor bicycling for fun or recreation or as part of club is appropriate

- Ask subjects to begin with last October (1989) and progress from left to right for each of listed activities.

  e.g. - begin with October 1989 for walking as exercise and end with this past September 1990.

- Ask subject to <u>estimate</u> the number of times they performed each exercise each month, and to provide an <u>estimate</u> of the average length of time spent per session for each activity.
- Ask subject to <u>estimate</u> the average intensity of each activity over the year long period. Light intensity would be barely above rest or normal conditions and would cause only a slight increase in heart rate and breathing. Moderate exercise would cause a noticeable increase in heart rate and breathing but would be tolerated quite easily by most individuals. Heavy exercise would cause a large and very noticeable increase in both heart rate and breathing. Breathing would be labored and heavy and the exercise would not be tolerated for a very long period of time.
- Ask subject to complete additional activity section at the bottom of the questionnaire. Let them refer to the physical activity reference card (attached) to help them with their recall.
- Review the questionnaire with each subject and clarify any responses which seem unrealistic.
- Ask the subject to print their name in the upper right hand corner of the questionnaire.
- Record your name under the subjects name.
- File this questionnaire with the monthly questionnaire data form under the subjects name.

B. Activities In The Past Month (September 1990)

- Mention the importance of obtaining more detailed information about the past month's activity than was provided in the 1st questionnaire.
- Explain the 2 examples at the top of the questionnaire.
- Ask subjects to complete each open activity space indicating the type of activity, the number of times they did the activity during the past month, the average time per session of activity and the estimated intensity of the activity. Have the subject fill out this questionnaire as completely as possible from recall first of all and then let them refer to the activity reference card to complete the questionnaire. Include all activities that they can recall which were over and above normal levels of habitual activity.
- Review each item on the questionnaire.
- Ask subjects to print their names in the upper right hand corner of the questionnaire.
- Print your name below the subjects name.
- File the questionnaire with the yearly questionnaire under the subjects name.

ACTIVITIES IN THE LAST MONTH

Please refer to the reference card for a list of activities. Answer the following for the physical activities you have done at least once in the last month.

Gardening and cultivating such as spading, digging, weeding

Occasions in the last month	Average time actually spent on each occasion Hrs / Mins	Intensity: Light — Slight change from normal state	Intensity: Medium — Some perspiration, Above normal breathing	Intensity: Heavy — Heavy perspiration, Heavy breathing
☐☐	☐ / ☐☐	1	2	3

Shovelling snow

Occasions in the last month	Average time actually spent on each occasion Hrs / Mins	Intensity: Light — Slight change from normal state	Intensity: Medium — Some perspiration, Above normal breathing	Intensity: Heavy — Heavy perspiration, Heavy breathing
☐☐	☐ / ☐☐	1	2	3

Mowing the lawn (pushing a power mower)

Occasions in the last month	Average time actually spent on each occasion Hrs / Mins	Intensity: Light — Slight change from normal state	Intensity: Medium — Some perspiration, Above normal breathing	Intensity: Heavy — Heavy perspiration, Heavy breathing
☐☐	☐ / ☐☐	1	2	3

Name of activity ____________________

Occasions in the last month	Average time Hrs / Mins	Intensity: Light	Intensity: Medium	Intensity: Heavy	Organized in levels or in a league: Yes	No	Competitive: Yes	No
☐☐	☐ / ☐☐	1	2	3	1	2	1	2

Name of activity ____________________

Occasions in the last month	Average time Hrs / Mins	Intensity: Light	Intensity: Medium	Intensity: Heavy	Organized: Yes	No	Competitive: Yes	No
☐☐	☐ / ☐☐	1	2	3	1	2	1	2

Name of activity ____________________

Occasions in the last month	Average time Hrs / Mins	Intensity: Light	Intensity: Medium	Intensity: Heavy	Organized: Yes	No	Competitive: Yes	No
☐☐	☐ / ☐☐	1	2	3	1	2	1	2

Name of activity ____________________

Occasions in the last month	Average time Hrs / Mins	Intensity: Light	Intensity: Medium	Intensity: Heavy	Organized: Yes	No	Competitive: Yes	No
☐☐	☐ / ☐☐	1	2	3	1	2	1	2

Name of activity ____________________

Occasions in the last month	Average time Hrs / Mins	Intensity: Light	Intensity: Medium	Intensity: Heavy	Organized: Yes	No	Competitive: Yes	No
☐☐	☐ / ☐☐	1	2	3	1	2	1	2

Name of activity ____________________

Occasions in the last month	Average time Hrs / Mins	Intensity: Light	Intensity: Medium	Intensity: Heavy	Organized: Yes	No	Competitive: Yes	No
☐☐	☐ / ☐☐	1	2	3	1	2	1	2

over

PHYSICAL ACTIVITY IN YOUR SPARE TIME

The following activities refer to physical activities that are not related to work.
Have you done any of the following physical activities in the past 12 months?

Please indicate whether you have done each activity listed below. Then for those activities which you have done, please complete the number of times done each month, and the average time spent on each occasion (not counting travel time, changing etc.).

	No	Yes	Number of times each month: Jan Feb Mar Apr May June July Aug Sept Oct Nov Dec	Average time per occasion: hrs min	Intensity: Light Medium Heavy
walking for exercise	☐	☐→			
bicycling	☐	☐→			
jogging or running	☐	☐→			
home exercises	☐	☐→			
exercise class, aerobics	☐	☐→			

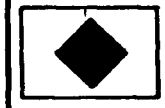
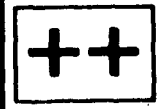
	No	Yes	J F M A M J J A S O N D	hrs min	
ice skating	☐	☐→			
cross-country skiing	☐	☐→			
downhill skiing	☐	☐→			
ice hockey	☐	☐→			
swimming	☐	☐→			

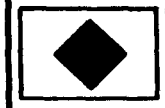
	No	Yes	J F M A M J J A S O N D	hrs min	
gardening, yard work	☐	☐→			
golf	☐	☐→			
tennis	☐	☐→			
weight training	☐	☐→			
baseball, softball	☐	☐→			

	No	Yes	J F M A M J J A S O N D	hrs min	
popular or social dance	☐	☐→			
ballet, modern dance	☐	☐→			
square or folk dance	☐	☐→			
bowling	☐	☐→			

Please refer to the Physical Activity Reference Card and list any other activities that you have done in the past 12 months.

	J F M A M J J A S O N D	hrs min	
_____			
_____			
_____			
_____			
_____			

	Daily Total
BREAKFAST	
SNACK	
LUNCH	
SNACK	
DINNER	
SNACK	

	Daily Total
BREAKFAST	
SNACK	
LUNCH	
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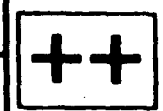
DINNER	
SNACK	

	Daily Total
BREAKFAST	
SNACK	
LUNCH	
SNACK	
DINNER	
SNACK