GYMNASTIC TRAINING AND BONE MASS IN PREPUBESCENT FEMALES

GYMNASTIC TRAINING AND BONE MASS IN PREPUBESCENT FEMALES: MAGNITUDE AND VOLUME EFFECTS OF IMPACT LOADING

By

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ABSTRACT

Nineteen elite (E) gymnasts (>15hours/week gymnastic training), 14 high recreation (HR) gymnasts (8-15hours/week), and 15 low recreation (LR) gymnasts (1-7.9hours/week) were investigated to determine the effects of varying volumes of gymnastic training on bone mineral density (BMD) in prepubescent girls. Two normoactive control groups were additionally investigated to determine whether there was a magnitude effect of mechanical loading on BMD: 16 controls (C) and 15 height- and weight-matched controls (M). The mother of each daughter was measured in order to control and investigate the familial component of bone mass. Areal bone mineral density at the left proximal femur, lumbar spine (LS), and whole body (WB) and % body fat were measured by dual energy x-ray absorptiometry (DXA), and volumetric BMD was measured at the distal radius by peripheral QCT (pQCT). DXA BMD measures were corrected for bone size and expressed as bone mineral apparent density (BMAD). The HR group was significantly younger (8.68 ± 0.844 y, mean ± SD) than both the E (10.02 ± 0.776 y) and C (9.96 ± 0.898 y) groups. The C group was significantly heavier (38.88 ± 4.868 kg) than the E (27.15 ± 2.819 kg), HR (25.44 ± 3.564 kg), LR (32.98 ± 5.786 kg), and M (26.95 ± 3.301 kg) groups. Additionally, the LR group was significantly heavier than all other groups, with the exception of the C group. Femoral neck (FN) BMD was only significantly different between the E (0.706 ± 0.051 $g \cdot cm^{-2}$) and LR (0.649 ± 0.069 $g \cdot cm^{-2}$) groups. FNBMAD was only greater in the E (0.232 ± 0.048 $g \cdot cm^{-3}$) group

compared to the C (0.191 ± 0.052 g•cm^{-3}) group. LSBMAD and WBBMAD were significantly greater in both E (0.233 ± 0.019 and 0.100 ± 0.008 g•cm^{-3}) and HR (0.239 ± 0.038 and 0.100 ± 0.006 g•cm^{-3}) groups when compared to the LR (0.212 ± 0.022 and 0.090 ± 0.008 g•cm^{-3}) and C (0.219 ± 0.020 and 0.085 ± 0.004 g•cm^{-3}) groups, respectively. Total radial and cortical radial BMD was greater in both E (360.50 ± 51.569 and 484.28 ± 70.179 mg•cm^{-3}) and HR (373.10 ± 45.318 and 480.66 ± 46.720 mg•cm^{-3}) groups compared to the C (296.61 ± 29.677 and 426.144 ± 37.652 mg•cm^{-3}) and M (306.42 ± 24.430 and 414.571 ± 25.194 mg•cm^{-3}) groups, respectively. Radial trabecular BMD was greater in both E (211.19 ± 38.202 mg•cm^{-3}) and HR (212.61 ± 44.299 mg•cm^{-3}) groups compared to the LR (175.89 ± 29.191 mg•cm^{-3}), C (162.68 ± 27.304 mg•cm^{-3}), and M (171.05 ± 30.639 mg•cm^{-3}) groups. There were no significant differences for any bone measure among the groups of mothers. Mother-daughter correlations were relatively weak, and often insignificant, for BMD measures (r = 0.10-0.37), but strong for radial morphometric measures (r = 0.43-0.55). Radial trabecular BMD (r = 0.37; $p<0.01$) was more significantly correlated with gymnastic training volume (hours/week) than radial cortical BMD (0.30; $p<0.05$). These results suggest that there is a volume of training effect on BMD and a magnitude effect of mechanical loading on BMD. It appears that trabecular bone at the distal radius may adapt more rapidly or be more sensitive than cortical BMD to the strains imposed by impact loading. Additionally, it appears that, during prepubescence in females, bone morphometric properties may be more genetically regulated than bone mineralization.

This thesis is dedicated to two women that have had a tremendous impact on who I am today:

To my mother, for all her unconditional love and support over the years, for instilling in me the value of learning, and for teaching me the principle that "might doesn't mean right." I love you, Mom.

and

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Her memory lives on in all of us.
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"Husk at elske
mens du tørdet.
Husk at leve
mens du gørdet."
- Piet Hein

"The woods are lovely, dark and deep.
But I have promises to keep,
And miles to go before I sleep,
And miles to go before I sleep."
- Robert Frost

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I hope that the world takes a look at itself and changes the way we treat one another.
I hope to see the day when women no longer fear men.
I hope for us all.

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1. INTRODUCTION

The skeleton serves to protect vital organs from injury, support the body's tissue, and facilitate movement by acting as a sequence of levers. The skeleton consists of the minimum amount of osseus material required to carry the greatest loads with the least strain. A further mechanical requirement is that the skeleton should not fracture or otherwise fail due to damage that may be caused by recurrent strains experienced in everyday activities (Carter, 1984).

In addition to providing mechanical support to the body, bone tissue serves as an active "ion reservoir" for maintaining physiologic concentrations of calcium and magnesium ions in the extracelluar fluid (Carter & Spengler, 1978). Bone tissue, therefore, is in a constant state of flux resulting from resorption and formation. In fact, bone is unique among connective tissues in that it is in a state of turnover throughout life, and, unlike other connective tissues, is mineralised with a calcium phosphate called hydroxyapatite. The presence of these mineral salts (calcium phosphate) account for the characteristic strength and hardness of bone (Carter & Spengler, 1978).

The growth and development of the chondro-osseus skeleton is realised by direct bone formation (intramembraneous ossification) and by the proliferation, maturation, degeneration and ossification of cartilage (endochondral ossification) (Carter, 1987). Bone that forms from intramembranous ossification, such as the bones of the skull, tend to maintain their mass and density throughout adulthood almost independent of strains placed

upon them. Conversely, endochondrally formed bone, found in the appendicular skeleton and most axial sites, actively remodels throughout life largely in response to mechanical stressors placed upon it (Carter, 1987). Additionally, once any functional level of bone mass has been achieved it is only maintained if the mechanically related stimulus continues (Lanyon, 1992).

A typical skeleton is subjected to millions of mechanical loading cycles throughout it's life. Repeated loadings over time at a specific site constitute a specific loading history. While it has been accepted by virtually all investigators that mechanical strain history is the primary modulator of bone form, the specific role of mechanical strain and the mechanism by which it regulates bone mass and architecture remain poorly understood.

Although profoundly influenced by mechanical loading, bone size and architecture are also influenced by other factors such as genetics, diet, and hormonal status. The basic structure and mass of each bone is genetically determined by evolutionary pressures and heredity (Pollitzer & Anderson, 1989; Krall & Dawson-Hughes, 1993; Carter, 1984). The specifics for the basic minimum structure of a weight loaded bone are determined genetically, but structural "normality" and the load-bearing competence of bone is only achieved by the effects of an adaptive response to load-bearing. Additionally, different sites in the body have variable threshold loading requirements so that what may be sufficient loading strain for one bone to invoke a rapid adaptive response may be insufficient for another (Carter, 1984).

2. EARLY RESEARCH ON BONE ADAPTATION TO MECHANICAL LOADING

The relationship between bone architecture and strength, and mechanical loading has been recognised for several hundreds of years. Early in the 17th century, Galileo (1638) first noted that body weight and activity were directly correlated to bone size. In 1834, Bell reported that bone adapted to imposed strain with the addition of the least possible amount of material, thus optimising it's strength to weight ratio. Four years later, Ward (1838) showed that increased compressive loads led to corresponding augmented levels of bone formation. Von Meyer (1867), concluded that the internal structure of bone was directly related to the magnitude and direction of the imposed load.

In 1892, Julius Wolff, published a paper titled "The Law of Bone Formation." In this paper he summarised more than 30 years of study, observation, and clinical work in osteology which resulted in the formation of Wolff's Law. Wolff was the first to integrate the concepts of the effects of mechanical loading on bone structure and morphology with the ability of bone to remodel itself. In its simplest translation (the original law was written in german) Wolff's Law states "*every change in the...function of bone...is followed by certain definite changes in...internal architecture and external conformation in accordance with mathematical laws.*" (Treharne, 1981). Form following function. Since its formulation, Wolff's Law has acted as a foundation for research relating the effects of mechanical stimuli to bone structure and adaptation.

While Wolff's Law appropriately stated that form followed function it failed to offer any succinct principles for the prediction of bone architecture or other adaptations that may occur in response to specific mechanical loading changes (Frost, 1983).

3. CONTEMPORARY THEORIES ON BONE ADAPTATION TO MECHANICAL LOADING

After Wolff formulated his law, research concerning bone and its response to mechanical stimuli remained generally uneventful until Frost published his book "The Laws of Bone Structure" in 1964, wherein he proposed the minimum effective strain (MES) hypothesis in relation to bone adaptation. The MES hypothesis has been the premiere hypothesis guiding research in this area for the past 30 years.

In 1979, Frost published a paper on the chondral modeling theory and although the paper did not directly address hard tissue adaptations to mechanical usage, the concepts presented were quickly applied to bone. Perhaps the most important new concept was that of the time-averaging property of chondral tissue. The time-averaged load was suggested to be some average of the magnitude of mechanical loads imposed on the tissue over a given period of time (Frost, 1979). The time-averaging property allowed the most common strains, rather than infrequent, atypical strains to dictate the bone adaptation response. Trivial loads and trivial changes in loading patterns were not presented as significant modulators of chondral modeling, an important concept considering that much of the previous research was preoccupied with the effects of trivial loading on bone

adaptation (Frost, 1979). However, Frost did not offer any hypothesis of how load was detected and averaged.

The concept of a magnitude effect in connective tissue adaptation was first addressed when Frost (1979) claimed that connective tissue put under increasing magnitudes of compression would result in an increased growth rate until a ceiling was reached, at which point further compression would retard growth.

In 1983, the MES model was readdressed by Frost and presented as an algorithm for predicting where and when adaptations in lamellar bone would occur under specified loading conditions. In simple terms, the MES model suggested that there was a strain level within bone that had to be exceeded for any changes in bone architecture to occur and that strains below the set-point range would not evoke a modification (Frost, 1983). Importantly, the MES was now described as encompassing a range of values, instead of a single set-point. Animal research has indicated that the MES is surprisingly similar between species, usually encompassing a range of between 0.0008-0.002 unit bone surface strain (0.08-0.2% change in length) [Rubin, 1984].

Frost suggested (1983) that strain rather than stress initiates the feedback mechanism within the MES model, which ultimately elicits bone adaptation. Strain is the deformation of a structure by an externally applied load, whereas stress is the resistance of the intermolecular bonds of matter to the deformation or strain induced by an applied load.

Activities creating loading situations in which the MES is regularly exceeded will induce architectural change that will affect the mechanical load so that the strains are again

brought below the MES level (Frost, 1983). In the updated MES theory, larger strain magnitudes invoke greater modifications in lamellar bone architecture than lesser magnitudes, provided they are over the MES set-point of values (Frost, 1983), much like what was suggested earlier in the chondral modeling theory (Frost, 1979). At this time (1983), Frost's MES hypothesis did not attempt to differentiate the effects of remodeling and modeling, and only applied to lamellar bone.

Later, in 1987(a,b) Frost differentiated between the three tissue-level processes responsible for the accretion and resorption of bone tissue: growth, modeling, and remodeling.

Growth

Growth is the attainment of size by increases in both the number of cells in a given tissue and by increased cell size. The genome and circulating systemic agents control general growth, while local factors modulate local growth (Frost, 1987a). Longitudinal bone growth contributes primary spongiosa and new length to corticies. Increased growth augments the bone bank and decreased growth rate slows, but cannot reverse the deposition of bone material (Frost, 1987a). During growth, bone architectural adjustments ensure that bone strain remains below approximately 1/10th of the fracture strain (fracture strain ~ 25,000 microstrain - μE) [Frost, 1990a].

Modeling

Modeling has the main function of providing functionally and mechanically purposeful architecture to fairly disorganised tissues in response to local modulators,

primarily local mechanical strain history (Frost, 1987a). Modeling drifts move bone surfaces to match mechanical usage. Osteoblasts in formation drifts add new bone and osteoclasts in resorption drifts remove bone over broad regions of a bone's surface (Frost, 1990a). Drifts occur almost exclusively on periostial and cortical-endostial bone surfaces (Frost, 1987a). Rapidly formed drifts typically involve the production of woven bone, whereas more gradually formed drifts are comprised of mostly lamellar bone (Frost, 1990a).

Growth and modeling normally cease at skeletal maturity, but some conditions such as the occurrence of fractures and specific pathologies in adulthood can reactivate both of these processes to a certain extent (Frost, 1991a).

Remodeling

Remodeling serves the maintenance, replacement, and microdamage repair functions of bone throughout life. The remodeling process is responsible for creating secondary osteons and replacing primary spongiosa beneath growth plates with lamellar bone (Frost, 1991a). Remodeling controls bone replacement through the turnover of small packets of bone cells referred to as basic multicellular units (BMU).

The remodeling process begins with an activation stimulus that gives rise to a resorption packet (which absorbs bone), followed by a formation packet (which deposits bone in the gully left from the resorption packet) [Frost, 1987a]. This is called the activation-resorption-formation sequence (ARF). In adults, about 80% of the trabecular bone surface and about 95% of the intracortical bone surface is quiescent in terms of

remodeling (Parfitt, 1984). In humans, a complete ARF sequence, referred to as sigma, usually lasts for a period of ~3 months. In the adult skeleton 2 x 10^6 BMUs are active at any moment and some 6 x 10^6 BMUs are completed annually (Frost, 1987b).

In bone tissue adjacent to marrow, more bone is absorbed than is formed per typical completed packet, in an approximate ratio of -20:+19 parts per packet (Frost, 1987a,b). As a consequence, increased remodeling usually leads to increased loss of trabecular and cortical-endostial bone. Various factors such as mechanical and non-mechanical influences acting locally or systemically can affect the ratio of resorption to formation, thereby increasing or decreasing the amount of bone tissue left at the completion of the AFR sequence (Frost, 1987a,b).

The co-ordination of cellular activity during remodeling must depend either on local signals or communication between adjacent cells (Parfitt, 1984). In the quiescent state, osteoclasts are separated from the bone surface by the lining cells. When a bone resorbing agent is introduced to the area, the lining cells retract and the osteoclasts extend onto the bone surface, form a ruffled border, and initiate bone resorption (Jones et al., 1985). It appears that the osteoclast is stimulated from a soluble signal released from the lining cell (Rodan & Martin, 1981).

Once the osteoclasts come into contact with bone they erode the surface to form a cavity of characteristic shape and dimensions (Parfitt, 1984). Bone resorption by osteoclasts is carried out by the release of proteolytic enzymes and hydrogen ions in the space beneath the ruffled border. Carbonic anhydrase type II produces hydrogen ions

within the cell which are transported across the ruffled border by a proton pump (Blair et al., 1989). Enzymes then released into the acidic environment by lysosomes are able to optimally degrade the underlying bone matrix (Mundy, 1985).

After the osteoclasts erode the bone site, a group of mononuclear cells "smooth" over the jagged surface left by the resorptive process and deposit a thin layer of highly mineralised, collagen-poor bone matrix termed the cement substance, in preparation for bone formation (Tran Van, 1982).

For successful resorption-formation coupling there must be both a stimulus for osteoblast cell division to ensure sufficient cells for matrix production and an attractor to bring the osteoblasts to the correct position (Parfitt, 1982). The release of human skeletal growth factor, thought to be part of the coupling process, stimulates osteoblast proliferation and causes bone formation (Farley et al., 1982). Mundy et al. (1982) observed that osteoblastic cells migrate in a unidirectional manner in response to a signal released by bone undergoing resorption. This chemotaxis may be an important link for the coupling of resorption to formation.

There are a few fundamental differences between modeling and remodeling. Remodeling is cyclical, usually results in a net loss of bone, and is characterised by simultaneous resorption and formation occurring on the same surface. At any time, about 20% of an average skeleton is undergoing remodeling (Parfitt, 1984; Burr et al., 1989a). In modeling the process is continuous, usually results in a net gain of bone, and the resorption and formation surfaces are different. With modeling, close to 100% of a given

skeletal region is undergoing modification (Parfitt, 1984; Burr et al., 1989a). Remodeling can lay down as much as 0.3-1.0 μm/d and modeling 2-10μm/day of new bone (Parfitt, 1984).

Frost claimed that mechanical loading during growth and adulthood can have differing effects on the three tissue-level mechanisms of bone apposition (1987a,b). During active growth (ie. before skeletal maturity) vigorous mechanical usage increases global bone deposits by modestly increasing longitudinal growth, increasing modeling drifts to increase cross-sectional area, depressing the recruitment of new remodeling packets, and reducing the loss per BMU, which conserves existing spongiosa and cortical bone (Frost, 1987a). In adults, increasing mechanical usage will also decrease recruitment of remodeling packets (although to a lesser degree in children), which also conserves existing spongiosa and cortical bone. Growth is absent in adults and modeling drifts are usually ineffective (Frost, 1987a,b). There is some evidence that vigorous mechanical usage can actually cause an increase in bone deposition through remodeling, but these increases in adults have yet to be greater than 5% of the previous bone volume (Frost, 1987a).

Frost (1987a) incorporated remodeling, along with modeling, in a further updated MES theory. He proposed that for modeling a MES set-point of 1500-2500μE (0.015-0.025% length change) existed, and that for remodeling events the threshold was set lower at 100-300μE (Frost, 1987a). Below a level of approximately 300μE,

remodeling levels are increased and above this level, remodeling is suppressed one-half to one-twentieth of the normal rate (Frost, 1987b).

Frost (1987b) coined the term mechanostat to describe the control of bone architectural changes in relation to it's particular loading environment. The mechanostat, working in a negative feedback loop, much the same way as a home thermostat, responds to any stimulus outside of the normal range to elicit bone modifications which bring the load to within an acceptable level of strain. The mechanostat monitors mechanical usage and adjusts bone architecture to match the site-specific mechanical loading history. Between birth and maturity, typical peak mechanical loads may increase by 50-fold, but the strains within the bone are kept to a level below the MES, which lends evidence that a MES exists (Frost, 1987b). Frost (1988) suggested that each skeletal tissue should have its own MES range for mechanically controlled modeling and remodeling. At this time, however, the MES and mechanostat theories did not account for the losses in bone associated with immobilisation or disuse.

Frost (1987a) concluded that the magnitude of mechanical loads appeared to be more important than frequency: a case in point was that marathon runners did not have the massive bone structures of weightlifters (barring, of course, selection).

In 1990(a,b), Frost developed a series of mathematical equations to predict changes that would occur in strained bone with modeling and remodeling. These equations accurately predicted where, when, and how much bone would be turned over by modeling and remodeling activities.

Frost also developed formulae to predict changes in bone in both children and adults (Frost 1991b). Accumulated bone mass in children (BMc) derives from the sum of three separate activities: growth (G) and modeling (M) which can add bone, and remodeling (R) which can remove it:

$BM_c = f(G) + f(M) + f(R)$

In adults, remodeling controls how well the bone mass (BM_a) accumulated during growth is conserved, since modeling and growth have a negligible effect after skeletal maturity:

$BM_a = f(R)$ (Frost, 1991b).

In 1992, Frost again made modifications to the MES theory to include a second, lower threshold strain to form a "mechanical usage window". In this new mechanical usage window, strains that were within the upper and lower set-point strain values would not initiate an adaptive response, while strains above or below the mechanical usage window would induce formation or resorption, respectively. The addition of the lower threshold explained observations of bone reduction during spaceflight, decreased usage, or immobilisation.

Frost subdivided the mechanical usage window into four constituent windows. The disuse window existed when peak strains fell below 0.5% fracture strain in the bone. At this time, BMU activation increased five-fold, each BMU absorbed more bone than it replaced, and no modeling drifts occurred (Frost, 1992). The bone in this state was remodeling to remove excess bone that was not required mechanically.

The second window, the adapted window, involved typical strains of 0.5% to 6% of the fracture strain. At this level, BMU activation proceeded normally, each BMU replaced approximately as much bone as resorbed, and modeling drifts did not occur (Frost, 1992). In this window, the skeleton was fully adapted to it's new loads.

The mild overload window occurred at strain ranges of approximately 6% to 12% of the fracture strain. Here, BMU activation proceeded normally, each BMU replaced approximately the same amount of bone resorbed (possibly a little more), and, in growing bone, modeling drifts were often switched on (Frost, 1992). These processes attempted to bring the strains encountered back into the range of the adapted window through modifications to architecture and bone mass.

The last window described by Frost (1992) was the pathologic overload window where typical strains usually exceeded 12% of the fracture strain. In this condition, all BMU functions in response to strain remained as in the preceding two windows, but BMU activity due to microdamage increased dramatically. In addition, there was often woven bone formation and massive resorption (Frost, 1992). In this window, bone has been overloaded to the point where it is no longer able to make modifications through normal processes and must turn to "faster" processes such as the production of woven bone or the initiation of a regional acceleratory phenomenon in an attempt to decrease the strain level of the area and avoid further damage.

The mechanostat and MES models are still driving much of the research in the field of bone adaptation to mechanical usage. Alternative models, however, have also been

proposed in recent years to describe the relationship between mechanical loading and bone adaptation.

Numerous researchers now believe that remodeling events occur in response to microdamage of the bone from repetitive loading. Martin & Burr (1982) and Carter et al. (1981) claim that the function of secondary osteonal remodeling is to repair microdamage caused by repetitive mechanical strains. Additionally, Carter et al. (1981) found that damage to bone due to mechanical loading has different effects depending on whether or not the bone is under compression or tension. There appears to be more extensive cellular insult to bone cells during compressive fatigue which may provide a greater stimulus for biological repair of microdamage. In contrast, Turner et al. (1994) did not find microcracks to be a causal factor in the increase of bone formation.

While it seems conceivable that microdamage may be a stimulus for the adaptation of bone to mechanical stimuli, this relationship is not universally accepted. Despite evidence that microdamage does accumulate and does influence internal modeling (Burr et al., 1985; Martin & Burr, 1982; Carter et al., 1981), there is no evidence that it also influences surface modeling to affect bone form. If functional adaptation was 'damage driven' one would expect surface adaptation to follow increased levels of internal replacement, which is not the case (Lanyon et al., 1982). Functional adaptations involving changes in bone form can be induced by strains of insufficient magnitude to induce appreciable microdamage (Lanyon et al., 1982; Rubin & Lanyon, 1984). In addition, microdamage usually only occurs at levels above 2000 microstrain (Eμ), therefore any

hypertrophy elicited by strains below this level must be caused by some other mechanism (Carter, 1984).

Turner (1992) disagrees with the mechanostat mechanism and homeostatic regulation of woven bone production, suggesting instead that bone adaptation may be under the influence of epigenetic regulation. Homeostatic regulation is controlled by negative feedback loops, whereas epigenetic regulation can be influenced by a number of factors, including positive feedback loops. With homeostatic regulation the system is driven to a steady-state point between two extremes, but with epigenetic regulation the system is driven to one of many steady-state levels, called attractors (Turner, 1992). It has been suggested that the rapid response of woven bone to mechanical loading may be a result of epigenetic regulation since this response would not occur rapidly enough under homeostatic regulation. Rubin et al. (1990) further suggested that epigenetic regulation may control both lamellar and woven bone production. Epigenetic regulation remains a plausible, but incompletely described mechanism in relation to bone formation.

Since bone is exposed to varying magnitudes of strains, in different orientations and in varying types (tension or compression), Carter (1984) suggested that the adaptation of bone to mechanical loading must be site specific. Immature bone may be more sensitive to alterations in cyclic strain than mature bone and structural adaptation due to cyclical loading may not be a very linear response. He proposes that bone hypertrophy and atrophy are controlled by two different stimuli, and proposes that bone mechanical microdamage may be one of many control stimuli affecting an increase in bone mass

(Carter, 1984). One of the most important differences in Carter's (1984) hypothesis compared to Frost's mechanostat (1992) is that the accretion response of bone to mechanical loading is curvilinear, whereas the mechanostat principle (Frost, 1992) predicts a linear response between the two variables.

In summary, many hypotheses have been put forth to account for the adaptations that occur in the bone due to mechanical loading. Both positive and negative feedback systems have been proposed, but further research is needed to more clearly elucidate the underlying mechanism or mechanisms. The extreme complexity of bone adaptation suggests that there may be many factors at work at any given time, working in synergy for a common goal: functionally adapted bone.

4. STRAIN SENSORS IN BONE

For a bone to adapt to a mechanical stimulus there must be some line of cells or subcellular components that are able to detect strain and communicate this information to the cell lines that are responsible for the accretion and resorption of bone. To date, four possible mechanisms have been described: the load is detected by collagen, bone mineral, extracellular fluid, and/or bone cells themselves.

Collagen theories

Mature collagen molecules put under strain can create electric potentials (streaming potentials) which may be a signal to increase osteogenesis. Increased loads

generate a greater magnitude of electrical potential which may result in increased bone formation (Treharne, 1981).

Bone Mineral Theories

Changes in the average applied load to bone may cause changes in the solubility of hydroxyapatite which may result in altered calcium concentration in the extracellular fluid. In tension the solubility of hydroxtapatite increases by 28%, which subsequently stimulates osteoclasts. In compression, the solubility of hydroxyapatite decreases, as does the calcium concentration in the extracellular fluid, stimulating osteoblastic activity. The changes in calcium concentration also cause streaming potentials within the bone matrix (Treharne, 1981), which may further potentiate osteoblast/osteoclast activity.

Extracellular Fluid Theories

The hydrostatic pressure of the extracelluar fluid in bone increases with increasing loads. This could cause a pressure to be directly transmitted to the cells, to the mineral phase of bone, to the collagen, or the changes in pressure could be converted to streaming potentials (Treharne, 1981). Bone remodeling due to tensile forces, however, is not explained by this theory.

Fluid pressure differentials, able to detect small deformations in bone, may be the load sensor mechanism. Pressure differentials caused by fluid displacements may interact directly with cell surfaces, and may also result in extracellular ion exchange which induces a corresponding production of electrokinetic currents (Rubin et al., 1990). As the magnitude of currents is strongly dependent on both the fluid's charge distribution and its

velocity through bone, changes in the mineral matrix or the viscosity of the interstitial fluid will change the magnitude of these currents. As mineral matures with age, the viscosity of the interstitial fluid increases, decreasing the magnitude of the charge, which may partially explain why there is a loss of bone with advancing age (Rubin et al., 1990). It has been further proven that strain application in older bone evokes a weaker current than the same levels of strain in immature or younger bone (Beretta & Pollack, 1986).

Cellular Theories

Lanyon (1987) proposes that the osteocytes (or some other population of bone cells) is sensitive to the distribution, rate of change, and magnitude of strain within the bone matrix. There are several lines of evidence supporting osteocytes as the primary cellular regulator mediating bone adaptations to mechanical strain. Osteocytes are distributed throughout the bone matrix, thus, it is like having a strain "sensor" in every part of the bone. Osteocytes have extensive processes which form large networks with each other and with the cells on the surface of the bone (Menton et al., 1984; Doty, 1981). These networks may relay chemical or electrical signals regarding the amount of loading experienced by their surrounding matrix relative to their genetic limit. If large differences between these two levels of strains exist, bone modifications will occur.

Recent evidence has suggested that osteoclasts are primarily controlled by osteoblasts, which are in turn controlled by osteocytes (Chambers, 1985). Therefore, the osteocyte is in position to control both bone resorption and formation aspects of remodeling in relation to the prevailing strain situation.

Osteocytes may be able to respond to the load applied to them through the collagen, bone mineral, or extracelluar fluid. The osteocytes may either respond directly to the load or are damaged by the load and indirectly stimulate the action of other cells. Directly, physical loads applied to cells can alter cell-membrane permeability (Treharne, 1981). And indirectly, cell death releases intra-cellular contents into the extracellular fluid which influences the behaviour of surrounding cell lines (Treharne, 1981).

Skerry (1987; 1988) was the first to investigate the behaviour of proteoglycans within mechanically loaded bone tissue. During short "osteogenic" periods of loading, proteoglycan molecules became reoriented for at least 24 hours, but reverted to normal orientation by 48 hours (Skerry, 1987). The orientation of these molecules may influence bone cell behaviour. Proteoglycan orientation may "capture" the strain presented to the bone and provide the "average" strain pattern accumulated over a 24-hour strain period. The mechanism underlying load induced proteoglycan orientation is not well known but it is hypothesised that it could be by the movement of the surrounding matrix itself, or by a strain induced flow of charged fluid through the tissue (Skerry et al, 1988; 1989). Lanyon (1987) observed a recoverable shift of proteoglycan orientation (again, returning to normal after approximately 48 hours), and found that the degree of recovery was dependent on the magnitude of the strain. These findings emphasise the possible role of the matrix in load transduction in bone.

In light of the non-linear response of bone to mechanical loading it may be very probable that there are separate mechanisms for both bone atrophy and hypertrophy (Carter, 1984).

5. ANIMAL WORK ON THE MECHANICAL ATTRIBUTES OF LOADING

5.1 Natural loading

The effects of natural loading (where the animal's body weight during activity provides the stimulus for adaptation) on bone in animals has been investigated primarily in rats. The results of these natural loading protocols have been fairly conclusive. In the majority of studies, training consisted of daily treadmill running at a variety of speeds and inclines for a prolonged period, usually between 6 weeks and 4 months. A number of studies have reported an increase in **bone size** in rats with run training (Raab et al., 1990; Steinberg et al., 1981; Yeh et al., 1993). Saville & Whyte (1969) concluded that running exercise caused muscle and bone to hypertrophy in exact proportion to one another in the hind limb of exercised rats, with no change in the BMD of the femur.

The effects of run training on **bone mineral density (BMD)** in rodents, however, are less positive, with most studies reporting no effect of run training (Salem et al., 1993; Raab et al., 1990; Li et al., 1991; Tuukanen et al., 1992). In one study, however, run training resulted in an increase in bone mass in mature rats only, and an increase in both density and cross-sectional area of the femur only in immature rats compared to controls (Steinberg & Trueta, 1981). Unfortunately, in this study the BMD of the femur was

simply estimated from a radiograph and not measured quantitatively, so its accuracy is questionable. Furthermore, 10 weeks of exercise training on a treadmill resulted in an increase in bone mineral content at the middiaphysis of the tibia in immature rats, but negative effects at the second metatarsus (Li et al., 1991). These latter findings suggest that immature bone of rodents responds in a bone-specific manner to local loading stressors imposed during strenuous exercise.

Woo et al. (1981) subjected five immature swine to 12 months of exercise training and concluded that the exercise had resulted in significant increases in the cross sectional area of the femur (17% increase), but did not change the mechanical properties of the cortical bone. This finding suggests that prolonged exercise has an effect on bone quantity but not bone quality. Tommerup et al. (1993) exercised sows on a treadmill for 20 weeks and found an increase in femoral but not rib BMD, leading to the conclusion that the bone response to weight bearing exercise appears to be specific to the loaded skeleton.

Additional studies utilising dogs, swine, and primates have found both positive (Martin et al., 1981; Woo et al., 1981; Matsuda et al., 1986; Biewener & Bertram, 1994) and negative effects (Bourrin et al., 1992) of exercise training on bone mass or morphological characteristics.

5.2 Immobilisation

The greatest rate of change in bone mass is observed after the elimination of loading forces. In this condition the loss of bone can be as high as 1% per week for trabecular bone, and somewhat less for cortical bone (Hogan, 1985).

Immobilisation resulted in striking losses of bone mass in mature rats (Swissa-Sivan et al., 1989; Li et al., 1990), and even more devastating effects in immature rats, including a decrease in overall body weight and length, a decrease in the size and weight of the long bones, a distortion in the shape of the long bones, and retarded epiphyseal ossification, compared to controls (Swissa-Sivan et al., 1989). Beagles subjected to a state of immobilisation showed a 40% loss in bone volume after 40 weeks (Jaworski et al., 1990). The above studies confirm that immobilisation leads to rapid and severe atrophy of bone tissue.

5.3 Artificial loading protocols

There are three major problems with artificial loading *in vivo*. The first is that bone remodeling is sensitive to both artificial loading and the effects of trauma from associated surgical procedures. It is difficult in such preparations to separate the effects of loading from trauma on the bone adaptive response. Secondly, when an artificial load is applied to a bone which is also subjected to normal functional loads, it is difficult to isolate the adaptive response to the different types of strains (Lanyon & Rubin, 1984). Lastly, surgery performed on one limb will almost certainly affect the usage of the contralateral limb, thereby jeopardising its use as a control.

Using the functionally isolated turkey ulna preparation, Rubin et al. (1992) displayed a differential response to loading between young and old bone. Following 8 weeks of artificial loading the cross-sectional area (CSA) in the younger animals (1 yr-old) increased by 30.2% compared with their functional contralateral ulna, whereas the CSA of

the older animals (3 yr-old) remained virtually unchanged. It was suggested that the osteogenic signal that causes apposition in younger animals is either non-existent or substantially deteriorated in the older animals or that there is a failure to respond to the signal (Rubin et al., 1992).

The isolated turkey preparation was also used to examine the influence of static and dynamic loads on bone accretion (Lanyon & Rubin, 1984). Three groups of birds with different loading patterns were utilised: unloaded, statically loaded, and dynamically loaded. Total bone area decreased by 13% in the unloaded group and in the statically loaded birds the remodeling changes were similar to those seen in disuse. In the dynamically loaded birds the CSA increased, mostly on the periosteum, by a mean of 24% over the experimental period (Lanyon & Rubin, 1984). Similar results were reported in sheep by Churches et al. (1979) and Churches & Howlett (1982) using variable dynamic loads and identical experimental procedures.

One of the simplest methods of artificially loading the skeleton in an animal model is the removal of one of a pair of bones (osteotomy) that typically share in load bearing. The remaining bone has an increased level of strain placed upon it immediately, to which it must adapt. After ulnar osteotomy in swine, the principle compressive strain on the radius increased 2 to 2.5 times normal (Goodship et al., 1979). The osteotomy caused rapid remodeling of the radius; after three months it was as large as the combined radius and ulna of the contralateral limb, and the principal compressive strain in the overloaded radius was normalised and not different from the control limb. Lanyon et al. (1982) performed a

number of osteotomies in sheep and reported that after an initial period of increased strain, the bone actually over adapted to bring strain levels below that of the original condition. Burr et al. (1989b) demonstrated in beagles that osteotomy resulted in an immediate increase in woven bone deposition on the radial periosteum by 2 weeks and a corresponding increase in CSA. This was followed by a longer term adaptive response of 2-3 months which returned the strain level to within normal limits.

Meade et al. (1984) loaded dogs statically for two months over a range of strains and found that there was a positive correlation between the increase in cross-sectional area and the superimposed strain. However, they made no attempt to functionally isolate the limbs, and the new bone laid down on the periosteum was of the woven type. In contrast, Carter et al. (1981) observed no hypertrophic response to increased loading in dogs, even though the strain environment doubled (only 8 weeks).

5.4 Specific loading parameter effects on bone adaptation

5.4.1 Magnitude of strain

A few studies have attempted to elucidate the relationship of the magnitude of loading with functional adaptation. It is difficult to compare the results of these experiments because of the differences in animal species, levels of maturity, methods of loading, and loading volumes. Rubin and Lanyon (1984) showed that with intermittent loading sufficient to produce peak strains between 0 and 4000μE there was a fairly linear 'dose-response' relationship between the change in bone area and the peak strain magnitude. They investigated the effect of peak strain magnitude by utilising the isolated

ulnar turkey model, and keeping strain rate and frequency constant across all loading groups (100 cycles at 0.01/second) while varying strain magnitude. Functional isolation caused a reduction in the bone cross-sectional area which was not reversed even with loads of 500μE. Bone levels were maintained, however, at 1000μE and any strain above this level was associated with bone accretion.

Using the avian ulnar isolation procedure, Rubin & Lanyon (1985) confirmed that bone remodeling was responsive to different magnitude strains. Strains below 1000μE were associated with bone resorption, while strains above this were associated with increases in CSA. Turner et al. (1994) found that there was a linear relationship between strain magnitude and the formation of lamellar bone in rat tibiae subjected to four-point bending. Loading below approximately 1050μE was not associated with any bone accretion. Churches and Howlett (1982) reported a significant relationship between the magnitude of load imposed on sheep metacarpi via bone pins and CSA ($r=0.75$).

5.4.2 Strain rate

O'Connor et al. (1982) were the first to suggest that the rate of strain application may have some impact on the functional adaptation of bone. Peak strains and strain rate applied in their preparation could be varied independently, and results showed that the peak strain rate consistently correlated most highly with remodeling. Low strain rates were highly associated with either less osteogenesis or even with resorption. The association between the potency of the osteogenic stimulus and the rapidity of strain change may indicate an inherent, stimulus-rate related response within the cells

themselves, or it may indicate that some stage of the mechanically related stimulus is itself strain-rate dependent. Rubin et al. (1990) concluded that the dynamic aspect of the loading signal was essential to osteogenesis, providing further evidence that the temporal character of the applied load is as critical to remodeling as the magnitude of the strain. Frequency was found to be the least important characteristic of the applied load. Additionally, McLeod & Rubin (1989), using the isolated turkey ulna, demonstrated that 500μE loads were insufficient to initiate osteogenesis at a rate of 1 Hz, but when the strain rate was increased to 15Hz substantial new bone was formed.

A number of studies have investigated the effects of static versus dynamic loads on levels of bone accretion. Static loading can be characterised as an extremely slow rate of load application. Lanyon and Rubin (1984) showed that static loading of the turkey ulna led to losses in bone similar to those observed with immobilisation. When the same magnitude loads were applied dynamically however, there was a 24% increase in CSA over the same 8 week period. In contrast, Meade et al. (1984) found that static loading led to increases in bone formation in beagles. Unfortunately, in this study, the limb was not functionally isolated and thus the results may have been biased by functional loading.

5.4.3 Distribution of strain

Lanyon et al. (1982) discovered that the radius of sheep hypertrophied at strain levels normally encountered in functional loading after resectioning the ulna. It was hypothesised that the different load distribution caused the acute bone response. It would seem, therefore, that the peak strain levels in this experiment were less important to bone

remodeling than the disruption in strain distribution. Nevertheless, this showed for the first time, that changes in the distribution of loads, even if within the normal mechanical strain window, may cause bone accretion. Studies by Lanyon (1987) and Rubin and Lanyon (1985) also indicated that bone was capable of responding to load magnitudes that were within its normal range. It was suggested that the response was mediated by the manner in which the load was distributed within the bone. If an osteocyte encounters an unusually high load it will stimulate osteoblastic activity on the bone surface. If the strains are inappropriate then the pattern of strain related stimulation will differ from that to which the cells are accustomed. It is this mismatch which defines the nature of the remodeling stimulus (Lanyon, 1987).

5.4.4 Volume of strain

The number of strain cycles needed to induce remodeling has been investigated in avian models loaded artificially *in vivo*. Peak strain levels were similar to those experienced in wing flapping, but strain distribution was somewhat different than that experienced naturally. By applying as low as 36 load cycles/day at 0.5 Hz, bone mineral at the midshaft was significantly increased (33%) over a six-week period (Rubin & Lanyon, 1984). No additional bone mass acquisition was observed after loading the bone up to 1800 cycles/day. Additionally, it was observed that 4 loading cycles/day was enough to prevent the resorptive modeling associated with disuse (Rubin & Lanyon, 1984). In contrast, Raab-Cullen et al. (1994a;b) found no relationship between the volume of loading and the magnitude of the bone response in rats submitted to four-point bending for

a different number of times per week. Strafford et al. (1989) demonstrated that cyclic loading of osteoblasts in culture stimulated a 50% increase in proliferation. The magnitudes of these changes were identical regardless of the strain duration (number of cycles). Intense running protocols (20,000 loading cycles/day), after a period of 10 days, displayed a decrease in bone apposition compared to controls (Foorwood & Parker, 1991). It appears from the few studies which have investigated this issue that a small amount of loading is necessary to maintain bone mass, and that greater volume may or may not lead to increased bone deposition and may in some situations even lead to resorption.

Lanyon (1987) suggested that the concept of minimum effective strain needs to be adjusted to include not only strain magnitude, but also strain distribution, strain rate, and non-mechanical influences on remodeling control. He proposed the concept of the Minimum Effective Strain-related Stimulus (MESS) to replace the MES, and suggested that the adaptive response of bone is pre-emptive to prevent damage, rather than a reparative response to damage already produced in the tissue.

5.5 Differential responses of mature and immature bone to loading

Bone, a dynamic tissue, experiences many changes in its material properties throughout the lifespan. Not surprisingly, the material property differences are similarly reflected in the mechanical properties of immature and mature bone.

Curry et al. (1975) concluded, after putting human bones of different ages under loading, that children had a lower modulus of elasticity, a lower bending strength, and a

lower ash content in their bones, compared to adults. Similarly, Frost (1983) stated that the compliance of human lamellar bone decreases significantly between infancy and skeletal maturity. Therefore, equal unit loads and stresses cause higher strains in more compliant immature bone than in stiffer mature ones. These higher strains will invoke faster bone modeling in younger individuals than in mature individuals (Frost, 1983), which may account for the greater modeling potential evident in rapidly growing mammals, as compared with the much reduced or virtually absent potential in adults. Indrekvam et al. (1991) showed that bone becomes stiffer in rats with increasing age, due to increased CSA, rather than due to changes in bone material quality. They concluded that young bones depend on elastic deformation to avoid fractures, whereas older bones depend on larger bone size to distribute the encountered loads.

Rubin & Bain (1989) showed that mechanical signals that were osteogenic in young turkeys were unable to elicit adaptive responses in older animals. This suggests that some aspect of the cell's sensitivity to its physical environment, or the tissue's ability to respond to these stimuli becomes depressed with increasing age. These studies suggest that mature and immature bone respond differently to mechanical usage.

6. HUMAN WORK ON THE MECHANICAL ATTRIBUTES OF LOADING

The methods utilised to study mechanical loading parameters in animals are for the most part invasive, or involve extreme levels of exercise interventions which are either unethical or not feasible in humans. Additionally, the isolation of a particular loading

parameter is often difficult to achieve and has never been attempted with the human model. Consequently, most of our understanding of the relationship between bone adaptation and mechanical attributes of loading in humans have been derived indirectly from correlation or comparative studies.

6.1 Magnitude of loading

Magnitude effects of mechanical loading in humans usually are best exemplified by comparing bone adaptations in athletes who experience different magnitudes of loading inherent in their sports. Across groups, athletes who incorporate heavy resistance training (high magnitude loading) as part of their training regimen typically have the highest BMD (Heinrich et al., 1990; Heinonen et al., 1993), followed by athletes who perform weight-bearing activities (medium magnitude loading) such as throwing, running, or playing soccer (Risser et al., 1990; Heinonen et al., 1993), followed by normoactive controls. Non-weight-bearing activity (lowest magnitude loading), such as swimming, where buoyancy counteracts gravity is typically associated with the lowest BMD (Risser et al., 1990; Cassell et al., 1993; Grimston et al., 1993; McColluch et al., 1992; Slemenda et al., 1991a; Taaffe et al., 1995).

While there has been no direct study of the effects of different magnitudes of loading on BMD in humans, Whalen et al. (1988), modeling data from numerous studies, concluded that the stress magnitude is a more important factor in the determination of BMD than the volume of loading (defined as the number of loading cycles). In summary,

the above studies suggest that there is indeed a magnitude response of bone to mechanical loading.

6.2 Volume of loading

Volume of training is a complex variable consisting of the product of training intensity, frequency, and duration. It is often incorrectly considered to be synonymous with training magnitude, which in animal studies is more appropriately described as strain load or intensity. Numerous studies have concluded that athletes who perform greater volumes of active loading generally have greater levels of BMD than non-athletes (e.g. Slemenda & Johnston, 1993; McCulloch et al., 1992; Young et al., 1994, Nichols et al., 1993; Cassell et al., 1993; Grimston et al., 1993). A positive curvilinear relationship was found in male powerlifters in terms of vertebral BMC and training volume (Granhed, 1987). However, Slemenda & Johnston (1993) found a significant negative correlation between the volume of practice and BMD in elite figure skaters. Lower leg BMD was found to have an inverted U relationship with training volume in male distance runners (MacDougall et al., 1992). These studies suggest that their may be a ceiling effect as to how much volume of exercise is optimal to skeletal adaptation.

6.3 Rate of loading

No study to date has investigated the effects of the rate of loading on bone accretion in humans. Sports differ in the nature of their loading patterns. Gymnastics, which is typically very dynamic, is characterised by extremely fast loading and unloading patterns on weight-bearing bones. Other weight-bearing activities, such as running, are

also characterised by rapid loading and unloading cycles (Heinrich et al., 1990), whereas in weight-lifting bones are loaded and unloaded at a relatively slow rate (Nilsson & Westlin, 1971). Although the rates of loading differ among these activities, the activities also differ tremendously in terms of their magnitudes, frequencies, and volumes of loading. These differences preclude isolation of the rate loading effect on bone in these comparative studies.

6.4 Frequency of loading

The effect of frequency of loading on BMD status has not been extensively or prospectively studied in humans. One study found that a higher frequency of run training per week was associated with insignificantly higher BMD at the femur in collegiate runners compared to recreational runners (Heinrich et al., 1990). Obviously, the comparison of elite to recreational runners, in itself, is not valid for the study of frequency effects since they almost certainly will have differing volumes and intensities of training.

In summary, there have been extremely few investigations of the different loading parameters on the functional adaptation of bone in humans. Results that have been obtained in this area, are often confounded by the inability to isolate specific loading parameters. The only trend that appears to have emerged is that of a possible ceiling effect for both the magnitude and volume of loading on skeletal adaptation in humans.

7. FACTORS AFFECTING BONE MASS IN CHILDHOOD

7.1 Introduction

Peak bone mass is believed to be largely genetically determined (Pollitzer & Anderson, 1989; Slemenda et al., 1991b), but other factors including diet and physical activity (PA) can influence the realisation of this genetic potential. These two factors, along with others, interact with hormonal status in a synergistic fashion to influence bone development. For example, low levels of endogenous estrogen may negate the effects of mechanical loading and may even bring about a state of bone loss (Young et al., 1994). Similarly, if dietary calcium is insufficient there will be abnormal accretion of bone, even if the mechanical stimulus for it is present.

The effects of calcium and PA may have their largest lifetime impact on bone during adolescence and early adulthood, when bone mass is being accumulated at its most rapid rate (Ott, 1991). Any action which increases bone mass during childhood and adolescence may have a dramatic impact on bone health in later life, a period when bone mass, and structural integrity, are characteristically waning.

7.2 Development

The bone mass that is present in later life is the product of three primary influences: the absolute amount of bone mass that was acquired by the time of peak bone mass, the ability of the individual to maintain that bone mass through adulthood, and the ability of the individual to resist the accelerated loss of bone mass with increasing age, especially after menopause for women. It is extremely important to attain the highest

possible peak bone mass early in life, since this is the level from which bone mass is gradually eroded with increasing age. A maximal bone mass at skeletal maturity is considered the best protection against age-related bone loss and subsequent fracture risk (Matkovic, 1992).

Numerous cross-sectional and longitudinal studies have investigated the timing of peak bone mass. It is now generally accepted that bone achieves its maximum mass sometime during late adolescence or early adulthood, although there appears to be bone and region specific differences in the attainment of timing of peak bone mass (Mazess, 1982; Recker et al., 1992; Gordon & Webber, 1993; Kelly et al., 1990; Rico et al., 1992; Blimkie et al., in press). It has been suggested that at least 90% of peak bone mass will be achieved by 20 years of age (Glastre et al., 1990; Matkovic et al., 1990; Matkovic et al., 1994). Hormonal status, especially the circulating level of estrogen in women, plays an important role in attaining and maintaining optimal peak bone mass both during adolescence and adulthood (Pollitzer & Anderson, 1989).

7.3 Genetic influences on bone mass

Three methodologies have been used to examine the heritability of bone mass: examining the bone mass in daughters of osteoporotic women, examining the bone mass in parents and offspring, and examining the resemblance of bone mass between twins.

Seeman et al. (1989) concluded that daughters of women with osteoporosis had lower than normal bone mass in the lumbar spine compared to daughters of mothers without osteoporosis, and suggested that the low bone mass in the daughters may be a

result of a failure to reach a high peak bone mass, rather than excessive loss of bone during early adulthood. Evans et al. (1988) concluded that osteoporotic mothers' vertebral BMD was significantly correlated with their daughters' BMD. Seeman et al. (1994) stated that the daughters of women with hip fractures are likely to be at increased risk for hip fractures themselves because of reduced femoral neck bone density.

In an investigation of postmenopausal mothers and their young adult daughters, Lutz (1986) found significant mother-daughter correlations for radial BMC ($r = 0.40$) and BMD ($r = 0.50$). Tylavsky et al. (1989) concluded that hereditary contributions from the mothers plays an overwhelmingly important role in the accrual of bone mass by their daughters by ages 18-22, but that non-genetic factors take on relatively more importance with increasing age. Lutz & Tesar (1990) found significant correlations between mother-daughter pairs for lumbar and femoral areal BMD and suggested that the inheritance of bone mass in females may have at least two components, one influencing the level of peak bone mass, and one related to the loss of bone at menopause.

Krall & Dawson-Hughes (1993) concluded that 46-62% of the variability in BMD could be accounted for by genetics, and that the other 38-54% was attributable to non-hereditary factors of measurement error and individual environment. Recently, McKay et al. (1994) studied daughter-mother-grandmother groups and found correlations of 0.41-0.57 for mother-daughter and mother-grandmother BMD at the proximal femur and lumbar spine. Matkovic et al. (1990) found significant correlations ($r = 0.4$-0.7)

between mean parental, maternal, and paternal BMD and BMD of their adolescent daughters.

Smith et al. (1973) studied monozygotic and dizygotic twins in an attempt to determine the heritability of bone mass and width at the radius. Intra-pair differences were smaller in MZ than DZ twins, indicating that bone mass and width have significant genetic determinants. It was also recognised that these intra-pair differences increased with age suggesting that there may be a genetic-environment interaction that contributes to the observed variation in bone mass. Slemenda et al. (1991b) found the same trends in intra-twin variability in BMD between MZ ($r = 0.71$-0.85) and DZ ($r = 0.19$-0.51) twins at a number of axial and appendicular skeletal sites. Adjustments for height, weight, age, and environmental characteristics did not reduce heritability estimates. It was suggested that with increasing age, an individual's environment becomes more influential on bone and the familial association becomes weaker (Slemenda et al, 1991b), as evidenced by the increasing within-MZ pair variability in older women.

It appears, from these studies, that genetics account for up to and perhaps over 50% of the variability in bone mass and size. It also appears that genetics plays a greater role in the axial skeleton (spine, proximal femur) (Dequecker et al., 1987), and is less important at the appendicular sites (forearm) (Pocock et al., 1987; Kelly et al., 1993), based on data gathered from twin-studies.

8. EXERCISE AND BONE ADAPTATION IN CHILDREN

8.1 Introduction

Weight-bearing exercise in mature adults has been found to either help in preventing bone loss, have no effect on bone status, or marginally increase bone mass by 1-5% (e.g. Krall & Dawson Hughes, 1993; Metz et al., 1993; Aliola et al., 1988; Recker et al., 1992). During adolescence, exercise intervention may account for 10-30% higher bone mass, as displayed by comparisons of dominant and non-dominant arms in young adult tennis athletes (Huddleston et al., 1980; Jacobson et al., 1984; Jones et al., 1977).

Numerous cross-sectional, but fewer longitudinal studies have investigated the relationship between PA and bone mineral status in adults (e.g. Schoutens et al., 1989; Whalen et al., 1988; Bailey & McCulloch, 1990; Blimkie et al., in press). The relationship between PA and bone mineral in children and adolescents, however, has not been extensively investigated. This is surprising since childhood and adolescence may be the period during which PA has its most significant effect on the acquisition of bone mass. In adults, the effects of PA have been modest with short term activity, but quite beneficial when performed for an extended duration at high levels of muscular loading (Marcus et al., 1992). The effectiveness of different types and durations of PA on bone density have not yet been adequately addressed in the pediatric and adolescent populations. Additionally, while it is generally accepted that weight-bearing activity leads to higher levels of BMD in children, the effect of varying the frequency, duration, magnitude and rate of loading on bone mineral adaptation remains to be described for this population.

8.2 Retrospective Studies of PA and Bone Mass.

Studies that utilised retrospective questionnaires to correlate current bone measures with childhood and adolescent activity levels have not been conclusive, owing, in part, to ambiguity in question construction and to accuracy of recall of historical events in childhood.

Talmage and Anderson (1987) found that 25 year-old women who participated in secondary school sports or had heavy farm chores as a child had higher BMD than those who did not do these activities. In addition, Tylavsky et al. (1992) reported a significant relationship between the amount of activity that the subjects did as a child, and present BMD levels. Kriska et al. (1988) investigated historical PA patterns in postmenopausal women and found a significant relation between historical PA and dimensions of current bone at the radius, particularly bone area, with the association being strongest for activity levels during the period of 14-21 years of age. Similarly, os calcis density assessed in healthy adult women was found to be significantly positively correlated with childhood levels of physical activity (McCulloch et al., 1990). Fehily et al. (1992) reported that time spent in sports activity during the first years of high school was positively associated with BMC measures at the radius in women: interestingly, this association was stronger than for current sport involvement, suggesting that the largest effect of interventions may be during the adolescent years while the bones are still growing.

It appears that activity levels during childhood may be positively correlated to bone mass during adulthood. These findings point to the importance of PA, especially

during adolescence, for the attainment of optimal peak bone mass, and the retention of high bone mass during the adult years.

8.3 Unilateral Studies of PA And Bone Mass

Activities performed during childhood and adolescence that stress one limb to a greater extent than the other provide a good internal control from which to assess the effects of predominantly unilateral activity on bone mineral status. The advantage of this model is that genetic, dietary, and endocrine influences are similar for both limbs and are controlled as possible confounding factors for the bone adaptation response.

A number of recent studies have investigated the differences in bone mass between dominant and non-dominant limbs of both normal and athletic groups of children. Faulkner et al. (1993) examined BMC and BMD in the dominant and non-dominant limbs of a group of children aged 8-16 years. BMD and BMC were significantly higher in the dominant arm. Greater BMD in the dominant arm was suggested to be a product of greater tensile loading compared to the non-dominant arm, which was evident even at the youngest ages tested (8-9 years). The lack of difference between the legs was attributed to the equally distributed weight-bearing functions by both limbs.

In a study of 18-22 year-old female tennis players, BMC was found to be significantly greater (16%) in the dominant compared to the non-dominant arm (Jacobson et al., 1984). Another study by Jones et al. (1977) found substantially increased cortical thickness in the playing arm of female professional tennis players compared with the non-playing arm (28.4% larger). In a normal non-athletic population, the difference

between arms has been found normally to be <3% (Awbrey et al., 1984). A recent study of the Finnish women's national squash team reported significantly higher bone density (15.6%) in the dominant arm of the players compared to controls (Haapasalo et al., 1994). The number of training years was significantly correlated with bone mass, and athletes who began playing before menarche had significantly higher BMDs (22% higher) than athletes who began at least one year after menarche. Another recent study of male Finnish tennis players by Kannus (1995) showed similar results. These latter studies point to the importance of physical activity early in childhood prior to puberty, to maximise increases in bone density.

The effects of immobility and inactivity on the skeleton have been studied extensively in adults, but not in children. In a group of children that had previous fractures to either the tibia or femur, significantly lower BMD was found at the hip of the injured leg compared to the hip from the uninjured side (Henderson et al., 1992). In addition, children who were immobilised longer, had larger deficits in bone mass than children who were immobilised for shorter periods. Bailey (1992) found significant differences between hips within the same individual in unilateral Legg-Calve-Perthes disease. Both of these studies illustrate the negative consequences of inadequate amounts of weight-bearing activity on the proximal femur and hip.

A summary of the unilateral studies discussed in this section is presented in Table 1.

Table 1. ***Unilateral studies on physical activity and bone density.***

Study	n	Age (y)	Method	Activity/Measurement Site	Bone Results
Jones et al. (1977)	tennis=84	14-50y	roentgen-ograms	Effects of preferential use of limbs in professional tennis players/humerus	It was concluded that the humerus of the playing arm was greater by 34.9% in the males and 28.4% in the females, indicating a highly signif. hypertrophy of bone in response to exercise.
Jacobson et al. (1984)	E=11	18-22y	SPA	Elite tennis performance/ measures taken at the distal radius	BMD was significantly higher (16%) at the dominant radius when compared to the non-dominant radius. In a normal population the difference between limbs ~3%
Henderson et al. (1992)	girls=14 boys=24	2-15y	DXA	Recent uncomplicated fracture to one tibia or femur/hip - measured 2.3 years after fracture	An average 3.3% difference between hips and that if the time of immobilization was over 8 weeks, the deficit to the bone was even larger (4.3%). In addition, if there was immobilization for less than 4 weeks there was no difference in BMD
Bailey et al. (1992)	girls=4 boys=14	8-16y	DXA	Compromised weight bearing with unilateral Legg-Calve-Perthes Disease/ hip	The mean difference found between the hips was 5.6%, with the side being afflicted having lower BMD
Faulkner et al. (1993)	girls=124 boys=110	8-16y	DXA	comparison of bone in weight-bearing and non-weight-bearing limbs	BMD and BMC were significantly higher in the dominant arm at all age groups. There was no difference between the dominant and non-dominant legs for any of the measures taken.
Haapasalo et al. (1994)	E=19 C=19	18-28y	DXA	Elite squash performance/ proximal humerus, ulnar shaft	BMD was significantly higher (15.6%) at the dominant humerus compared to the non-dominant (BMC=17.8%). Ulnar shaft showed the same trends. Those athletes that began training at or before menarche had significantly higher BMD (22%)
Kannus et al. (1995)	female tennis and squash=105 C=50	16-50y	DXA	Determine the difference in dominant and -non-domin. arms (BMC)/ humerus & radius	Racquet-sport athletes had greater BMC differences between arms (8.5-16.2%) than controls (3.2-4.6%). Additionally, the difference was 2 to 4 times greater in the athletes who began their careers at or before menarche.

E = Exercise group; C = Control group; DXA = Dual Energy X-ray Absorptiometry; SPA = Single Photon Absorptiometry;
BMD = Bone Mineral Density; BMC = Bone Mineral Content; PA = physical activity
Significance at least $p<0.05$

8.4 Cross-Sectional Observational Studies

Most research investigating the relationship between PA and bone adaptation in young females has been cross-sectional in nature and have incorporated mostly elite athletic populations. Differences in bone mass between athletic and control populations permit inferences about the effect a particular activity may have on bone accretion, provided that other covariables, such as weight, selection bias, or hormonal status, have been controlled.

8.4.1 General Physical Activity Levels And Bone Mass.

Several studies have correlated general PA levels of children with measures of bone mass. Children's poor recall, sporadic activity, unrealistic estimates of physical activity, and diverse range of activities make it difficult to precisely quantify their activity levels (Saris, 1986).

Ruiz et al. (1995) determined that greater weekly duration of sports activity led to higher BMD at the LS and FN. Physical activity during the adolescent years, as determined by questionnaire, had a positive effect on hip, but not LS, BMD in 138 high school girls (Turner et al., 1992). Similarly, Kroger et al. (1992) found significantly higher mean femoral BMD in study subjects who were physically active (lumbar measures showing the same trend, but failing to reach significance), after adjusting for age, body weight, and height. In contrast, after correcting for weight and pubertal stage, Rubin et al. (1993) found that there was a contribution of exercise to LSBMD ($p = 0.036$), but not proximal femur BMD.

Slemenda et al. (1991a) found significantly higher BMD measures in children who were more active, after adjusting for age and gender. They further concluded that, depending on the specific skeletal site, an individual who was active throughout childhood could emerge from adolescence with 5-10% higher BMD than an inactive individual. In contrast, Southard et al. (1991) found no effect of PA on bone mass in a group of 218 healthy children, after adjusting for Tanner stage and weight. In summary, higher levels of PA in children are generally associated with higher BMD, especially in the weight-bearing bones.

A summary of the general PA studies discussed in this section is presented in Table 2.

8.4.2 Athletic Populations And Bone Mass.

Numerous studies have examined the effect of weight-bearing athletic activities on bone mass acquisition. Running has been shown to produce skeletal impact forces 3-5 times body weight (Engsberg et al, 1991; Nigg, 1985), and jumping and high impact activities up to 7-10 times body weight (Lees, 1981; Nigg, 1985). These high impact and weight-bearing activities may impart the stimulus needed to accelerate the already rapid modeling process within growing adolescent bone. In most instances, athletic populations have been categorised into one of three classes of activity: non-weight-bearing activities or active loading (swimmers), weight-bearing activities (figure skaters, runners), and high impact loaded activities (gymnasts).

Table 2. ***General Physical Activity Levels and Bone Mass***

Study	n	Age(y)	Method	Activity/Measurement Site	Bone Results
Slemenda et al. (1991)	118 (59 twins)	5.3-14y	SPA & DPA	Tried to determine whether childhood activity was associated with bone mass/radius, lumbar spine & femur	Adjusted for age and gender, there was a significant effect of activity on bone mass. The children with higher activity levels had higher BMD. Concluded that an active child may emerge from childhood with 5-10% higher BMD
Southard et al. (1991)	girls=134 boys=84	1-19y	DXA	Healthy white children/lumbar spine	No effect of PA on any bone measure when controlled for Tanner stage
Kroger et al. (1992)	girls=44 boys=40	6-19y	DXA	Healthy white children/ FN & LS	When corrected for age, body weight and height, sig. higher mean femoral density was found in children who were physically active
Turner et al. (1992)	girls=138	mean=16.4	DXA	Cohort of healthy high-school girls/ lumbar spine (L2-L4), proximal femur	Concluded that PA had a significant positive effect on BMD at the proximal femur; this was not found to be true for the lumbar vertebrae. PA accounted for between 4-5% of the variability at the proximal femur
Rubin et al. (1993)	299	6-18y	SPA & DPA	Healthy white children/ BMD at the lumbar vertebrae; BMC at the distal third of the radius	A significant positive effect of PA at the lumbar vertebrae, but no relationship for the distal radius
Ruiz et al. (1995)	n=151	7-15.3y	DXA	Effects of general PA on BMD/ lumbar spine (L2-L4), proximal femur	The weekly duration of sports activity influenced both vertebral and femoral sites especially in girls and during puberty.

DXA = Dual Energy X-Ray Absorptiometry; DPA = Dual Energy Absorptiometry; SPA = Single Photon Absorptiometry;
BMD = Bone Mineral Density; PA = Physical Activity;
Significance at least $p<0.05$

Effects of Active Loading On Bone Mass. Children who swim as their primary form of PA consistently have lower bone densities than children involved in weight-bearing sports, and quite often lower densities than children who are sedentary (Risser et al., 1990; Cassell et al., 1993; Grimston et al., 1993; McColluch et al., 1992; Taaffe et al., 1995). It seems that the active loading of the muscles pulling on the bones is not enough to resist bone loss in the lower gravity environment of swimming. The relative weightlessness during many hours of swimming may decrease bone density, as does zero gravity in astronauts. Alternatively, swimming may select athletes with lower bone density at certain sites for increased buoyancy.

A summary of the studies discussed in terms of active loading and bone mass is presented in Table 3.

Effects Of Weight-Bearing Activity On Bone Mass. Several studies have concluded that weight-bearing activity in adolescents and children leads to higher bone densities. McCulloch et al. (1992) investigated the differences in BMD in adolescent soccer players, actively loaded swimmers, and sedentary individuals. There was a trend for the soccer players to have higher BMDs than the swimmers ($p = 0.08$) at the os calcis, but there were no differences at the distal radius, a nonweight-bearing site, between any of the groups. When assessing the BMD of the calcaneus in competitive swimmers, volleyball players, basketball players and controls, it was concluded that the athletes, aside from the swimmers, possessed significantly higher densities than the control subjects (Risser et al., 1990).

Table 3. ***Effects of Active Loading on Bone Mass***

Study	n	Age(y)	Method	Activity/Measurement Site	Bone Results/Conclusions
Risser et al. (1990)	V=12 B=9 swim=10 C=13	18-20y	DPA	Differences in BMD between actively loaded and impact or weight-bearing activities/calcaneous & lumbar spine	Adjusted for height and weight, the swimmers had sig. lower BMD in the lumbar spine than the other athletes and the controls (11-20%).The V and B had sig. higher calcaneal BMD (31-50%)
McCulloch et al. (1992)	soc=23 swim=20 C=25	13-17y	CT & SPA	Differences in BMD between soccer players, actively loaded athletes, and controls/os clacis & distal radius	Trend (P=0.08) for the soccer players to have higher BMD than the swimmers at the os calcis. There were no differences in the distal radius between groups
Cassell et al. (1993)	gym=25 swim=21 C=10	7-10y	DXA	Differences in BMD between gymnasts, swimmers, and controls/ total body BMD segmented	When corrected for weight, lean body mass, and peak torque, total body BMD was higher in gymnasts than both controls and swimmers (4-5%)
Grimston et al. (1993)	I=17 swim=17	10-16y	DPA	The effect of impact activities vs. the effect of swimming on BMD/ lumbar spine & femoral neck	I group had higher BMD at the femoral neck, but this failed to reach significance (P=0.057). There were no significant differences between the lumbar measures.
Taaffe et al. (1995)	gym=13 swim=26 C=19	18-22y	DXA	The effects of impact loading vs. the effect of swimming on BMD/ LS, FN, trochanter, & whole body	Gymnasts - greater FN, trochanteric, and weight-corrected whole-body and LS BMD when compared to C and swim groups. Swimming confers no beneficial effects on bone mass; impact loading is a powerful osteogenic stimulus.

DXA = Dual Energy X-Ray Absorptiometry; DPA = Dual Energy Absorptiometry; SPA = Single Photon Absorptiometry; M = Male;
F = Female; C = Controls; I = Impact Loaded group; swim = swimmers; WT = Weight Trainers; BMD = Bone Mineral Density; PA = Physical Activity;
run = long-distance runners; gym = gymnasts; V = Volleyball players; B = Basketball players; ballet = ballet dancers;
fig = figure skaters; soc = soccer players
Significance at least $p<0.05$

The effects of weight-bearing activity on bone density are exemplified in a study by Young et al. (1994). BMD was determined in weight-bearing and nonweight-bearing skeletal sites in elite ballet dancers (average age 17 years), many of whom had irregular menses. The dancers were compared with similarly aged groups of regularly menstruating girls and amenorrheic anorectic girls. BMD at weight-bearing sites was not affected by prolonged oligomenorrhea and reduced body weight, and after adjusting for body weight was actually 5-10% higher than the reference groups. In the nonweight-bearing sites, however, BMD was decreased similar to that found in anorexia nervosa. The higher density in the lower limbs of the dancers may have resulted from several years of practice and increased accumulation of bone during the prepubertal years.

Slemenda and Johnston (1993) studied site-specific bone mass effects among elite female figure skaters, forty percent of whom had irregular menses. When densities were adjusted for age and weight, there was no difference in the upper body measures between skaters and controls. There were, however, significant difference in lower body densities (leg 5.5% and pelvis 11% higher in skaters) which supported the conclusion of site-specific adaptations of bone. Furthermore, regularly menstruating skaters had an approximately 2% greater BMD than the ammenorrheic athletes. It was concluded that menstrual irregularities had only slight (2%) negative effects on the skaters' skeletons, and that the jumping motions used in ice skating may be the stimulus responsible for the positive effect on bone density. The activity levels in this study, however, were extreme and not typical of normal athletes.

A summary of all the studies discussed in this section is presented in Table 4.

Effects Of Impact Loading On Bone Mass. A number of researchers have recently looked at gymnastic activity and more specifically the effects of repetitive high impact loading on bone accretion. High impact activities impart large compressive strains to the skeleton, which is believed to be the stimulus for increased levels of bone deposition.

Grimston et al. (1993) compared the differences in bone mineral density in children who participated in competitive sports which involved high levels of impact loading or placed high weight-bearing stress on the skeleton (running, gymnastics, tumbling, and dance) to elite swimmers who actively loaded their bones through their nonweight-bearing activity. FNBMD measures were higher in the young females in the impact loaded and weight-bearing groups compared to matched actively-loaded swimmers, although the relationship failed to meet significance ($p = 0.057$). This trend was evident even though most of the swimmers engaged in a low-resistance, high-repetition weight training program in the off-season. None of the lumbar spine measures were significantly different between the groups of females. The relative lack of effect in the lumbar region and the trend for higher densities in the proximal femur may be due to preferential loading of the FN region in these weight-bearing and impact activities. Interestingly, there was no significant correlation between BMD at the femoral neck or lumbar spine and total weight-bearing hours, which is in contrast to the findings of Slemenda et al. (1991a).

Table 4. ***Effects of Weight-Bearing Activity on Bone Mass***

Study	n	Age(y)	Method	Activity/Measurement Site	Bone Results
Risser et al. (1990)	V=12 B=9 swim=10 C=13	18-20y	DPA	Differences in BMD between actively loaded and impact or weight-bearing activities/calcaneous & lumbar spine	Adjusted for height and weight, the swimmers had sig. lower BMD in the lumbar spine than the other athletes and the controls (11-20%).The V and B had sig. higher calcaneal BMD (31-50%)
McCulloch et al. (1992)	soc=23 swim=20 C=25	13-17y	CT & SPA	Differences in BMD between soccer players, actively loaded athletes, and controls/os clacis & distal radius	Trend (P=0.08) for the soccer players to have higher BMD than the swimmers at the os calcis. There were no differences in the distal radius between groups
Slemenda & Johnson (1993)	fig=22 C=22	10-23y	DEXA	Looked at site-specific bone mass in figure skaters and compared them to a group of sedentary controls/ head, arms, legs, pelvis, & trunk	Skaters had a significantly higher (5.5-11%) BMD in the lower body measures when compared to the controls. Skaters with regular menses had ~2% higher BMD on average (failed to reach significance)
Young et al. (1994)	ballet=44 C=23	mean=17y	DEXA	Studied weight-bearing and non-weight-bearing sites in elite ballet dancers vs. eumenorrheic controls/ lumbar spine, ribs, arms, head, proximal femur	BMD at the weight-bearing sites was 5-10% higher in the dancers when adjusted for mass. But, in the non weight-bearing sites, the BMD in the dancers was similar to that found in anorexics

DXA = Dual Energy X-Ray Absorptiometry; DPA = Dual Energy Absorptiometry; SPA = Single Photon Absorptiometry; M = Male;
F = Female; C = Controls; I = Impact Loaded group; swim = swimmers; WT = Weight Trainers; BMD = Bone Mineral Density; PA = Physical Activity;
run = long-distance runners; gym = gymnasts; V = Volleyball players; B = Basketball players; ballet = ballet dancers;
fig = figure skaters; soc = soccer players
Significance at least $p<0.05$

Recently, Padro et al. (1995) reported that prepubertal gymnasts displayed significantly greater LS and tibial, but not radial BMD, when compared to matched controls. Additionally, predominantly trabecular regions of the skeleton exhibited higher densities than the predominantly cortical regions, indicating a differential response of the two bone compartments to impact loading. In another recent study of prepubertal gymnasts (Bass et al., 1995), BMD at the arms, legs, and LS was found to be ~15% higher than matched controls. Bass et al. (1995) further stated that prepubescence may be the most opportune time during the lifespan to increase BMD.

Taaffe et al. (1995) found that young adult gymnasts (mean age 19 years) had higher FN, and trochanteric BMD than both an active control group and a group of swimmers. Additionally, when BMD was corrected for body mass, gymnasts also displayed higher TBBMD (than swimmers only) and LSBMD. Leg and arm BMD was significantly higher in the gymnasts compared to the swimmers. These findings suggested that the impact loading associated with gymnastics imparted a powerful osteogenic stimulus, and that long-term non-weight-bearing training (swimming) that incorporates powerful muscular contractions confers no beneficial skeletal effects on bone mass of young women.

Significantly higher levels of BMD were found in the lumbar spine of both controls and gymnasts compared to long-distance runners (Robinson et al., 1993). In addition, femoral neck density was highest in the gymnasts and lowest in the runners. In

another study, elite gymnasts had significantly higher lumbar spine and femoral neck BMDs compared to active controls (Nichols et al., 1994).

Cassell et al. (1993) compared total body BMD in young (7 to 10 year-old) female gymnasts, swimmers and a group of controls. When controlled for mass and peak torque, gymnasts displayed higher BMD measures than both other groups. Strain imparted on the skeleton from gymnastic activity increased bone mass independent from body weight and lean mass which suggested that there were forces apart from body mass that effected the acquisition of bone.

In another study of runners, gymnasts and controls (average age 20.4 years) who had a similar prevalence of amenorrhea and oligomenorrhea, Robinson et al. (1995) reported that gymnasts had higher densities in the femoral neck than both the other groups. When corrected for bone size and expressed as BMAD, bone density differed for the FN and LS among all groups: gymnasts>controls>runners. The sample sizes were too small, however, for statistical comparisons. The high impact forces inherent to gymnastics were implied to have caused greater bone mass accretion than the lower impact forces of running, and thus were able to override the negative effects of disturbed menstrual status.

These findings support the animal models of Rubin and Lanyon (1985) which conclude that the absolute magnitude of the stimulus is much more important to the formation of additional bone than is the frequency of loading. This leads to the hypothesis that a larger magnitude stress at a lower frequency may be better for bone accretion than a lower level impact stress with a higher frequency (such as running), as suggested by Frost

(1988) with the MES theory. It seems that the high impact stressors placed on the bones through gymnastics participation lead to higher bone densities than other sports studied (basketball, volleyball, swimming, running, soccer). The femur and hip area in gymnasts, two impact-bearing regions, seem to be the areas most consistently found to exhibit higher densities.

A summary of all the studies involving gymnastic activity and BMD discussed in this section is presented in Table 5.

8.5 Controlled trials, longitudinal, and prospective observational studies

Only one prospective study has assessed the influence of high impact load training through gymnastics on bone mass and density in humans. Nichols et al. (1994) examined changes of BMD in the hip and spine following 27 weeks of gymnastics training in 11 intercollegiate gymnasts, and compared the results to 11 normally active sedentary controls. At the beginning of training, the gymnasts displayed significantly higher BMDs than the controls in both the FN (7.8%) and the LS (7.8%), which suggested that gymnastic activity up to that time had a positive effect on bone accretion. At the conclusion of the training period, BMD was increased in the gymnasts at the LS by 1.3%, with no significant increase at the FN; the control group showed no change in bone mass at any site (Nichols et al., 1994).

Interestingly, among the gymnasts, the veteran (elite) gymnasts increased their lumbar densities by only 0.9% whereas the density of the freshman gymnasts increased by 1.5% over the training period. These results suggest that the elite gymnasts were closer to

Table 5. ***Effects of Impact Loading on Bone Mass***

Study	n	Age(y)	Method	Activity/Measurement Site	Bone Results/Conclusions
Robinson et al. (1993)	run=20 gym=12 C=19	17-27y	DXA	Differences in bone mass between gymnasts, runners and controls/ femoral neck & lumbar spine	Runners had significantly lower lumbar BMD (12-17%) than the gymnasts and the controls.Femoral neck density was highest in the gymnasts and lowest in the runners
Cassell et al. (1993)	gym=25 swim=21 C=10	7-10y	DXA	Differences in BMD between gymnasts, swimmers, and controls/ total body BMD segmented	When corrected for weight, lean body mass, and peak torque, total body BMD was higher in gymnasts than both controls and swimmers (4-5%)
Grimston et al. (1993)	I=17 swim=17	10-16y	DPA	The effect of impact activities vs. the effect of swimming on BMD/ lumbar spine & femoral neck	I group had higher BMD at the femoral neck, but this failed to reach significance (P=0.057). There were no significant differences between the lumbar measures.
Nichols et al. (1994)	gym=11 C=11	18-22y	DXA	Effects of a season of gymnastic training on BMD/ lumbar spine & femoral neck	Gymnasts has significantly greater LS and FN BMD when compared to the controls. Additionally, the gymnasts LS BMD increased by 1.3% over the 27 wk training period
Padro et al. (1995)	gym=13 C=13	9-11y	DXA	Effects of impact loading on BMD in prepubertal females/ lumbar spine, radius, & tibia	Gymnast group (20h/week) had greater BMD at the lumbar spine and tibia, but not the radius. Concluded that impact loading may effect trabecular bone to a greater degree than cortical bone.
Bass et al. (1995)	gym=34 C=37	8-9y	DXA	The effects of elite gymnastic activity on BMD and bone growth/ arms, legs, & lumbar spine	BMD at arms, legs and spine was 10-15% higher in the gym. Also concluded that intense gymnastic activity prepubertally may result in both increased BMD and shorter stature.
Taaffe et al. (1995)	gym=13 swim=26 C=19	18-22y	DXA	The effects of impact loading vs. the effect of swimming on BMD/ LS, FN, trochanter, & whole body	Gymnasts - greater FN, trochanteric, and weight-corrected whole-body and LS BMD when compared to C and swim groups. Swimming confers no beneficial effects on bone mass; impact loading is a powerful osteogenic stimulus.
Robinson et al. (1995)	gym=21 run=20 C=19	mean=20.4	DXA	Differences in BMD between gymnasts, runners, and controls/femoral neck, lumbar spine & whole body	The gymnasts exhibited higher densities (11-23%) in the femoral neck than the runners or the controls, despite similar prevalence of oligomenorrhea in the athletes

their genetic potential for peak bone mass than the freshman gymnasts. It was surprising to find such significant changes over such a short training interval, since trabecular bone remodelling usually requires 16-18 weeks, and cortical bone much longer (Snow-Harter & Marcus, 1991). It was concluded that this may have been why there was a significant effect at the LS, a largely trabecular area, and not at the FN, a more cortical area (Nichols et al., 1994). In addition, the initial high densities of the gymnasts may have diminished the effect of the exercise intervention and their mature skeletons may not have responded in the same manner as a growing skeleton. A study performed with two groups of skeletally immature sedentary individuals, one acting as a control, and the other acting as the intervention group would perhaps allow for the effects of the intervention to be most dramatic.

There have been only two investigations of the effects of resistance training on bone mass in adolescent females and the results have been less than conclusive. Blimkie et al. (1993) trained 17 (18 control) girls between the ages of 14 and 18 with resistance machines for 26 weeks. Strength increased over the training period, but their was only a transient increase in lumbar spine BMD and no significant increases in bone mass in any of the other measured areas. Snow-Harter et al. (1992) randomly placed a group of young women (19.9 years average) into either a resistance training, running, or control group. After a training period of 8 months no BMD differences between the groups at the proximal femur were seen, and the increases at the lumbar spine were minimal (+1.3% runners, +1.2% resistance trainers, -0.8% controls). Although statistically significant,

many of the participants were already fairly active, so the interventions may not have provided a sufficiently novel stimulus for greater bone adaptation.

Kroger et al. (1993) performed a prospective study investigating the development of bone mass in the LS and the FN in children and was unable to find strong associations between PA and bone measures. BMD was corrected for bone size, since bone size is known to influence the BMD measures determined by DXA (Katzman et al., 1991). While there was a trend for the most physically active children to gain more BMD, it failed to reach significance. The small sample size was cited as a possible factor for the lack of significant correlations.

In summary, these longitudinal and prospective studies have been largely inconclusive and have not addressed the effects of specific loading parameters on bone adaptation. Additional studies of longer duration with larger sample sizes are needed to adequately describe the effects of different load magnitudes, rates, intensities, and frequencies on bone adaptation.

9. SUMMARY OF SPECIFIC LOADING PARAMETER EXPERIMENTATION IN CHILDREN

9.1 Deficiencies in experiments to date

There has been no systematic or controlled attempt to elucidate the effects of specific loading parameters of PA or exercise on bone adaptation in children. Grimston et al. (1993) have been the lone group to suggest that there may be a load magnitude effect

on BMD in weight-bearing regions of the skeleton. In their study, however, impact-loaded athletes (gymnasts) were grouped with weight-bearing athletes (runners and dancers), so a distinction could not be made between these different types of loading regimens. The impact strains inherent to these activities vary 2- to 3- fold and thus any positive effects that many have been realised from loading during impact activity may have been offset by the inclusion of athletes experiencing substantially less magnitude loads. The extremely small sample (n=17) of athletes also made it difficult to establish any conclusive relationships between loading and bone adaptation. In addition, the "impact" loaded group was compared with a group of elite swimmers, who consistently have been shown to have lower BMD than even normal sedentary controls. Age- and size-matched (height and weight) normal sedentary girls would have been a more appropriate control for these types of comparisons.

In summary, there is an obvious lack of studies involving children that have attempted to isolate the effects of specific loading parameters of exercise on BMD.

9.2 Rationale for study

While there is general support of the positive effects of weight-bearing exercise on bone in children, adolescents, and adults, there has been no systematic attempt to isolate and distinguish between the effects of load magnitude, frequency, distribution, volume, or rate of strain application in humans. Experimental evidence in animals suggests that the magnitude and volume of loading may have positive effects on bone adaptation. Evidence for magnitude and volume effects of loading has also been supported through correlative

studies in humans. Interestingly, the little research that has implied that there is a volume and magnitude response to loading has also shown a trend for a ceiling effect for both of these variables.

Many studies have concluded that impact loads associated with gymnastics activity can impart forces of 10-14 times body weight on the skeleton (Hall, 1986; Panzer et al., 1988; Miller & Nissenen, 1987). In addition, the contact forces within the body have been estimated to be as high as 25 times body weight (O'Connor, 1992). It seems appropriate, therefore, to use gymnastics training as a suitable exercise model in the study of bone adaptation to high-impact mechanical loading (magnitude effect). A few studies performed with adult and adolescent gymnasts have concluded that the high impact loading associated with this activity leads to greater and positive bone adaptation as compared to both controls and other athletes who participate in lower impact sports. The effect of high impact loading in prepubescent children has only been investigated in one other study (Dyson et al., 1995), and positive associations were reported between impact magnitude (hours/week) and BMD, at most skeletal regions assessed.

While the effects of impact loading have been assessed commonly at the LS and FN, measured effects at the distal radius have been scarce. The distal radius may be an ideal site to observe the effects of impact loading through gymnastic activity since it is not involved in normal, active weight-bearing as are most other sites commonly measured, and because this region also experiences high-impact loading inherent in training and competition manoeuvres.

No study to date has attempted to directly investigate the differential response of impact loading on trabecular and cortical bone compartments. With new peripheral quantitative computerised tomography technology it is possible to differentiate between these two bone compartments at the distal radius and describe differential effects that may be caused by impact loading.

Additionally, there has been no attempt to establish whether there is a volume response of high-impact loading on bone adaptation in any age group of gymnasts. Lastly, many studies have failed to control for the genetic potential of these athletes, and the possibility of self-selection.

PURPOSE

This study investigated the effects of the magnitude of load on BMD, the effects of the volume of high-impact loading on BMD, and the differential response of trabecular and cortical bone to impact loading. The magnitude effect of loading was determined by comparing BMD of high-impact loaded gymnasts with normal non-impact loaded controls. The volume effect of impact-loading was determined by comparing the BMD of three gymnast groups who participated in different volumes (hours/week) of gymnastic training, over a 1 year period. The effects of diet, habitual physical activity, muscle strength and maternal influence on bone density during prepubescence were also addressed in this investigation.

HYPOTHESES

The following hypotheses were tested in this investigation:

1. Gymnasts would exhibit higher bone densities than normo-active controls at all sites measured, thus exhibiting a magnitude effect of loading on skeletal adaptation;
2. BMD would increase among gymnast groups with increasing levels of gymnastic activity (hours/week), thus exhibiting a volume effect of loading on skeletal adaptation;
3. Within the gymnastic groups, distal radial trabecular bone would be more responsive to impact loading than cortical bone; and
4. The positive effects of impact loading on the skeleton will be evident even after correction for familial influences.

METHODOLOGY

Subjects

Seventy-nine healthy, biological mother(28-50 year-old)-daughter(8-11 year-old) pairs were recruited for the study. The mother-daughter pairs were subdivided into one of five categories depending on the daughter's level of gymnastic involvement: elite gymnasts with >15 hours training per week; high recreation gymnasts with 8-15 hours of training per week; low recreation gymnasts with <8 hours of training per week; normally active controls who received no formal gymnastics training; and normally active height- and weight-matched (to the elite gymnasts) controls who received no formal gymnastics training. Within the club setting, the elite gymnasts were termed provincial-level gymnasts, the high recreation gymnasts were termed regional-level gymnasts, and the low recreation were termed either invitational of recreational gymnasts. For each of the skill levels within the gymnastic club setting, a predetermined level of competency in gymnastic manoeuvres had to be displayed before acceptance was given into a higher level. Identical measurements were made on mothers and daughters.

Study Terminology

In this study, magnitude is defined as the absolute amount of mechanical strain imparted to a given weight-bearing site by typical impact loading gymnastic manoeuvres independent of the frequency of such manouvuers. The magnitude of the strain is dependent largely upon body mass and the product of body mass and displacement.

Volume is defined as the overall amount of impact loading within a prescribed period, and is the product of strain magnitude, frequency, intensity, and duration. In this study, volume is defined as the number of hours per week of gymnastic training. Additionally, this study only addresses the volume of impact loading for the one-year period preceding the bone mineral measurements, since activity at this time would be the primary determinant of extant or current bone mineral status.

Recruitment

Gymnasts were recruited from gymnastics clubs within a one-hour drive of the testing facility. A member of the research team visited the clubs and provided information to club executive members and coaching staff regarding the details of the project, and at a few of the clubs a member of the research team gave a presentation to the gymnasts and their parents. Information pamphlets (Appendix A) were left at each club describing the purpose of the study, measurements to be made, time commitment, and potential risks. Gymnast mother-daughter pairs who felt they met the inclusion criteria and were interested in the study contacted either the project co-ordinator directly by telephone or the club personnel who then gave the names to the co-ordinator. Participants were then contacted by telephone, at which time the details of the study were explained and the research co-ordinator briefly interviewed the mother to ensure that all the inclusion criteria were met. If the mother-daughter pair was still interested in the study an initial appointment for testing was made. Similar procedures were used for control subjects who were recruited through advertisements in local newspapers, radio, and television, by

posting information in public areas thought to be appropriate, and by word-of-mouth (Appendix A).

Measurement Procedures

All subjects came in for testing twice: once to the Department of Nuclear Medicine at Chedoke-McMaster Hospital, and once at a tertiary care osteoporosis clinic. Dual energy x-ray absorptiometry (DXA) data were collected at the Department of Nuclear Medicine. All anthropometric measurements and the peripheral quantitative computerised tomography (pQCT) scan of the non-dominant distal radius were made at the tertiary care osteoporosis clinic. At the beginning of the first session the purpose and procedures of the study were again explained to the participants both verbally by the primary investigator and in writing via both an information leaflet (different for mother and daughter) and a written informed consent (Appendix B). The radiation dose and risk associated with the scanning procedures was also explained and discussed at this time. All procedures were carried out after signed consent was given by both the mother and daughter.

During the first visit, the mother-daughter pair was given all the appropriate questionnaires to complete. If questionnaires were not fully completed on site, they were taken home, completed and returned on the second visit. When the questionnaires were returned, they were reviewed for completion and clarity of responses.

At the end of the second testing session the daughters were given a $10.00 gift certificate in appreciation for their participation, and the mothers were reimbursed for travel and parking expenses.

Ethical Approval and Informed Consent

The study was performed with the approval of the McMaster University Research Advisory Board. Written, informed consent was obtained from both the mother and daughter, following a verbal explanation of the form, during the first visit before any measurements were taken. Subjects were then informed that they had the right to withdraw from the study at any time without penalty.

Inclusion Criteria

Daughters

All gymnasts must have been involved in some level of formal gymnastics training for at least two consecutive years before the testing occurred. Control subjects must never have been involved in any form of gymnastics training at any time in the past. All girls in this study were premenarchal and between the ages of eight and 11 years.

Mothers

All mothers were premenopausal (10 or more menses per year) and the biological mothers of the daughters studied. Gynaecological status was attained with the aid of a medical questionnaire (Appendix C).

Exclusion Criteria

Subjects were excluded from the study if any of the inclusion criteria were not met. In addition, the presence of metabolic disorders known to affect bone status, or the presence of any condition that would negatively affect activity levels (determined from the medical questionnaires) were grounds for exclusion. Sexual maturity status of daughters

was self-determined and confirmed by their mothers from a series of diagrams of breast and pubic hair development based on the criteria of Tanner (1962). The method of maturity self-assessment by Tanner staging has been found to be reliable in previous pediatric research (Duke et al., 1980; Caine & Broekoff, 1987; Matsudo & Matsudo, 1994). Tanner stages were determined and girls who were above Tanner stage 2 (pubic hair) were excluded from the study. Mothers who were either pregnant or lactating were also excluded.

Measures

Bone Mass and Body Composition

Areal bone mineral density (BMD), and projected area (Ap) of the lumbar spine (L2-L4), left proximal femur (femoral neck, greater trochanter, and Ward's triangle), and whole body, along with body composition, were measured by DXA (Hologic 1000W, Waltham, Mass.) for each mother and daughter. Areal BMD is calculated by dividing bone mineral content by the projected area of the bone in the region of interest and is expressed in units of $g \cdot cm^{-2}$. BMD measures were also normalised for differences in bone size utilising equations developed by Katzman et al. (1991). Normalised bone mineral measures were expressed as bone mineral apparent density (BMAD) in units of $g \cdot cm^{-3}$.

Areal BMD measures are problematic, when comparing individuals of different size, because they tend to systematically underestimate bone density in smaller compared to larger individuals. Carter et al. (1992) claimed that normalisation for bone size by the BMAD technique offered an important advantage in cross-sectional studies which

compare different sized subjects. The justification for normalisation for bone size is best exemplified in the investigation of BMD changes during rapid periods of growth. The near linear relationship reported between areal BMD and stature until the age of 13-14 years (Glastre et al., 1990; Bonjour et al., 1991; Kroger et al., 1992), contrasts with the findings of Gilsanz et al. (1988) and Schonau et al. (1993) who measured true volumetric bone density by quantitative computed tomography and described a relatively stable BMD during this period of growth. It can, therefore, be concluded that the inability of DXA areal BMD measures to correct for bone size led to incorrect conclusions concerning the relationship of linear growth and true BMD.

It should be noted that the normalisation procedure utilised in this study was developed from the data of adult subjects, and therefore, the relationships between bone variables may not hold as true in children as in the adults the normalisation was developed with. This procedure, however, has been utilised with success in the pediatric population (Katzman et al., 1991).

The radiation source in the Hologic 1000W consists of an x-ray tube which emits pulses of alternating rays at 70 and 140 kV. Entrance radiation dosage to the subject varies between 2-5 mrem, which is approximately equivalent to one-tenth of a standard chest x-ray (manufacturers specifications). Because each patient received three DXA scans, the cumulative radiation dosage was less than 10 mrem. Normally, one receives 300 mrems of natural background radiation from the environment annually, so the scans represented only a negligible radiation risk to the participants. *In vivo* precision (i.e.

measurement reliability expressed as a coefficient of variation) of DXA has been reported as 1-2%, and *in vitro* accuracy (where accuracy is measured by comparison of DXA density values with those obtained directly from measurements of phantoms or anatomical bone sections) as 3-5% (Alhava, 1991).

For DXA scanning, subjects were instructed to remove all metallic objects such as watches or jewellery and wore only light clothing without metallic objects (e.g. buttons or zippers).

For the lumbar spine scan, subjects were supine with their lower legs placed on a padded box supplied by Hologic, to allow the spine to flatten on the bed (to minimise natural lordosis). The lower legs were placed on the box so that the knees and hips were in approximately 45 degrees of flexion. At this point the operator identified the L5 region and allowed the scan path to proceed upward until L2 was fully displayed. During analysis, the operator, through a computer software program, indicated the vertebrae to be included in the analysis and labelled the vertebrae accordingly.

For the hip scan, subjects were again placed in a supine position with the left foot rotated slightly inward and secured in place with a foot fixation board supplied by the manufacturer. Positioning the foot in such a manner causes the head of the femur to rotate outward to allow for a larger portion of its surface to be measured. Once the participant was correctly positioned, the operator selected a region just lateral to the greater trochanter and allowed the scan to proceed medially until the acetabulum has been fully scanned. For the femoral neck, the operator selected the appropriate region of the

femur (between the greater trochanter and the base of the head of the femur), after which time all other measurements were performed automatically by the computer.

For the whole body scans, the subjects were motionless in a supine position while the densitometer scanned from head to toe in a sweeping motion. At the end of the measurement the operator delineated different regions of the body for further analysis. A phantom tissue bar was included adjacent to the subject for the whole body scans. This allowed the operator to determine tissue composition of various areas of the body, including body fat % and lean body mass.

Peripheral Quantitative Computerized Tomography

Bone mineral density scans of the distal radius were performed by peripheral quantitative computerised tomography (pQCT - Stratec XCT 960, Norland Corporation, WI.). The pQCT allows for measurement of the different compartments of bone found within the radius (total, cortical, and trabecular bone), in addition to selected morphometric (e.g. bone area) and biomechanical (e.g. moment of inertia) measures. The pQCT utilises a single x-ray beam of 45kV producing a radiation dosage of approximately 6 mrem per scan (manufacturers specifications). In vivo precision for adults is reportedly ± 5, 3, and 9 $mg \cdot cm^{-3}$ for total, trabecular, and cortical density measures, respectively (manufacturer's specifications). An unpublished pilot study in this laboratory reported in vivo reproducibility of 7.9%, 5.8%, and 14.7% (CV's) for total, trabecular and cortical density, respectively for children and 3.8%, 2.1%, and 6.0%, respectively, for adults.

Forearm length, from the tip of the olecranon process to the most distal point of the ulnar styloid with the arm flexed at 90 degrees, was measured to the closest millimetre with a flexible plastic ruler supplied by the manufacturer. The pQCT was then adjusted to the length of the subject's forearm. Participants were seated next to the scanner and inserted their non-dominant arm into the aperture. At this time a short "scout" scan of variable length (between 15 and 30 mm.) was taken in order to determine the location of the distal-most point of the radius. Once this point was attained the scout scan was halted and the CT scan was initiated at a proximal point 4% of the length of the forearm in the mothers and 6% of the length of the forearm in the daughters. It was necessary to add the extra 2% in length for the daughters' arms to ensure that measurements were made beyond the undermineralized growth plate zone, which varied in thickness between subjects dependent on their bone age. The scout scan and CT scans were obtained with the shoulder abducted and elbow flexed at 90 degrees, and with the hand pronated. Scans were performed by two experienced technicians and analysed by one technician. Measurement included total, trabecular, and cortical volumetric BMD ($mg \cdot cm^{-3}$), and axial moment of inertia (a measure of mechanical strength).

Anthropometric Measurements.

Body height was determined with subjects in stocking feet, by a free-standing Harpenden stadiometer accurate to 0.1cm. Subjects stood erect and inhaled deeply while their height was taken. Body mass was measured on a standard balance scale (accurate to

0.1kg) with subjects wearing only light clothing and without shoes. Body composition was determined by DXA.

Local Muscle strength.

Grip strength was measured for all subjects with a standard dynamometer (Lafayette Instrument Co.) for both hands, and the best of two trials was used as the criterion maximum grip strength. There was at least a one-minute rest given between repeat tests of the same hand. The dynamometer was adjusted by the examiner to fit each hand correctly (held between the distal phalangeal joints and the proximal thumb joint), to ensure a maximum grip and standardisation of positioning between subjects. Participants were also asked to indicate their dominant leg (preferred leg to kick with) and arm (same as writing hand).

Questionnaires

Several questionnaires were administered to both the mother and daughter. An extensive questionnaire (Appendix C) that detailed past and present dietary, medical health, exercise, and gynaecological status was given to all participants. These medical/health questionnaires were slightly different for mother and daughter in that the daughters' questionnaire did not examine gynaecological or reproductive status.

There were two questionnaires of physical activity level. One, which was incorporated into the medical/health questionnaire, examined the level of physical activity of the subject in the past 2 weeks. A second, more extensive, questionnaire required subjects to estimate participation in all physical activities carried out within a one year

period (Appendix D). There were several activities listed on both questionnaires, with blank spaces on the bottom of the forms to allow for additional activities. This information was tabulated and expressed as weight bearing hours of activity and non-weight bearing hours of activity for the measured periods.

Dietary Analysis

Current dietary intake was determined by a three-day food diary (Appendix E) whereby subjects recorded all the food and liquids consumed over two weekdays and one weekend day. Subjects were given written and verbal instructions on the proper completion of the diaries and were encouraged to be as accurate as possible when identifying and quantifying the foods ingested. Subjects were instructed to maintain their normal dietary habits during the collection period. A nutritional analysis program (Nutrient Analysis/Dietary Programs, version 4.26.2 - June 1994 by Elizabeth Warwick, UPEI) was used to estimate the total number of calories, protein, fat, carbohydrates, vitamin D, and calcium that was ingested during the three day period. All dietary analyses were performed by one researcher.

Statistical Analyses

Pearson product-moment correlations were determined between mothers and daughters for selected anthropometric, bone density and content, and bone morphometric variables. Forward stepwise multiple regression analyses were computed to determine the relationship between bone density and bone morphological characteristics and selected descriptive characteristics of subjects. Differences between groups for all variables were

analysed by ANOVA (1 level, 1 factor). ANCOVA was also used to determine the differences between groups for BMD and BMAD. Covariates in the ANCOVA included body mass, lean body mass, height, age, and the mother's bone mineral measure. All bone measurements for the girl gymnasts were regressed (univariate regression) against number of hours of gymnastic training to determine the relationship between training volume per week and bone mineral status. For these analyses, data were collapsed across groups of gymnasts.

Pearson product-moment correlation analyses were used to determine the relationship between gymnastic training volume (hours/week) and adaptations of specific cortical and trabecular bone components at the wrist. The bone compartment with the highest positive correlations was deemed to have been the most influenced by the impact loading.

Differences between groups for all analyses and the strength of the relationships among variables were considered significant at $p < 0.05$. A Tukey Honest Significant Difference post hoc analysis was performed to determine differences between groups.

RESULTS

PHYSICAL CHARACTERISTICS

Daughters

Physical characteristics for all gymnastic and control groups are presented in Table 6.

Table 6. Physical Characteristics - Daughters

Group	n	Age (years)	Height (cm)	Mass (kg)	LBM	Body Fat (%)
Elite gymnasts (E)	19	10.02[b] (0.78)	130.81[c,d] (4.25)	27.15[c,d] (2.82)	22.99[c,d] (2.19)	15.23[c,d,e] (1.83)
High recreation gymnasts (HR)	14	8.69[a,d] (0.84)	126.14[c,d,e] (7.96)	25.44[c,d] (3.56)	21.11[c,d] (2.96)	17.01[c,d] (1.56)
Low recreation gymnasts (LR)	15	9.56 (0.87)	136.89[a,b] (4.52)	32.99[a,b,d,e] (5.79)	25.80[a,b,e] (20.24)	21.23[a,b,d] (4.20)
Controls (C)	16	9.96[b] (0.90)	142.25[a,b] (7.14)	38.55[a,b,c,e] (4.87)	28.50[a,b,e] (2.89)	25.76[a,b,c,e] (4.73)
Matched controls (M)	15	9.76 (0.80)	136.33[b] (5.51)	26.95[c,d] (3.31)	21.88[c,d] (2.87)	18.79[a,d] (3.72)

Values represent mean and (SD)
Superscripts indicate a significant ($p<0.05$) mean difference between groups as follows:
[a]significantly different from E
[b]significantly different from HR
[c]significantly different from LR
[d]significantly different from C
[e]significantly different from M

The HR group was significantly younger than both the E and C groups. There were no other significant differences in age among any of the other groups. The C group was significantly heavier than all other groups and the LR group was significantly heavier than the E, HR, and M groups. There were no other significant body mass differences among the

groups. LBM displayed the same pattern as body mass with the exception that the C and LR groups were not significantly different from each other.

The E and HR groups were both shorter than the LR and C groups. In addition, the HR group was significantly shorter than the M group. There were no additional differences in stature between the groups. The E and HR groups had significantly less body fat than the LR and C groups. The M group had a significantly higher percent body fat than the E group. The LR group had a significantly lower percent body fat than the C group, but had a significantly higher percent body fat than the M group. There were no additional differences in percent body fat between the groups.

The pubertal status of the girls is summarized in Table 7.

Table 7. Pubertal Status of Daughters (based on Tanner stages).

	Mean Tanner Stage Score			
	Breast		**Pubic Hair**	
	mean	SD	mean	SD
Elite gymnasts	1.053†	0.229	1.000	0.000
High recreation gymnasts	1.071	0.267	1.000	0.000
Low recreation gymnasts	1.267	0.458	1.200	0.414
Controls	1.563†	0.814	1.313	0.602
Matched controls	1.200	0.414	1.133	0.352

† indicates a significant mean difference between groups at $p<0.05$

When pubertal status was determined by breast development, the mean score for the C group was significantly higher than the E group. There were no additional differences among

the groups in terms of breast development. There were no significant differences among groups, however, when pubertal status was expressed as a function of pubic hair development.

Mothers

The physical characteristics for all groups of mothers are presented in Table 8.

Table 8. Physical Characteristics - Mothers.

Group	n	Age (years)	Height (cm)	Mass (kg)	LBM	Body Fat (%)
Elite gymnasts	19	38.55 (3.10)	159.28† (5.17)	60.50 (10.05)	44.07 (3.10)	26.63 (5.69)
High recreation gymnasts	14	37.54† (5.58)	164.41 (5.25)	67.44 (15.89)	47.98 (6.82)	27.40 (7.28)
Low recreation gymnasts	15	37.62 (3.12)	165.20† (5.23)	69.41 (16.58)	49.54 (7.43)	27.59 (5.98)
Controls	16	42.57† 4.32)	162.31 (6.68)	68.68 (12.47)	46.28 (6.10)	30.81 (6.38)
Matched controls	15	39.00 (5.43)	161.99 (4.35)	62.45 (6.87)	45.77 (3.77)	26.39 (4.48)

Values represent mean and (SD)
† indicates a significant mean difference between groups at $p<0.05$.

There were no statistically significant differences among the groups in terms of body mass, LBM, or % body fat. Control mothers were significantly older than mothers of HR gymnasts and mothers of the LR gymnasts were significantly taller than mothers of the E gymnasts.

CORRELATIONAL ANALYSES

Correlational Analyses of All Mother-Daughter Pairs

A summary of the correlational analyses between mother-daughter pairs for selected anthropometric, bone mineral density, and bone morphometric variables is presented in Table 9. Significant positive correlations were found between daughters and mothers for all variables except height, FNBMAD, trochanteric BMD, LSBMAD, radial trabecular BMD, and radial cortical BMD.

Correlational Relationships with Gymnastics Training Volume - Gymnasts Only

Significant positive correlations were obtained between the number of hours spent in gymnastic training in the past year and FNBMD, FNBMAD, trochanteric BMD, Ward's triangle BMD, LSBMAD, and radial cortical CSA. Hours of gymnastic training was significantly negatively correlated with weight, LBM, percent body fat, and height. At the distal radius, the number of hours of gymnastic training in the past year was more significantly correlated with trabecular ($r = 0.373$; $p<0.01$) than cortical BMD ($r = 0.301$; $p<0.05$). Complete correlational matrices for the gymnasts and their mothers can be found in Appendix F.

Correlational Relationships with General Weight Bearing Activity - Gymnast and Control Groups Combined

The number of weight bearing hours per year was significantly correlated with FNBMD ($r = 0.322$, $p>0.05$), trochanteric BMD ($r = 0.398$, $p<0.01$), TBMAD ($r = 0.411$, $p<0.01$), total radial BMD ($r = 0.560$, $p<0.001$), radial trabecular BMD ($r = 0.380$, $p<0.01$), and radial

cortical BMD ($r = 0.569$. $p<0.001$). The number of weight bearing hours over a two-week period was significantly correlated with FNBMD ($r = .377$, $p<0.01$), trochanteric BMD ($r = 0.491$, $p<0.001$), Ward's triangle BMD ($r = 0.318$, $p<0.05$), LSBMD ($r = 0.305$, $p<0.05$), LSBMAD ($r = 0.507$, $p<0.001$), WBBMAD ($r = 0.434$, $p<0.001$), total radial BMD ($r = 0.650$, $p<0.001$), radial trabecular BMD ($r = 0.489$, $p<0.001$), and radial cortical BMD ($r = 0.616$, $p<0.001$). Non-weight bearing hours per year was not significantly correlated with any bone measure.

Table 9. Daughter-Mother Correlations for Measured Variables - All Groups.

Variable	Pearson product-moment correlation (r)	Significance
WEIGHT	0.441	p<0.001
LBM	0.352	p<0.01
HEIGHT	0.175	NS
% BODY FAT	0.539	p<0.001
FNBMD	0.280	p<0.01
FNBMAD	0.102	NS
TROCHANTER BMD	0.200	NS
WARD'S TRIANGLE BMD	0.374	p<0.01
LSBMD	0.340	p<0.01
LSBMAD	0.124	NS
WHOLE BODY BMD	0.287	p<0.01
WHOLE BODY BMAD	0.270	p<0.01
TOTAL RADIAL BMD	0.285	p<0.01
RADIAL TRABECULAR BMD	0.128	NS
RADIAL CORTICAL BMD	0.174	NS
TOTAL RADIAL CROSS-SECTIONAL AREA	0.494	p<0.001
RADIAL TRABECULAR CROSS-SECTIONAL AREA	0.431	p<0.001
CORTICAL TRABECULAR CROSS-SECTIONAL AREA	0.549	p<0.001
RADIAL MOMENT OF INERTIA	0.529	p<0.001

NS=not statistically significant

REGRESSION ANALYSES

The results of all regressional analyses for the bone mineral density and morphometric variables are summarized in Tables 10 and 11.

Table 10. Regression Results - All Daughters.

	Percent of Variance Explained by Variable (r^2)						Cumul Exp.
Measure	Age	Weight	LBM	Height	GS	MVS	**Var.%**
FNBMD	4.621					5.898	10.519
FNBMAD				10.519*			10.519
Troch BMD	7.085				5.230		12.315
Ward's BMD					5.679	12.076*	17.755
LSBMD						13.593**	13.593
LSBMAD				8.245		4.670	12.915
Whole Body BMD			25.608**			3.864	29.472
Total Radial BMD	6.349			24.527**		4.811	35.687
Trabecular Radial BMD				7.407			7.407
Cortical Radial BMD	14.263**			12.133*			26.396
Total Radial X-Sectional Area			13.383**			23.828**	28.742
Trabecular Radial X-Sectional Area			20.216**			15.359**	35.575
Cortical Radial X-Sectional Area			4.001		4.721	30.043**	38.765
Radial Moment Of Inertia			12.191**			27.013**	39.191

Blank cells = did not contribute significantly to explained variance.
GS = grip strength.
MVS = mother's individual variable score for analysed measure.
Cuml. Exp. Var.% = cumulative explained variance in percent
All results are $p<0.05$, except *=$p<0.01$ and **=$p<0.001$.

For the daughters, the mother's bone measures consistently accounted for the greatest proportions of the explained variance in the dependent variable compared to the other independent variables, with significant contributions ranging from 12.07% to 30.04%. LBM was the second most consistent significant predictor, followed by height and age. The various combinations of independent variables accounted for between 7.4% to 39.1% of the total explained variance in the daughter's bone measures.

Table 11. Regression Results - Gymnasts Alone.

	Percent of Variance Explained by Variable						Cumul. Ex. Var.
Measure	Age	Weight	LBM	GS	MVS	Hours	(%)
FNBMD				10.810		21.517**	32.327
FNBMAD				16.527**	8.158		24.685
Troch BMD				8.748		25.993**	34.741
Ward's BMD					9.676		9.676
LSBMD	6.889				18.674*		25.563
LSBMAD						15.950*	15.950
Whole Body BMD			35.039**	7.571		8.775	51.385
Total Radial BMD		16.766*			12.970		29.736
Trabecular Radial BMD						13.929*	13.929
Cortical Radial BMD	9.034	12.004					21.038
Total Radial X-Sectional Area			10.785		26.941**	10.194	47.920
Trabecular Radial X-Sectional Area			21.195*		10.756	7.301	39.252
Cortical Radial X-Sectional Area			5.765		34.911**	10.450*	51.126
Radial Moment Of Inertia			10.071*		29.929**	8.599	48.599

Blank cells = did not contribute significantly to explained variance; GS = grip strength.
MVS = mother's individual variable score for analysed measure.
All results are $p<0.05$, except *=$p<0.01$ and **=$p<0.001$.

When only the gymnasts' data were used for regression analysis, hours of gymnastics training consistently accounted for the greatest proportion of the explained variance in the dependent variables, with significant contributions ranging from 10.4% to 25.9%. The mother's bone measure was the second most consistent independent variable, followed by

LBM, weight, and grip strength. The various combinations of independent variables accounted for between 9.6% to 51.4% of the total explained variance in the daughter's bone measures.

Univariate Analyses

Hours of gymnastic training was regressed separately against all bone measures. Hours of gymnastic training accounted for significant variance in FNBMD ($r^2 = 0.215$, $p<0.001$), FNBMAD ($r^2 = 0.096$, $p<0.05$), trochanteric BMD ($r^2 = 0.260$, $p<0.001$), LSBMAD ($r^2 = 0.159$, $p<0.01$), total radial BMD ($r^2 = .099$, $p<0.05$), radial trabecular BMD ($r^2 = 0.139$, $p<0.001$), radial cortical BMD ($r^2 = 0.090$, $p<0.05$), and radial cortical CSA ($r^2 = 0.103$, $p<0.05$).

All regression analyses performed can be found in Appendix G.

ANOVA ANALYSES

Grip Strength

Results of grip strength measurements for daughters and mothers for both right and left hands are given in Table 12.

Table 12. Mean Grip Strength of Mother and Daughter Groups.

Group	Daughter-Left	Daughter-Right	Mother-Left	Mother-Right
	Grip Strength (kg)	Grip Strength (kg)	Grip Strength (kg)	Grip Strength (kg)
Elite gymnasts	16.34 (2.74)	16.82 (2.86)	32.53† (5.69)	36.47 (6.40)
High rec. gymnasts	15.79 (2.21)	16.18 (2.55)	36.50 (4.77)	39.04 (4.08)
Low rec. gymnasts	17.90 (2.52)	19.03 (2.94)	37.83† (7.40)	40.43† (5.50)
Controls	17.34 (3.23)	18.25 (3.48)	33.09 (6.15)	34.00† (5.50)
Matched controls	17.80 (3.83)	18.40 (3.64)	32.40 (5.00)	35.07 (5.59)

Values represent means and (SD).
† indicates statistically significant difference at $p<0.05$.

There were no statistically significant main effects for left or right hand and no differences in grip strength among groups for daughters. Left hand grip strength was significantly greater for the LR compared to the E mothers, and right hand grip strength was greater for LR versus C mothers. No other differences were evident among groups for grip strength.

DXA Bone Density Measures

Hip Measures.

All DXA measures for the hip region are displayed in Figures 1-4 for the daughters.

Figure 1. Effect of various levels of high impact loading on FNBMD of prepubescent girls.

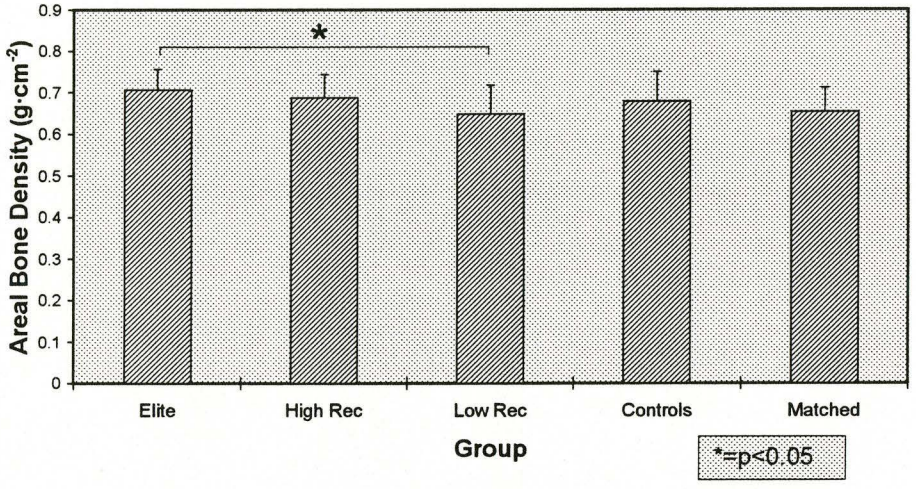
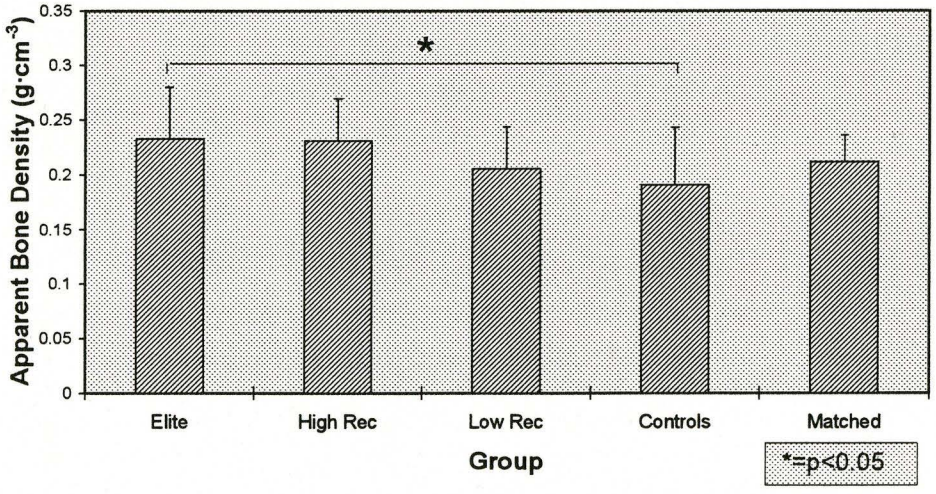


Figure 2. The effect of various levels of high impact loading on FNBMAD of prepubescent girls.



FNBMD was significantly higher in the E versus the LR gymnasts group. There were no other differences among groups for FNBMD (Figure 1). When FNBMD was corrected for bone size and expressed as apparent BMD (BMAD) the E group had significantly higher density than the C group (Figure 2).

Figure 3. The effect of various levels of high impact loading on Ward's Triangle BMD of prepubescent girls.

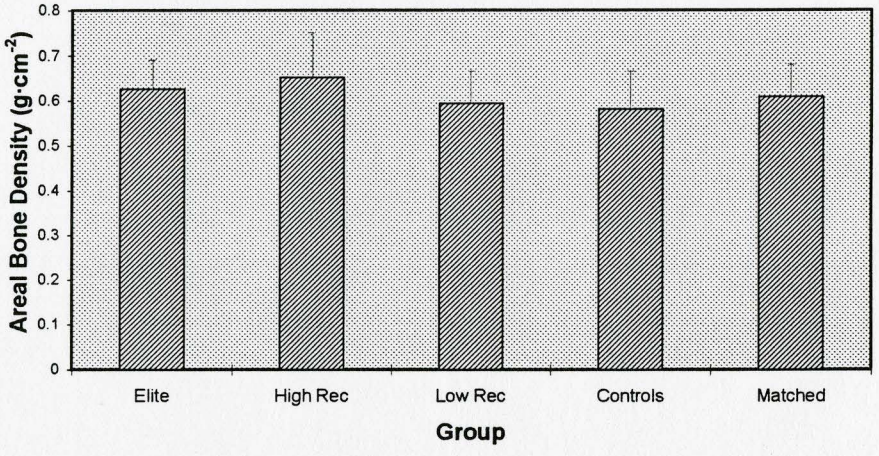
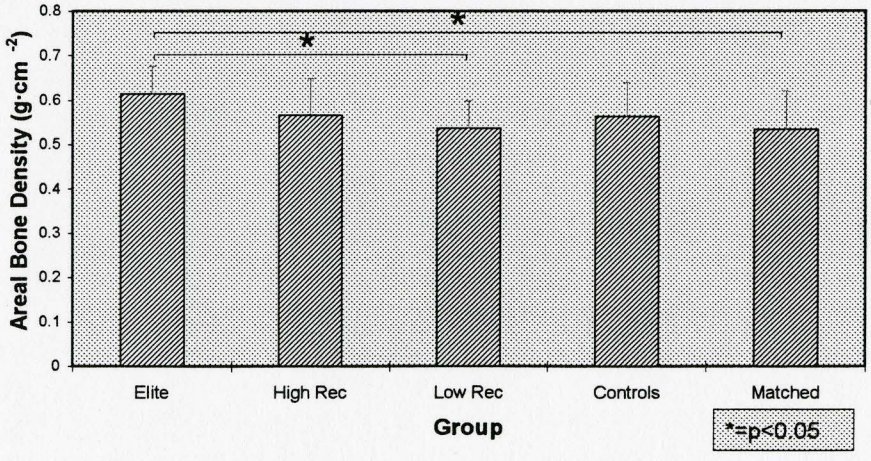


Figure 4. The effect of various levels of high impact loading on trochanter BMD of prepubescent girls.



There were no significant differences among groups of daughters for Ward's triangle BMD (Figure 3). The E gymnasts had significantly higher trochanteric BMD than the LR gymnasts and size-matched controls (Figure 4). There were no other significant differences in trochanteric BMD among groups.

DXA measures for the hip region for the mothers is summarised in Table 13.

Table 13. DXA Measures for Hip Region in Mothers.

		Elite	High Rec	Low Rec	Control	Matched
FNBMD ($g \cdot cm^{-2}$)	mean	0.853	0.867	0.827	0.832	0.839
	SD	0.099	0.097	0.110	0.087	0.079
FNBMAD ($g \cdot cm^{-3}$)	mean	0.203	0.174	0.160	0.198	0.190
	SD	0.073	0.027	0.042	0.041	0.024
TROCH BMD ($g \cdot cm^{-2}$)	mean	0.717	0.719	0.712	0.775	0.713
	SD	0.084	0.078	0.089	0.187	0.082
WARD'S BMD ($g \cdot cm^{-2}$)	mean	0.681	0.716	0.667	0.641	0.690
	SD	0.113	0.114	0.119	0.101	0.087

There were no statistically significant differences for any of the hip measures among groups of mothers.

Lumbar Spine Measures.

Lumbar spine BMD and BMAD of the daughters are presented in Figures 5 and 6, respectively.

Figure 5. The effect of various levels of high impact loading on LSBMD of prepubescent girls.

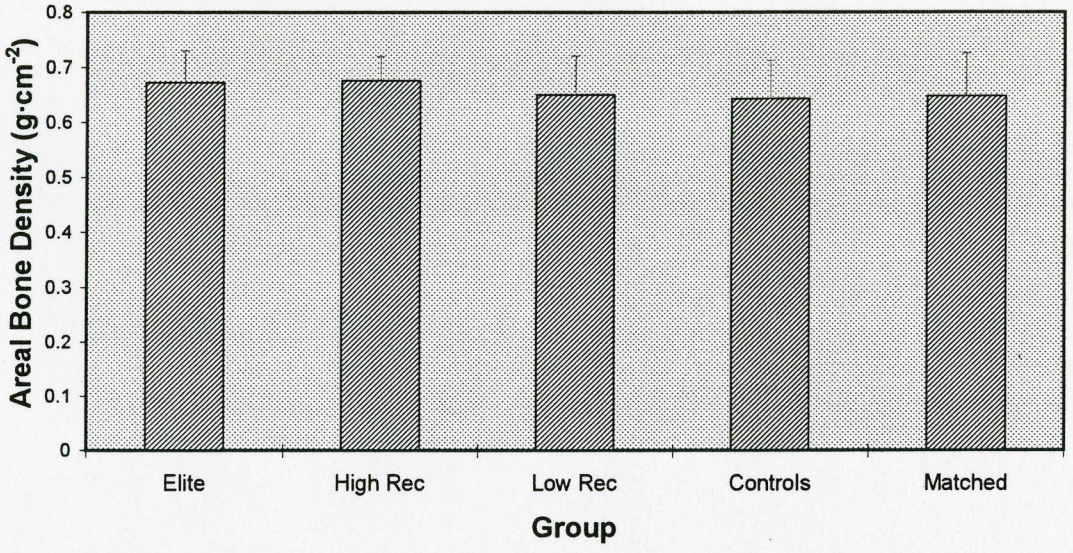
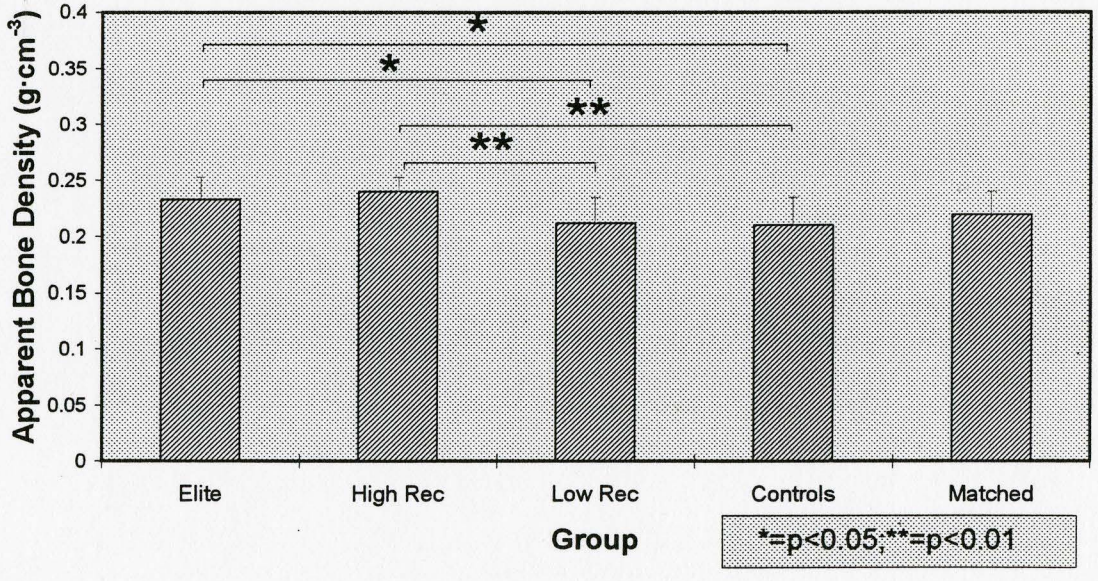


Figure 6. The effect of various levels of high impact loading on LSBMAD of prepubescent girls.



There were no significant differences among groups of daughters for LSBMD (Figure 5.). Both the E and the HR groups had significantly higher LSBMAD than the LR, and C groups (Figure 6). There were no other significant differences among groups for LSBMAD.

Lumbar spine bone mineral measures for the mothers are presented in Table 14. There were no significant differences among groups for either LSBMD or LSBMAD.

Table 15. Mean Lumbar Spine Measures - Mothers.

		Elite	High Rec	Low Rec	Control	Matched
LSBMD ($g \cdot cm^{-2}$)	mean	1.089	1.105	1.102	1.090	1.152
	SD	0.100	0.061	0.149	0.140	0.172
LSBMAD ($g \cdot cm^{-3}$)	mean	0.297	0.286	0.288	0.288	0.301
	SD	0.026	0.013	0.033	0.036	0.042

Whole Body Measures

Whole body BMD and BMAD for the girls are presented in Figures 7 and 8, respectively.

Figure 7. The effect of various levels of high impact loading on whole-body BMD of prepubescent girls.

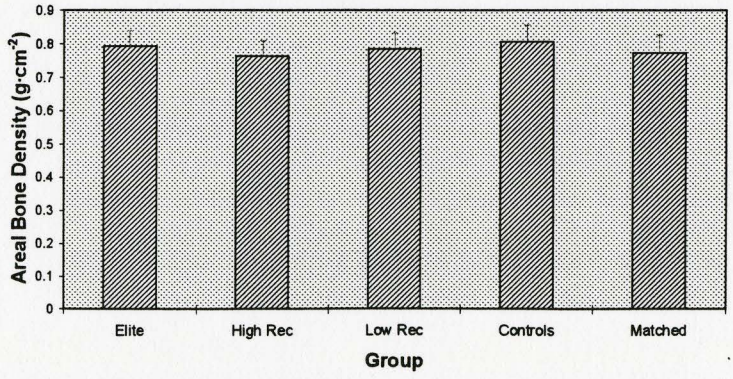
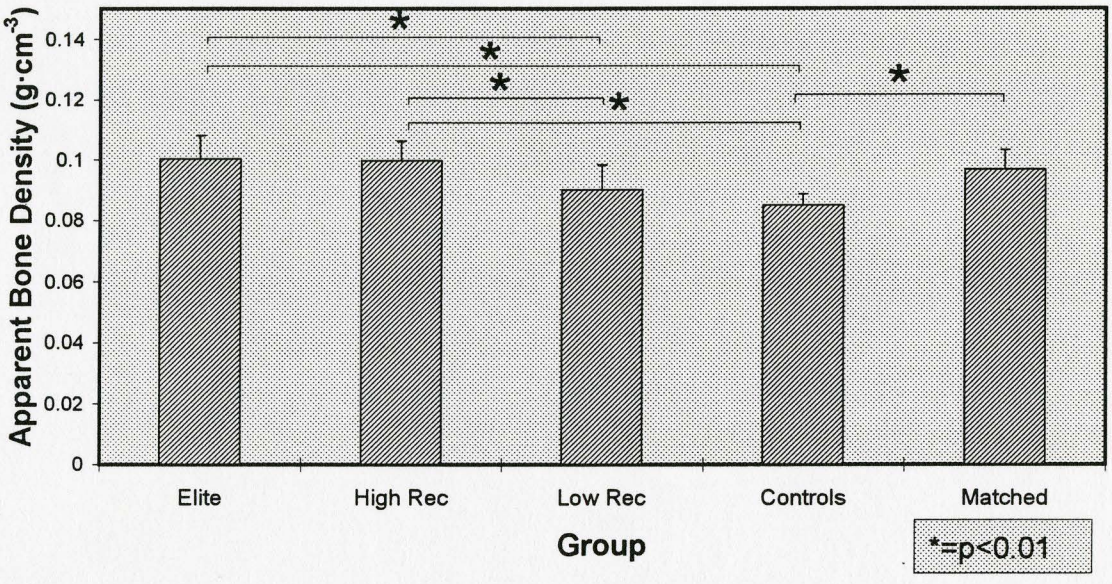


Figure 8. The effect of various levels of high impact loading on whole-body BMAD of prepubescent girls.



There were no statistically significant differences in whole body BMD among the groups of daughters. After correcting for bone size, BMAD was significantly greater in the E and HR groups than the LR and C groups, and the M group had significantly higher BMAD than the C group.

Whole body measures for the mothers are summarized in Table 15. There were no significant differences among groups for either whole body BMD or BMAD.

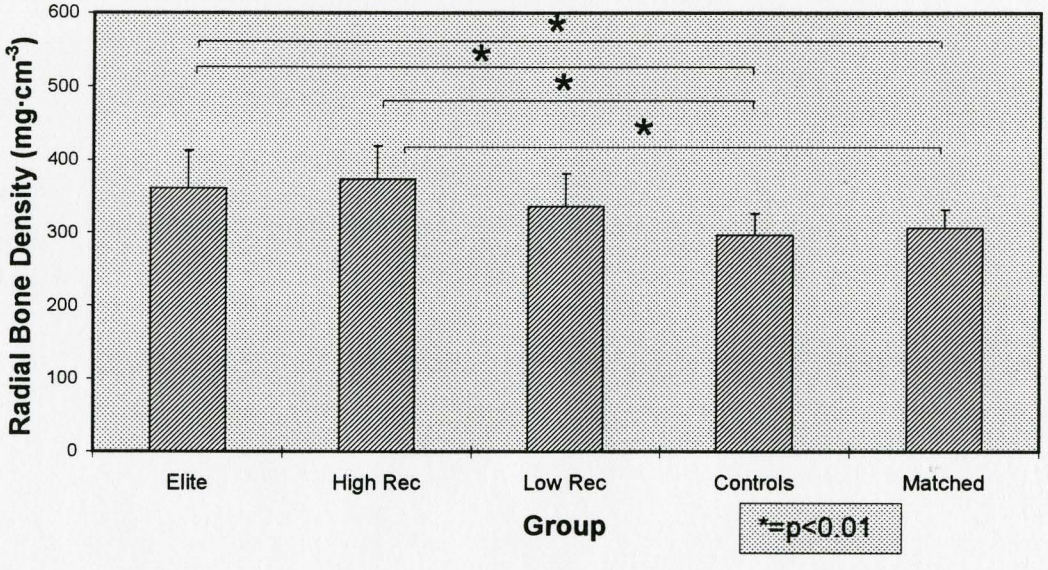
Table 15. Mean Whole Body Bone Mineral Measures - Mothers.

		Elite	High Rec	Low Rec	Control	Matched
BMD ($g \cdot cm^{-2}$)	mean	0.792	0.764	0.785	0.807	0.773
	SD	0.047	0.046	0.047	0.049	0.054
BMAD ($g \cdot cm^{-3}$)	mean	1.101	1.107	1.099	1.099	1.095
	SD	0.053	0.071	0.064	0.050	0.056

Radial Bone Mineral Density

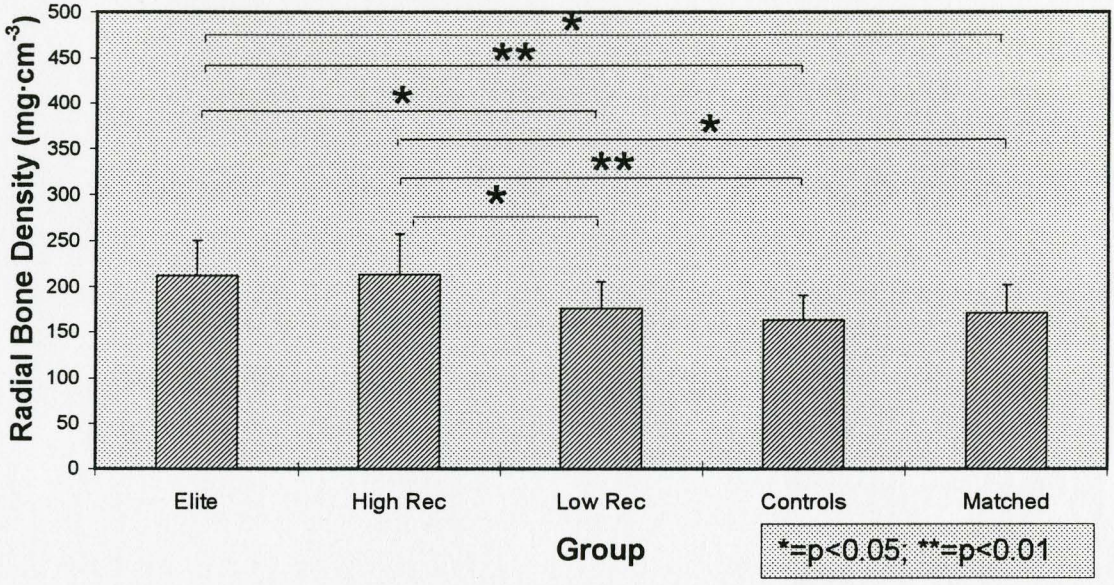
Bone mineral measures of the distal radius for daughters are summarised in Figures 9. through 11.

Figure 9. The effect of various levels of high impact loading on total radial BMD of prepubescent girls.



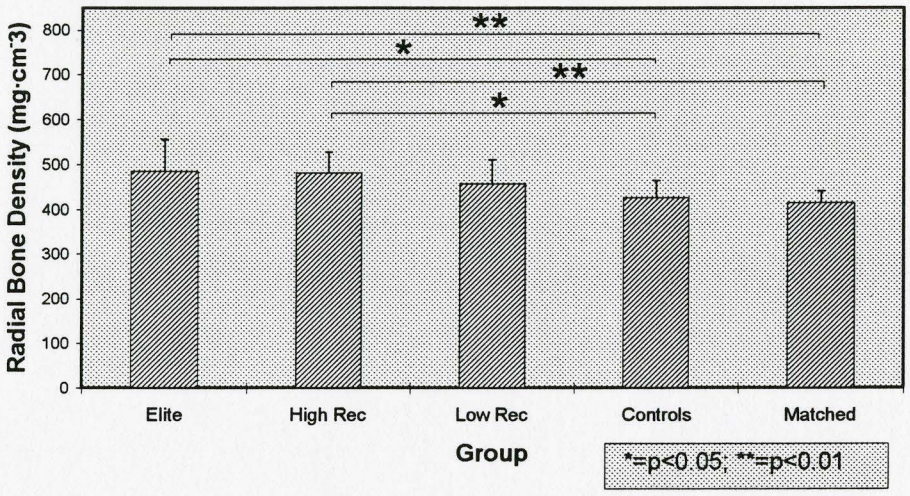
The E and HR groups had significantly higher total BMD than both the C and M groups (Figure 9.). There were no significant differences among any of the groups of gymnasts, and no difference among LR, C, and M groups.

Figure 10. The effect of various levels of high impact loading on radial trabecular BMD of prepubescent girls.



Radial trabecular BMD was significantly higher in the E and HR groups than all other groups (Figure 10.). There were no significant differences among E and HR groups and no differences among LR, C, and M groups.

Figure 11. The effect of various levels of high impact loading on radial cortical BMD of prepubescent girls.



Radial cortical BMD was significantly higher in the E and HR groups compared to the C and M groups (Figure 11). There were no differences among the gymnastics groups, or between C and M groups.

Table 16 summarizes the BMD measures groups of the mothers at the distal radius as determined by pQCT.

Table 16. pQCT BMD Measures - Mothers.

	Group	Elite	High Rec	Low Rec	Control	Matched
Total Radial BMD ($mg \cdot cm^{-3}$)	mean	398.172	406.371	411.193	410.150	412.357
	SD	64.156	81.412	70.757	61.824	50.913
Trabecular BMD ($mg \cdot cm^{-3}$)	mean	191.283	178.510	196.407	183.056	191.571
	SD	41.729	49.057	36.038	38.454	44.734
Cortical BMD ($mg \cdot cm^{-3}$)	mean	628.894	634.221	630.173	639.056	641.064
	SD	88.074	92.871	98.087	84.454	71.785

There were no significant differences found among the groups of mothers for any of the BMD measures at the distal radius.

Radial Morphological and Biomechanical Variables

Statistically significant mechanical and morphometric measures of the distal radius for the daughters are shown in Figures 12 through 15.

Figure 12. Total radial cross-sectional area of prepubescent girls

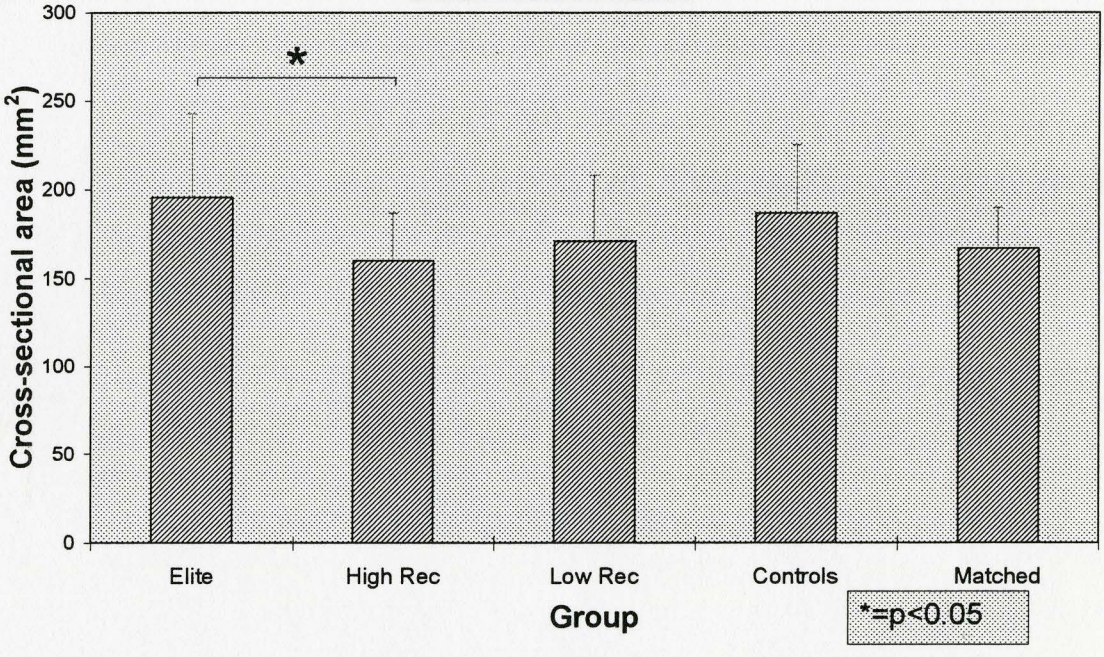


Figure 13. Radial trabecular cross-sectional area of prepubescent girls.

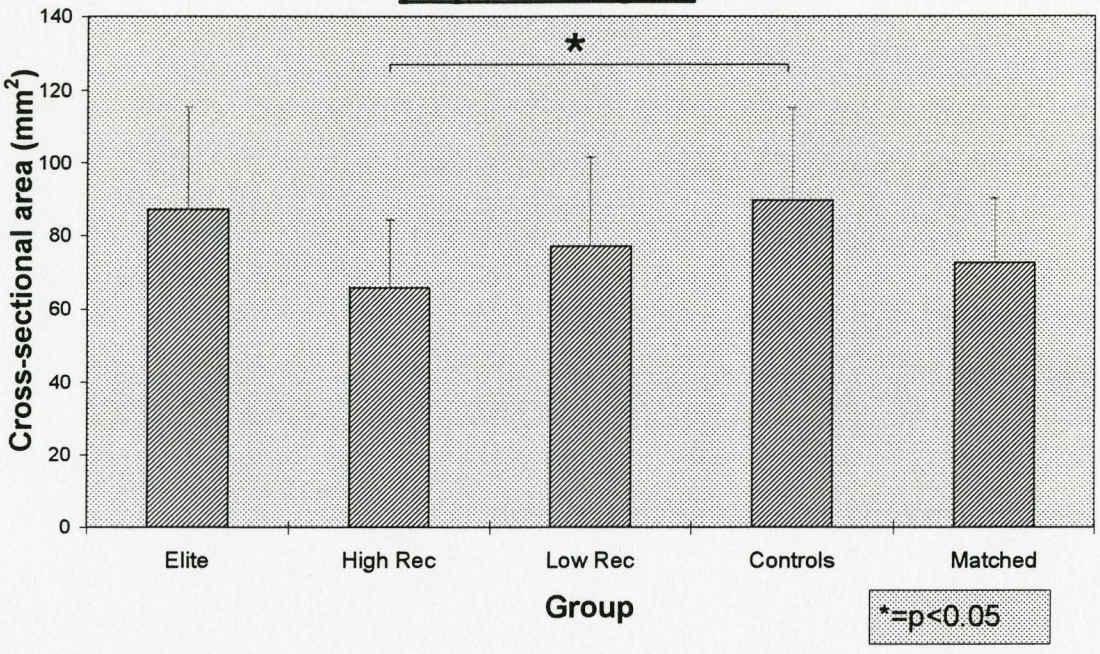


Figure 14. Radial cortical cross-sectional area of prepubescent girls

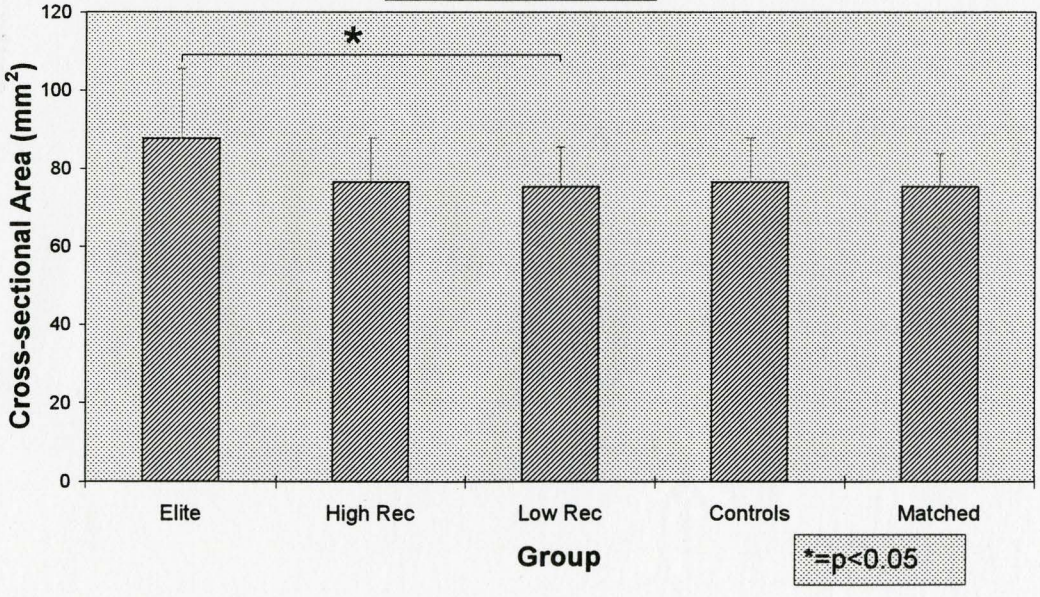
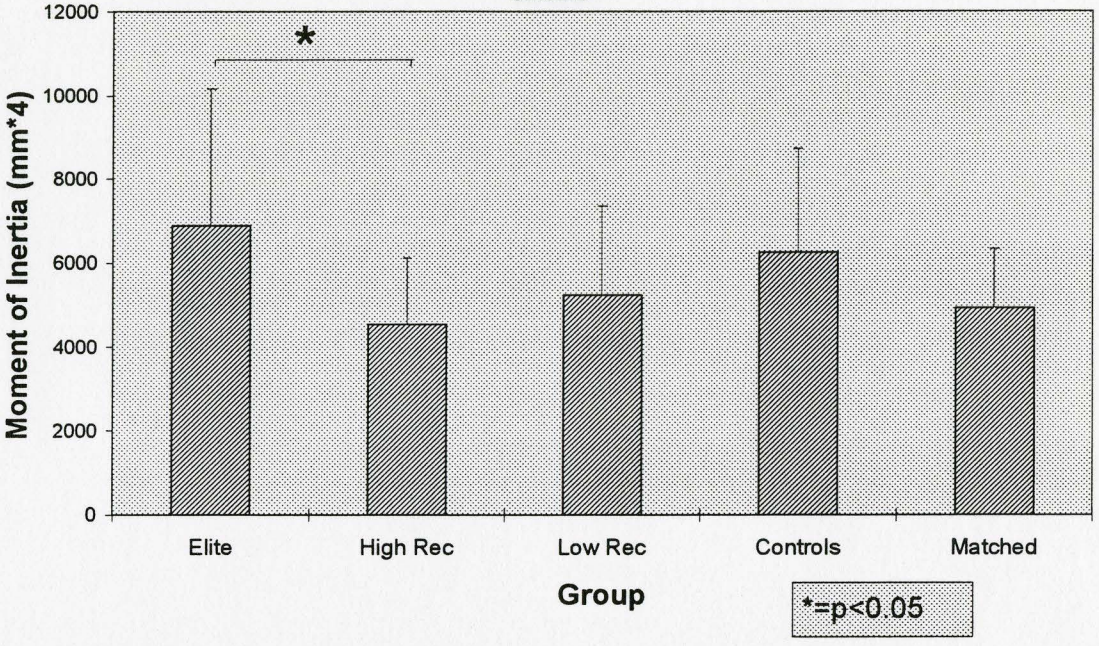


Figure 15. Radial moment of inertia of prepubescent girls.



Radial trabecular CSA was significantly larger in C compared to HR gymnasts, and E had significantly larger radial cortical CSA then the LR gymnasts. The E group had a significantly greater total radial CSA and moment of inertia than the HR group (Figures 12 and 15). There were no other significant differences for either of the variables among other groups.

The mothers mechanical and morphological characteristics of the distal radius are presented in Table 17.

Table 17. pQCT Mechanical and Morphometric Measures - Mothers.

	Group	Elite	High Rec	Low Rec	Control	Matched
Total Radial Area (mm^2)	mean	293.970	281.637	284.745	267.646	286.3
	SD	69.580	39.324	37.199	57.872	29.5
Trabecular Area (mm^2)	mean	147.903	143.070	147.897	134.258	144.5
	SD	45.569	31.368	28.347	41.977	23.7
Cortical Area (mm^2)	mean	117.154	110.920	109.326	106.278	114.0
	SD	20.199	10.252	10.117	12.851	6.8
Moment of Inertia (mm*4)	mean	17,139.400	15,096.800	15,116.400	13,980.900	15,542.2
	SD	10,988.600	4,128.110	3,738.530	6,562.720	3,111.4

There were no statistically significant differences in bone morphometric or biomechanical measures at the distal radius among the groups of mothers.

Habitual Physical Activity

Annual Activity Levels

Results for non-weight-bearing and weight-bearing hours of activity, inclusive of gymnastic training time, are presented for the daughters in Tables 18 and 19, respectively.

Table 18. Annual Non-weight-bearing Activity - Daughters.

	Non-weight-bearing Activity (hours/year)	
	mean	SD
Elite Gymnasts	37.750	20.162
High Recreation Gymnasts	43.636	57.366
Low Recreation Gymnasts	43.500	44.588
Controls	43.950	32.862
Matched Controls	44.100	24.365

There were no significant differences observed between groups for non weight-bearing hours of activity per year.

Table 19. Annual Weight-bearing Activity - Daughters.

	Weight-bearing Activity (hours/year)	
	mean	SD
Elite Gymnasts	410.127†,‡	378.824
High Recreation Gymnasts	450.602¥,ß	216.439
Low Recreation Gymnasts	246.450	157.235
Controls	137.792†,¥	95.491
Matched Controls	118.239‡,ß	54.477

†,‡,¥,ß indicate a significant mean difference between groups at $p<0.05$

Annual weight-bearing activity differed significantly between groups with the E and HR groups having a significantly greater number of hours of weight bearing activity than both of the control groups. There were no other significant differences among groups for annual weight-bearing activity.

The results of the mothers annual non-weight-bearing and weight-bearing activity are summarized in Tables 20 and 21, respectively.

Table 20. Annual Non-weight-bearing Activity - Mothers.

	Non-weight-bearing Activity (hours/year)	
	mean	SD
Elite Gymnasts	67.632	46.668
High Recreation Gymnasts	28.589	38.213
Low Recreation Gymnasts	34.838	72.396
Controls	53.791	47.687
Matched Controls	44.311	44.220

Table 21. Annual Weight Bearing Activity - Mothers.

	Weight Bearing Activity (hours/year)	
	mean	SD
Elite Gymnasts	147.466	194.763
High Recreation Gymnasts	122.907	64.708
Low Recreation Gymnasts	205.611	117.493
Controls	244.370	260.516
Matched Controls	183.244	99.495

There were no significant differences for either annual non-weight bearing or annual weight bearing activity between the groups of mothers.

Two-week Activity Levels

The results of the two-week activity assessments for the daughters and mothers are found in Tables 22 and 23, respectively.

Table 22. Two-week Activity - Daughters.

	Weight Bearing Activity (hours/two weeks)	
	mean	SD
Elite Gymnasts	$41.011^{\dagger,\ddagger,¥}$	14.654
High Recreation Gymnasts	$26.818^{ß}$	3.459
Low Recreation Gymnasts	$11.622^{\dagger}$	5.842
Controls	$5.029^{\ddagger,ß}$	5.223
Matched Controls	$16.497^{¥}$	33.958

$^{\dagger,\ddagger,¥,ß}$ indicate a significant mean difference between groups at $p<0.05$

The E group had significantly higher levels of weight-bearing activity for the two-week period investigate compared to the LR, C, and M groups. The HR group had significantly higher levels of weight-bearing activity compared to the C group. There were no other significant differences between groups.

Table 23. Two-week Activity - Mothers.

	Weight Bearing Activity (hours/2 weeks)	
	mean	SD
Elite Gymnasts	5.479	4.059
High Recreation Gymnasts	4.565	2.767
Low Recreation Gymnasts	6.611	4.794
Controls	5.039	2.450
Matched Controls	5.467	3.210

There were no significant differences between any of the groups of mothers in terms of two-week weight-bearing activity.

Dietary Intake

The dietary intakes for all daughter groups are presented in Table 24. There were no statistically significant differences between the groups for any of the nutritional variables studied. Mean calcium and Vitamin D intakes exceeded the RDI for each daughter group.

Table 24. Three-day Dietary Analyses - Daughters.

	Elite Gymnasts	High Rec. Gymnasts	Low Rec. Gymnasts	Controls	Matched Controls
Protein (grams)	67.017 (10.682)	67.436 (12.031)	69.433 (18.754)	66.006 (13.823)	70.390 (18.741)
Fat (grams)	65.567 (22.421)	65.464 (15.758)	62.880 (25.694)	69.687 (18.546)	64.340 (22.213)
Carbohydrate (grams)	275.833 (47.944)	266.214 (40.075)	281.600 (84.928)	264.188 (73.063)	290.800 (37.838)
Energy (kC)	1940.25 (362.128)	1870.643 (269.221)	1929.000 (579.800)	1916.500 (465.205)	1979.600 (370.706)
Calcium (mg.)	872.917 (246.478)	1019.000 (372.911)	945.133 (370.146)	975.500 (309.516)	938.000 (351.097)
% RDI Calcium	109.583 (39.530)	141.571 (54.413)	117.733 (52.506)	118.563 (46.588)	114.700 (58.929)
Phosphorus (mg.)	1038.417 (206.935)	1171.786 (354.329)	1077.800 (337.453)	1080.938 (279.693)	1115.800 (230.664)
Vitamin D (µg)	3.457 (1.961)	5.316 (3.071)	4.212 (2.775)	4.537 (2.369)	4.602 (2.148)
% RDI Vitamin D	138.333 (78.492)	212.643 (122.666)	168.600 (111.317)	181.313 (94.675)	184.000 (85.907)

values represent the mean and (SD)

All dietary analyses for the mother groups are summarized in Table 25. The C group mothers consumed significantly less carbohydrate than the mothers of the HR, LR, and M groups, and had significantly lower dietary energy intake than the mothers from the LR and M groups.

Table 25. Three-day Dietary Analyses - Mothers

	Elite Gymnasts	High Rec. Gymnasts	Low Rec. Gymnasts	Controls	Matched Controls
Protein (grams)	64.354 (16.042)	72.936 (19.306)	79.860 (17.903)	63.867 (13.615)	72.627 (19.797)
Fat (grams)	53.746 (19.510)	68.786 (28.640)	69.507 (25.179)	49.907 (12.117)	61.882 (30.581)
Carbohydrate (grams)	234.769 (67.034)	248.786† (53.716)	258.200‡ (64.206)	186.333†,‡,¥ (51.064)	283.455¥ (55.311)
Energy (kC)	1682.538 (417.368)	1872.143 (471.142)	1973.600‡ (426.113)	1476.333‡,¥ (252.101)	2007.182¥ (431.560)
Calcium (mg.)	666.539 (305.490)	834.571 (318.373)	975.000 (336.623)	707.333 (419.654)	874.727 (459.956)
% RDI Calcium	98.846 (38.000)	119.143 (45.533)	139.333 (48.025)	101.067 (60.121)	124.909 (65.887)
Phosphorus (mg.)	934.615 (223.369)	1120.643 (332.766)	1212.867 (303.982)	953.467 (394.321)	1120.727 (382.632)
Vitamin D (µg.)	2.786 (1.779)	4.702 (3.146)	4.269 (2.100)	3.069 (1.989)	3.045 (2.899)
% RDI Vitamin D	111.307 (71.064)	187.929 (125.526)	170.733 (83.639)	122.733 (79.531)	121.818 (115.969)

values represent mean and (SD)

†, ‡, and ¥ indicate statistically significant differences at $p<0.05$.

Full ANOVA tables for all analyses performed can be found in Appendix H.

ANCOVA RESULTS

Mothers' Bone Values Used as the Covariate

Differences between groups of daughters derived from ANOVA were re-assessed with ANCOVA, using the mother's measure as the covariate. Results from the ANCOVA analyses for the bone density and bone morphometric variables are provided in Appendix I. When adjusted for their mothers' values, there were no significant differences among groups for FNBMD. With ANCOVA, the E group had significantly higher radial trabecular area than the HR group. The differences between groups in radial cortical area that were present with ANOVA were lost when ANCOVA analysis was performed. After adjusting for the mother's stature, the M group was significantly taller than the E group and all other relationships between groups remained unchanged.

Age and Anthropometric Variables of Children as the Covariate

All bone mineral and morphometric variables for the girls were analyzed using ANCOVA with age and the anthropometric variables weight, LBM, and height as covariates. When the effect of a covariate on a particular variable is not discussed, it can be assumed that it did not change the statistical significance of the outcome, compared to the results from the ANOVA.

Hip Measures

There were no significant covariate effects of age, weight, LBM, or height for FNBMD or trochanteric BMD. There was a trend, however, for increased significance of pre-existing differences (ANOVA) among groups for FNBMD. After controlling for differences in body

weight, LBM, and height, (each independently) Ward's triangle BMD was significantly higher in the HR group compared to the C group. There were no covariate effects of age, for Ward's triangle BMD.

Lumbar Spine Measures

LSBMD was statistically significantly higher in the E and HR groups compared to the C group when controlled for height, weight, or LBM. There was no significant covariate effect of age on LSBMD.

Whole Body Measures

Whole body BMD of the E and HR groups was significantly higher than the C group after adjusting for weight. There were no significant covariate effects of age or height on whole body BMD.

Radial Measures

There are no significant covariant effects for age, weight, LBM, or height for radial total, trabecular or cortical BMD. There was a trend, however, towards a reduced level of significance among groups compared to the ANOVA, after these covariate analyses.

There was a significant covariate effect of age on total radial CSA. The difference between the E and HR groups was eliminated after controlling for age differences. Differences among groups for radial trabecular CSA were eliminated after either age, weight, LBM, or height were controlled. Radial CSA became significantly greater in the E group compared to the M group after controlling for the effects of either weight or height.

Differences in the radial moment of inertia were eliminated when adjusted for age, and there was a trend for increased significance of differences for moment of inertia values after controlling for weight, LBM, and height.

CLINICAL SIGNIFICANCE OF BMD MEASURES

The majority of research that attempts to determine the relationship between fracture rate and BMD levels reports bone density in standard deviations greater or less than average. Table 26 presents the BMD results of this study in a clinical perspective.

Table 26. Percentage Differences Between Each Group With the Control Group

	Percentage of Control Value				
Measures	Elites	High Recs	Low Recs	Matched	1 SD of Control Value*
FNBMD	104.0%	101.4%	96.0%	96.1%	10.5%
FNBMAD	121.9%	121.2%	107.7%	111.0%	27.4%
TROCHANTER BMD	109.1%	100.3%	95.2%	94.6%	13.3%
WARD'S BMD	107.8%	112.4%	102.4%	104.8%	14.5%
LSBMD	104.6%	105.2%	101.1%	100.7%	10.9%
LSBMAD	110.9%	114.1%	10.9%	104.4%	11.5%
WBBMD	98.1%	94.6%	97.3%	95.8%	6.1%
WBBMAD	118.0%	117.5%	106.2%	113.8%	4.3%
TOTAL RADIAL BMD	121.5%	125.8%	113.0%	103.3%	10.0%
TRABECULAR BMD	129.8%	130.7%	108.1%	105.1%	16.8%
CORTICAL BMD	113.6%	112.8%	106.9%	97.3%	8.8%
TOTAL RADIAL CSA	104.7%	85.6%	91.5%	89.3%	20.4%
TRABECULAR CSA	97.1%	73.2%	85.7%	80%	28.5%
CORTICAL CSA	114.8%	100.2%	98.6%	98.5%	11.3%
MOMENT OF INERTIA	110.0%	72.6%	83.8%	78.8%	28.2%

* Calculated by dividing the SD of the control group by the mean of the control group for the given measure, multiplied by 100. For each measure, the 1 SD of Control Value can be compared with the same variable in the other groups to examine the differences among groups in terms of control group's SD.

DISCUSSION

Young adult and adolescent gymnasts involved in high impact loading have been shown to possess higher levels of BMD in weight-bearing regions of the skeleton than either normo-active controls or other elite athletic populations (Grimston et al., 1991; Cassell et al., 1993; Robinson et al., 1993; Nichols et al., 1994; Padro et al., 1995; Bass et al., 1995). To date, only two studies have described positive effects of impact loading on BMD in prepubescent females (Padro et al., 1995; Bass et al., 1995). However, no study to date has attempted to describe the relationship between the volume of impact loading and the resulting changes in BMD, or whether there is a differential response of trabecular and cortical bone to impact loading in prepubescent children.

All the previously mentioned studies of impact loading utilised DXA to determine areal BMD measures at selected skeletal sites. Areal BMD measurements, however, are problematic when comparing skeletons of different sized athletes, due to the inability of DXA to compensate for bone thickness (Katzman et al., 1991; Blimkie et al, in press). Systematic underestimation of bone density in smaller skeletons may have led to incorrect conclusions concerning the relationship of BMD and impact loading in these studies. Additionally, none of these impact loading studies attempted to control for familial influences on BMD.

To overcome the limitations of these previous studies, DXA areal BMD measures in this study were corrected for estimates of bone thickness (Katzman et al., 1991) and

expressed as apparent volumetric BMD (BMAD), and the maternal influence on BMD was considered by correcting the girl's BMD by their mother's BMD utilising analysis of covariance. Furthermore, most of the previous studies measured changes in BMD at sites which were predominantly weight-bearing. A unique aspect of the present study was the inclusion of BMD measures at the wrist, a region which is not habitually exposed to weight-bearing, and which might, therefore, be more sensitive to periodic weight-bearing activity inherent to gymnastic training. Lastly, the typical measures of whole body BMD and BMAD used in previous studies may be too insensitive to site specific adaptations. An additional strength of the present study is the inclusion of site-specific weight-bearing regions of interest which increases the chances of detecting regions of bone adaptation to mechanical loading.

The major findings of this study were, 1) that BMAD was generally greater in the gymnast groups compared to the control group, suggesting a magnitude effect of mechanical loading, 2) that bone density increased in a relatively stepwise fashion with increasing volume, and 3) that radial trabecular BMD was more responsive to impact loading than radial cortical BMD.

The magnitude of load effect

The results of the present study generally support our hypothesis of a positive relationship between load magnitude and bone density adaptation. These results are consistent with Frost's MES theory (1983), and with the results of Rubin and Lanyon (1983; 1985) from mechanical loading studies in animals.

The gymnasts (E group) in the present study only exhibited significantly higher areal BMD values than the controls (M group) at one site measured by DXA (trochanteric BMD). The general unresponsiveness of impact loading in the present study agrees with results from Grimston et al. (1993) for LSBMD, and Taafee et al. (1995) for whole body BMD, for similarly aged children. In contrast, numerous studies have found higher BMD at several weight-bearing sites (proximal femur, lumbar spine, whole body) in prepubertal (Padro et al., 1995; Bass et al., 1995), pubertal (Grimston et al., 1991), and young adult (Robinson et al., 1995; LaRiviere et al., 1995; Cassell et al., 1993; Robinson et al., 1993; Nichols et al., 1994) gymnasts compared to other athletes or normo-active controls. Thus, past findings lend indirect support that higher magnitude loading leads to higher levels of BMD, notwithstanding other confounding influences, such as hormonal status and selection bias. The results of the present study, however, do not support a load magnitude effect for most measures of areal BMD in our prepubertal girls. While areal BMD differences between gymnasts and controls were not always significant in the present study, the trend for insignificantly higher values in the gymnast groups suggests that with a larger sample, additional significant effects may have been realised. Additionally, in contrast to this study, most of the past studies have investigated the relationship between impact loading and areal BMD in subjects who have undergone or are experiencing puberty. Puberty has been identified as a critical time for bone growth and accretion which, therefore, may account for some of the differences in the findings between the present and past studies.

When areal BMD measures were adjusted for bone thickness and expressed as BMAD, gymnast groups (E and HR) had higher values at all sites (FN, LS, WB) compared to the control group (C). Results from a recent study (Robinson et al., 1995) of young adult gymnasts which also accounted for bone size, agreed with the results of the current study. In the study by Robinson et al. (1995) gymnasts had significantly greater density at the LS and FN than both control and running groups, after converting BMD measures to BMAD. Encouragingly, the patterns that emerged from the BMAD measures in the current study followed the same trends as the true volumetric measures determined by the pQCT, which indirectly lended support to the BMAD adjustment.

The results from the pQCT measurements provided the strongest support for a load magnitude effect. These measures of true volumetric density showed controls having dramatically lower BMD than the two upper-level gymnast groups for total, trabecular, and cortical radial BMD. The LR group displayed higher radial BMD values than the control groups, but they failed to reach significance. These findings are in agreement with the findings of Taaffe et al. (1995) and Bass et al. (1995), but in contrast to Padro et al. (1995), all of whom examined radial BMD of prepubertal gymnasts by DXA. Unlike the pQCT, the DXA is not sensitive to the different bone tissue compartments and this may, in part, explain some of the discrepancy between studies. Additionally, the other studies, because of their small samples may not have had sufficient power to detect the effect of impact loading at the radius.

There were no apparent load magnitude effects on the biomechanical or morphometrical properties at the radius in the present study. There are no other published studies including similar measurements against which to compare these results. Additionally, the loading conditions observed in this study appeared to fall within Frost's acceptable window (1992) since there was no evidence of extensive bone resorption and pathologic bone adaptation.

In summary, the results of this study lend support to the theory that there is a magnitude response of bone mineralisation to mechanical loading, as suggested by Frost (1983). This was more evident for BMAD and volumetric BMD as assessed by pQCT, than areal BMD.

The volume of impact loading effect

The significant correlations between hours of gymnastic training per week and most measures of BMD and BMAD in the present study support our hypothesis and suggest a positive volume of loading effect on bone mineral adaptation in prepubertal female gymnasts. The highly significant positive correlations indicate a strong volume relationship at all measured sites, with the exception of Ward's triangle. There are no comparable studies of variable impact loading volume in humans against which to compare our results. Nevertheless, areal BMD has been shown to vary in a positive curvilinear fashion with annual training volume in male weight-lifters (Granhed, 1987), in a negative curvilinear fashion with weight-bearing loading in figure skaters (Slemenda & Johnston, 1993), and in an inverted U fashion with running mileage (MacDougall et al., 1992).

These variable responses to volume of mechanical loading may be explained by differences in the nature of the loads, and perhaps the magnitude and intensities of these loads across studies.

True volumetric density at the distal radius determined by pQCT indicated that there was a strong relationship between the volume of training and BMD. The E and HR gymnast groups both had higher total radial density and radial cortical BMD than the two control groups. The pattern was similar for radial trabecular BMD; however, radial trabecular BMD was lower in the LR compared to the other gymnastics groups. This latter observation suggested a threshold of training effect below which no adaptation in bone occurred. This threshold seemed to be between the level of the LR and HR gymnasts. No other investigations have examined the effects of variable volumes of impact loading on the radius, therefore comparison with other studies is not possible.

Interestingly, there were no differences in BMD between the HR and E groups for any site measured. In fact, the HR group often exhibited insignificantly higher radial densities than the E group. This suggests that the volume of impact loading performed by the HR group was optimal for bone accretion and that higher levels had no additional beneficial effect on BMD. The sometimes lower BMD in the E compared to the HR gymnast group suggests that excessive training may in fact have a detrimental effect on bone density. Detrimental effects of high intensity gymnastic training on skeletal growth (Theintz et al., 1993; Albanese et al., 1989; Carter & Aldridge, 1988) have been reported previously, and our results could be interpreted as being consistent with these findings.

Alternatively, these results could suggest that these two groups are really from the same population of gymnasts whose training volume is purposefully differentiated by age; the HR gymnasts were slightly younger than the E gymnasts and their training volume was strictly limited by the gymnastics federation and local club regulations, to below 15 hours per week. From this perspective, the similarities in densities among these two groups might reflect a common selection bias acting on bone density, rather than an insensitivity to training volume.

In summary, a volume response of impact loading on BMD is apparent. The threshold appears to be between the loading levels inherent to the LR and HR gymnasts: between 3-11 hours per week. Additionally, it seems that, at some sites, the high volume of impact loading performed by the E gymnasts may in fact be less beneficial than the loading performed by the HR gymnasts. These latter observations suggest that there may be an optimal level of impact loading for bone adaptation, that if exceeded, may have detrimental effects on skeletal adaptations in prepubescent athletes.

Differential responses of radial trabecular and cortical bone to impact loading

This is the first study, to my knowledge, to attempt to separately differentiate the effects of impact loading on the isolated cortical and trabecular bone compartments of the skeleton in children.

In this study, hours of gymnastic activity was more highly correlated with trabecular BMD at the radius than cortical BMD at the same site, suggesting that the

volume of loading had a more profound effect on the trabecular bone compartment. Additionally, the finding that trabecular BMD was more highly correlated with hours of gymnastic training suggests that it is the impact force that is important to the accretion of bone and not the local muscular strains induced by the activity, since trabecular bone which is centrally localised is not directly affected by local muscle insertions.

Padro et al. (1995) came to a similar conclusion in their study of elite, prepubertal gymnasts. However, their conclusion was based on BMD measures made by DXA at regions of primarily trabecular (lumbar spine) or cortical bone (radius, tibia) and not isolated bone tissue compartments as in the present study. In this study, differences in BMD were also greater at other sites with predominantly trabecular bone (LS; $p<0.001$), compared to sites with proportionately less trabecular bone (FN; $p<0.01$), further supporting the hypothesis of a differential response from the different compartments of radial bone to impact loading.

It has been well documented that trabecular bone is turned over more rapidly than cortical bone (Snow-Harter & Marcus, 1991). The faster turnover of trabecular bone is attributed to a greater sensitivity of this type of bone to systemic hormone regulation (Seeman, 1982). In addition, the trabecular bone compartment has an increased surface area per unit of bone volume compared to cortical bone, thereby potentially allowing for a greater number of activated BMUs. Since trabecular bone is turned over at a much faster rate than cortical bone, adaptations to mechanical perturbations may therefore, be evident first at sites of predominantly trabecular bone composition.

The prepubescent gymnasts in the present study had not been involved in serious gymnastic training for an extended period, owing to their young age. They may, therefore, have had a larger physiological window for bone adaptation. Since the girls in this study were prepubertal it is unlikely that variability among groups in the sex hormone levels contributed to the observed group differences in BMD. Additionally, since there were no differences in nutritional status among groups, it appears that variability in mechanical loading history is the primary determinant of the observed differences in BMD among groups in this study.

Genetic/familial components of bone mass and physical characteristics

Bone mineral density, bone size, and bone shape are thought to be largely genetically determined (Krall & Dawson-Hughes, 1993; Pollitzer & Anderson, 1989; Slemenda et al., 1991). In this study, significant positive correlations were found at a few sites for unadjusted DXA measures of BMD, but surprisingly, after corrections for bone size were made, there were only two significant correlations between mothers and daughters BMD or BMAD (Ward's triangle, total radial BMD). Additionally, when total radial BMD was separated into its component trabecular and cortical compartments, the previously significant mother-daughter correlation was lost, suggesting a weaker and non-significant hereditary/familial influence at this region, than was implied by the total radial BMD measure.

These results generally are in contrast to those of Tylavsky et al. (1989) and Lutz (1986) for radial bone mineral density. Tylavsky et al. (1989), suggested that maternal

genetic influence played an overwhelmingly critical role in the accrual of radial bone mass in their 18-22 year-old daughters, and that after this time environmental influences had a greater impact on ultimate BMD than previously. Lutz (1986) reported significant correlations between mother and daughter radial BMC and BMC/bone width measures. These studies both used SPA to measure BMD and investigated young adult women with their mothers, who were, in the case of Lutz, (1986) menopausal. In contrast, the present study utilised pQCT and investigated prepubertal daughters and their premenopausal mothers. The differences in the method of BMD assessment and the different age groups investigated may explain the discrepant results between these studies and the present study.

Our findings for other sites besides the wrist are also in contrast with a number of studies that have found significant positive correlations for BMD between mother-daughter pairs (e.g. Matkovic et al., 1990, Seeman et al., 1989; Evans et al., 1988; Seeman et al., 1994). The discrepancy may be explained in part by differences in mother-daughter variability in PA across studies. Variability was much greater in the present study due to the intense training of the young gymnasts and the relative inactivity of their mothers. The extreme differences in PA level will attenuate familial influences and contribute to lower correlations between mother-daughter pairs. Activity differences between mothers and daughters was not as large in other studies as in our study. Therefore, other studies tend to inflate the mother-daughter correlations and overestimate the heritability of BMD. Additionally, there may be different relative genetic and

environmental contributions to BMD in skeletally immature and mature individuals (e.g. genetic influence may not be realised until postpubertally).

In the present study, total radial and trabecular CSA and moment of inertia differed between a few groups, but when adjustments for age were made, these differences became insignificant. Cortical CSA was significantly higher in the E group compared to the LR group and this difference persisted even after adjusting for various anthropometric influences. Differences in cortical CSA among groups disappeared, however, when adjusted for maternal values. From these observations it can be concluded that impact loading doesn't have much effect on CSA or the moment of inertia in these young subjects.

The highly significant ($p<0.001$) correlations between all mother-daughter CSA and moment of inertia measures, and the elimination of group differences after adjusting for maternal values suggest that these dimensional and biomechanical variables may be under strong genetic control and may be relatively insensitive to mechanical loading. The lack of differences among groups for dimensional measurements also suggests that they might be under different genetic regulatory control than the bone mineralisation process. This finding is similar to the results of Matkovic et al. (1990) where a highly significant and strong positive mother-daughter correlation ($r = 0.50$) was found for cortical CSA at the radius. Additionally, Malina & Bouchard (1991) state that the estimated genotypic contribution to variation in bone dimensions is about 60% in adults of both sexes. There may be a ceiling level of strain that is appropriate for bone size (CSA) at the radius which

was attained by all subjects in this study, owing to their relatively high activity. Additionally, PA above this level may not affect the CSA to any noticeable extent.

The pQCT was used in the present study to provide measures of gross bone morphometry and biomechanical measures of moment of inertia. Present developments in the application of this technology (Gordon, personal communication), however, should permit non-invasive evaluation of trabecular bone microarchitecture in addition to these traditional measures. In the future this technology should be used to assess not only the quantitative and dimensional changes in bone, but also the qualitative architectural changes in response to various mechanical loading regimes.

These findings suggest that BMD may be more amenable to adaptation due to mechanical loading than previously believed, and less under the influence of genetics than morphometric characteristics such as CSA, which appear to be more strongly genetically controlled.

Dietary and habitual physical activity considerations

A number of studies and reviews have stressed the importance of proper nutrition for skeletal health (e.g. Matkovic et al., 1991; Johnston et al., 1992). Encouragingly, vitamin D and calcium intakes for all groups in this study were, on average, above the RDI. There were no statistically significant differences among any of the groups of daughters for dietary intake as determined by a three-day food diary. While the three-day food diary is probably the most common method of assessing the dietary status of an individual it can be problematic. Inaccuracies arise in the estimation of food quantities and

types and the completion of the diary itself may influence the dietary choices of the individuals. Since there were no discernible differences in diet among the gymnast or control groups, it can be assumed that the observed differences in BMD were not due to differing dietary intakes.

Past-year and two-week weight-bearing activity was found to be significantly associated with BMD at a number of skeletal sites in the daughters. This finding agrees with results from Slemenda et al. (1991). The two upper-level gymnasts groups exhibited significantly higher weight-bearing hours both during the past year and during a current two-week period.

In this study weight-bearing hours of PA were determined over two time periods. One period was thought to give a current assessment of PA levels, while the other was thought to provide a general idea of activity levels over the past year. Unfortunately, the intensity of the activities performed, and the magnitude of loading associated with each activity, were not objectively assessed in this study. The activity measures (hours/week) are insensitive to the nature and magnitude of mechanical loading. For example, it is possible to achieve similar scores for weight-bearing activity from activities which provide dramatically different mechanical load magnitude to the skeleton (e.g. gymnastics training compared to walking). The activity measures in the present study are insensitive to the potential osteogenic influences of these different types and magnitudes of loads. In the future, a scale that takes the magnitude of load into consideration may be beneficial. Therefore, the measure of hours of weight-bearing activity in this study should only be

used as a rough approximation of prevailing relationships between PA and BMD. Compared to other published studies where the subjects were simply placed into one of three categories based on their level of PA (Katzman et al., 1991; Kroger et al., 1992), the methods utilised here were fairly detailed.

While it is tempting to factor out gymnastic training hours to report the data as non-gymnastic weight-bearing hours, this is erroneous, since it is impossible to factor out the effects of the gymnastic training on the bone, as well. Since there were no significant differences in the mothers' activity patterns (weight-bearing and non weight-bearing), familial differences in PA patterns appear not to be a contributing factor to the differences observed in BMD among the groups of daughters in this study.

Strength Assessment

Grip strength did not differ among any of the groups investigated. It was concluded, from a separate examination of this data set, that the intense impact loading inherent in gymnastics has a local effect on BMD at the wrist that is unrelated to grip strength (Blimkie et al., 1995). This suggests that the bone is more responsive to impact forces rather than to tensile forces exerted on the bone surface by muscle activation.

Effects of gymnastic training on skeletal health

Over the past 20 years, evidence has accumulated that gymnastics training may have a negative effect on skeletal development during growth. Recently, Theintz et al. (1993) suggested that gymnasts' shorter stature may be due in part to activities inherent to the activity, and not due to genetic or familial factors. They presented evidence that growth stunting occurred primarily in the lower body and that it was most likely caused by the extreme dieting that occurs among elite gymnasts, or by prolonged exercise associated inhibition of the hypothalamic-pituitary-gonadal axis. These results were recently supported by Bass et al. (1995) who stated that stunting may occur in elite prepubescent gymnasts. Results in the present study support this conclusion since elite gymnasts were shorter than the other groups, despite a lack of difference in height among the mothers. Unlike previous studies, however, the shorter stature of gymnasts in the present study could not be attributed to dietary or nutritional differences among groups. Intense mechanical loading in gymnastics may have negative effects on growth plate development at the wrist (Albanese et al., 1989; Carter & Aldridge, 1988), and perhaps at other sites of intense loading, which could explain growth stunting. Alternatively, differences in height in the present study, could be due to selection bias. Despite the potential for growth stunting, however, gymnastic activity has been found, in this and other recent studies, to lead to striking increases in BMD (Grimston et al., 1991; Cassell et al., 1993; Robinson et al., 1993; Nichols et al., 1994; Padro et al., 1995; Bass et al., 1995; Robinson et al., 1995; LaRiviere, 1995).

The cross-sectional nature of this study precludes definitive cause-and-effect conclusions regarding the relationship between impact loading activity and bone adaptations. Additionally, the observed relationships in this study may not be generalizable to other ages and stages of development. Recent studies (Cassell et al., 1993; Nichols et al., 1994, Robinson et al., 1995; LaRiviere et al., 1995), however, suggest that the patterns of bone adaptation evident in this study, as early as prepuberty, may persist and become enhanced with continued high impact activity with increasing maturity. Lastly, it is not known if these positive effects afforded by impact loading will remain with the cessation of gymnastic training.

Clinical Significance of BMD Results

When compared to the C group, the E and HR groups had greater bone density at all sites measured: up to 22% higher FNBMAD measures, 9% greater trochanteric BMD measures, 12% greater Ward's triangle BMD measures, 14% greater LSBMAD measures, 18% greater WBBMAD measures, 25% greater total radial BMD, 30% greater trabecular radial BMD, and 13% greater cortical radial BMD. These differences represent +0.8 ~ +2.0 standard deviation unit increases in the E and HR gymnasts compared to the control group. The clinical significance of these findings is evident when these differences are translated into risk reduction for fracture. Cummings et al. (1993) reported that a 1 SD decrease in bone density at the femoral neck led to a 160% increase in the chance of hip fracture, and a 55% increase of fracture risk at the distal radius (Cummings et al., 1990). Based on these relationships, the observed increased BMD in the E and HR groups in this

study would translate into a fracture risk reduction of 45-320%. These results suggest that these young, impact-loaded athletes may have significantly increased resistance to fracture, now, and possibly in the future if this level of bone mass is maintained. It should be noted, however, that these projections are based on the assumption that the relationship between bone mass and fracture risk is the same in children as in adults, since this relationship has not been thoroughly investigated in children.

CONCLUSION

This study demonstrated that there are positive skeletal adaptations associated with gymnastic training in prepubescent girls. A magnitude effect to mechanical loading was apparent, as was a volume effect. However, the data also suggested that there may be a volume at which further impact loading may be detrimental to BMD. Trabecular bone at the radius was found to be more sensitive to impact loading than cortical bone in these young gymnasts. The positive BMD adaptations observed from gymnastic activity appear to be independent of maternal bone density and current dietary intake.

BMD was not found to be highly correlated between mother-daughter pairs whereas bone morphometric measures were. These findings suggest that bone morphometric variables such as shape and size are strongly genetically determined and relatively unaffected by environmental influences, whereas quantitative aspects of bone development such as BMD may be less strongly determined, and may be more influenced by environmental factors, such as physical activity or exercise.

In summary, it appears that the high impact loading associated with gymnastics increases BMD in prepubertal athletes, which may eventually lead to higher peak bone mass and therefore a decreased risk of osteoporosis in later life. Longitudinal studies are needed to elucidate the positive and negative consequences of this activity on skeletal health in growing children, and to further describe the effects of different loading parameters, such as magnitude and volume, on bone mass.

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APPENDIX A

Mother/Daughter Physical Activity and Bone Density Study

Mother's General Information Form

Dear Parent:

It is believed that physical activity during the growth years may lead to stronger, healthier bones both during childhood and later on in adult life. The amount and type of physical activity, however, which is required to bring about favourable changes in bone during childhood is currently unknown. A group of researchers at McMaster University is interested in this question and have initiated a study involving prepubertal girls of varying activity backgrounds between 8 and 10 years of age. The purpose of this study is to investigate the relationship between physical activity, diet and bone density in young girls and their mothers. We would greatly appreciate your participation in this study.

Participation will require two visits, one to the McMaster University Medical Centre, and another to the Hamilton Osteoporosis Clinic. Bone density and body composition (the amount of bone, muscle and fat in the body) will be measured using special scanning devices. One device measures the bone density of the whole body, spine and hip while the subject lies still for between 12 to 20 minutes, and another measures the density of the hand/wrist region while the subject sits quietly for about 10 minutes. The bone scanning techniques are used extensively with individuals of all ages, and involve safe and very low doses of radiation (about the same as one would acquire in flying return across the Atlantic ocean). Subjects will also be asked to complete extensive questionnaires, with our help, about their past and current physical activity levels and medical histories. In addition, subjects will be required to complete a three day food diary for dietary analysis. Muscle strength of the left and right hands will be determined using a special testing device and height and weight will also be measured. All of the measurements in this study have been approved by a human ethics research committee at the University Medical Centre.

Bone density is in part genetically determined. To account for this influence it is hoped, wherever possible, also to make these measurements on the child's natural mother. Testing will be scheduled to minimize conflicts with school and other activities. Each visit, including measurements for both the mother and daughter, will last approximately 1.5 hours. Subjects will receive individual feedback about their personal results, compared to the group average, at the end of the study. All personal information will be kept strictly confidential, and known only to the researchers. You have the right to withdraw from the study at any time, even after you have agreed to participate.

We hope that you and your daughter will take part in the study, and we think that you will both find it enjoyable and interesting. Please do not hesitate to contact either Dr. Cameron (Joe) Blimkie or Shannon Frazer (study coordinator) at the numbers below if you have any questions. We thank you for your interest in and support of our study, and look forward to meeting you.

Dr. C.J. Blimkie 905-525-9140 ext. 24465
Ms. Shannon Frazer 905-528-6243

Sincerely,

Cameron (Joe) Blimkie, Ph.D.,
Associate Professor,
Department of Kinesiology,
McMaster University,
Hamilton, Ontario.

==========

WE NEED YOUR HELP

Does your daughter have any close friends of the same age who are not involved in Gymnastics (and who are either short for their age or normal height), who you think, along with her mother, might be interested in participating in this study? Yes ☐ No ☐

If yes, could you please provide us with their names and phone numbers so we can contact them regarding their possible involvement in this study.

Name	Phone Number	City
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

Do you mind if we use your name as a reference when contacting these individuals? Yes ☐ No ☐

.....
.....

Mother/Daughter Physical Activity and Bone Density Study

Child's General Information Form

You probably already know that muscles get stronger if you exercise. The purpose of this study is to determine if physical activity and diet influence your bones in the same way.

To find this out we use special scanning machines like video cameras which take pictures of your bones. One machine takes a picture of your entire skeleton, your back and your hip while you are lying still for between 12 to 20 minutes. A second machine takes pictures of your hand/wrist while you sit resting on a chair for about 10 minutes. Images of your skeleton appears on a TV screen and a computer extracts important information from these pictures about the health of your bones.

Since food is important for bone growth and health, we will also ask you, with your parents help, to try to write down all the food that you eat each day for three days in a special diary. We will also measure how strong your right and left hands are with a special testing device, and ask you to complete a questionnaire with our help, about your physical activity or exercise habits.

Hopefully your mother also will be involved in the study. This means that you will be doing all the measurements together if you decide to take part. All of your results will be kept secret and told only to you. You will get a personal report on your test results at the end of the study and will receive a small gift for your participation.

NONE OF THESE TESTS HURTS. If you decide that you would like to take part, and then change your mind, you can drop out of the study at any time. We think that you will find this study interesting and that you will enjoy being part of this important project. HOPE YOU DECIDE TO TAKE PART, AND WE ARE LOOKING FORWARD TO MEETING YOU!

Sincerely,

Dr. Cameron (Joe) Blimkie, Ph.D.
Associate Professor,
Department of Kinesiology,
McMaster University,
Hamilton, Ontario.

APPENDIX B

Mother/Daughter Physical Activity and Bone Density Study

Mother's Consent Form

I, _____, consent to participate in a study designed to investigate the relationship between physical activity, diet and bone density in mother/daughter pairs. The investigator has explained that I will be invited once to both the McMaster University Medical Centre and the Hamilton Osteoporosis Clinic, as outlined in the information sheet overleaf.

I understand that no known harmful effects will occur as a result of the measurements explained to me in the information sheet. I further understand that there are no direct benefits to myself from taking part in this study. I also understand that I can withdraw at any time from the study, even after I have signed this form. Any information which is collected will be kept confidential, and will not identify me in any way. This will also apply if the results are published.

_____	_____	_____
Name (print)	Signature	Date
_____	_____	_____
Witness (print)	Signature	Date

I have explained the nature of this study to the subject above, and believe that she fully understands the terms of this agreement.

_____	_____	_____
Investigator	Signature	Date

Mother/Daughter Physical Activity and Bone Density Study

Child's Consent Form

I, _____, consent to allow my daughter _____ to participate in a study designed to investigate the relationship between physical activity, diet and bone density in mother/daughter pairs. The investigator has explained that my daughter will be invited once to both the McMaster University Medical Centre and the Hamilton Osteoporosis Clinic, as outlined in the information sheet overleaf.

I understand that no known harmful effects will occur to my daughter as a result of the measurements explained in the information sheet. I further understand that there are no direct benefits to my daughter from taking part in this study. I also understand that my daughter can withdraw at any time from the study, even after she has signed this form. Any information which is collected will be kept confidential, and will not identify my daughter in any way. This will also apply if the results are published.

Daughter's Name (print)	Signature	Date
Mother's Name (print)	Signature	Date
Witness (print)	Signature	Date

I have explained the nature of this study to the subject above, and believe that she fully understands the terms of this agreement.

Investigator	Signature	Date

APPENDIX C

Mother/Daughter Physical Activity and Bone Density Study

Medical/Health Questionnaire

MOTHER'S FORM

The questions in this survey are directed towards events in childhood, adolescence and adult life which may have some influence on your bone mineral density. Please read the questions carefully and mark the appropriate response with a check mark (✓). Answer questions which are <u>not</u> relevant or to which you are unable to respond with an N/A. All information will be kept strictly confidential.

Interviewer:_____ Date:_____

1. **MOTHER'S** IDENTIFICATION

1.1 Surname:_____ Given Name(s):_____

1.2 Address:_____

1.3 City or Town:_____ Postal Code:_____

1.4 Telephone (Home):_____ Other:_____

1.5 Date of Birth: Day_____ Month_____ Year_____

2. **DAUGHTER'S** IDENTIFICATION

2.1 Surname:_____ Given Name(s):_____

2.2 Date of Birth: Day_____ Month_____ Year_____

2.3 Gym (>15 hr/wk) ☐ Gym (>8hr/wk) ☐

Gym (<8hr/wk) ☐ Control ☐

2.3.1 During the **current** Gymnastic season, how many hours per week on average does your daughter participate in gymnastics training and/or competition? _____ hours per week

2.3.2 How many years has your daughter been training/competing at her **current** number of hours? _____ years

2.3.3 How many years has your daughter been involved in training/competing in Gymnastics? _____ years

2.3.4 How many hours per week (on average) did your daughter train/compete in gymnastics during the following years?

1989 - 1990 _____ hours/week
1990 - 1991 _____ hours/week
1991 - 1992 _____ hours/week
1992 - 1993 _____ hours/week
1993 - 1994 _____ hours/week

2.3.5 Does your daughter participate in any other organized sport besides gymnastics? Yes ☐ No ☐

2.3.6 If yes, which sports does she participate in regularly, and at what level of competition?

Sports	Level of Participation Recreational	Competitive	Hours/Week
_____	☐	☐	_____
_____	☐	☐	_____
_____	☐	☐	_____
_____	☐	☐	_____
_____	☐	☐	_____

3. MOTHER'S LIFESTYLE INFORMATION - SMOKING, ALCOHOL, AND DIET

3.1 Have you ever smoked? Yes ☐
No ☐ go to question 3.6

3.2 Have you ever smoked for 6 months or more?
Yes ☐ How many years did you smoke? ____
No ☐ go to question 3.6

3.3 Do you still smoke? Yes, daily ☐
Yes, occasionally ☐
No, not at all ☐

3.4 When you are/were smoking, how many cigaretts do/did you usually smoke per day? About _____ per day

3.5 At about what age did you start to smoke daily? _____ yrs.

3.6 At about what age did you stop smoking? _____ yrs of age.

3.7 Do you eat a special diet? Yes ☐ No ☐

3.8 If yes, please specify the type of diet:

Vegetarian ☐
Low sodium ☐
Low cholesterol ☐
Other (please specify) _____

3.9 Do you take a calcium supplement? Yes ☐ No ☐

3.10 If yes, how many times a day do you take it? _____ times/day

3.11 What is the name of the supplement? _____

3.12 How many milligrams of calcium does it contain? _____ mgs.

3.13 Do you take a multivitamin supplement? Yes ☐ No ☐

3.14 If yes, how many times a day do you take it? _____ times/day

3.15 What is the name of the supplement? _____

3.16 How many milligrams of calcium does it contain? _____ mgs.

3.17 Do you take any of the following antacids on a daily basis?

Rolaids, Tums, Yes ☐ No ☐

3.18 If yes, how many times a day do you take it? _____ times/day

3.19 Do you take a bran or fiber supplement? Yes ☐ No ☐

3.20 If yes, how many times a day do you take it? _____ times/day

3.21 What is the name of the supplement? _____

3.22 How many grams of fiber does it contain? _____ gm/serving.

3.23 During **your early childhood** (up to 13 years of age) how often did you eat/drink the following foods?

Food	Never	1-2 Times Daily	3 + Times Daily	1-2 Times Weekly	3 + Times Weekly	1-2 Times/ Month	3+ Times/ Month
			Frequency				
Alcohol	☐	☐	☐	☐	☐	☐	☐
Tea/Coffee	☐	☐	☐	☐	☐	☐	☐
Cola/Pop	☐	☐	☐	☐	☐	☐	☐
Milk	☐	☐	☐	☐	☐	☐	☐
Yogurt	☐	☐	☐	☐	☐	☐	☐
Cheese	☐	☐	☐	☐	☐	☐	☐
Cottage Cheese	☐	☐	☐	☐	☐	☐	☐
Pizza w cheese	☐	☐	☐	☐	☐	☐	☐
Sour Cream	☐	☐	☐	☐	☐	☐	☐
Ice Cream/Milk	☐	☐	☐	☐	☐	☐	☐
Beans	☐	☐	☐	☐	☐	☐	☐
Beets	☐	☐	☐	☐	☐	☐	☐
Broccoli	☐	☐	☐	☐	☐	☐	☐
Red Meat	☐	☐	☐	☐	☐	☐	☐
White Meat	☐	☐	☐	☐	☐	☐	☐
Foul eg. Chicken	☐	☐	☐	☐	☐	☐	☐
Shell Fish	☐	☐	☐	☐	☐	☐	☐
Fish	☐	☐	☐	☐	☐	☐	☐
Organ Meat	☐	☐	☐	☐	☐	☐	☐

3.24 During **your adolescence** (13 to 18 years of age) how often did you eat/drink the following foods?

Food	Never	1-2 Times Daily	3 + Times Daily	1-2 Times Weekly	3 + Times Weekly	1-2 Times/ Month	3+ Times/ Month
			Frequency				
Alcohol	☐	☐	☐	☐	☐	☐	☐
Tea/Coffee	☐	☐	☐	☐	☐	☐	☐
Cola/Pop	☐	☐	☐	☐	☐	☐	☐
Milk	☐	☐	☐	☐	☐	☐	☐
Yogurt	☐	☐	☐	☐	☐	☐	☐
Cheese	☐	☐	☐	☐	☐	☐	☐
Cottage Cheese	☐	☐	☐	☐	☐	☐	☐
Pizza w Cheese	☐	☐	☐	☐	☐	☐	☐
Sour Cream	☐	☐	☐	☐	☐	☐	☐
Ice Cream/Milk	☐	☐	☐	☐	☐	☐	☐
Beans	☐	☐	☐	☐	☐	☐	☐
Beets	☐	☐	☐	☐	☐	☐	☐
Broccoli	☐	☐	☐	☐	☐	☐	☐

Red Meat	☐	☐	☐	☐	☐	☐	☐
White Meat	☐	☐	☐	☐	☐	☐	☐
Foul eg. Chicken	☐	☐	☐	☐	☐	☐	☐
Shell Fish	☐	☐	☐	☐	☐	☐	☐
Fish	☐	☐	☐	☐	☐	☐	☐
Organ Meat	☐	☐	☐	☐	☐	☐	☐

3.25 During **your adulthood** (18 years of age to present) how often did you eat/drink the following foods?

		Frequency					
Food	Never	1-2 Times Daily	3 + Times Daily	1-2 Times Weekly	3 + Times Weekly	1-2 Times/ Month	3+ Times/ Month
Alcohol	☐	☐	☐	☐	☐	☐	☐
Tea/Coffee	☐	☐	☐	☐	☐	☐	☐
Cola/Pop	☐	☐	☐	☐	☐	☐	☐
Milk	☐	☐	☐	☐	☐	☐	☐
Yogurt	☐	☐	☐	☐	☐	☐	☐
Cheese	☐	☐	☐	☐	☐	☐	☐
Cottage Cheese	☐	☐	☐	☐	☐	☐	☐
Pizza w Cheese	☐	☐	☐	☐	☐	☐	☐
Sour Cream	☐	☐	☐	☐	☐	☐	☐
Ice Cream/Milk	☐	☐	☐	☐	☐	☐	☐
Beans	☐	☐	☐	☐	☐	☐	☐
Beets	☐	☐	☐	☐	☐	☐	☐
Broccoli	☐	☐	☐	☐	☐	☐	☐
Red Meat	☐	☐	☐	☐	☐	☐	☐
White Meat	☐	☐	☐	☐	☐	☐	☐
Foul eg. Chicken	☐	☐	☐	☐	☐	☐	☐
Shell Fish	☐	☐	☐	☐	☐	☐	☐
Fish	☐	☐	☐	☐	☐	☐	☐
Organ Meat	☐	☐	☐	☐	☐	☐	☐

4. MOTHER'S LIFESTYLE INFORMATION - PHYSICAL ACTIVITY

4.1 Rate (cirle one) your overall level of physical activity as a **child and youth** (up to 18 years of age).

1	2	3	4	5
seldom active	sometimes active	active	moderately active	very active

4.2 How would you describe the games you played most often as a **child** and **youth**? (Circle one)

1	2	3
games such as board games, drawing, puzzles, etc.	games requiring some running, jumping, climbing, throwing, etc.	mostly running, jumping, climbing, and throwing etc.

4.3 During which years were you physically active? (Circle more than one if need be)

1	2	3	4	5
5-10 yrs	10-15 yrs	15-20 yrs	20-30 yrs	30-40 yrs

4.4 During which years were you the **MOST** physically active? (Circle one only)

1	2	3	4	5
5-10 yrs	10-15 yrs	15-20 yrs	20-30 yrs	30-40 yrs

4.5 Did you participate in organized sport as a child or youth?
Yes ☐ No ☐

4.6 If yes, please list below, the sports you participated in during your **YOUTH (before 18 years of age)**, and the approximate number of years of participation.

Sport	Age While Participating (yrs)	# of Years of Participation (yrs)

4.7 **Since the age of 18 years**, did you participate regularly in sport (e.g. tennis, soccer, basketball)?

Yes ☐ No ☐

4.8 If yes, please list below, the sports in which you participated, your age while participating, and the approximate number of years of participation.

Sport	Age While Participating (yrs)	# of Years of Participation (yrs)

4.9 Were you regularly involved in heavy physical work (e.g. farm chores or heavy lifting) as a child or youth?

Yes ☐ No ☐

4.10 How would you rate your current level of **PHYSICAL ACTIVITY** compared to others your age (Check one only)

very low ☐ low ☐ average ☐ high ☐ very high ☐

4.11 How would you rate your current level of **PHYSICAL FITNESS** compared to others your age (Check one only)

very low ☐ low ☐ average ☐ high ☐ very high ☐

4.12 During the last week, how many times did you do any of the activities listed below, about how much time (average) did you spend doing the various activities on each occasion, and how difficult or strenuous was the activity on average

For difficulty or strenuousness of the activity, use the following guidelines:

Light: slight sweating and slight increase in breathing
Moderate: noticeable sweating and above normal breathing
Heavy: heavy sweating and heavy breathing

ACTIVITY	# Of Times	MINUTES EACH TIME 1-15	16-30	31-59	60 +	STRENUOUSNESS LIGHT	MOD	HEAVY
Walking For Exercise	___	☐	☐	☐	☐	☐	☐	☐
Calisthenics	___	☐	☐	☐	☐	☐	☐	☐
Aerobics	___	☐	☐	☐	☐	☐	☐	☐
Weight Lifting	___	☐	☐	☐	☐	☐	☐	☐
Stat Cycling/ Bicycling	___	☐	☐	☐	☐	☐	☐	☐
Jogging/ Running	___	☐	☐	☐	☐	☐	☐	☐
Bowling	___	☐	☐	☐	☐	☐	☐	☐
Social Dancing	___	☐	☐	☐	☐	☐	☐	☐
Modern/ Jazz Dancing	___	☐	☐	☐	☐	☐	☐	☐
Racquet Sports	___	☐	☐	☐	☐	☐	☐	☐
Golf	___	☐	☐	☐	☐	☐	☐	☐
Swimming	___	☐	☐	☐	☐	☐	☐	☐
Gardening/ Yard Work	___	☐	☐	☐	☐	☐	☐	☐
House Work	___	☐	☐	☐	☐	☐	☐	☐
Baseball/ Softball	___	☐	☐	☐	☐	☐	☐	☐
Basketball	___	☐	☐	☐	☐	☐	☐	☐
Volleyball	___	☐	☐	☐	☐	☐	☐	☐
Curling	___	☐	☐	☐	☐	☐	☐	☐
Skipping	___	☐	☐	☐	☐	☐	☐	☐

Skating/ Rollerblading ___	☐	☐	☐	☐	☐	☐	☐
Skiing/ Down Hill ___	☐	☐	☐	☐	☐	☐	☐
Skiing/ Cross-Country ___	☐	☐	☐	☐	☐	☐	☐
Ringette/ Ice Hockey ___	☐	☐	☐	☐	☐	☐	☐
Tag ___	☐	☐	☐	☐	☐	☐	☐
Others (Please specify)							
_____ ___	☐	☐	☐	☐	☐	☐	☐
_____ ___	☐	☐	☐	☐	☐	☐	☐
_____ ___	☐	☐	☐	☐	☐	☐	☐

OR, I DID NOTHING LIKE THIS IN THE LAST WEEK ☐

==========

4.13 Approximately how many hours of television do you watch each day?

_____ average hours per day from Monday-Friday

_____ average hours per day on Saturday and Sunday

4.14 Please provide any other comments regarding your lifestyle or physical activity which you think we should know about.

_____

_____

_____

_____

_____

_____

5. MOTHER'S REPRODUCTIVE HISTORY AND GYNECOLOGICAL STATUS

5.1 Are you pregnant? ____yes ____ no

5.2 To the nearest half-year, at what age did you have your first menstrual period?

_____ years of age.

5.3 Do you menstruate regularly? Yes ☐ No ☐

5.4 If yes, please indicate the approximate time in days between periods: _____ days

5.5 If no,

a) what is the shortest time you experienced between periods: ____ days

b) what is the longest time you experienced between periods : ____ days

5.6 How many periods do you usually have in a year? (Circle one only)

1	2	3
11-17	4-10	less than 4

5.7 When was your last period? ____ day ____ mo. ____ yr.

5.8 Other than when you were pregnant or lactating, have you ever had an absence or loss of periods? Yes ☐ No ☐

5.9 If yes, at what age did these missed periods occur?

1st time: ____ years old

2nd time: ____ years old

5.10 For how long did your periods stop during these occasions?

1st time: _____ mo. ____ yrs.

2nd time: _____ mo. ____ yrs.

5.11 Have you had a hysterectomy? Yes ☐ No ☐

5.12 If yes, were the ovaries removed? Yes ☐ No ☐

5.13 When was this surgery performed? ____ mo. ____ year

5.14 Has your menopause begun (no periods for a year or more after your last period)? Yes ☐ No ☐

5.15 At what age did you begin to experience menopausal symptoms e.g. hot flashes, irregular menstrual cycles?
_____ years old.

5.16 How many times have you been pregnant? ____

5.17 How many children have you given birth to? ____

5.18 How old were you at the birth of your children?

1st child: ____ yrs old 2nd child: ____ yrs old

3rd child: ____ yrs old 4th child: ____ yrs old

5.19 Did you breast feed one or more of your children?

____ no none of them

Yes, I breast fed _____ (number) of them

5.20 List the number of months spent breast feeding each child

1st child: ____ mos. 2nd child: ____ mos.

3rd child: ____ mos. 4th child: ____ mos.

5.21 Do you now or have you ever used oral contraceptives?

Yes ☐ No ☐

5.22 If yes, for how many years? ____ years

5.23 Please indicate the brand name of the contraceptive:

_____

5.24 If you previously used oral contraceptives but are no longer using them, what was the last approximate date of use?

____ mo. ____ yr.

5.25 Do you now or have you ever taken estrogen supplements other than oral contraceptives?

Yes ☐ No ☐

5.26 If yes, what medication did you or are you taking?
_____ (brand name)

5.27 When did you begin taking this medication?
____ mo. ____ yr.

5.28 When did you stop taking this medication?
____ mo. ____ yr.

6. **MOTHER'S DETAILED MEDICAL HISTORY AND STATUS**

6.1 Have you seen a doctor in the last 6 months for a medical concern? Yes ☐ No ☐

6.2 If yes, what was the reason for your visit?

_____

_____

_____

_____

6.3 Has there been any change in your general health during the last 6 months? Yes ☐ No ☐

6.4 If yes, please describe the nature of the change:

_____

_____

6.5 Have you been hospitalized in the last year? ____ yes ____ no

6.6 If yes, please indicate the medical condition(s) which was being treated:

_____

6.7 Have you had any surgery in the past 2 years? Yes ☐ No ☐

6.8 If yes, list the procedure and approximate date of surgery.

Type of Surgery	Date of Surgery	Length of Hospital Stay
_____	_____	_____
_____	_____	_____
_____	_____	_____

6.9 Have you ever been treated for any of the following conditions? [hyper = excess; hypo = deficiency]

food allergies	yes ☐	no ☐	asthma	yes ☐	no ☐
other allergies	yes ☐	no ☐	kidney disease	yes ☐	no ☐
back pain	yes ☐	no ☐	liver problems	yes ☐	no ☐
scoliosis	yes ☐	no ☐	gastrointestinal		
epilepsy	yes ☐	no ☐	disease	yes ☐	no ☐
osteoporosis	yes ☐	no ☐	muscular		
rheumatoid			dystrophy	yes ☐	no ☐
arthritis	yes ☐	no ☐	osteoarthritis	yes ☐	no ☐
diabetes	yes ☐	no ☐	anemia	yes ☐	no ☐
excess urinary			malabsorption	yes ☐	no ☐
calcium	yes ☐	no ☐	excess blood		
hyperparathyroid	yes ☐	no ☐	calcium	yes ☐	no ☐
hyperthyroidism	yes ☐	no ☐	hypoparathyroid	yes ☐	no ☐
hypothyroidism	yes ☐	no ☐	other (specify):	_____	

6.10 Have you had a bone scan or a diagnostic X-ray in the last year? Yes ☐ No ☐

6.11 If yes, what body part was X-rayed? _____

6.12 Have you ever had a fractured bone? Yes ☐ No ☐

6.13 If yes, please indicate which bone(s) was/were fractured and when the fractures occurred.

1st fracture: body part_____ ____ mo. ____ yr.
2nd fracture: body part_____ ____ mo. ____ yr.
3rd fracture: body part_____ ____ mo. ____ yr.

6.14 Have you ever been hospitalized or confined to bed for any reason, or had a limb immobilized (e.g. arm in a cast) for 21 days or longer?

Yes ☐ No ☐

6.15 If yes, list the condition, approximate date it occurred, and the length of time you were hospitalized or immobolized.

	Injury type	Date of Injury	Time Immobolized
e.g.	wrist fracture	July, 1982	6 weeks
	_____	_____	_____
	_____	_____	_____
	_____	_____	_____

6.16 Is there a history of wrist, hip or spine fracture in your immediate family? Yes ☐ No ☐

6.17 If yes, please indicate who was affected.

biological mother ☐
maternal grandmother ☐
maternal grandfather ☐
biological father ☐
paternal grandmother ☐
paternal grandfather ☐

6.18 Is there a history of osteoporosis in your family?
Yes ☐ No ☐

6.19 If yes, indicate who was affected.

biological mother ☐
maternal grandmother ☐
maternal grandfather ☐
biological father ☐
paternal grandmother ☐
paternal grandfather ☐

6.20 Is there a history of any other bone disease in your family?

Yes ☐ No ☐

6.21 If yes, indicate which family member is/was affected and the name of the condition.

Family Member	Condition
_____	_____
_____	_____
_____	_____

7. **MOTHER'S MEDICATIONS**

7.1 Are you curently taking any prescription medications?
Yes ☐ No ☐

7.2 If yes, which medications are you taking?

_____

_____

7.3 What are these medications for?

_____

_____

7.4 Have you ever taken any of the following medications? Please indicate at what age you began to use them, and for how long you used them.

Medication	Currently Using	Age at Start	Duration of Use
e.g. Insulin	_____	15 yrs old	20 years
Calcium preparations	_____	_____	_____
Antacids	_____	_____	_____
Inhaled steroids	_____	_____	_____
Anabolic steroids	_____	_____	_____
Fluoride	_____	_____	_____
Vitamin D compounds	_____	_____	_____
Calcitonin	_____	_____	_____
Diuretics	_____	_____	_____
Heparin	_____	_____	_____
Cortisone (oral)	_____	_____	_____
Corticosteroids	_____	_____	_____
Anti-inflammatories	_____	_____	_____
Thyroid preparations	_____	_____	_____

8. MOTHER'S MEDICAL DECLARATION

8.1 Do you have any medical/health condition which might prevent you from participating in this study?
Yes ☐ No ☐

8.2 **I certify that the information provided on this form is correct.**

Signature: _____

Date: _____

Mother/Daughter Physical Activity and Bone Density Study

Medical/Health Questionnaire

DAUGHTER'S FORM

The questions in this survey are directed towards events in childhood which may have some influence on your bone mineral density. Please read the questions carefully and mark the appropriate response with a check mark (✓). Answer questions which are <u>not</u> relevant or to which you are unable to respond with an N/A. All information will be kept strictly confidential.

INSTRUCTIONS: It is probably best if the mother assists her daughter in completing this questionnaire.

Interviewer:_____ Date:_____

1. **DAUGHTER'S** IDENTIFICATION

1.1 Surname:_____ Given Name(s):_____

1.2 Date of Birth: Day_____ Month_____ Year_____

1.3 Gym (>15 hr/wk) ☐ Gym (>8hr/wk) ☐

Gym (<8hr/wk) ☐ Control ☐

2. **DAUGHTER'S LIFESTYLE INFORMATION - DIET**

2.1 During **your daughter's childhood** (from birth to present) how often did she eat/drink the following foods?

		Frequency					
Food	Never	1-2 Times Daily	3 + Times Daily	1-2 Times Weekly	3 + Times Weekly	1-2 Times/ Month	3+ Times/ Month
Alcohol	☐	☐	☐	☐	☐	☐	☐
Tea/Coffee	☐	☐	☐	☐	☐	☐	☐
Cola/Pop	☐	☐	☐	☐	☐	☐	☐
Milk	☐	☐	☐	☐	☐	☐	☐
Yogurt	☐	☐	☐	☐	☐	☐	☐
Cheese	☐	☐	☐	☐	☐	☐	☐
Cottage Cheese	☐	☐	☐	☐	☐	☐	☐

Pizza w Cheese	☐	☐	☐	☐	☐	☐	☐
Sour Cream	☐	☐	☐	☐	☐	☐	☐
Ice Cream/Milk	☐	☐	☐	☐	☐	☐	☐
Beans	☐	☐	☐	☐	☐	☐	☐
Beets	☐	☐	☐	☐	☐	☐	☐
Broccoli	☐	☐	☐	☐	☐	☐	☐
Red Meat	☐	☐	☐	☐	☐	☐	☐
White Meat	☐	☐	☐	☐	☐	☐	☐
Foul eg. Chicken	☐	☐	☐	☐	☐	☐	☐
Shell Fish	☐	☐	☐	☐	☐	☐	☐
Fish	☐	☐	☐	☐	☐	☐	☐
Organ Meat	☐	☐	☐	☐	☐	☐	☐

2.2 Does your daughter eat a special diet? Yes ☐ No ☐

2.3 If yes, please specify the type of diet:

Vegetarian ☐
Low sodium ☐
Low cholesterol ☐
Other (please specify) _____

2.4 Does your daughter take a calcium supplement?
Yes ☐ No ☐

2.5 If yes, how many times a day does she take it?
_____ times/day

2.6 What is the name of the supplement? _____

2.7 How many milligrams of calcium does it contain? _____ mgs.

2.8 Does your daughter take a multivitamin supplement?
Yes ☐ No ☐

2.9 If yes, how many times a day does she take it?
_____ times/day

2.10 What is the name of the supplement? _____

2.11 How many milligrams of calcium does it contain? _____ mgs.

2.12 Does your daughter take any of the following antacids on a daily basis?

Rolaids, Tums, Yes ☐ No ☐

2.13 If yes, how many times a day does she take it?
_____ times/day

2.14 Does your daughter take a bran or fiber supplement?
Yes ☐ No ☐

2.15 If yes, how many times a day does she take it?
_____ times/day

2.16 What is the name of the supplement? _____

2.17 How many grams of fiber does it contain? _____ gm/serving.

3. DAUGHTER'S LIFESTYLE INFORMATION - PHYSICAL ACTIVITY

3.1 Rate (cirle one) your daughter's overall level of physical activity **compared to her friends.**

1	2	3	4	5
seldom active	sometimes active	active	moderately active	very active

3.2 How would you describe the types of games your daughter plays **in her free time**? (Circle one)

1	2	3
games such as board games, drawing, puzzles, etc.	games requiring some running, jumping, climbing, throwing, etc.	mostly running, jumping, climbing, and throwing etc.

3.3 Is your daughter regularly involved in heavy physical work (e.g. farm chores or heavy lifting) outside of sports?

Yes ☐ No ☐

3.4 How would you rate your daughter's current level of **PHYSICAL ACTIVITY** compared to others her age (Check one only)

very low ☐ low ☐ average ☐ high ☐ very high ☐

3.5 How would you rate your daughter's current level of **PHYSICAL FITNESS** compared to others her age (Check one only)

very low ☐ low ☐ average ☐ high ☐ very high ☐

3.6 During **the last week**, how many times did your daughter do any of the activities listed below, about how much time (average) did she spend doing the various activities on each occasion, and how difficult or strenuous was the activity on average

For difficulty or strenuousness of the activity, use the following guidelines:

Light: slight sweating and slight increase in breathing

Moderate: noticeable sweating and above normal breathing

Heavy: heavy sweating and heavy breathing

ACTIVITY	# Of Times	MINUTES EACH TIME 1-15	16-30	31-59	60 +	STRENUOUSNESS LIGHT	MOD	HEAVY
Walking For Exercise	___	☐	☐	☐	☐	☐	☐	☐
Calisthenics	___	☐	☐	☐	☐	☐	☐	☐
Aerobics	___	☐	☐	☐	☐	☐	☐	☐
Weight Lifting	___	☐	☐	☐	☐	☐	☐	☐
Stat Cycling/ Bicycling	___	☐	☐	☐	☐	☐	☐	☐
Jogging/ Running	___	☐	☐	☐	☐	☐	☐	☐
Bowling	___	☐	☐	☐	☐	☐	☐	☐
Social Dancing	___	☐	☐	☐	☐	☐	☐	☐
Modern/ Jazz Dancing	___	☐	☐	☐	☐	☐	☐	☐
Racquet Sports	___	☐	☐	☐	☐	☐	☐	☐
Golf	___	☐	☐	☐	☐	☐	☐	☐
Swimming	___	☐	☐	☐	☐	☐	☐	☐
Gardening/ Yard Work	___	☐	☐	☐	☐	☐	☐	☐
House Work	___	☐	☐	☐	☐	☐	☐	☐
Baseball/ Softball	___	☐	☐	☐	☐	☐	☐	☐

Basketball	___	☐	☐	☐	☐	☐	☐	☐
Volleyball	___	☐	☐	☐	☐	☐	☐	☐
Curling	___	☐	☐	☐	☐	☐	☐	☐
Skipping	___	☐	☐	☐	☐	☐	☐	☐
Skating/ Rollerblading	___	☐	☐	☐	☐	☐	☐	☐
Skiing/ Down Hill	___	☐	☐	☐	☐	☐	☐	☐
Skiing/ Cross-Country	___	☐	☐	☐	☐	☐	☐	☐
Ringette/ Ice Hockey	___	☐	☐	☐	☐	☐	☐	☐
Tag	___	☐	☐	☐	☐	☐	☐	☐
Intra-murals	___	☐	☐	☐	☐	☐	☐	☐
Others (Please specify)								
_____	___	☐	☐	☐	☐	☐	☐	☐
_____	___	☐	☐	☐	☐	☐	☐	☐
_____	___	☐	☐	☐	☐	☐	☐	☐

OR, I DID NOTHING LIKE THIS IN THE LAST WEEK ☐

==========

3.7 Approximately how many hours does your daughter spend watching television or playing video/computer games each day?

_____ average hours <u>per</u> day from Monday-Friday

_____ average hours <u>per</u> day on Saturday and Sunday

3.8 Please provide any other comments regarding your daughter's lifestyle or physical activity which you think we should know about.

_____

_____

3.9 How does your daughter usually get to and from school?

Fall and Spring

Walk ☐ Bike ☐ Car ☐ Bus ☐ Other:_____

Winter

Walk ☐ Bike ☐ Car ☐ Bus ☐ Other:_____

3.12 How far is it from home to your daughter's school?

_____ kilometers.

3.13 Does your daughter usually come home for lunch? Yes ☐ No ☐

3.14 How does your daughter usually get to and from school at lunch and after school?

Fall and Spring

Walk ☐ Bike ☐ Car ☐ Bus ☐ Other:_____

Winter

Walk ☐ Bike ☐ Car ☐ Bus ☐ Other:_____

3.15 After eating lunch, what type of activity, if any, does your daughter do? _____

3.16 How long are your daughter's lunch breaks? _____ minutes.

3.17 Does your daughter take Physical Education at school?

Yes ☐ No ☐

3.18 How many times per week does she have Physical Education classes? _____ times per week.

3.19 How long are Physical Education classes usually? ____ minutes.

4. DAUGHTER'S DETAILED MEDICAL HISTORY AND STATUS

4.1 Has your daughter seen a doctor in the last 6 months for a medical concern? Yes ☐ No ☐

4.2 If yes, what was the reason for her visit?

_____

_____

_____

_____

4.3 Has there been any change in your daughter's general health during the last 6 months? Yes ☐ No ☐

4.4 If yes, please describe the nature of the change:

_____

_____

4.5 Has your daughter been hospitalized in the last year? Yes ☐ No ☐

4.6 If yes, please indicate the medical condition(s) which was being treated:

_____

4.7 Has your daughter had any surgery in the past 2 years? Yes ☐ No ☐

4.8 If yes, list the procedure and approximate date of surgery.

Type of Surgery	Date of Surgery	Length of Hospital Stay
_____	_____	_____
_____	_____	_____
_____	_____	_____

4.9 Has your daughter ever been treated for any of the following conditions? [hyper = excess; hypo = deficiency]

food allergies	yes ☐	no ☐	asthma	yes ☐	no ☐
other allergies	yes ☐	no ☐	kidney disease	yes ☐	no ☐
back pain	yes ☐	no ☐	liver problems	yes ☐	no ☐
scoliosis	yes ☐	no ☐	gastrointestinal		
epilepsy	yes ☐	no ☐	disease	yes ☐	no ☐
osteoporosis	yes ☐	no ☐	muscular		
rheumatoid			dystrophy	yes ☐	no ☐
arthritis	yes ☐	no ☐	osteoarthritis	yes ☐	no ☐
diabetes	yes ☐	no ☐	anemia	yes ☐	no ☐
excess urinary			malabsorption	yes ☐	no ☐
calcium	yes ☐	no ☐	excess blood		
hyperparathyroid	yes ☐	no ☐	calcium	yes ☐	no ☐
hyperthyroidism	yes ☐	no ☐	hypoparathyroid	yes ☐	no ☐
hypothyroidism	yes ☐	no ☐	other (specify):	_____	

4.10 Has your daughter had a bone scan or a diagnostic X-ray in the last year? Yes ☐ No ☐

4.11 If yes, what body part was X-rayed? _____

4.12 Has your daughter ever had a fractured bone? Yes ☐ No ☐

4.13 If yes, please indicate which bone(s) was/were fractured and when the fractures occurred.

1st fracture: body part_____ ____ mo. ____ yr.
2nd fracture: body part_____ ____ mo. ____ yr.
3rd fracture: body part_____ ____ mo. ____ yr.

4.14 Has your daughter ever been hospitalized or confined to bed for any reason, or had a limb immobilized (e.g. arm in a cast) for 21 days or longer?

Yes ☐ No ☐

4.15 If yes, list the condition, approximate date it occurred, and the length of time she was hospitalized or immobolized.

	Injury type	Date of Injury	Time Immobolized
e.g.	wrist fracture	July, 1982	6 weeks
	_____	_____	_____
	_____	_____	_____
	_____	_____	_____

5. DAUGHTER'S MEDICATIONS

5.1 Is your daughter currently taking any prescription medications?
Yes ☐ No ☐

5.2 If yes, which medications is she taking?

_____

_____

5.3 What are these medications for?

_____

_____

5.4 Has your daughter ever taken any of the following medications? Please indicate at what age she began to use them, and for how long she used them.

Medication	Currently Using	Age at Start	Duration of Use
e.g. Insulin	_____	6 yrs old	2 years
Calcium preparations	_____	_____	_____
Antacids	_____	_____	_____
Inhaled steroids	_____	_____	_____
Anabolic steroids	_____	_____	_____
Fluoride	_____	_____	_____
Vitamin D compounds	_____	_____	_____
Calcitonin	_____	_____	_____
Diuretics	_____	_____	_____
Heparin	_____	_____	_____
Cortisone (oral)	_____	_____	_____
Corticosteroids	_____	_____	_____
Anti-inflammatories	_____	_____	_____

Thyroid preparations _____ _____ _____

==========

6. DAUGHTER'S MEDICAL DECLARATION

6.1 Does your daughter have any medical/health condition which might prevent her from participating in this study?
Yes ☐ No ☐

6.2 **I (mother) certify that the information provided on this form is correct.**

Signature of mother: _____

Signature of daughter: _____

Date: _____

Mother/Daughter Physical Activity and Bone Density Study

Personal Descriptive and Anthropometric Data

Mother's Form

Name:_____ Birth Date:_____
(m/d/y)

Decimal Age Birth Day:_____
(m/d/y)

Date of Test:_____ Tester:_____
(m/d/y)

---

1) Anthropometry

Stature (cm): _____

Weight (kg) : _____

2) Hand Preference (To Write): Left ☐ Right ☐

Leg Preference (To Kick) : Left ☐ Right ☐

3) Grip Strength

	Trial 1	Trial 2	Average
Left Hand:	_____	_____	_____
Right Hand:	_____	_____	_____

4) Skinfold Thickness

	Trial 1	Trial 2	Average
Triceps Skf	_____	_____	_____
Subscapular Skf	_____	_____	_____

5) Bioelectrical Impedance

	Trial 1	Trial 2	Average
Reactance	_____	_____	_____
Resistance	_____	_____	_____
% Body Fat	_____	_____	_____
Lean Body Mass (kg)	_____	_____	_____

Mother/Daughter Physical Activity and Bone Density Study

Personal Descriptive and Anthropometric Data

Child's Form

Name:_____ Birth Date:_____
(m/d/y)

Decimal Age Birth Day:_____
(m/d/y)

Date of Test:_____ Tester:_____
(m/d/y)

---

1) Anthropometry

Stature (cm): _____

Weight (kg) : _____

2) Hand Preference (To Write): Left ☐ Right ☐

Leg Preference (To Kick) : Left ☐ Right ☐

3) Grip Strength

	Trial 1	Trial 2	Average
Left Hand:	_____	_____	_____
Right Hand:	_____	_____	_____

4) Skinfold Thickness

	Trial 1	Trial 2	Average
Triceps Skf	_____	_____	_____
Subscapular Skf	_____	_____	_____

5) Bioelectrical Impedance

	Trial 1	Trial 2	Average
Reactance	_____	_____	_____
Resistance	_____	_____	_____
% Body Fat	_____	_____	_____
Lean Body Mass (kg)	_____	_____	_____

APPENDIX D

Directions For Activity Questionnaire Completion

A. Activities In The Past Year

- Mention importance of accuracy of detail.
- Record activities which subject did besides 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 January 1994 and progress from left to right for each of listed activities.

  e.g. - begin with January 1994 for walking as exercise and end with this past December, 1994

- Ask subject to estimate the number of times they performed each exercise each month, and to provide an estimate of the average length of time spent per session for each activity.
- Ask subject to estimate 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.

PHYSICAL ACTIVITY IN YOUR SPARE TIME

Subject's Name: _____

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	
_____			
_____			
_____			
_____			
_____			

Activities To Consider When Completing The Activity Form

Activity	Activity
Walking for pleasure	Softball
Walking to and from work	Badminton
Walking during work break	Paddle ball
Using stairs when elevator is available	Racket ball
Cross-country hiking	Basketball: non-game
Back packing	Basketball: game play
Mountain climbing	Basketball: officiating
Bicycling to work and/or for pleasure	Touch football
Dancing—Ballroom and/or square	Handball
Home exercise	Squash
Health club	Soccer
Jogging and walking	Golf: riding a power cart
Running	Golf: walking, pulling clubs on cart
Weight lifting	Golf: walking and carrying clubs
Water skiing	Mowing lawn with riding mower
Sailing	Mowing lawn walking behind power mower
Canoeing or rowing for pleasure	Mowing lawn pushing hand mower
Canoeing or rowing in competition	Weeding and cultivating garden
Canoeing on a camping trip	Spading, digging, filling in garden
Swimming (at least 50 ft) at a pool	Raking lawn
Swimming at the beach	Snow shoveling by hand
Scuba diving	Carpentry in workshop
Snorkeling	Painting inside of house, includes paper hanging
Snow skiing, downhill	Carpentry outside
Snow skiing, cross country	Painting outside of house
Ice (or roller) skating	Fishing from river bank
Sledging or tobogganing	Fishing in stream with wading boots
Bowling	Hunting pheasants or grouse
Volley ball	Hunting rabbits, prairie chickens, squirrels, raccoon
Table tennis	Hunting large game: deer, elk, bear
Tennis, singles	
Tennis, doubles	

APPENDIX E

DIETARY DIARY
INSTRUCTIONS FOR COMPLETION

The idea behind the diary is that it enables us to construct an accurate picture of the quantities of many nutrients that you consume each day. For this reason please be as detailed and specific as possible.

E.g. If you ate a sandwich for lunch please add more detail than telling us what the filling was. We would like to know what sort of bread was used, whether you had butter or margarine, if you had salt or pepper, whether it contained other dressings such as mayonnaise and any additional information you feel that you would like to include. In short, please tell us exactly what you ate in as much detail as possible.

In addition to knowing what you ate, we also want to know how much you ate.

E.g. If you drank a glass of milk, please tell us what kind of milk (whole, skimmed, partly skimmed, chocolate etc), and also whether you had a small, medium or large glass.

It is important to stress that this is not a "test" of what you and your children eat, and we do not want you to change your eating habits because you are completing the diary.

Please also record what you drink during the day (other than water), because this is valuable information as well.

E.g. If you drank tea in the morning, please tell us the size of the serving, whether you added whole, partly skimmed or skimmed milk, and how much sugar (or artificial sweetener) you added.

IN SUMMARY

- **Record everything you and your daughter eat.**
- **Record items in as much detail as possible.**
- **Record the quantity of each item that you record.**
- **Try not to let the diary influence your eating habits.**

Please do not hesitate to contact Shannon Frazer at 905-528-6243 if you have any questions regarding the completion of this diary.

FOOD DIARY

Instructions:

1) Record all food and beverages you eat or drink over the 3 days and where and when eaten

   It's best to write down what you've eaten/drank immediately afterward so that you won't forget any item

2) **List the amount**
   - cups (250 mL), ounces or mL
   - teaspoons or tablespoons (level or heaping)
   - slices or ounces/grams
   - dimensions for meat, fish, cheese,... (for example 2"x1"x2")
   - scale weight - weigh container then container with food

3) Give **method of preparation**
   - broiled, fried, boiled, baked...

4) Give **Brand Names / Restaurant names**
   - for example - 2 cups of Kraft dinner (not macaroni and cheese)

5) Note anything **added to food or drink**
   - for example - 1 1/2 cups of coffee with 1 teaspoon of sugar and 1 tablespoon of homogenized milk

6) For combination foods (casseroles, sandwiches, sauces...) record the main ingredients and amounts of each and total amount of recipe along with the portion size that you had

   for example:
   - egg salad sandwich -2 pieces of whole wheat bread
     -2 teaspoons Becel margarine
     -half of egg salad recipe

   egg salad recipe - 3 large eggs, boiled, 3 tablespoons Miracle Whip salad dressing, 1 green onion

7) Include all supplement and vitamin preparations

8) Please record your medications and amounts

Try to be as descriptive as possible when recording your food diary AND most of all, try not to change what you eat or drink for this study - we're interested in you !

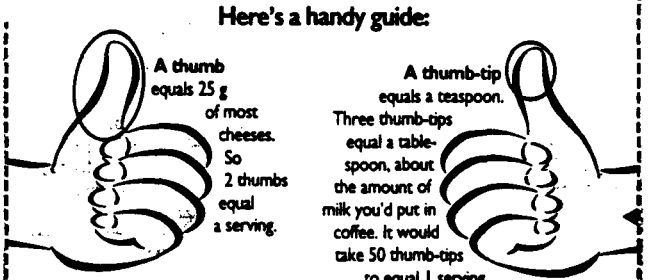
POCKET SERVING SIZER AND FOOD GUIDE

We all want to enjoy a balanced diet. But what does a serving size actually look like? This Pocket Serving Sizer shows you. Used with Canada's Food Guide to Healthy Eating, it can make sure you're enjoying a variety of foods from the 4 Food Groups. We've included a synopsis of the Food Guide so you can keep track of meals eaten outside the home. (It's on the back of the Serving Sizer.)

HANDY SERVING SIZER

(With special thanks to the originator, Jane Kirby, R.D. of Glamour Magazine)

What's a serving size actually look like? Here's a handy guide:



A thumb equals 25 g of most cheeses. So 2 thumbs equal a serving.

A thumb-tip equals a teaspoon. Three thumb-tips equal a tablespoon, about the amount of milk you'd put in coffee. It would take 50 thumb-tips to equal 1 serving of milk.

A palm equals a serving of meat, fish or poultry. That's without fingers and thumb!

A fist equals a cup. A fist would be 1½ servings of yogourt. A fist size of raw leafy greens would be a serving of lettuce.

DAIRY BUREAU OF CANADA
1981 McGILL COLLEGE AVENUE, SUITE 1330
MONTREAL, QC H3A 2X9

September 1994

DIETARY DIARY SHEET

Name: _____ **Date** _____

Breakfast:-

Snacks:-

Lunch:-

Snacks:-

Dinner:-

Snacks:-

If you need extra space, please continue on another sheet of paper.

DIETARY DIARY SHEET

Name:_____ **Date**_____

Breakfast:-

Snacks:-

Lunch:-

Snacks:-

Dinner:-

Snacks:-

If you need extra space, please continue on another sheet of paper.

DIETARY DIARY SHEET

Name: _____ **Date** _____

Breakfast:-

Snacks:-

Lunch:-

Snacks:-

Dinner:-

Snacks:-

If you need extra space, please continue on another sheet of paper.

APPENDIX F

PEARSON PRODUCT MOMENT CORRELATION RESULTS

All Daughters

Significance levels with n=77 based on two-tailed test

p<.05 at 0.225
p<0.01 at 0.265
p<0.001 at 0.38

N=77

	HOURS	AGE	WEIGHT	LBM	HEIGHT	FNBMD	FNBMAD	TROCH	WARD	LSBMD	LSBMAD	TBMD	% BF	TOTAL	TRAB	CORT	TOT CSA	TRAB CSA	CORT CSA	INERTIA
HOURS TRAINING	1	-0.05819	-0.48351	-0.37626	-0.54235	0.348627	0.333356	0.399185	0.29237	0.20363	0.440223	-0.02216	-0.61828	0.563739	0.507288	0.505956311	0.10849493	-0.004413	0.3121663	0.11976
AGE	-0.05819	1	0.327764	0.428732	0.588045	0.236758	-0.22261	0.274384	0.017484	0.071686	-0.16299	0.289143	0.00811	-0.08742	-0.14233	0.100629866	0.19969222	0.2396637	0.0666452	0.18175
WEIGHT	-0.48351	0.327764	1	0.954343	0.72048	0.084911	-0.22072	-0.0014	-0.03701	0.100613	-0.2845	0.554122	0.760646	-0.4143	-0.20766	-0.32055771	0.29993573	0.386802	0.0571397	0.278897
LBM	-0.37626	0.428732	0.954343	1	0.760418	0.213558	-0.21323	0.120481	0.059546	0.184674	-0.2377	0.575467	0.541033	-0.36379	-0.17616	-0.24560109	0.38479491	0.4496219	0.1648169	0.362741
HEIGHT	-0.54235	0.588045	0.72048	0.760418	1	0.062606	-0.31609	0.03304	-0.07038	0.16207	-0.29704	0.431234	0.439247	-0.49525	-0.27216	-0.34833091	0.32494777	0.4001652	0.0923988	0.289594
FNBMD	0.348627	0.236758	0.084911	0.213558	0.062606	1	0.210224	0.743328	0.716904	0.59093	0.536917	0.507562	-0.25022	0.249918	0.426319	0.223642745	0.3016471	0.2438828	0.3454696	0.28528
FNBMAD	0.333356	-0.22261	-0.22072	-0.21323	-0.31609	0.210224	1	0.025378	0.217877	0.096016	0.170234	-0.02817	-0.22042	0.202252	0.287999	0.16419385	-0.01605684	-0.055849	0.0617933	-0.01709
TROCH BMD	0.399185	0.274384	-0.0014	0.120481	0.03304	0.743328	0.025378	1	0.590005	0.456473	0.407334	0.403415	-0.3041	0.311377	0.313394	0.345516393	0.23895664	0.2023981	0.261561	0.23459
WARD'S BMD	0.29237	0.017484	-0.03701	0.059546	-0.07038	0.716904	0.217877	0.590005	1	0.522858	0.544795	0.342687	-0.24862	0.349128	0.49112	0.21775578	0.09313961	0.0151112	0.2295717	0.085909
LSBMD	0.20363	0.071686	0.100613	0.184674	0.16207	0.59093	0.096016	0.456473	0.522858	1	0.810147	0.519334	-0.12217	0.029181	0.466659	-0.07806883	0.4303789	0.3914714	0.4041293	0.405532
LSBMAD	0.440223	-0.16299	-0.2845	-0.2377	-0.29704	0.536917	0.170234	0.407334	0.544795	0.810147	1	0.248804	-0.31774	0.235256	0.594129	0.052177181	0.25173833	0.1604326	0.3693411	0.229331
TBMD	-0.02216	0.289143	0.554122	0.575467	0.431234	0.507562	-0.02817	0.403415	0.342687	0.519334	0.248804	1	0.317554	-0.03003	0.328963	-0.05589366	0.39695485	0.3963035	0.3036979	0.384994
% BODY FAT	-0.61828	0.00811	0.760646	0.541033	0.439247	-0.25022	-0.22042	-0.3041	-0.24862	-0.12217	-0.31774	0.317554	1	-0.42132	-0.23151	-0.41265627	0.00189406	0.1081888	-0.206873	-0.00903
RAD TOT BMD	0.563739	-0.08742	-0.4143	-0.36379	-0.49525	0.249918	0.202252	0.311377	0.349128	0.029181	0.235256	-0.03003	-0.42132	1	0.424864	0.888558919	-0.4821128	-0.583769	-0.146446	-0.4499
RAD TRAB BMD	0.507288	-0.14233	-0.20766	-0.17616	-0.27216	0.426319	0.287999	0.313394	0.49112	0.466659	0.594129	0.328963	-0.23151	0.424864	1	0.165933965	0.27851212	0.1616763	0.4384014	0.280188
RAD CORT BMD	0.505956	0.10063	-0.32056	-0.2456	-0.34833	0.223643	0.164194	0.345516	0.217756	-0.07807	0.052177	-0.05589	-0.41266	0.888559	0.165934	1	-0.46065786	-0.476642	-0.298004	-0.44336
RAD TOT CSA	0.108495	0.199692	0.299936	0.384795	0.324948	0.301647	-0.01606	0.238957	0.09314	0.430379	0.251738	0.396955	0.001894	-0.48211	0.278512	-0.46065786	1	0.9580291	0.8286589	0.990258
RAD TRAB CSA	-0.00441	0.239664	0.386802	0.449622	0.400165	0.243883	-0.05585	0.202398	0.015111	0.391471	0.160433	0.396303	0.108189	-0.58377	0.161676	-0.47664244	0.95802912	1	0.6344048	0.934513
RAD CORT CSA	0.312166	0.066645	0.05714	0.164817	0.092399	0.34547	0.061793	0.261561	0.229572	0.404129	0.369341	0.303698	-0.20687	-0.14645	0.438401	-0.29800366	0.82865893	0.6344048	1	0.848128
MOM OF INERTIA	0.11976	0.18175	0.278897	0.362741	0.289594	0.28528	-0.01709	0.23459	0.085909	0.405532	0.229331	0.384994	-0.00903	-0.4499	0.280188	-0.44335947	0.99025812	0.9345128	0.8481285	1
GRIP LEFT	-0.2064	0.366267	0.325162	0.414746	0.460963	0.182749	-0.2472	0.220114	0.200453	0.171391	-0.05531	0.182924	0.038074	-0.20309	-0.17379	-0.10280153	0.13339754	0.1995112	-0.031593	0.101455
GRIP RIGHT	-0.27956	0.418613	0.321886	0.408586	0.471144	0.14764	-0.33707	0.210264	0.123565	0.104552	-0.13125	0.126459	0.038827	-0.15811	-0.231	-0.01491093	0.07723141	0.1556403	-0.098338	0.046141

All Mothers

N=76

	GS L	GS R	AGE	WEIGHT	LBM	HEIGHT	FNBMD	FNBMAD	TROCH	WARD	LSBMD	LSBMAD	TBMD	% BF	TOTAL	TRAB	CORT	TOT CSA	TRAB CSA	CORT CSA	INERTIA
GRIP LEFT	1	0.840673	-0.06888	0.520695	0.623515	0.434437	0.24373	-0.07311	0.077629	0.172785	0.250471	0.002405	0.383546	0.090391	0.030644	0.151610408	0.04614092	0.1995129	0.219118	0.128374	0.134607
GRIP RIGHT	0.840673	1	-0.1823	0.358928	0.517883	0.361779	0.246734	-0.09803	-0.08319	0.157498	0.24109	0.009667	0.271169	-0.07649	0.06577	0.093749401	0.08419546	0.1131655	0.1325966	0.059563	0.056112
AGE	-0.06888	-0.1823	1	0.013329	-0.02186	-0.08834	-0.0244	0.035785	0.161312	-0.19125	-0.21417	-0.24394	-0.05608	0.054531	0.147009	-0.02694261	0.19703374	-0.187278	-0.2286	-0.06013	-0.12513
WEIGHT	0.520695	0.358928	0.013329	1	0.899226	0.47643	0.424628	0.02179	0.238555	0.2081	0.242264	0.045137	0.484679	0.730029	0.044971	0.006051776	0.10718423	0.0467517	0.0653606	-0.00753	0.036829
LBM	0.623515	0.517883	-0.02186	0.899226	1	0.60238	0.458517	-0.07847	0.248371	0.219007	0.310207	-0.00721	0.550855	0.377317	0.049633	0.065300465	0.11092656	0.1824709	0.177131	0.169952	0.151292
HEIGHT	0.434437	0.361779	-0.08834	0.47643	0.60238	1	0.172823	-0.35283	0.200015	0.054644	0.293173	-0.0414	0.24878	0.104295	0.110742	0.123731863	0.13866491	0.2028112	0.1912855	0.198867	0.181456
FNBMD	0.24373	0.246734	-0.0244	0.424628	0.458517	0.172823	1	0.364542	0.537821	0.738273	0.392544	0.315836	0.682858	0.171245	0.25464	0.433808232	0.21194532	0.1158224	0.076162	0.201873	0.110045
FNBMAD	-0.07311	-0.09803	0.035785	0.02179	-0.07847	-0.35283	0.364542	1	0.197831	0.466897	0.119692	0.259168	0.269734	0.119464	0.011702	0.169916344	0.01402031	0.0075569	0.0095863	-0.00278	-0.03883
TROCH BMD	0.077629	-0.08319	0.161312	0.238555	0.248371	0.200015	0.537821	0.197831	1	0.497621	0.36631	0.243804	0.520936	0.100734	0.19527	0.351641724	0.19266404	0.119845	0.0743502	0.213522	0.123184
WARD'S BMD	0.172785	0.157498	-0.19125	0.2081	0.219007	0.054644	0.738273	0.466897	0.497621	1	0.441197	0.439059	0.673288	0.045378	0.231267	0.428956467	0.12826221	0.1250249	0.0950468	0.192302	0.096354
LSBMD	0.250471	0.24109	-0.21417	0.242264	0.310207	0.293173	0.392544	0.119692	0.36631	0.441197	1	0.799835	0.584249	0.003645	0.151956	0.273018638	0.16110511	0.2223815	0.196455	0.262781	0.190205
LSBMAD	0.002405	0.009667	-0.24394	0.045137	-0.00721	-0.0414	0.315836	0.259168	0.243804	0.439059	0.799835	1	0.509606	0.088789	0.226848	0.33653714	0.17712622	0.0319826	0.008197	0.101399	0.02779
TBMD	0.383546	0.271169	-0.05608	0.484679	0.550855	0.24878	0.682858	0.269734	0.520936	0.673288	0.584249	0.509606	1	0.139322	0.195184	0.379700893	0.18231934	0.2073197	0.1769062	0.2608	0.166333
% BODY FAT	0.090391	-0.07649	0.054531	0.730029	0.377317	0.104295	0.171245	0.119464	0.100734	0.045378	0.003645	0.088789	0.139322	1	-0.00839	-0.11839445	0.03736882	-0.17104	-0.124722	-0.26788	-0.13605
RAD TOT BMD	0.030644	0.06577	0.147009	0.044971	0.049633	0.110742	0.25464	0.011702	0.19527	0.231267	0.151956	0.226848	0.195184	-0.00839	1	0.512966925	0.90135223	-0.592044	-0.683595	-0.2268	-0.51773
RAD TRAB BMD	0.15161	0.093749	-0.02694	0.006052	0.0653	0.123732	0.433808	0.169916	0.351642	0.428956	0.273019	0.336537	0.379701	-0.11839	0.512967	1	0.22849274	-0.035524	-0.089137	0.111669	-0.00555
RAD CORT BMD	0.046141	0.084195	0.197034	0.107184	0.110927	0.138665	0.211945	0.01402	0.192664	0.128262	0.161105	0.177126	0.182319	0.037369	0.901352	0.228492741	1	-0.557948	-0.640873	-0.21922	-0.51815
RAD TOT CSA	0.199513	0.113166	-0.18728	0.046752	0.182471	0.202811	0.115822	0.007557	0.119845	0.125025	0.222381	0.031983	0.20732	-0.17104	-0.59204	-0.03552445	-0.55794789	1	0.9796805	0.855041	0.967943
RAD TRAB CSA	0.219118	0.132597	-0.2286	0.065361	0.177131	0.191285	0.076162	0.009586	0.07435	0.095047	0.196455	0.008197	0.176906	-0.12472	-0.68359	-0.08913675	-0.64087265	0.9796805	1	0.734572	0.922341
RAD CORT CSA	0.128374	0.059563	-0.06013	-0.00753	0.169952	0.198867	0.201873	-0.00278	0.213522	0.192302	0.262781	0.101399	0.2608	-0.26788	-0.2268	0.111669223	-0.21921602	0.8550411	0.7345716	1	0.885864
MOM OF INERTIA	0.134607	0.056112	-0.12513	0.036829	0.151292	0.181456	0.110045	-0.03883	0.123184	0.096354	0.190205	0.02779	0.166333	-0.13605	-0.51773	-0.00554995	-0.51815499	0.9679428	0.9223406	0.885864	1

Gymnast Daughters Alone

Significance levels with n=47 based on two-tailed test

p<.05 at 0.288
p<0.01 at 0.372
p<0.001 at 0.465

N=47

	HOURS	AGE	WEIGHT	LBM	HEIGHT	FNBMD	FNBMAD	TROCH	WARD	LSBMD	LSBMAD	TBMD	% BF	TOTAL	TRAB	CORT	TOT CSA	TRAB CSA	CORT CSA	INERTIA
HOURS	1	0.183463	-0.48174	-0.3516	-0.39357	0.478708	0.298587	0.523905	0.275181	0.156564	0.431847	0.084104	-0.71071	0.314467	0.373214	0.300676814	0.19733331	0.1061916	0.3206986	0.204479
AGE	0.183463	1	0.26288	0.377271	0.532701	0.222134	-0.1641	0.206131	-0.00276	0.040239	-0.09609	0.280127	-0.12132	0.038294	-0.07405	0.198907002	0.17889365	0.1762002	0.1405937	0.157468
WEIGHT	-0.48174	0.26288	1	0.961286	0.774585	-0.04523	-0.11774	-0.1803	-0.00773	0.242419	-0.25726	0.53891	0.726399	-0.40947	-0.14496	-0.34647346	0.3438825	0.4008973	0.1867117	0.308388
LBM	-0.3516	0.377271	0.961286	1	0.839242	0.11393	-0.18122	-0.03532	0.08065	0.319554	-0.17741	0.595809	0.519382	-0.37146	-0.11352	-0.28990459	0.41919693	0.4603848	0.2756305	0.379931
HEIGHT	-0.39357	0.532701	0.774585	0.839242	1	0.011358	-0.30185	-0.07803	-0.01561	0.302662	-0.18256	0.468307	0.380816	-0.3893	-0.14293	-0.3089032	0.31419263	0.3613691	0.16991	0.270783
FNBMD	0.478708	0.222134	-0.04523	0.11393	0.011358	1	-0.03449	0.688756	0.735028	0.518161	0.554774	0.445536	-0.46821	0.1944	0.44572	0.136782343	0.35656711	0.2899239	0.4163469	0.327871
FNBMAD	0.298587	-0.1641	-0.11774	-0.18122	-0.30185	-0.03449	1	-0.14985	0.058357	-0.12286	-0.03415	-0.08051	-0.02542	0.031177	0.187714	0.040231382	0.00143314	0.0166467	-0.030945	-0.0128
TROCH BMD	0.523905	0.206131	-0.1803	-0.03532	-0.07803	0.688756	-0.14985	1	0.632804	0.311122	0.399104	0.363828	-0.50673	0.386664	0.321548	0.353501466	0.15391065	0.0843744	0.2601381	0.152069
WARD'S BMD	0.275181	-0.00276	-0.00773	0.08065	-0.01561	0.735028	0.058357	0.632804	1	0.508457	0.562751	0.363222	-0.23751	0.204741	0.543309	0.066994754	0.26469559	0.2027304	0.33366	0.236995
LSBMD	0.156564	0.040239	0.242419	0.319554	0.302662	0.518161	-0.12286	0.311122	0.508457	1	0.761492	0.498016	-0.01304	-0.22921	0.373929	-0.32306418	0.49757947	0.4958644	0.4112156	0.445882
LSBMAD	0.431847	-0.09609	-0.25726	-0.17741	-0.18256	0.554774	-0.03415	0.399104	0.562751	0.761492	1	0.188555	-0.3655	0.016264	0.504115	-0.10836907	0.34390446	0.2860535	0.3816842	0.296053
TBMD	0.084104	0.280127	0.53891	0.595809	0.468307	0.445536	-0.08051	0.363828	0.363222	0.498016	0.188555	1	0.196125	-0.02727	0.332065	-0.08071625	0.39864741	0.3690552	0.3839337	0.377566
% BODY FAT	-0.71071	-0.12132	0.726399	0.519382	0.380816	-0.46821	-0.02542	-0.50673	-0.23751	-0.01304	-0.3655	0.196125	1	-0.37505	-0.15488	-0.40441988	0.01877555	0.0935977	-0.111585	0.01156
RAD TOT BMD	0.314467	0.038294	-0.40947	-0.37146	-0.3893	0.1944	0.031177	0.386664	0.204741	-0.22921	0.016264	-0.02727	-0.37505	1	0.222316	0.908971467	-0.58438924	-0.675334	-0.289263	-0.5497
RAD TRAB BMD	0.373214	-0.07405	-0.14496	-0.11352	-0.14293	0.44572	0.187714	0.321548	0.543309	0.373929	0.504115	0.332065	-0.15488	0.222316	1	-0.02395909	0.33065131	0.2449187	0.4217094	0.320471
RAD CORT BMD	0.300677	0.198907	-0.34647	-0.2899	-0.3089	0.136782	0.040231	0.353501	0.066995	-0.32306	-0.10837	-0.08072	-0.40442	0.908971	-0.02396	1	-0.60680756	-0.626365	-0.440885	-0.59115
RAD TOT CSA	0.197333	0.178894	0.343882	0.419197	0.314193	0.356567	0.001433	0.153911	0.264696	0.497579	0.343904	0.398647	0.018776	-0.58439	0.330651	-0.60680756	1	0.9628972	0.8688681	0.98949
RAD TRAB CSA	0.106192	0.1762	0.400897	0.460385	0.361369	0.289924	0.016647	0.084374	0.20273	0.495864	0.286053	0.369055	0.093598	-0.67533	0.244919	-0.62636532	0.96289718	1	0.7038406	0.936337
RAD CORT CSA	0.320699	0.140594	0.186712	0.27563	0.16991	0.416347	-0.03095	0.260138	0.33366	0.411216	0.381684	0.383934	-0.11159	-0.28926	0.421709	-0.44088542	0.86886806	0.7038406	1	0.889356
MOM OF INERTIA	0.204479	0.157468	0.308388	0.379931	0.270783	0.327871	-0.0128	0.152069	0.236995	0.445882	0.296053	0.377566	0.01156	-0.5497	0.320471	-0.59114962	0.98948976	0.9363373	0.8893561	1

Control Daughters Alone

Significance levels with n=30 based on two-tailed test

p<.05 at 0.349
p<0.01 at 0.4487
p<0.001 at 0.5541

N=30

	AGE	WEIGHT	LBM	HEIGHT	FNBMD	FNBMAD	TROCH	WARD	LSBMD	LSBMAD	TBMD	% BF	TOTAL	TRAB	CORT	TOT CSA	TRAB CSA	CORT CSA	INERTIA
AGE	1	0.302103	0.427504	0.634297	0.366871	-0.21129	0.525085	0.179319	0.219248	-0.12117	0.28009	-0.09048	0.016747	-0.03109	0.343499	0.257670727	0.32789928	0.0196687	0.2533571
WEIGHT	0.302103	1	0.945825	0.549317	0.371068	-0.17949	0.353395	0.104904	0.126324	-0.11975	0.596204	0.705815	-0.04875	0.072047	0.131507	0.31509102	0.38047951	0.0572579	0.3200604
LBM	0.427504	0.945825	1	0.627429	0.45578	-0.12025	0.442706	0.18758	0.181764	-0.14453	0.559307	0.446197	-0.05435	0.021573	0.202788	0.398021852	0.44059636	0.1675838	0.4076435
HEIGHT	0.634297	0.549317	0.627429	1	0.36304	-0.12334	0.466589	0.112094	0.274735	-0.16541	0.421357	0.171496	-0.14357	0.03575	0.250727	0.48536176	0.50420062	0.2933096	0.4674832
FNBMD	0.366871	0.371068	0.45578	0.36304	1	0.514941	0.803884	0.673034	0.660095	0.479938	0.634855	0.044083	0.276457	0.350928	0.327888	0.217850622	0.21091214	0.1607322	0.219363
FNBMAD	-0.21129	-0.17949	-0.12025	-0.12334	0.514941	1	0.172106	0.37015	0.288753	0.312342	0.096024	-0.23938	0.259619	0.274755	0.129325	-0.04969844	-0.13147058	0.1482517	-0.031508
TROCH BMD	0.525085	0.353395	0.442706	0.466589	0.803884	0.172106	1	0.48925	0.589593	0.340764	0.511422	-0.00406	-0.04808	0.162742	0.202367	0.413459313	0.44563154	0.2073874	0.4023547
WARD'S BMD	0.179319	0.104904	0.18758	0.112094	0.673034	0.37015	0.48925	1	0.50847	0.45055	0.379531	-0.12089	0.5733	0.281127	0.382569	-0.23382992	-0.26220511	-0.094604	-0.211253
LSBMD	0.219248	0.126324	0.181764	0.274735	0.660095	0.288753	0.589593	0.50847	1	0.868107	0.598398	-0.07543	0.245178	0.603121	0.102477	0.36913965	0.31160453	0.3855427	0.3807985
LSBMAD	-0.12117	-0.11975	-0.14453	-0.16541	0.479938	0.312342	0.340764	0.45055	0.868107	1	0.425214	-0.05182	0.281955	0.639619	-0.12177	0.128581139	0.05162804	0.265986	0.1399591
TBMD	0.28009	0.596204	0.559307	0.421357	0.634855	0.096024	0.511422	0.379531	0.598398	0.425214	1	0.431095	0.141738	0.572069	0.137039	0.412023876	0.43259228	0.2310393	0.4189533
% BODY FAT	-0.09048	0.705815	0.446197	0.171496	0.044083	-0.23938	-0.00406	-0.12089	-0.07543	-0.05182	0.431095	1	-0.01751	0.108688	-0.03994	-0.02474914	0.06600257	-0.23057	-0.033976
RAD TOT BMD	0.016747	-0.04875	-0.05435	-0.14357	0.276457	0.259619	-0.04808	0.5733	0.245178	0.281955	0.141738	-0.01751	1	0.421929	0.581575	-0.55485998	-0.60209384	-0.283392	-0.525665
RAD TRAB BMD	-0.03109	0.072047	0.021573	0.03575	0.350928	0.274755	0.162742	0.281127	0.603121	0.639619	0.572069	0.108688	0.421929	1	-0.06769	0.248106188	0.15321586	0.3745264	0.2665065
RAD CORT BMD	0.343499	0.131507	0.202788	0.250727	0.327888	0.129325	0.202367	0.382569	0.102477	-0.12177	0.137039	-0.03994	0.581575	-0.06769	1	-0.22590523	-0.12949837	-0.390622	-0.206525
RAD TOT CSA	0.257671	0.315091	0.398022	0.485362	0.217851	-0.0497	0.413459	-0.23383	0.36914	0.128581	0.412024	-0.02475	-0.55486	0.248106	-0.22591	1	0.9619743	0.7719084	0.992368
RAD TRAB CSA	0.327899	0.38048	0.440596	0.504201	0.210912	-0.13147	0.445632	-0.26221	0.311605	0.051628	0.432592	0.066003	-0.60209	0.153216	-0.1295	0.961974298	1	0.5699746	0.9483382
RAD CORT CSA	0.019669	0.057258	0.167584	0.29331	0.160732	0.148252	0.207387	-0.0946	0.385543	0.265986	0.231039	-0.23057	-0.28339	0.374526	-0.39062	0.771908385	0.56997457	1	0.7808602
MOM OF INERTIA	0.253357	0.32006	0.407643	0.467483	0.219363	-0.03151	0.402355	-0.21125	0.380798	0.139959	0.418953	-0.03398	-0.52566	0.266507	-0.20653	0.992368046	0.94833817	0.7808602	1

Mother-Daughter Correlations

Significance levels with n=77 based on two-tailed test

p<.05 at 0.225
p<0.01 at 0.265
p<0.001 at 0.38

N=76

	DAUGHTI																	
MOTHERS	WEIGHT	LBM	HEIGHT	FNBMD	FNBMAD	TROCH	WARD	LSBMD	LSBMAD	TBMD	% BF	TOTAL	TRAB	CORT	TOT CSA	TRAB CSA	CORT CSA	INERTIA
WEIGHT	0.440862	0.352621	0.060654	0.076207	0.155186	0.238474	-0.03537	0.160884	0.008511	0.202088	0.380472	0.022354	0.045663	0.019584	-0.03655	0.011058137	-0.15089595	-0.037528
LBM	0.370078	0.352425	0.076948	0.123087	0.134755	0.272332	-0.02617	0.134302	-0.00244	0.198159	0.23627	-0.01233	0.031545	0.009343	0.038529	0.073666531	-0.05810547	0.0301573
HEIGHT	0.187361	0.199103	0.174813	-0.04957	0.077448	0.228954	-0.15206	0.122284	0.112391	0.077378	0.119729	-0.08835	0.002163	-0.03468	0.074008	0.103919086	-0.01366125	0.0447095
FNBMD	0.022663	0.114112	-0.08206	0.279877	0.150279	0.247647	0.282245	0.10963	-0.01465	0.229228	-0.13373	0.065079	0.000145	0.136992	0.106002	0.052719645	0.22944201	0.1129269
FNBMAD	-0.02544	-0.00462	-0.12855	0.086106	0.102487	0.163861	0.16751	0.030175	0.08009	0.191454	-0.07027	0.104966	0.229658	0.091325	0.106318	0.06675471	0.19001846	0.1101204
TROCH BMD	-0.05837	0.008443	-0.07553	0.200642	0.084057	0.199514	0.184944	0.003327	-0.17163	0.077618	-0.1192	0.007358	-0.07201	0.105758	0.13769	0.09094736	0.24113709	0.1319206
WARD'S BMD	-0.00753	0.097978	-0.00369	0.251875	0.143373	0.149965	0.37436	0.110381	0.181325	0.272511	-0.16491	0.104298	0.058029	0.122976	0.027111	-0.0140659	0.13515509	0.0044254
LSBMD	0.06597	0.110423	0.028858	0.081037	0.117653	0.246136	0.21662	0.34032	0.17239	0.207491	-0.04838	-0.02773	-0.16046	0.061362	0.082976	0.049574801	0.16163087	0.0604332
LSBMAD	-0.14082	-0.07952	-0.08912	0.101761	0.065292	0.092756	0.22303	0.251787	0.124389	0.103327	-0.20429	0.029918	-0.09466	0.08158	0.032563	-0.02298443	0.17372592	0.0203719
TBMD	0.317232	0.318172	-0.0183	0.08527	0.156327	0.257272	0.088745	0.251384	0.0868	0.286918	0.18698	-0.07933	0.007195	-0.05958	0.182698	0.172392641	0.17967174	0.1707841
% BODY FAT	0.395386	0.201664	0.008613	-0.08707	0.101776	0.072216	-0.08035	0.124843	0.021241	0.103362	0.528548	0.052803	0.022377	-0.00399	-0.16717	-0.11035529	-0.27937864	-0.155103
RAD TOT BMD	-0.07755	-0.01113	0.076968	0.235081	-0.0939	-0.08199	0.267782	-0.06318	0.071902	0.107183	-0.17987	0.284644	0.188028	0.188765	-0.09712	-0.14834729	0.06608019	-0.081758
RAD TRAB BMD	0.013559	0.063926	-0.05597	0.210345	0.171589	0.064416	0.247186	0.185453	0.164742	0.23579	-0.08459	0.037673	0.127731	0.017595	0.239607	0.147394135	0.42967874	0.2679849
RAD CORT BMD	-0.06777	0.000103	0.086617	0.235702	-0.06257	-0.00238	0.191474	-0.15747	-0.10233	0.075303	-0.17228	0.22315	0.168687	0.174411	-0.11855	-0.14117452	-0.02775955	-0.1292
RAD TOT CSA	0.094187	0.086691	-0.01692	0.023866	0.127815	0.216144	-0.07428	0.175145	-0.08561	0.095743	0.066269	-0.36574	-0.11389	-0.22862	0.493997	0.477785862	0.41983731	0.4982954
RAD TRAB CSA	0.105107	0.081589	-0.05246	-0.01288	0.180327	0.214401	-0.10799	0.158554	-0.17504	0.082306	0.100369	-0.36866	-0.13446	-0.22236	0.421443	0.431174811	0.29633695	0.4069835
RAD CORT CSA	0.051128	0.077093	0.052907	0.086211	-0.0132	0.161868	0.012162	0.172444	0.100439	0.103309	-0.01852	-0.27371	-0.05268	-0.18758	0.511802	0.452015103	0.54934575	0.5484949
MOM OF INERTIA	0.102142	0.083914	-0.01967	0.030813	0.087579	0.215862	-0.07747	0.160772	-0.05678	0.089106	0.09517	-0.36785	-0.10172	-0.24613	0.506856	0.486091278	0.4394275	0.5287399

APPENDIX G

MULTIPLE REGRESSION RESULTS

All Daughters

FNBMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
FN	1	0.24286	0.058981	0.058981	4.826201	0.031119	1
AGE	2	0.324334	0.105193	0.046211	3.92494	0.051241	2
GS_L	3	0.351695	0.123689	0.018497	1.583077	0.212221	3

FNBMAD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HEIGHT	1	0.324335	0.105193	0.105193	9.052079	0.003571	1
GS_R	2	0.375641	0.141106	0.035913	3.177822	0.078691	2
FNBMAD	3	0.396795	0.157447	0.01634	1.454523	0.231594	3

TROCHANTER BMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
AGE	1	0.266175	0.070849	0.070849	5.871354	0.017835	1
TROCH	2	0.313774	0.098454	0.027605	2.327112	0.1314	2
HEIGHT	3	0.356391	0.127014	0.02856	2.453646	0.121519	3
GS_L	4	0.423462	0.17932	0.052306	4.71641	0.033081	4

WARD'S TRIANGLE BMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
WARD_S	1	0.347503	0.120758	0.120758	10.57548	0.001718	1
GS_L	2	0.421367	0.177551	0.056792	5.24798	0.024783	2
HEIGHT	3	0.436316	0.190372	0.012821	1.187685	0.279286	3

LSBMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
AVE__L_	1	0.368685	0.135929	0.135929	12.113	0.000833	1
GS_L	2	0.406871	0.165544	0.029615	2.697264	0.104652	2

LSBMAD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HEIGHT	1	0.287137	0.082447	0.082447	6.918893	0.010371	1
LSBMAD	2	0.359366	0.129144	0.046696	4.075219	0.04714	2
WEIGHT	3	0.387337	0.15003	0.020886	1.842965	0.178729	3
LBM	4	0.442711	0.195993	0.045963	4.230423	0.043231	4

WHOLE BODY BMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
LBM	1	0.506045	0.256082	0.256082	26.16179	2.44E-06	1
TBMD	2	0.542886	0.294725	0.038643	4.109353	0.046299	2
GS_R	3	0.557901	0.311253	0.016528	1.775807	0.18681	3
AGE	4	0.573222	0.328583	0.01733	1.884254	0.174054	4

TOTAL RADIAL BMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HEIGHT	1	0.495248	0.245271	0.245271	24.37336	4.86E-06	1
AGE	2	0.555666	0.308765	0.063494	6.797364	0.011063	2
TOTAL	3	0.597388	0.356873	0.048108	5.460634	0.022197	3

TRABECULAR RADIAL BMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HEIGHT	1	0.272162	0.074072	0.074072	5.999854	0.016673	1
TRAB_	2	0.299741	0.089845	0.015773	1.282385	0.261112	2

CORTICAL RADIAL BMD

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HEIGHT	1	0.348331	0.121334	0.121334	10.35671	0.001925	1
AGE	2	0.513774	0.263963	0.142629	14.3397	0.000311	2
CORT_	3	0.528123	0.278914	0.014951	1.513557	0.222547	3

TOTAL RADIAL CSA

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
TOTAL_A	1	0.488134	0.238275	0.238275	23.46068	7.08E-06	1
LBM	2	0.610005	0.372106	0.133832	15.77266	0.000167	2
WEIGHT	3	0.621874	0.386728	0.014621	1.740416	0.191266	3
GS_R	4	0.633303	0.401073	0.014346	1.724559	0.193277	4

TRABECULAR RADIAL CSA

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
LBM	1	0.449622	0.20216	0.20216	19.0038	4.15E-05	1
TRAB_A	2	0.596447	0.355749	0.153589	17.64151	7.35E-05	2

CORTICAL RADIAL CSA

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
CORT_A	1	0.548116	0.300432	0.300432	32.20897	2.72E-07	1
LBM	2	0.583476	0.340444	0.040013	4.489278	0.037559	2
WEIGHT	3	0.609623	0.37164	0.031196	3.624187	0.060942	3
GS_R	4	0.647189	0.418853	0.047213	5.849359	0.018113	4

MOMENT OF INERTIA - RADIAL

Summary of Stepwise Regression (mom&daug.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
INERTIA	1	0.519737	0.270127	0.270127	27.75757	1.37E-06	1
LBM	2	0.626125	0.392032	0.121905	14.83794	0.000252	2
WEIGHT	3	0.637898	0.406914	0.014881	1.831683	0.180163	3
GS_R	4	0.651476	0.424422	0.017508	2.190102	0.143263	4

Gymnasts Only

FNBMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HOURS	1	0.463868	0.215174	0.215174	12.61171	0.000928	1
GS_R	2	0.568569	0.323271	0.108097	7.18804	0.010292	2
FN	3	0.591772	0.350194	0.026924	1.82308	0.183852	3

FNBMAD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
GS_R	1	0.406537	0.165272	0.165272	9.107802	0.00418	1
FNBMAD	2	0.496839	0.246849	0.081576	4.874094	0.032402	2

TROCHANTER BMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HOURS	1	0.509828	0.259925	0.259925	16.15585	0.00022	1
GS_R	2	0.589413	0.347408	0.087483	6.03246	0.01797	2

WARD'S TRIANGLE BMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
WARD_S	1	0.311059	0.096758	0.096758	4.927658	0.031883	1
HOURS	2	0.389729	0.151888	0.05513	2.925162	0.094587	2
GS_L	3	0.464309	0.215583	0.063694	3.572782	0.065648	3
AGE	4	0.483134	0.233419	0.017836	1.00047	0.322925	4
HEIGHT	5	0.513508	0.26369	0.030272	1.726727	0.195961	5

LSBMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
AVE__L_	1	0.432137	0.186742	0.186742	10.56261	0.002276	1
GS_L	2	0.497737	0.247742	0.061	3.649025	0.062946	2
HOURS	3	0.551074	0.303683	0.055941	3.534881	0.067038	3
HEIGHT	4	0.581968	0.338687	0.035004	2.276035	0.138874	4
AGE	5	0.638421	0.407581	0.068894	4.884322	0.032605	5

LSBMAD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HOURS	1	0.399369	0.159495	0.159495	8.72902	0.005014	1
AGE	2	0.45964	0.211269	0.051774	2.953899	0.0927	2
GS_R	3	0.5162	0.266463	0.055193	3.310678	0.075639	3

WHOLE BODY BMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
LBM	1	0.591941	0.350394	0.350394	24.81211	1.03E-05	1
HOURS	2	0.661922	0.438141	0.087747	7.027803	0.011113	2
GS_L	3	0.716831	0.513847	0.075706	6.851916	0.012094	3

TOTAL RADIAL BMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
WEIGHT	1	0.409465	0.167662	0.167662	9.064563	0.004499	1
TOTAL	2	0.545312	0.297365	0.129703	8.122214	0.00688	2
LBM	3	0.567303	0.321833	0.024468	1.551394	0.220177	3
GS_L	4	0.614573	0.377699	0.055867	3.770522	0.05923	4
GS_R	5	0.65411	0.42786	0.050161	3.594559	0.065209	5
HEIGHT	6	0.694146	0.481838	0.053978	4.166888	0.047857	6

TRABECULAR RADIAL BND

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
HOURS	1	0.373214	0.139289	0.139289	7.282351	0.010066	1
TRAB_	2	0.403587	0.162882	0.023594	1.240107	0.271937	2
AGE	3	0.43919	0.192887	0.030005	1.598558	0.213248	3
HEIGHT	4	0.473461	0.224166	0.031278	1.693253	0.200438	4
GS_L	5	0.496059	0.246075	0.021909	1.191448	0.281411	5

CORTICAL RADIAL BMD

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
WEIGHT	1	0.346473	0.120044	0.120044	6.138913	0.017435	1
AGE	2	0.458672	0.21038	0.090336	5.03378	0.030322	2
HEIGHT	3	0.531513	0.282506	0.072126	4.322593	0.043906	3
CORT_	4	0.551668	0.304338	0.021832	1.31806	0.257595	4
LBM	5	0.570835	0.325852	0.021514	1.308458	0.259309	5

TOTAL RADIAL CSA

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
TOTAL_A	1	0.519046	0.269408	0.269408	16.59391	0.000195	1
LBM	2	0.614209	0.377253	0.107845	7.619738	0.008452	2
HOURS	3	0.692236	0.47919	0.101937	8.416292	0.00584	3

TRABECULAR RADIAL CSA

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
LBM	1	0.460385	0.211954	0.211954	12.10328	0.001166	1
TRAB_A	2	0.565257	0.319515	0.107561	6.954894	0.011587	2
HOURS	3	0.626522	0.392529	0.073014	5.168309	0.028055	3

CORTICAL RADIAL CSA

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
CORT_A	1	0.590856	0.349111	0.349111	24.13626	1.35E-05	1
LBM	2	0.637777	0.406759	0.057648	4.275651	0.044712	2
HOURS	3	0.715022	0.511256	0.104497	9.193735	0.004104	3

MOMENT OF INERTIA - RADIAL

Summary of Stepwise Regression (mom&dau2.sta)

	Step +in/-out	Multiple R	Multiple R-square	R-square change	F - to entr/rem	p-level	Variabls included
INERTIA	1	0.54707	0.299286	0.299286	19.22017	7.38E-05	1
LBM	2	0.632454	0.399998	0.100712	7.385535	0.009438	2
HOURS	3	0.697126	0.485985	0.085987	7.19323	0.01034	3

APPENDIX H

ANALYSIS OF VARIANCE RESULTS

Tukey HSD post-hoc analysis included where necessary

LEGEND
{1} = ELITE GYMNAST GROUP
{2} = HIGH RECREATION GYMNAST GROUP
{3} = LOW RECREATION GYMNAST GROUP
{4} = CONTROL GROUP
{5} = MATCHED CONTROL GROUP

Daughters

Anthropometric Variables

AGE

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	4.305777	74	0.70033	6.148213	0.000251

Tukey HSD test; variable AGE (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		10.01749	8.693319	9.559982	9.962438	9.758533
1	{1}		0.000348	0.51308	0.999713	0.897596
2	{2}	0.000348		0.051296	0.000939	0.008747
3	{3}	0.51308	0.051296		0.668586	0.966212
4	{4}	0.999713	0.000939	0.668586		0.960656
5	{5}	0.897596	0.008747	0.966212	0.960656	

WEIGHT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	470.9945	74	17.37103	27.11379	7.25E-14

Tukey HSD test; variable WEIGHT (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		27.14737	25.44286	32.98667	38.55000	26.95333
1	{1}		0.773293	0.001228	0.000125	0.999932
2	{2}	0.773293		0.000175	0.000125	0.865569
3	{3}	0.001228	0.000175		0.00362	0.001635
4	{4}	0.000125	0.000125	0.00362		0.000125
5	{5}	0.999932	0.865569	0.001635	0.000125	

% BODY FAT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	277.7827	74	11.75184	23.63737	1.29E-12

Tukey HSD test; variable %_BF (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		15.23158	17.01429	21.22667	25.75625	18.79333
1	{1}		0.580929	0.000148	0.000125	0.028758
2	{2}	0.580929		0.012426	0.000125	0.631875
3	{3}	0.000148	0.012426		0.004066	0.303856
4	{4}	0.000125	0.000125	0.004066		0.000126
5	{5}	0.028758	0.631875	0.303856	0.000126	

Tanner Staging

BREAST ASSESSMENT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.68799	74	0.231713	2.969146	0.024817

Tukey HSD test; variable WB_2WK (bmddat8b.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CAT

		{1}	{2}	{3}	{4}	{5}
		41.01105	26.81786	11.62200	5.028750	16.49733
1	{1}		0.12986	0.000149	0.000125	0.000777
2	{2}	0.12986		0.120316	0.006384	0.473129
3	{3}	0.000149	0.120316		0.812392	0.932339
4	{4}	0.000125	0.006384	0.812392		0.330848
5	{5}	0.000777	0.473129	0.932339	0.330848	

LEAN BODY MASS

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	144.0191	74	7.957688	18.09811	2.1E-10

Tukey HSD test; variable LBM (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		22.99225	21.11039	25.80007	28.50389	21.87931
1	{1}		0.32975	0.040226	0.000126	0.783633
2	{2}	0.32975		0.000364	0.000125	0.948042
3	{3}	0.040226	0.000364		0.068928	0.002718
4	{4}	0.000126	0.000125	0.068928		0.000125
5	{5}	0.783633	0.948042	0.002718	0.000125	

HEIGHT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	584.3096	74	35.46124	16.47742	1.07E-09

Tukey HSD test; variable HEIGHT (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		130.8053	126.1357	136.8867	142.2563	136.3333
1	{1}		0.181657	0.033018	0.000126	0.065548
2	{2}	0.181657		0.000178	0.000125	0.000268
3	{3}	0.033018	0.000178		0.099723	0.999108
4	{4}	0.000126	0.000125	0.099723		0.053849
5	{5}	0.065548	0.000268	0.999108	0.053849	

PUBIC HAIR ASSESSMENT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.290836	74	0.129336	2.248693	0.071867

Grip Strength

LEFT HAND

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	13.11967	74	8.77355[illegible]2	1.495366	0.21231

RIGHT HAND

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	21.5249	74	9.733003	2.211538	0.075888

DXA Proximal Femur

FNBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.009654	74	0.00374[illegible]8	2.575498	0.044426

Tukey HSD test; variable FNBMD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .7058421	{2} .6884286	{3} .6488000	{4} .6789375	{5} .6524667
1	{1}		0.927592	0.064013	0.695089	0.09636
2	{2}	0.927592		0.415326	0.993199	0.514473
3	{3}	0.064013	0.415326		0.648847	0.999852
4	{4}	0.695089	0.993199	0.648847		0.74969
5	{5}	0.09636	0.514473	0.999852	0.74969	

FNBMAD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.005101	74	0.00174	2.93111	0.026255

Tukey HSD test; variable FNBMAD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .2323985	{2} .2311983	{3} .2054184	{4} .1907030	{5} .2116945
1	{1}		0.999991	0.34123	0.034	0.606186
2	{2}	0.999991		0.462991	0.071355	0.717414
3	{3}	0.34123	0.462991		0.862844	0.993884
4	{4}	0.034	0.071355	0.862844		0.629597
5	{5}	0.606186	0.717414	0.993884	0.629597	

TROCHANTER BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.0188	74	0.005377	3.496185	0.01138

Tukey HSD test; variable TROCH (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .6147895	{2} .5651429	{3} .5362000	{4} .5634375	{5} .5327333
1	{1}		0.314975	0.022187	0.246866	0.015073
2	{2}	0.314975		0.825214	0.999997	0.757479
3	{3}	0.022187	0.825214		0.839079	0.999942
4	{4}	0.246866	0.999997	0.839079		0.771106
5	{5}	0.015073	0.757479	0.999942	0.771106	

WARD'S TRIANGLE BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.011862	74	0.006007	1.974535	0.107197

DXA Lumbar Spine

LSBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.00369	74	0.004244	0.869478	0.486428

LSBMAD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.002602	74	0.000409	6.364097	0.000186

Tukey HSD test; variable LSBMAD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .2325002	{2} .2392879	{3} .2116354	{4} .2097384	{5} .2189931
1	{1}		0.87496	0.030389	0.012026	0.308805
2	{2}	0.87496		0.004017	0.001492	0.063437
3	{3}	0.030389	0.004017		0.999013	0.856137
4	{4}	0.012026	0.001492	0.999013		0.708113
5	{5}	0.308805	0.063437	0.856137	0.708113	

DXA Whole Body

WHOLE BODY BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.004218	74	0.002343	1.800673	0.137754

WHOLE BODY BMAD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.00071	74	4.50E-05	15.8837	2.00E-09

Tukey HSD test; variable TBMAD (bmddat8b.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CAT

		{1} 0.100254	{2} 0.099807	{3} 0.090203	{4} 0.084928	{5} 0.096667
1	{1}		0.99974	0.00051	0.00012	0.53121
2	{2}	0.99974		0.00225	0.00012	0.71346
3	{3}	0.00051	0.00225		0.19265	0.07189
4	{4}	0.00012	0.00012	0.19265		0.00017
5	{5}	0.53121	0.71346	0.07189	0.00017	

pQCT Distal Radius

TOTAL RADIAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	16760.61	72	1692.113	9.905138	1.92E-06

Tukey HSD test; variable TOTAL (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		360.5000	373.1000	335.1933	296.6063	306.4214
1	{1}		0.910642	0.404945	0.000332	0.003964
2	{2}	0.910642		0.106783	0.000147	0.000628
3	{3}	0.404945	0.106783		0.079224	0.336264
4	{4}	0.000332	0.000147	0.079224		0.965774
5	{5}	0.003964	0.000628	0.336264	0.965774	

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	15158.52	72	2556.263	5.929952	0.00035

Tukey HSD test; variable CORT_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		484.2833	480.6643	455.5133	426.1437	414.5714
1	{1}		0.99967	0.485037	0.011161	0.002273
2	{2}	0.99967		0.66829	0.034122	0.008007
3	{3}	0.485037	0.66829		0.492138	0.19949
4	{4}	0.011161	0.034122	0.492138		0.970575
5	{5}	0.002273	0.008007	0.19949	0.970575	

RADIAL TRABECULAR CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	1534.974	72	561.816	2.732165	0.035468

Tukey HSD test; variable TRAB_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.04333	65.61643	76.83733	89.61375	72.47929
1	{1}		0.093893	0.733084	0.997847	0.425916
2	{2}	0.093893		0.707947	0.054233	0.939543
3	{3}	0.733084	0.707947		0.566049	0.987677
4	{4}	0.997847	0.054233	0.566049		0.288506
5	{5}	0.425916	0.939543	0.987677	0.288506	

MOMENT OF INERTIA - RADIAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	15001699	72	5440156	2.757586	0.034163

Tukey HSD test; variable INERTIA (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		6883.640	4545.872	5245.907	6257.395	4933.094
1	{1}		0.048293	0.272561	0.935242	0.142398
2	{2}	0.048293		0.927521	0.274143	0.992183
3	{3}	0.272561	0.927521		0.747596	0.996363
4	{4}	0.935242	0.274143	0.747596		0.533079
5	{5}	0.142398	0.992183	0.996363	0.533079	

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8647.679	72	1189.413	7.270544	5.64E-05

Tukey HSD test; variable TRAB_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		211.1889	212.6071	175.8867	162.6750	171.0500
1	{1}		0.999964	0.035843	0.001114	0.014103
2	{2}	0.999964		0.042241	0.001741	0.017613
3	{3}	0.035843	0.042241		0.823324	0.995659
4	{4}	0.001114	0.001741	0.823324		0.963544
5	{5}	0.014103	0.017613	0.995659	0.963544	

TOTAL RADIAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3459.271	72	1312.594	2.635445	0.0409

Tukey HSD test; variable TOTAL_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		195.5533	159.9264	170.8940	186.7325	166.6907
1	{1}		0.055177	0.302624	0.953955	0.178653
2	{2}	0.055177		0.92538	0.266313	0.987751
3	{3}	0.302624	0.92538		0.741969	0.997936
4	{4}	0.953955	0.266313	0.741969		0.558513
5	{5}	0.178653	0.987751	0.997936	0.558513	

RADIAL CORTICAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	488.1386	72	157.8831	3.091773	0.020869

Tukey HSD test; variable CORT_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.74000	76.55714	75.32800	76.40875	75.29000
1	{1}		0.102625	0.046747	0.076552	0.052362
2	{2}	0.102625		0.998979	1	0.998924
3	{3}	0.046747	0.998979		0.999324	1
4	{4}	0.076552	1	0.999324		0.999264
5	{5}	0.052362	0.998924	1	0.999264	

Weight-Bearing Activity

NON-WEIGHT-BEARING ACTIVITY - ANNUAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	62.12955	48	1597.118	0.038901	0.997019

WEIGHT-BEARING ACTIVITY - ANNUAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	365163.4	74	50223.97	7.2707	5.4E-05

Tukey HSD test; variable WB_YEAR (bmddat8b.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CAT

		{1}	{2}	{3}	{4}	{5}
		410.1274	450.6021	246.4500	137.7919	118.2393
1	{1}		0.985913	0.225036	0.00546	0.003035
2	{2}	0.985913		0.113404	0.002654	0.001505
3	{3}	0.225036	0.113404		0.661748	0.52326
4	{4}	0.00546	0.002654	0.661748		0.999286
5	{5}	0.003035	0.001505	0.52326	0.999286	

WEIGHT-BEARING ACTIVITY - 2 WEEKS

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3446.705	74	284.4904	12.11536	1.26E-07

Tukey HSD test; variable WB_2WK (bmddat8b.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CAT

		{1}	{2}	{3}	{4}	{5}
		41.01105	26.81786	11.62200	5.028750	16.49733
1	{1}		0.12986	0.000149	0.000125	0.000777
2	{2}	0.12986		0.120316	0.006384	0.473129
3	{3}	0.000149	0.120316		0.812392	0.932339
4	{4}	0.000125	0.006384	0.812392		0.330848
5	{5}	0.000777	0.473129	0.932339	0.330848	

Dietary Intakes

PROTEIN

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	57.68961	62	226.2069	0.25503	0.905523

ENERGY

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	26737.65	62	186167.3	0.143622	0.965153

FAT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	71.04656	62	446.9239	0.158968	0.958197

CALCIUM

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	41855.23	62	111651.1	0.374875	0.8257

CARBOHYDRATES

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	1773.364	62	3856.5	0.459838	0.764877

VITAMIN D

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	7.21932	62	6.33989	1.138714	0.346691

DIETARY FIBRE

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3.561939	62	20.83428	0.170965	0.952435

PHOSPHORUS

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	59107.82	62	84611.98	0.698575	0.595856

Mothers

Anthropometric Variables

AGE

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	66.55576	74	19.01804	3.499612	0.011323

Tukey HSD test; variable AGE (momsstat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CATEGORY

		{1}	{2}	{3}	{4}	{5}
		38.55305	37.53536	37.61500	42.57344	38.99854
1	{1}		0.96376	0.971042	0.061025	0.998377
2	{2}	0.96376		0.999999	0.019091	0.894976
3	{3}	0.971042	0.999999		0.018727	0.907426
4	{4}	0.061025	0.019091	0.018727		0.16283
5	{5}	0.998377	0.894976	0.907426	0.16283	

HEIGHT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	90.48453	74	29.15994	3.103042	0.020354

Tukey HSD test; variable HEIGHT (momsstat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CATEGORY

		{1}	{2}	{3}	{4}	{5}
		159.2789	164.4143	165.2000	162.3125	161.9933
1	{1}		0.063619	0.018157	0.467523	0.594422
2	{2}	0.063619		0.994979	0.824509	0.747717
3	{3}	0.018157	0.994979		0.573628	0.485761
4	{4}	0.467523	0.824509	0.573628		0.999851
5	{5}	0.594422	0.747717	0.485761	0.999851	

WEIGHT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	264.5316	74	161.3896	1.639087	0.17345

LEAN BODY MASS

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	72.38992	73	37.62021	1.92423	0.115457

% BODY FAT
Summary of all Effects; design: (momsstat.sta)
1-CATEGORY

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	48.42332	73	35.94064	1.347314	0.260705

Grip Strength

LEFT HAND

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	98.04087	74	34.65243	2.829264	0.030527

Tukey HSD test; variable GS_L (momsstat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CATEGORY

		{1}	{2}	{3}	{4}	{5}
		32.52632	36.50000	37.83333	33.09375	32.40000
1	{1}		0.317916	0.078922	0.998614	0.999997
2	{2}	0.317916		0.973227	0.514135	0.340283
3	{3}	0.078922	0.973227		0.176696	0.095556
4	{4}	0.998614	0.514135	0.176696		0.997503
5	{5}	0.999997	0.340283	0.095556	0.997503	

RIGHT HAND

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	109.886	74	33.65994	3.264593	0.016026

Tukey HSD test; variable GS_R (momsstat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: CATEGORY

		{1}	{2}	{3}	{4}	{5}
		36.47368	39.03571	40.43333	34.00000	35.06667
1	{1}		0.720015	0.287908	0.71833	0.955439
2	{2}	0.720015		0.966493	0.134756	0.358461
3	{3}	0.287908	0.966493		0.023292	0.094358
4	{4}	0.71833	0.134756	0.023292		0.986036
5	{5}	0.955439	0.358461	0.094358	0.986036	

DXA Proximal Femur

FNBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.003934	74	0.009038	0.435282	0.782708

FNBMAD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.005038	74	0.002189	2.301558	0.066504

TROCHANTER BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.011704	74	0.012666	0.924059	0.454689

WARD'S TRIANGLE BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.011514	74	0.011571	0.99502	0.415763

DXA Lumbar Spine

LSBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.010168	74	0.016813	0.604755	0.660426

LSBMAD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.008844	74	0.006476	1.365745	0.254057

DXA Whole Body

WHOLE BODY BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.000265	73	0.003454	0.076816	0.989107

WHOLE BODY BMAD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	6.14E-05	73	3.7E-05	1.661293	0.168237

pQCT Distal Radius

TOTAL RADIAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	556.7651	72	4406.315	0.126356	0.972461

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	445.6461	72	7675.901	0.058058	0.993612

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	505.5212	72	1293.021	0.390961	0.814459

MOMENT OF INERTIA - RADIAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	22350730	72	45077600	0.495828	0.73881

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	769.3937	72	1767.564	0.435285	0.782691

TOTAL RADIAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	1540.784	72	2547.25	0.604881	0.66037

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	294.0515	72	178.215	1.649982	0.171123

Weight-Bearing Activity

NON-WEIGHT-BEARING ACTIVITY - ANNUAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3973.236	74	2608.758	1.523037	0.204253

WEIGHT-BEARING ACTIVITY - 2 WEEKS

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8.471183	69	12.78411	0.662634	0.620071

WEIGHT-BEARING ACTIVITY - ANNUAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	35502.78	74	28204.09	1.258781	0.293902

Dietary Intake

PROTEIN

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	648.2603	63	300.5501	2.156913	0.084121

CARBOHYDRATE

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	17594.46	63	3432.462	5.125902	0.001221

Tukey HSD test; variable CHO (dietary2.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: VAR1

		{1} 234.7692	{2} 248.7857	{3} 258.2000	{4} 186.3333	{5} 283.4546
1	{1}		0.971227	0.828378	0.200128	0.264738
2	{2}	0.971227		0.992612	0.043058	0.586376
3	{3}	0.828378	0.992612		0.011341	0.813164
4	{4}	0.200128	0.043058	0.011341		0.000967
5	{5}	0.264738	0.586376	0.813164	0.000967	

CALCIUM

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	222085.6	63	136589.3	1.625937	0.178772

PHOSPHORUS

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	203052.7	63	110680	1.834592	0.133279

FAT

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	974.4592	63	563.7188	1.728626	0.154797

ENERGY

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	686083.8	63	163019.8	4.208591	0.0044

Tukey HSD test; variable ENERGY (dietary2.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: VAR1

		{1} 1682.538	{2} 1872.143	{3} 1973.600	{4} 1476.333	{5} 2007.182
1	{1}		0.740343	0.326856	0.662791	0.296152
2	{2}	0.740343		0.960909	0.075641	0.920352
3	{3}	0.326856	0.960909		0.010905	0.999607
4	{4}	0.662791	0.075641	0.010905		0.01298
5	{5}	0.296152	0.920352	0.999607	0.01298	

VITAMIN D

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	9.982892	63	5.838405	1.709867	0.158936

DIETARY FIBRE

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	39.27724	63	18.62392	2.108968	0.090113

APPENDIX I

ANALYSIS OF COVARIANCE RESULTS

Tukey HSD post-hoc analysis and adjusted means included where necessary

LEGEND
{1} = ELITE GYMNAST GROUP
{2} = HIGH RECREATION GYMNAST GROUP
{3} = LOW RECREATION GYMNAST GROUP
{4} = CONTROL GROUP
{5} = MATCHED CONTROL GROUP

Daughters

Variables Controlled for Age

FNBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.01045	73	0.003534	2.956626	0.025376

Adjusted means (bmd7stat.sta)
F(4,73)=2.96; p<.0254

	FNBMD
1	0.697737
2	0.705931
3	0.649542
4	0.671897
5	0.649369

Tukey HSD test; variable FNBMD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .7058421	{2} .6884286	{3} .6488000	{4} .6789375	{5} .6524667
1	{1}		0.920009	0.052573	0.671305	0.081088
2	{2}	0.920009		0.385133	0.992388	0.484875
3	{3}	0.052573	0.385133		0.623018	0.999834
4	{4}	0.671305	0.992388	0.623018		0.728809
5	{5}	0.081088	0.484875	0.999834	0.728809	

TROCHANTER BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.017331	73	0.005072	3.416893	0.012859

Adjusted means (bmd7stat.sta)
F(4,73)=3.42; p<.0129

	TROCH
1	0.605109
2	0.586046
3	0.537086
4	0.555029
5	0.529034

Tukey HSD test; variable TROCH (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .6147895	{2} .5651429	{3} .5362000	{4} .5634375	{5} .5327333
1	{1}		0.286496	0.017205	0.220828	0.011467
2	{2}	0.286496		0.80934	0.999996	0.737206
3	{3}	0.017205	0.80934		0.824209	0.999935
4	{4}	0.220828	0.999996	0.824209		0.751651
5	{5}	0.011467	0.737206	0.999935	0.751651	

WARD'S TRIANGLE BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.013643	73	0.005992	2.276937	0.069095

LSBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.004782	73	0.004221	1.133006	0.347745

WHOLE BODY BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.002181	73	0.002282	0.955677	0.437103

TOTAL RADIAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	16817.16	71	1692.429	9.9367	1.92E-06

Adjusted means (bmd7stat.sta)
F(4,71)=9.94; p<.0000

	TOTAL
1	358.3621
2	378.218
3	335.2778
4	294.3533
5	305.6097

Tukey HSD test; variable TOTAL (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} 360.5000	{2} 373.1000	{3} 335.1933	{4} 296.6063	{5} 306.4214
1	{1}		0.910656	0.405146	0.000336	0.003998
2	{2}	0.910656		0.107	0.000149	0.000636
3	{3}	0.405146	0.107		0.07942	0.336486
4	{4}	0.000336	0.000149	0.07942		0.965777
5	{5}	0.003998	0.000636	0.336486	0.965777	

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8059.063	71	1205.023	6.687892	0.000126

Tukey HSD test; variable TRAB_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		211.1889	212.6071	175.8867	162.6750	171.0500
1	{1}		0.999964	0.0378	0.001221	0.015059
2	{2}	0.999964		0.044428	0.001901	0.018761
3	{3}	0.0378	0.044428		0.826735	0.995775
4	{4}	0.001221	0.001901	0.826735		0.964378
5	{5}	0.015059	0.018761	0.995775	0.964378	

Adjusted means (bmd7stat.sta)
F(4,71)=6.69; p<.0001

	TRAB_
1	211.6601
2	211.4791
3	175.868
4	163.1716
5	171.2289

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	17609.33	71	2419.295	7.278706	5.7E-05

Tukey HSD test; variable CORT_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		484.2833	480.6643	455.5133	426.1437	414.5714
1	{1}		0.999631	0.457015	0.008508	0.001614
2	{2}	0.999631		0.644901	0.027468	0.006027
3	{3}	0.457015	0.644901		0.464188	0.177295
4	{4}	0.008508	0.027468	0.464188		0.967457
5	{5}	0.001614	0.006027	0.177295	0.967457	

Adjusted means (bmd7stat.sta)
F(4,71)=7.28; p<.0001

	CORT_
1	478.4851
2	494.545
3	455.7425
4	420.0335
5	412.37

TOTAL RADIAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	2503.601	71	1324.071	1.890836	0.121493

RADIAL TRABECULAR CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	974.4129	71	563.6181	1.728853	0.153171

RADIAL CORTICAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	473.4299	71	160.1022	2.957048	0.025557

Tukey HSD test; variable CORT_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.74000	76.55714	75.32800	76.40875	75.29000
1	{1}		0.106852	0.049258	0.080067	0.055086
2	{2}	0.106852		0.999006	1	0.998952
3	{3}	0.049258	0.999006		0.999342	1
4	{4}	0.080067	1	0.999342		0.999297
5	{5}	0.055086	0.998952	1	0.999297	

Adjusted means (bmd7stat.sta)
F(4,71)=2.96; p<.0256

	CORT_A
1	87.76994
2	76.48546
3	75.32681
4	76.44031
5	75.30137

MOMENT OF INERTIA - RADIAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	11519280	71	5502817	2.093343	0.090668

Variables Controlled for Weight

FNBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.013257	73	0.003577	3.706007	0.008402

Adjusted means (bmd7stat.sta)
F(4,73)=3.71; p<.0084

	FNBMD
1	0.716752
2	0.705399
3	0.63895
4	0.649308
5	0.664067

Tukey HSD test; variable FNBMD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.7058421	.6884286	.6488000	.6789375	.6524667
1	{1}		0.921621	0.054817	0.676277	0.084116
2	{2}	0.921621		0.391334	0.992561	0.490996
3	{3}	0.054817	0.391334		0.628403	0.999838
4	{4}	0.676277	0.992561	0.628403		0.733189
5	{5}	0.084116	0.490996	0.999838	0.733189	

TROCHANTER BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.019835	73	0.005393	3.677652	0.008759

Adjusted means (bmd7stat.sta)
F(4,73)=3.68; p<.0088

	TROCH
1	0.620334
2	0.573766
3	0.531194
4	0.548381
5	0.538628

Tukey HSD test; variable TROCH (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6147895	.5651429	.5362000	.5634375	.5327333
1	{1}		0.316592	0.022556	0.248388	0.01535
2	{2}	0.316592		0.82599	0.999997	0.758488
3	{3}	0.022556	0.82599		0.839803	0.999943
4	{4}	0.248388	0.999997	0.839803		0.772072
5	{5}	0.01535	0.758488	0.999943	0.772072	

WARD'S TRIANGLE BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.019936	73	0.005634	3.538461	0.01075

Adjusted means (bmd7stat.sta)
F(4,73)=3.54; p<.0108

	WARD_S
1	0.641923
2	0.677062
3	0.580642
4	0.538364
5	0.625127

Tukey HSD test; variable WARD_S (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6263158	.6527857	.5947334	.5807500	.6085333
1	{1}		0.854056	0.740926	0.387826	0.95898
2	{2}	0.854056		0.239384	0.076813	0.510826
3	{3}	0.740926	0.239384		0.985325	0.986844
4	{4}	0.387826	0.076813	0.985325		0.840778
5	{5}	0.95898	0.510826	0.986844	0.840778	

LSBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.011951	73	0.003813	3.134527	0.019509

Adjusted means (bmd7stat.sta)
F(4,73)=3.13; p<.0195

	AVE__L_S
1	0.686983
2	0.699852
3	0.634062
4	0.597506
5	0.663088

Tukey HSD test; variable AVE__L_S (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6708070	.6746905	.6486667	.6414375	.6458889
1	{1}		0.999793	0.836867	0.628564	0.769262
2	{2}	0.999793		0.788016	0.584088	0.719168
3	{3}	0.836867	0.788016		0.997565	0.999953
4	{4}	0.628564	0.584088	0.997565		0.999672
5	{5}	0.769262	0.719168	0.999953	0.999672	

WHOLE BODY BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.005237	73	0.001558	3.36001	0.013985

Adjusted means (bmd7stat.sta)
F(4,73)=3.36; p<.0140

	TBMD
1	0.812418
2	0.796067
3	0.765804
4	0.750013
5	0.795479

Tukey HSD test; variable TBMD (bmd7stat.sta)

		{1}	{2}	{3}	{4}	{5}
1	{1}	.7915263	.7635714	.7846667	.8067500	.7732667
2	{2}		0.27148	0.986885	0.786734	0.667962
3	{3}	0.27148		0.605553	0.030398	0.964079
4	{4}	0.986885	0.605553		0.52985	0.932549
5	{5}	0.786734	0.030398	0.52985		0.138346
		0.667962	0.964079	0.932549	0.138346	

TOTAL RADIAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	9473.05	71	1669.897	5.672836	0.000509

Tukey HSD test; variable TOTAL (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		360.5000	373.1000	335.1933	296.6063	306.4214
1	{1}		0.908639	0.398248	0.000314	0.003704
2	{2}	0.908639		0.103089	0.000146	0.000587
3	{3}	0.398248	0.103089		0.076204	0.329763
4	{4}	0.000314	0.000146	0.076204		0.964943
5	{5}	0.003704	0.000587	0.329763	0.964943	

Adjusted means (bmd7stat.sta)
F(4,71)=5.67; p<.0005

	TOTAL
1	355.1611
2	365.2892
3	339.6201
4	310.0577
5	301.6928

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8314.42	71	1151.917	7.217899	6.18E-05

Tukey HSD test; variable TRAB_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		211.1889	212.6071	175.8867	162.6750	171.0500
1	{1}		0.999961	0.031733	0.000922	0.012171
2	{2}	0.999961		0.037551	0.001413	0.015288
3	{3}	0.031733	0.037551		0.814675	0.99538
4	{4}	0.000922	0.001413	0.814675		0.961386
5	{5}	0.012171	0.015288	0.99538	0.961386	

Adjusted means (bmd7stat.sta)
F(4,71)=7.22; p<.0001

	TRAB_
1	216.9836
2	221.0849
3	171.082
4	148.0749
5	176.1824

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	10940.78	71	2475.757	4.419168	0.003018

Tukey HSD test; variable CORT_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		484.2833	480.6643	455.5133	426.1437	414.5714
1	{1}		0.999648	0.468818	0.009563	0.001894
2	{2}	0.999648		0.654822	0.030158	0.006815
3	{3}	0.468818	0.654822		0.475964	0.186499
4	{4}	0.009563	0.030158	0.475964		0.968785
5	{5}	0.001894	0.006815	0.186499	0.968785	

Adjusted means (bmd7stat.sta)
F(4,71)=4.42; p<.0030

	CORT_
1	475.7911
2	468.2401
3	462.5547
4	447.5404
5	407.0499

TOTAL RADIAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	4205.672	71	1151.752	3.651543	0.009212

Tukey HSD test; variable TOTAL_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		195.5533	159.9264	170.8940	186.7325	166.6907
1	{1}		0.03429	0.24092	0.942109	0.13103
2	{2}	0.03429		0.907102	0.207725	0.984342
3	{3}	0.24092	0.907102		0.693016	0.997336
4	{4}	0.942109	0.207725	0.693016		0.493839
5	{5}	0.13103	0.984342	0.997336	0.493839	

Adjusted means (bmd7stat.sta)
F(4,71)=3.65; p<.0092

	TOTAL_A
1	206.0891
2	175.3403
3	162.1582
4	160.1871
5	176.0222

RADIAL TRABECULAR CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	1185.829	71	491.2202	2.414048	0.05678

RADIAL CORTICAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	703.562	71	147.3577	4.774517	0.001811

Tukey HSD test; variable CORT_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.74000	76.55714	75.32800	76.40875	75.29000
1	{1}		0.084136	0.036263	0.061464	0.04095
2	{2}	0.084136		0.998831	1	0.998767
3	{3}	0.036263	0.998831		0.999196	1
4	{4}	0.061464	1	0.999196		0.999142
5	{5}	0.04095	0.998767	1	0.999142	

Adjusted means (bmd7stat.sta)
F(4,71)=4.77; p<.0018

	CORT_A
1	90.54918
2	80.66698
3	72.99876
4	69.33089
5	77.77808

MOMENT OF INERTIA - RADIAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	17330076	71	4890747	3.543442	0.010793

Tukey HSD test; variable INERTIA (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		6883.640	4545.872	5245.907	6257.395	4933.094
1	{1}		0.032478	0.223948	0.922372	0.108059
2	{2}	0.032478		0.913284	0.225412	0.990401
3	{3}	0.223948	0.913284		0.708738	0.995511
4	{4}	0.922372	0.225412	0.708738		0.47973
5	{5}	0.108059	0.990401	0.995511	0.47973	

Adjusted means (bmd7stat.sta)
F(4,71)=3.54; p<.0108

	INERTIA
1	7506.139
2	5456.589
3	4729.761
4	4688.979
5	5484.439

Variables Controlled for Lean Body Mass

FNBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.015729	73	0.003298	4.769962	0.001786

Tukey HSD test; variable FNBMD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.7058421	.6884286	.6488000	.6789375	.6524667
1	{1}		0.910179	0.040945	0.641823	0.065139
2	{2}	0.910179		0.34972	0.991305	0.449423
3	{3}	0.040945	0.34972		0.591286	0.99981
4	{4}	0.641823	0.991305	0.591286		0.702682
5	{5}	0.065139	0.449423	0.99981	0.702682	

Adjusted means (bmd7stat.sta)
F(4,73)=4.77; p<.0018

	FNBMD
1	0.714245
2	0.711682
3	0.635047
4	0.643848
5	0.669652

TROCHANTER BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.022076	73	0.0052	4.245119	0.003818

Tukey HSD test; variable TROCH (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6147895	.5651429	.5362000	.5634375	.5327333
1	{1}		0.29861	0.019247	0.231865	0.012933
2	{2}	0.29861		0.816247	0.999996	0.746007
3	{3}	0.019247	0.816247		0.830683	0.999938
4	{4}	0.231865	0.999996	0.830683		0.760102
5	{5}	0.012933	0.746007	0.999938	0.760102	

Adjusted means (bmd7stat.sta)
F(4,73)=4.25; p<.0038

	TROCH
1	0.620725
2	0.581567
3	0.526486
4	0.538654
5	0.544872

WARD'S TRIANGLE BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.025067	73	0.005352	4.683822	0.002022

Tukey HSD test; variable WARD_S (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .6263158	{2} .6527857	{3} .5947334	{4} .5807500	{5} .6085333
1	{1}		0.84202	0.72231	0.361512	0.955067
2	{2}	0.84202		0.216587	0.065211	0.484892
3	{3}	0.72231	0.216587		0.983833	0.985509
4	{4}	0.361512	0.065211	0.983833		0.827869
5	{5}	0.955067	0.484892	0.985509	0.827869	

Adjusted means (bmd7stat.sta)
F(4,73)=4.68; p<.0020

	WARD_S
1	0.6365
2	0.680966
3	0.578066
4	0.538225
5	0.629361

LSBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.013103	73	0.003659	3.580881	0.010099

Tukey HSD test; variable AVE__L_S (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .6708070	{2} .6746905	{3} .6486667	{4} .6414375	{5} .6458889
1	{1}		0.999775	0.826413	0.610062	0.755616
2	{2}	0.999775		0.775182	0.564543	0.703518
3	{3}	0.826413	0.775182		0.997362	0.999949
4	{4}	0.610062	0.564543	0.997362		0.999644
5	{5}	0.755616	0.703518	0.999949	0.999644	

Adjusted means (bmd7stat.sta)
F(4,73)=3.58; p<.0101

	AVE__L_S
1	0.680314
2	0.700996
3	0.633108
4	0.601743
5	0.66533

WHOLE BODY BMD

Summary of all Effects; design: (bmd7stat.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.002797	73	0.001654	1.690668	0.161366

TOATL RADIAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	11131.11	71	1681.033	6.621592	0.000138

Tukey HSD test; variable TOTAL (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} 360.5000	{2} 373.1000	{3} 335.1933	{4} 296.6063	{5} 306.4214
1	{1}		0.909649	0.401668	0.000325	0.003848
2	{2}	0.909649		0.105017	0.000147	0.000611
3	{3}	0.401668	0.105017		0.077788	0.333093
4	{4}	0.000325	0.000147	0.077788		0.965359
5	{5}	0.003848	0.000611	0.333093	0.965359	

Means (bmd7stat.sta)
F(4,71)=6.62; p<.0001

	TOTAL
1	360.5
2	373.1
3	335.1933
4	296.6063
5	306.4214

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8298.522	71	1173.287	7.072886	7.5E-05

Tukey HSD test; variable TRAB_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} 211.1889	{2} 212.6071	{3} 175.8867	{4} 162.6750	{5} 171.0500
1	{1}		0.999963	0.034106	0.001034	0.013288
2	{2}	0.999963		0.040248	0.001584	0.016631
3	{3}	0.034106	0.040248		0.819669	0.995541
4	{4}	0.001034	0.001584	0.819669		0.962633
5	{5}	0.013288	0.016631	0.995541	0.962633	

Adjusted means (bmd7stat.sta)
F(4,71)=7.07; p<.0001

	TRAB_
1	213.6984
2	218.7068
3	172.4154
4	153.6857
5	174.9014

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	12335.21	71	2543.448	4.849797	0.001626

Adjusted means (bmd7stat.sta)
F(4,71)=4.85; p<.0016

	CORT_
1	481.2254
2	473.2317
3	459.7432
4	437.0975
5	409.8784

Tukey HSD test; variable CORT_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		484.2833	480.6643	455.5133	426.1437	414.5714
1	{1}		0.999666	0.48256	0.010948	0.002226
2	{2}	0.999666		0.666216	0.033567	0.007848
3	{3}	0.48256	0.666216		0.489666	0.197561
4	{4}	0.010948	0.033567	0.489666		0.970298
5	{5}	0.002226	0.007848	0.197561	0.970298	

TOTAL RADIAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3807.703	71	1085.506	3.50777	0.011372

Adjusted means (bmd7stat.sta)
F(4,71)=3.51; p<.0114

	TOTAL_A
1	202.4119
2	176.5966
3	161.4071
4	162.1649
5	177.2165

Tukey HSD test; variable TOTAL_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		195.5533	159.9264	170.8940	186.7325	166.6907
1	{1}		0.027052	0.214684	0.93587	0.11214
2	{2}	0.027052		0.897597	0.183246	0.982484
3	{3}	0.214684	0.897597		0.668961	0.997011
4	{4}	0.93587	0.183246	0.668961		0.463703
5	{5}	0.11214	0.982484	0.997011	0.463703	

RADIAL TRABECULAR CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	956.151	71	469.68	2.03575	0.098565

RADIAL CORTICAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	761.5121	71	139.6092	5.454598	0.000691

Adjusted means (bmd7stat.sta)
F(4,71)=5.45; p<.0007

	CORT_A
1	89.72149
2	81.37327
3	72.58717
4	69.31098
5	78.33099

Tukey HSD test; variable CORT_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.74000	76.55714	75.32800	76.40875	75.29000
1	{1}		0.071193	0.029311	0.05112	0.033311
2	{2}	0.071193		0.99869	1	0.998618
3	{3}	0.029311	0.99869		0.999106	1
4	{4}	0.05112	1	0.999106		0.999046
5	{5}	0.033311	0.998618	1	0.999046	

MOMENT OF INERTIA - RADIAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	15781426	71	4635737	3.404298	0.013237

Adjusted means (bmd7stat.sta)
F(4,71)=3.40; p<.0132

	INERTIA
1	7294.447
2	5544.363
3	4677.67
4	4785.867
5	5563.559

Tukey HSD test; variable INERTIA (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		6883.640	4545.872	5245.907	6257.395	4933.094
1	{1}		0.026153	0.200939	0.91507	0.092885
2	{2}	0.026153		0.905224	0.202333	0.989376
3	{3}	0.200939	0.905224		0.687756	0.995016
4	{4}	0.91507	0.202333	0.687756		0.452363
5	{5}	0.092885	0.989376	0.995016	0.452363	

Variables Controlled for Height

FNBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.014654	73	0.003506	4.180054	0.004198

Tukey HSD test; variable FNBMD (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.7058421	.6884286	.6488000	.6789375	.6524667
1	{1}		0.91891	0.051103	0.667933	0.079098
2	{2}	0.91891		0.380964	0.992269	0.480746
3	{3}	0.051103	0.380964		0.61937	0.999831
4	{4}	0.667933	0.992269	0.61937		0.725834
5	{5}	0.079098	0.480746	0.999831	0.725834	

Adjusted means (bmd7stat.sta)
F(4,73)=4.18; p<.0042

	FNBMD
1	0.716363
2	0.712305
3	0.641926
4	0.656705
5	0.647175

TROCHANTER BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.023984	73	0.005157	4.650715	0.002121

Tukey HSD test; variable TROCH (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6147895	.5651429	.5362000	.5634375	.5327333
1	{1}		0.29454	0.018547	0.22815	0.012426
2	{2}	0.29454		0.813958	0.999996	0.743086
3	{3}	0.018547	0.813958		0.828538	0.999937
4	{4}	0.22815	0.999996	0.828538		0.757297
5	{5}	0.012426	0.743086	0.999937	0.757297	

Adjusted means (bmd7stat.sta)
F(4,73)=4.65; p<.0021

	TROCH
1	0.625305
2	0.589008
3	0.529329
4	0.541216
5	0.527445

WARD'S TRIANGLE BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.016627	73	0.005804	2.864658	0.02907

Tukey HSD test; variable WARD_S (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6263158	.6527857	.5947334	.5807500	.6085333
1	{1}		0.860657	0.751279	0.403114	0.961095
2	{2}	0.860657		0.252989	0.084123	0.525648
3	{3}	0.751279	0.252989		0.98612	0.987559
4	{4}	0.403114	0.084123	0.98612		0.847881
5	{5}	0.961095	0.525648	0.987559	0.847881	

Adjusted means (bmd7stat.sta)
F(4,73)=2.86; p<.0291

	WARD_S
1	0.636683
2	0.676314
3	0.58796
4	0.558842
5	0.603319

LSBMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.015842	73	0.003511	4.512079	0.002591

Tukey HSD test; variable AVE__L_S (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.6708070	.6746905	.6486667	.6414375	.6458889
1	{1}		0.999756	0.815352	0.590973	0.741304
2	{2}	0.999756		0.761707	0.544485	0.687196
3	{3}	0.815352	0.761707		0.997141	0.999944
4	{4}	0.590973	0.544485	0.997141		0.999614
5	{5}	0.741304	0.687196	0.999944	0.999614	

Adjusted means (bmd7stat.sta)
F(4,73)=4.51; p<.0026

	AVE__L_S
1	0.688062
2	0.713851
3	0.637393
4	0.604974
5	0.637211

WHOLE BODY BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.002825	73	0.001962	1.439414	0.229652

TOTAL RADIAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	6389.654	71	1647.754	3.877796	0.006619

Tukey HSD test; variable TOTAL (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		360.5000	373.1000	335.1933	296.6063	306.4214
1	{1}		0.906598	0.391386	0.000293	0.003431
2	{2}	0.906598		0.099285	0.000143	0.000543
3	{3}	0.391386	0.099285		0.073083	0.323063
4	{4}	0.000293	0.000143	0.073083		0.964093
5	{5}	0.003431	0.000543	0.323063	0.964093	

Adjusted means (bmd7stat.sta)
F(4,71)=3.88; p<.0066

	TOTAL
1	355.6544
2	361.7617
3	338.4891
4	307.211
5	308.7047

RADIAL TRABECULAR BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	6708.97	71	1189.957	5.637993	0.000534

Tukey HSD test; variable TRAB_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		211.1889	212.6071	175.8867	162.6750	171.0500
1	{1}		0.999964	0.036017	0.001129	0.014201
2	{2}	0.999964		0.042432	0.001762	0.017726
3	{3}	0.036017	0.042432		0.823435	0.995661
4	{4}	0.001129	0.001762	0.823435		0.963565
5	{5}	0.014201	0.017726	0.995661	0.963565	

Adjusted means (bmd7stat.sta)
F(4,71)=5.64; p<.0005

	TRAB_
1	213.5513
2	218.1349
3	174.2799
4	157.5048
5	169.9368

RADIAL CORTICAL BMD

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8425.599	71	2553.436	3.299711	0.015436

Tukey HSD test; variable CORT_ (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		484.2833	480.6643	455.5133	426.1437	414.5714
1	{1}		0.999669	0.48455	0.01116	0.002279
2	{2}	0.999669		0.66785	0.034084	0.008009
3	{3}	0.48455	0.66785		0.49165	0.199192
4	{4}	0.01116	0.034084	0.49165		0.970509
5	{5}	0.002279	0.008009	0.199192	0.970509	

Adjusted means (bmd7stat.sta)
F(4,71)=3.30; p<.0154

	CORT_
1	480.6267
2	472.1082
3	458.0004
4	434.1463
5	416.2945

TOTAL RADIAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3980.136	71	1140.608	3.489486	0.011682

Tukey HSD test; variable TOTAL_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		195.5533	159.9264	170.8940	186.7325	166.6907
1	{1}		0.033013	0.23653	0.941126	0.127808
2	{2}	0.033013		0.905597	0.203609	0.984047
3	{3}	0.23653	0.905597		0.689147	0.997285
4	{4}	0.941126	0.203609	0.689147		0.488919
5	{5}	0.127808	0.984047	0.997285	0.488919	

Adjusted means (bmd7stat.sta)
F(4,71)=3.49; p<.0117

	TOTAL_A
1	203.6518
2	178.876
3	165.3858
4	169.0088
5	162.8746

RADIAL TRABECULAR CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	1137.273	71	487.0548	2.335	0.063747

RADIAL CORTICAL CSA

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	693.9506	71	146.91	4.723642	0.001948

Adjusted means (bmd7stat.sta)
F(4,71)=4.72; p<.0019

	CORT_A
1	89.87167
2	81.54502
3	73.87814
4	71.74354
5	74.28553

Tukey HSD test; variable CORT_A (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.74000	76.55714	75.32800	76.40875	75.29000
1	{1}		0.083369	0.035839	0.060845	0.04049
2	{2}	0.083369		0.998824	1	0.99876
3	{3}	0.035839	0.998824		0.999191	1
4	{4}	0.060845	1	0.999191		0.999137
5	{5}	0.04049	0.99876	1	0.999137	

MOMENT OF INERTIA - RADIAL

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	16423808	71	4903115	3.349668	0.014343

Adjusted means (bmd7stat.sta)
F(4,71)=3.35; p<.0143

	INERTIA
1	7343.314
2	5621.463
3	4933.257
4	5251.384
5	4716.49

Tukey HSD test; variable INERTIA (bmd7stat.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		6883.640	4545.872	5245.907	6257.395	4933.094
1	{1}		0.032802	0.22506	0.922702	0.108811
2	{2}	0.032802		0.913649	0.226526	0.990456
3	{3}	0.22506	0.913649		0.709704	0.995533
4	{4}	0.922702	0.226526	0.709704		0.481014
5	{5}	0.108811	0.990456	0.995533	0.481014	

Daughter's Measure Controlled for Mother's Measure

WEIGHT

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	393.0335	73	12.99104	30.25421	7.61E-15

Adjusted means (mom&daug.sta)
F(4,73)=30.25; p<.0000

	WEIGHT
1	28.02047
2	25.15069
3	32.36224
4	38.04857
5	27.49825

Tukey HSD test; variable WEIGHT (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		27.14737	25.44286	32.98667	38.55000	26.95333
1	{1}		0.665776	0.00023	0.000124	0.999879
2	{2}	0.665776		0.000126	0.000124	0.791447
3	{3}	0.00023	0.000126		0.00061	0.000284
4	{4}	0.000124	0.000124	0.00061		0.000124
5	{5}	0.999879	0.791447	0.000284	0.000124	

LBM

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	118.8518	72	6.013199	19.76516	5.15E-11

Adjusted means (mom&daug.sta)
F(4,72)=19.77; p<.0000

	LBM
1	23.50238
2	20.87053
3	25.26065
4	28.11219
5	22.0623

Tukey HSD test; variable LBM (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		22.99225	21.11039	25.80007	28.02604	21.87931
1	{1}		0.199603	0.012239	0.000125	0.683518
2	{2}	0.199603		0.000142	0.000125	0.915997
3	{3}	0.012239	0.000142		0.105316	0.000476
4	{4}	0.000125	0.000125	0.105316		0.000125
5	{5}	0.683518	0.915997	0.000476	0.000125	

HEIGHT

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	584.618	73	32.03873	18.24723	1.99E-10

Tukey HSD test; variable HEIGHT (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} 130.8053	{2} 126.1357	{3} 136.8867	{4} 142.2563	{5} 136.3333
1	{1}		0.143618	0.021797	0.000124	0.04638
2	{2}	0.143618		0.000143	0.000124	0.00018
3	{3}	0.021797	0.000143		0.073743	0.99891
4	{4}	0.000124	0.000124	0.073743		0.037319
5	{5}	0.04638	0.00018	0.99891	0.037319	

Adjusted means (mom&daug.sta)
F(4,73)=18.25; p<.0000

	HEIGHT
1	132.0273
2	125.4905
3	135.9557
4	142.3753
5	136.5684

% BODY FAT

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	190.4949	72	8.52043	22.35743	4.98E-12

Tukey HSD test; variable %_BF (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} 15.23158	{2} 17.01429	{3} 21.22667	{4} 25.43333	{5} 18.79333
1	{1}		0.420168	0.000125	0.000125	0.006407
2	{2}	0.420168		0.002176	0.000125	0.477296
3	{3}	0.000125	0.002176		0.001792	0.162463
4	{4}	0.000125	0.000125	0.001792		0.000125
5	{5}	0.006407	0.477296	0.162463	0.000125	

Adjusted means (mom&daug.sta)
F(4,72)=22.36; p<.0000

	%_BF
1	15.56802
2	17.12053
3	21.27764
4	24.53055
5	19.20246

FNBMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.008428	73	0.003612	2.333539	0.063592

FNBMAD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.005067	73	0.001731	2.927519	0.026491

Tukey HSD test; variable FNBMAD (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .2323985	{2} .2311983	{3} .2054184	{4} .1907030	{5} .2116945
1	{1}		0.999991	0.338577	0.033376	0.603695
2	{2}	0.999991		0.460253	0.070252	0.715347
3	{3}	0.338577	0.460253		0.861646	0.993817
4	{4}	0.033376	0.070252	0.861646		0.62718
5	{5}	0.603695	0.715347	0.993817	0.62718	

Adjusted means (mom&daug.sta)
F(4,73)=2.93; p<.0265

	FNBMAD
1	0.23022
2	0.232504
3	0.208516
4	0.1891
5	0.211073

TROCHANTER BMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.018828	73	0.005236	3.59587	0.009879

Tukey HSD test; variable TROCH (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1} .6147895	{2} .5651429	{3} .5362000	{4} .5634375	{5} .5327333
1	{1}		0.301971	0.019839	0.23494	0.013362
2	{2}	0.301971		0.818113	0.999996	0.748392
3	{3}	0.019839	0.818113		0.83243	0.999939
4	{4}	0.23494	0.999996	0.83243		0.76239
5	{5}	0.013362	0.748392	0.999939	0.76239	

Adjusted means (mom&daug.sta)
F(4,73)=3.60; p<.0099

	TROCH
1	0.616129
2	0.566204
3	0.538192
4	0.557199
5	0.534579

WARD'S TRIANGLE BMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.007383	73	0.005521	1.337246	0.264319

LSBMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.00483	73	0.003627	1.331661	0.266343

LSBMAD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.002652	73	0.000402	6.592426	0.000138

Tukey HSD test; variable LSBMAD (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		.2325002	.2392879	.2116354	.2097384	.2189931
1	{1}		0.871696	0.028545	0.011168	0.300978
2	{2}	0.871696		0.003687	0.00136	0.06024
3	{3}	0.028545	0.003687		0.998981	0.852467
4	{4}	0.011168	0.00136	0.998981		0.701868
5	{5}	0.300978	0.06024	0.852467	0.701868	

Adjusted means (mom&daug.sta)
F(4,73)=6.59; p<.0001

	LSBMAD
1	0.232658
2	0.23991
3	0.21217
4	0.210266
5	0.217151

WHOLE BODY BMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	0.003268	72	0.002049	1.594636	0.185026

TOTAL RADIAL BMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	17775	71	1443.129	12.31699	1.16E-07

Tukey HSD test; variable TOTAL (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		360.5000	373.1000	335.1933	296.6063	306.4214
1	{1}		0.884062	0.324068	0.000175	0.001528
2	{2}	0.884062		0.066361	0.00013	0.000257
3	{3}	0.324068	0.066361		0.046782	0.258924
4	{4}	0.000175	0.00013	0.046782		0.954537
5	{5}	0.001528	0.000257	0.258924	0.954537	

Adjusted means (mom&daug.sta)
F(4,71)=12.32; p<.0000

	TOTAL
1	362.8416
2	373.4156
3	334.3175
4	295.9882
5	305.258

TRABECULAR RADIAL BMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	8808.471	71	1170.306	7.526636	4.11E-05

Tukey HSD test; variable TRAB_ (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		211.1889	212.6071	175.8867	162.6750	171.0500
1	{1}		0.999962	0.03377	0.001018	0.013129
2	{2}	0.999962		0.039868	0.00156	0.01644
3	{3}	0.03377	0.039868		0.818985	0.995519
4	{4}	0.001018	0.00156	0.818985		0.962463
5	{5}	0.013129	0.01644	0.995519	0.962463	

Adjusted means (mom&daug.sta)
F(4,71)=7.53; p<.0000

	TRAB_
1	210.7479
2	213.9728
3	174.721
4	163.3977
5	170.5683

CORTICAL RADIAL BMD

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	15714.75	71	2457.632	6.394265	0.000188

Adjusted means (mom&daug.sta)
F(4,71)=6.39; p<.0002

	CORT_
1	485.0445
2	480.7249
3	456.1063
4	425.5685
5	413.7321

Tukey HSD test; variable CORT_ (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		484.2833	480.6643	455.5133	426.1437	414.5714
1	{1}		0.999643	0.465063	0.009216	0.001812
2	{2}	0.999643		0.651683	0.02928	0.006555
3	{3}	0.465063	0.651683		0.472218	0.183542
4	{4}	0.009216	0.02928	0.472218		0.968368
5	{5}	0.001812	0.006555	0.183542	0.968368	

TOTAL RADIAL CSA

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	3494.462	71	965.4991	3.619332	0.009657

Adjusted means (mom&daug.sta)
F(4,71)=3.62; p<.0097

	TOTAL_A
1	191.3746
2	160.387
3	170.1856
4	192.457
5	165.3928

Tukey HSD test; variable TOTAL_A (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		195.5533	159.9264	170.8940	186.7325	166.6907
1	{1}		0.016274	0.166944	0.92172	0.080011
2	{2}	0.016274		0.8763	0.139441	0.978238
3	{3}	0.166944	0.8763		0.618145	0.996239
4	{4}	0.92172	0.139441	0.618145		0.403441
5	{5}	0.080011	0.978238	0.996239	0.403441	

TRABECULAR RADIAL CSA

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	1749.012	71	439.0839	3.983321	0.005675

Adjusted means (mom&daug.sta)
F(4,71)=3.98; p<.0057

	TRAB_A
1	85.66223
2	65.76097
3	75.45834
4	92.53976
5	72.16884

Tukey HSD test; variable TRAB_A (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		87.04333	65.61643	76.83733	89.61375	72.47929
1	{1}		0.041866	0.634065	0.996511	0.300975
2	{2}	0.041866		0.603677	0.020859	0.9082
3	{3}	0.634065	0.603677		0.442733	0.980426
4	{4}	0.996511	0.020859	0.442733		0.179249
5	{5}	0.300975	0.9082	0.980426	0.179249	

CORTICAL RADIAL CSA

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	278.1896	71	115.5717	2.407075	0.057363

MOMENT OF INERTIA - RADIAL

Summary of all Effects; design: (mom&daug.sta)
1-NUMBER

	df Effect	MS Effect	df Error	MS Error	F	p-level
1	4	12981693	71	3912050	3.318387	0.015018

Adjusted means (mom&daug.sta)
F(4,71)=3.32; p<.0150

	INERTIA
1	6553.08
2	4598.027
3	5294.376
4	6518.631
5	4901.792

Tukey HSD test; variable INERTIA (mom&daug.sta)
Probabilities for Post Hoc Tests
MAIN EFFECT: NUMBER

		{1}	{2}	{3}	{4}	{5}
		6883.640	4545.872	5245.907	6257.395	4933.094
1	{1}		0.01224	0.136184	0.887741	0.054236
2	{2}	0.01224		0.875223	0.137321	0.985356
3	{3}	0.136184	0.875223		0.615191	0.993069
4	{4}	0.887741	0.137321	0.615191		0.365125
5	{5}	0.054236	0.985356	0.993069	0.365125	