RETIRED COLLEGIATE ARTISTIC GYMNASTS RETAIN HIGH BONE MASS

by

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(Under the Direction of RICHARD D. LEWIS)

ABSTRACT

Fifteen years after cessation of the sport, we found that retired collegiate artistic gymnasts (GYM; n=18) had higher measures of areal bone mineral density (aBMD; g/cm^2) at all skeletal sites compared to nongymnast controls (CON; n=15) of similar age (years), height (cm) and body weight (BW; kg). It is unknown, however, if the aBMD differences in GYM and CON observed at that time are maintained into the years approaching menopause. A nine-year follow-up study was conducted to compare aBMD in GYM (n=16; age=45.3 ± 3.3 years) and CON (n=13; age=45.4 ± 3.8 years) and the changes over time. Total body fat mass (FM; kg), percent fat (%FAT), fat-free soft tissue (FFST; kg) and aBMD of the total body, lumbar spine, non-dominant proximal femur (PF), femoral neck and Ward's triangle were assessed using dual-energy X-ray absorptiometry (DXA; Hologic QDR-1000W). Past physical activity was estimated using a self-report, study-designed questionnaire. Independent samples *t*-tests were employed to compare aBMD in GYM and CON at baseline and at the nine-year follow-up. Analysis of covariance was used to compare the changes (Δ) in aBMD between GYM and CON and to quantify the magnitude of the effects (i.e., partial eta-squared; η^2; where 0.06 and 0.13 are medium and large effects, respectively). GYM had significantly lower BW, FM, and %FAT ($p<0.05$; $\eta^2>0.14$), and higher measures of FFST/BW and aBMD at all skeletal sites compared to

CON ($p<0.05$; $\eta^2>0.14$) at both time points. Over time, changes in GYM and CON did not differ significantly with respect to BW ($p=0.12$; $\eta^2=0.09$), FM ($p=0.38$; $\eta^2=0.03$), %FAT ($p=0.92$; $\eta^2=0.00$), or aBMD at any skeletal site ($p>0.05$; $\eta^2<0.08$). CON had greater gains in FFST than GYM (8.68 ± 1.80% vs. 3.22 ± 0.92%; $p=0.01$; $\eta^2=0.23$); however, when FFST was corrected for BW, no significant difference was found between GYM and CON. Additionally, there were no significant differences in the total minutes of physical activity per week reported over the past nine years between groups. In conclusion, the higher aBMD observed in GYM compared to CON fifteen years after the cessation of the sport, was maintained over the following nine years, regardless of less physical activity since competitive gymnastics training.

INDEX WORDS: GYMNASTICS, FORMER GYMNASTS, RETIRED GYMNASTS, AREAL BONE MINERAL DENSITY, PAST ATHLETIC PARTICIPATION AND BONE

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DEDICATION

This thesis is dedicated to my parents, Norman Jesse and Maria Valladares Pollock, for all their support and love. Thank you for all your encouragement, positive attitude, and guidance throughout my life.

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CHAPTER 1

INTRODUCTION

Osteoporosis, a systemic skeletal disorder, can be defined conceptually as a condition of generalized skeletal fragility such that fractures occur with minimal trauma, often no more than is applied by routine daily activity. In the United States, osteoporosis currently affects approximately 10 million people,[1] and the National Osteoporosis Foundation[2] estimates that an additional 34 million more people have low bone mass (osteopenia), placing them at increased risk for osteoporosis. Fractures due to bone disease are common, costly, and often become a chronic burden on individuals and society. One in two women and one in four men over age 50 will have an osteoporosis-related fracture in her/his remaining lifetime.[2] It was estimated that national direct expenditures (hospitals and nursing homes) for osteoporotic and associated fractures was $17 billion in 2001.[2] As the aging population increases, if effective prevention methods are not implemented, the impact of osteoporosis will continue to intensify leading to more fractures and higher national expenditures.

The etiology of osteoporosis is multifactorial with age, gender, physical activity, diet, hormone status, and lifestyle all playing roles.[3] Of the modifiable lifestyle factors that influence the skeleton, such as nutrition, tobacco use, and exercise, it is believed that exercise during growth that has vast potential to reduce the public health burden of osteoporosis.[4] The use of exercise in building and maintaining bone health throughout the lifespan and ultimately preventing osteoporosis-related fractures has been the focus of considerable research.

Skeletal responses to exercise vary with age, hormone status, nutritional status, and nature of the exercise. It has been hypothesized that attaining a high peak bone mass early in life may prevent unwanted fractures later in life, particularly when fracture risk is high. Scientists have been examining the role of exercise during growth for promoting maximal peak bone mineral density accrual. However, there is no clear evidence of a persisting benefit of exercise during growth on bone structure or strength in old age when falls and fracture risk increases. Part of the problem is that researchers cannot, without crossing ethical boundaries and considering the financial burden, conduct prospective intervention studies following children into late adulthood tracking every bone-influencing factor until the point of fracture. Currently, the best evidence we have linking childhood exercise and bone health in late adulthood lies in cross-sectional and short-term prospective studies in former competitive athletes.

Cross-sectional studies of active adults and former competitive athletes who started training in their youth are evocative, but uncertain, with respect to whether bone gains achieved during the younger years are maintained into adulthood. Comparisons of active collegiate gymnasts[5-7] with nongymnast athletes or controls have demonstrated that gymnasts have significantly higher areal bone mineral density (aBMD) values, ranging from 5% to 36%. Competitive college-age soccer,[8, 9] tennis,[10] volleyball players,[11, 12] and weightlifters[13] have also been shown to have significantly higher aBMD compared to nonathletic controls, ranging from 1% to 24%. Furthermore, former artistic gymnasts,[14-16] soccer players,[8, 9] and weightlifters[13] retired from competitive training less than 20 years, were found to have significantly higher aBMD values compared to nonathlete controls with differences ranging from 5% to 22%. The aBMD differences observed in these studies of retired athletes would imply that potential bone gains from participation in high-impact youth sports persist into adulthood, yet some studies

suggest otherwise. Areal BMD of former soccer players[8, 9] and weightlifters[17, 18], some 60 years of age and older and retired from their competitive training for more than 20 years, was not different when compared to nonathlete controls of the same age, leaving questionable the sustainability of skeletal benefits from earlier sports participation.

Only three studies have evaluated changes in bone prospectively following cessation of intensive training in former competitive athletes.[19-21] In those studies, retired college gymnasts and tennis players maintained significantly higher aBMD or BMC values when compared to controls or when observing side-to-side arm comparisons in racquet sports. However, the former athletes in these studies were still relatively young (mean age range 19 to 35 years) and were retired from their sport for five years or less. Whether this is the case in older former competitive athletes for longer periods of time since retirement is unclear.

The purpose of this present investigation was to determine if the higher aBMD of former gymnasts compared to controls, previously reported by Kirchner et al.,[25] is still present in the same cohort of former gymnasts approaching menopause and approximately 25 years since the cessation of college gymnastics training and competition. The specific aim was to examine changes in aBMD and related factors including body composition, physical activity, and selected nutrient intakes in the former female college gymnasts and controls approximately nine years after baseline measurements. It was hypothesized that the higher bone mass observed in the former artistic gymnasts compared with controls will be maintained over nine years.

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CHAPTER 2

REVIEW OF THE LITERATURE

The prevalence of osteoporosis is on the rise in the United States (U.S.), due primarily to the aging of the population. By the year 2020, 14 million individuals over the age of 50 years are expected to develop osteoporosis and another 47 million are projected to have low bone mass.[1] The impact of osteoporotic-related fractures can be devastating to our economy, the lives of the individuals who suffer from the disorder, as well as friends and family members. However, evidence clearly suggests that individuals still can do a great deal to promote their own bone health. In this review, the following topics will be described: bone biology, determinants of bone including genetics, hormonal status, nutrition, physical activity, and body composition. In particular, the effects of artistic gymnastics and other sports on bone will be addressed, with the primary focus being on bone mineral retention resulting from past participation from these sports. Finally, the gaps in the literature related to the influence of sports participation on bone will be discussed.

Bone Biology

Bone is a unique living tissue with the main responsibility of supporting loads applied on it. The bony skeleton serves the function of movement, acts as a protector of vital organs, provides an environment for storage of calcium and phosphorus, and serves as a site for blood cell formation.[2] Ninety-eight percent of bone is an organic matrix made up of type I collagen and noncollagenous proteins, while the remaining 2% is composed of inorganic material, consisting of enmeshed hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$] containing primarily calcium and phosphorus.[3]

The architecture of the skeleton adapts to provide adequate strength and mobility so that bones do not break when subjected to substantial impact, even the loads placed on bone during vigorous physical activity.

There are two types of bone in the human body: trabecular (cancellous) bone and cortical (compact) bone. Trabecular bone, a soft spongy bone consisting of horizontal and vertical cross-links found primarily at the ends of long bones and within vertebrae, has a large surface area and is susceptible to accelerated bone turnover.[4] Comprising about 20% of the skeletal mass, trabecular bone's turnover rate is approximately 26% per year.[5] Cortical bone, which makes up the remaining 80% of skeletal mass, is found primarily in the shafts of the long bones, has a slow turnover rate at about 3% per year, and has strength as its primary function.[3] Figure 2.1 illustrates trabecular bone and cortical bone.

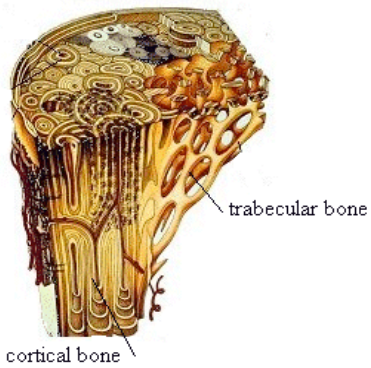

Figure 2.1. **Inner Structure of Bone,** Adapted from Marieb, 1998.[2]

Bone undergoes both formation and resorption throughout the lifecycle. Three types of bone cells, osteoblasts, osteocytes, and osteoclasts, are primarily involved with either formation or resorption. To form bone, mesenchymal stem cells produce osteoprogenitor blood cells that likely differentiate into single nucleated, bone-forming osteoblasts.[6] Later, these cells mature into osteocytes and lose some of their cell organelles once incorporated into the bone matrix within

the lacunae.[6] Once established within the cell matrix, bone formation by these cells ceases and the tissue becomes highly mineralized. The osteocytes facilitate communication between adjacent cells within the mineralized matrix via gap junctions.[6]

Bone resorption occurs primarily as a function of the multi-nucleated, large cell osteoclast that may originate from circulating mononuclear progenitor cells.[7] The characteristic feature of the osteoclast is the ruffled border surrounded by a ring of contractile protein. This border serves to attach the osteoclast to the bone surface and create what is known as the extracellular bone-resorbing compartment.[8] Lysosomal enzymes are actively synthesized in the osteoclast and then secreted, via the ruffled border, into the extracellular bone-resorbing compartment where a high concentration of enzymes develops to resorb bone.[9] The acidic environment digests the noncollagenenous link between hydroxyapatite crystals and collagen, allowing calcium to be released from the skeleton.[9]

Together, the osteoblast, osteocyte, and osteoclast comprise the small basic multicellular units (BMU) where the process of remodeling occurs within the cortical and trabecular bone.[10] The fact that osteoclastic bone resorption and osteoblastic bone formation follow each other is fundamental to the concept of the BMU, which describes a packet of bone being resorbed or rebuilt.[11] As a result of coupling of osteoclast and osteoblast function, bone resorption initiates bone formation (activation), which, under balanced conditions, restores lost bone (Figure 2.2).[9]

These active bone cells, which are known to act in response to various environmental signals including chemical, mechanical, electrical, and magnetic stimuli, are essential for the modeling and remodeling processes within bone.[3, 12, 13] The balance between modeling and remodeling differs between the growing and adult skeleton. In the former, modeling is the dominant mode, whereas in the latter, remodeling is dominant.[14] Modeling, seen in early

childhood up to early adulthood, is the process in which bones become larger, heavier, and denser; hence, osteoblastic activity exceeds osteoclastic activity. This uncoupled process with osteoblasts and osteoclasts improves bone strength not only by adding mass, but also by expanding the periosteal and endocortical diameters of bone.[15]

Bone remodeling begins to take over in adulthood, where bone mass undergoes constant and equal removal of old bone and renewal with newly formed bone.[16] An equilibrium exists between bone resorption and formation until the fourth or fifth decade of life, when bone resorption begins to supercede the continually declining bone formation process.[14] In the situations of an aging skeleton, with estrogen withdrawal, or even a lack of physical activity, the balance between the amount of bone resorbed and formed is shifted in favor of resorption, thereby resulting in a net loss of bone.[17, 18] Bone remodeling supports response and adaptation to mechanical stresses and metabolic demands of the body, as well as repairing skeletal damage, preserving bone strength, and maintaining mineral homeostasis throughout adulthood.[4, 12, 17] This process is regulated by complex interactions between genetic, hormonal, and environmental factors working to preserve the mechanical structure of the skeleton.[12, 18]

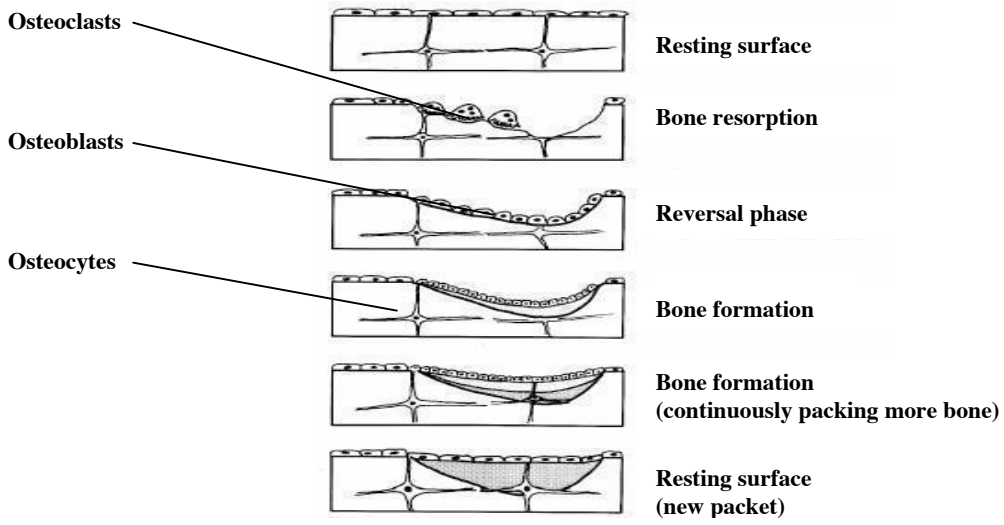

Figure 2.2. **Remodeling cycle in trabecular bone,** Adapted from Khan et al, 2001.[19]

Determinants of Bone Health

Genetics

The development of osteoporosis has a strong genetic component. Bone mass, osteoporosis, and fracture are complex traits subject to the influence of multiple physiologic and environmental factors. Twin and family studies have showed that genetic factors may account for up to 85% of interindividual bone mass variance.[20-22] Many genetic factors appear to influence areal bone mineral density (aBMD) development and are therefore related to fracture risk.[23] Some include body composition, age at menarche, age at menopause, serum parathyroid hormone level, and serum 25 dihydroxyvitamin D level.[24] Additionally, peak bone mass and rate of bone loss have separately been shown to be under genetic control.[24-27]

Multiple risk factors exist for fracture; many of these unrelated to bone strength. For example, a family history of fragility fracture has been shown to be a risk factor for fracture, independent of BMD.[21, 28] Bone size and structure are heritable traits and have been related to fracture risk.[26] In addition, hip-axis length and cortical thickness also show genetic variation.[25]

Normal genetic variation in complex traits is likely not due to deleterious mutations but to common polymorphisms at different sites.[23] This can lead to changes in gene-function or expression, gene-gene interaction, and gene-environment interaction.[26] Although there seems to be heritability of many components of bone strength, non-genetic factors are often of sufficient magnitude to mask the phenotype of these genetic differences.[29] The effects of genetics on bone traits are less obvious with age, as environmental factors play a large role.[29]

A gene-environment interaction would be present if genetic factors regulated the response of bone to physical activity. Thus, some individuals would respond more and some less to identical doses of bone loading, because of genetic differences. Athletes may be genetically

predisposed to having high BMD, or they may respond more positively to exercise intervention than nonathletes. This might explain why cross-sectional aBMD studies in athletes reveal substantial differences between subjects and controls, whereas intervention studies in general populations find a much smaller aBMD difference between the exercise and control groups.

It is important to understand that heredity and environment are not entirely separable. For example, genetic factors can influence such processes as the efficiency with which an individual utilizes and conserves the nutrients needed for bone building and maintenance. On marginal dietary intakes, an individual genetically equipped with efficient utilization will come closer to the ideal peak bone mass than one who utilizes nutrients inefficiently.[30] However, at high intakes the two individuals may be indistinguishable.[30] In this way, manipulation of an environmental factor (e.g., diet) can influence the expression of a genetic influence. For this reason, the high heritability often reported for bone mass should not be taken as grounds for a fatalistic attitude toward optimizing bone accumulation through lifestages.

Hormones

Hormones are chemical messengers released by glands and tissues throughout the body. They are vital for processes such as growth and maturation and for influences on energy balance, body weight and bone strength. From infancy to advanced age, a variety of hormones regulate bone growth and regeneration. The ability of hormones to do their job effectively depends on a number of factors, including diet, body weight, age and general health.[31] Moreover, their inability to function properly can create imbalances that impact bone integrity.

Many hormones are involved in the regulation of bone development. Table 2.1 lists some of the key hormones. Among those listed, the hormone estrogen, which it plays a more vital role in optimal bone health in women, will be discussed primarily.

Hormones Involved In Bone Development	Functions
estrogen	*regulates the rate of bone formation and bone resorption *prevents calcium loss and maintains levels of vitamin D
androgen	*stimulates bone growth
parathyroid hormone	*stimulates osteoclastic bone resorption indirectly to release calcium from bone *stimulates bone formation that is coupled to bone resorption *increases renal tubular reabsorption of calcium *stimulates the renal production of 1,25 dihydroxyvitamin D
calcitonin	*inhibits osteoclast resorption *delays calcium absorption from the intestine
1,25 dihydroxyvitamin D	*promotes gastrointestinal absorption of calcium and phosphorus
insulin-like growth factor-1	*increase rate of protein synthesis for bone formation *increases rate of mitosis of osteoblasts
thyroid hormone	*increases the rate of protein synthesis *controls energy production rate
glucocorticoid	*decreases calcium absorption from the intestines *inhibits bone formation *increases bone resorption *decreases sex steroid production
insulin	*increases energy production from glucose

Table 2.1. Key hormones in bone development. Adapted from Hurley et al (2004).[32]

Estrogen

The hormone, estrogen, plays a major role in bone health and the potential development of osteoporosis. Estrogen maintains bone mass by limiting bone resorption.[33] Directly, actions involving estrogen are mediated via the estrogen receptor on the osteoblast.[34] When estrogen binds to the receptor, it increases production of type I collagen and transforming growth factor-β which both are involved in the formation of bone.[35] Estrogen may indirectly increase renal calcium retention by stimulating an increase in renal synthesis of 1,25-dihydroxyvitamin D_3.[36]

A regular menstrual cycle is the external vital sign of a normally functioning reproductive system in the premenopausal female. It has been found that menarche, the start of the first

menstrual period, occurs later in athletes than in nonathletes, particularly in certain sports such as ballet, gymnastics, and running.[37] Later onset of menarche could theoretically be associated with a lower rate of bone mineral accretion during adolescence and therefore decreased peak bone mass.[38, 39] The relationship between age of menarche and aBMD in female athletes is unclear, however, some investigators have found statistically significant, but moderate to weak, negative correlations at a number of bone sites.[40-42]

Amenorrhea, defined as fewer than three cycles per year or no cycles for the past six months,[43] occurs in some premenopausal women after chronic intense exercise training. It occurs most often in female athletes where leanness is an advantage and very strenuous training is the norm. Disruption in the hypothalamic-pituitary axis from reduced energy availability is the determining factor of exercise-associated amenorrhea.[44] Because estrogen levels are reduced, the exercise-associated amenorrhea may result in bone loss.[45] In most cases, the positive effect of exercise on bone cannot offset the negative effects of inadequate energy-intake and high-intensity, high-volume exercise training.[45] However, the exceptions to this rule are gymnasts who, despite a high prevalence of menstrual disturbance, exhibit aBMD values well above normal.[42, 46, 47] In these cases, the magnitude and rate of bone loading is so great that it partially overrides the effect of hormonal disturbance.

Oral contraceptives, which contain variable amounts of estrogen and progesterone, are a common form of birth control method for women in the U.S. It has been estimated that 80% of women born since 1945 have used birth control pills at some point in their lives.[48] The factors that determine the effect of an oral contraceptive on bone health are the dose of estrogen and the age of the women.[49] The formulations of oral contraceptives have changed over the years, with older types having higher estrogen levels than do newer formulations. [50] Because of the varying

levels of estrogen found in the newer formulations, there is potential for varying effects on bone and fracture risk. However, both short- and long-term effects of oral contraceptives on aBMD are uncertain at this time.[49]

With the onset of menopause, circulating estrogen levels fall, therefore bone turnover is increased with bone resorption greater than bone formation and ultimately, bone mass declines.[51, 52] Although the premenopausal years are characterized by small changes in aBMD, the rate of aBMD loss is approximately 3% per year during the first five years following the onset of menopause and approximately 1% per year thereafter.[51] Early withdrawal of circulating estrogen levels or deficiency in estrogen production may exist in premenopausal women resulting in the inhibition of osteoclast apoptosis. [53] As a result, estrogen may prevent excessive bone loss before and after menopause by limiting osteoclast life span through promotion of apoptosis.

The role of estrogen in promoting the baseline level of mineralization in bones may be more important than the imbalance between the osteoblastic and osteoclastic activities.[54] Proposed by Jarvinen et al,[54] this estrogen-bone hypothesis includes four statements: " 1) in puberty the bones of females become stronger and attain more mineral content than those of males; 2) the puberty-associated extra packing of bone to females' skeleton is estrogen-driven; 3) the sex-related difference in skeleton mass or strength relative to locomotive needs is not limited to the most rapid period of skeletal growth but is a difference that persists throughout the entire fertile period; and 4) there is a reversal of the above mentioned estrogen-driven packing of the skeleton after menopause, that is a net increase in bone resorption caused by estrogen withdrawal." Thus, it is important to recognize that not only the function of estrogen in skeletal development is critical but also the timing of estrogen exposure.[54, 55]

Dietary and non-nutritional lifestyle factors

Diet can play an important role in constructing and maintaining bone mass throughout life, primarily by providing the key nutrients involved in the bone modeling and remodeling processes. Additionally, diet provides energy input, which together with the energy output from physical activity determines body weight. Maintenance of an optimal body weight can help promote better bone health.[56] A number of nutritional factors have been linked to bone health, including calcium, vitamin D, protein, sodium, zinc, magnesium, vitamin K, and vitamin A.[57] However, in this section we will focus primarily on calcium and vitamin D.

Calcium

While many nutrients play a role in bone health, calcium has been singled out as a major public health concern today not only because it is a critical nutrient for bone but also because of national survey data suggesting that the average calcium intake of individuals is far below the levels recommended for optimal bone health.[48] Calcium is absorbed by all parts of the small intestine, however the most rapid absorption after a meal occurs in the duodenum, where an acidic medium (pH < 7) prevails.[58] Adults absorb approximately 30% of ingested calcium, but some individuals may absorb as little as 10%.[59] The greater the body's need for calcium and the smaller the dietary supply, the more efficient the absorption of calcium becomes. Increased needs encountered during growth, pregnancy, lactation, and calcium-deficient states enhance calcium absorption.[58] Calcium is absorbed by two mechanisms: 1) active transport, and 2) passive transport. Active transport, which acts predominantly at low luminal concentrations of calcium ions, is controlled through the action of 1,25-dihydroxyvitamin D ($1,25[OH]_2D_3$).[58] Vitamin D increases calcium uptake at the brush border of the intestinal mucosal cell.[60] When calcium intakes are high, calcium is absorbed by unsaturable, passive diffusion, whereas the

active transport mechanism becomes much more important when calcium intakes are low and requirements are not being met.[58]

Regulation of blood calcium levels is a process utilizing parathyroid hormone (PTH), vitamin D, and calcitonin.[59] Parathyroid hormone is released from the parathyroid gland when blood calcium levels drop.[61] Parathyroid hormone acts to increase phosphate excretion and calcium reabsorption in the kidney.[61] Parathyroid also acts at the skeletal level to stimulate bone resorption by osteoclasts. Both vitamin D and PTH act to increase calcium reabsorption in the kidney and to free calcium from the skeleton.[60] As mentioned previously, calcitonin is a hormone released in response to high serum calcium. Calcitonin acts directly on osteoclasts to inhibit bone resorption in order to reduce the amount of calcium released into the bloodstream.[59]

As 99% of the total body calcium is in the skeleton, the rate of growth and skeletal development have a significant impact on calcium retention, which in turn has a profound effect on the dietary requirement for calcium. Current daily dietary recommendations or Adequate Intakes (AI) for calcium are 500 mg for children aged one to three years, 800 mg for children aged four to eight years, 1300 mg for adolescents aged nine to 18 years, 1000 mg for adults aged 19 to 50 years, and 1200 mg for adults aged 51 years and older.[62] Calcium requirements are much higher during skeletal modeling than during the bone remodeling phase when longitudinal bone expansion no longer exists and periosteal bone expansion is at its minimum.[63, 64] During the pubertal growth spurt, teenagers may develop a transient osteopenia, a reduced bone mass due to inadequate osteoid synthesis,[65] and increased intracortical porosity,[66] presumably resulting from a high demand for calcium. Endosteal bone apposition with a concomitant increase in bone mineral density proceeds during the bone remodeling phase.[66] However, this process occurring prominently in young and middle aged adults requires less calcium as compared to the bone

modeling phase with a concurrent decline in calcium requirement. In older adults, where increased remodeling and accelerated bone loss occurs, calcium recommendations are increased to accommodate these changes.

The major sources of calcium in the U.S. are dairy products, with small amounts coming from grains, fruits, and vegetables. Consuming the AI for calcium has been demonstrated to reduce the risk of osteoporosis.[67, 68] Unfortunately, calcium intakes of most population groups are below dietary recommendations. According to the Third National Health and Nutrition Examination Survey, most females of all race/ethnic groups over the age of 11 years fail to consume the recommended intakes of calcium.[69] Similarly, data from the 1994-1996 Continuing Survey of Food Intakes by Individuals reveal that many population groups, particularly adolescent and older females, and adults in later years, consume diets containing significantly less calcium than the amount recommended.[70]

Vitamin D

More recently, vitamin D has received attention along with calcium as an important dietary modifier for enhancing skeletal health.[71] Due to concerns that individuals do not get enough vitamin D through the primary source, sunlight. Recommendations for dietary intake are set at a high level to be adequate for individuals having no sun exposure.[72] However, Moore et al.[73] reported intakes of vitamin D from food sources and dietary supplements were not meeting recommended levels among the U.S. population, where the lowest levels were reported by female teenagers and female adults.

Vitamin D is a fat-soluble vitamin and aids calcium's effort in forming and maintaining stronger bones by promoting enhanced calcium absorption in the intestine. The main source of vitamin D is sunlight, and most people meet their requirement of vitamin D by the conversion of

precursors in the skin to the active form of vitamin D. With sustained exposure to sunlight, there is increased production of inactive vitamin D metabolites, providing a mechanism for preventing vitamin D intoxication.[71] Several factors can limit the production of vitamin D by the skin, including location (those who live in northern latitudes during the winter months do not get adequate exposure to sunlight),[74] how much body surface is covered by clothing or sunscreen, the degree of skin pigmentation (darker skin tends to take longer to make active vitamin D),[60] and age (the skin of older individuals is less efficient in making vitamin D).[60] Food sources of vitamin D include fish oils, egg yolk, butter and liver. Fortification with vitamin D in certain food products is common, particularly in dairy foods, orange juice, and cereals.

In the presence of ultraviolet light, provitamin D_3 (7-dehydrocholesterol) is cleaved to previtamin D_3.[71] Previtamin D_3 isomerizes to vitamin D_3 (cholecalciferol), which is carried to the liver or stored in fat by a vitamin D-binding protein.[72] Activation of vitamin D_3 requires two hydroxylation reactions making vitamin D more biologically effective. The first hydroxylation, though not tightly regulated, occurs in the liver, where vitamin D_3 becomes 25(OH)D.[72] The second hydroxylation occurs in the kidney, and is well-regulated. Further activation of 25(OH)D is accomplished by 25(OH)D-1$\propto$-hydroxylase, which is up-regulated by PTH and down-regulated by its biologically active product 1,25-$(OH)_2$D, or calcitriol.[72] When serum calcium levels drop, PTH is released and acts on the kidney to increase hydroxylation of 25(OH)D.[61]

The direct effect of 1,25-$(OH)_2$D on bone work in concert with those already described in the intestine, to maintain or increase serum calcium concentrations. 1,25-$(OH)_2$D acts on osteoblasts and their precursors, causing the production of receptor activator of NFkappaB ligand (RANK-L), which binds to osteoclasts to stimulate their development into osteoclasts.[75] This leads to an increase in osteoclastic bone resorption. 1,25-$(OH)_2$D also directly stimulates alkaline

phosphatase activity and the production of osteopontin and osteocalcin by osteoblasts.[75] While it has been suggested that 1,25-$(OH)_2D$ may directly influence skeletal mineralization, the balance of evidence suggests that this occurs indirectly, as a result of vitamin D effects on serum calcium and phosphate concentrations.[71]

Adequate intakes for vitamin D may be listed on food and dietary supplement labels in either micrograms (μg) or International Units (IU). The biological activity of 1 μg vitamin D is equal to 40 IU.[62] Adequate intakes for vitamin D in infants, children, and adults are listed in Table 2.2.

Table 2.2. ***Adequate Intake for vitamin D for infants, children, and adults.***

Age (years)	Children (μg/day)	Men (μg/day)	Women (μg/day)	Pregnancy (μg/day)	Lactation (μg/day)
Birth-13	5 (=200 IU)				
14-18		5 (=200 IU)	5 (=200 IU)	5 (=200 IU)	5 (=200 IU)
19-50		5 (=200 IU)	5 (=200 IU)	5 (=200 IU)	5 (=200 IU)
51-70		10 (=400 IU)	10 (=400 IU)		
71+		15 (=600 IU)	15 (=600 IU)		

Adapted from Institute of Medicine, Food & Nutrition Board. (1997)

Non-nutritional lifestyle factors

A number of non-nutritional lifestyle factors have been linked to bone health with the most attention focused on tobacco, alcohol, and physical activity. Smoking tobacco has been found to have a negative cumulative effect on bone mineral.[76, 77] Smoking increases bone resorption and decreases formation.[78] This may be due to the direct effects of nicotine on

osteoblasts[79] as well as to decreased production and increased degradation of estrogen.[80] Alcohol abuse has been found to be associated with numerous factors that contribute to low bone mass, along with poor nutrition, leanness, liver disease, malabsorption, vitamin D deficiency, hypogonadism, and parathyroid dysfunction.[81] Excessive alcohol consumption has found to depress osteoblast function.[82] On the other hand, moderate alcohol consumption is unlikely to be associated with lower bone density.[83] Surprisingly, researchers have even shown a beneficial effect of moderate alcohol consumption.[84] However, a potential confounder in this association is that higher socioeconomic status (and thus perhaps better nutrition) may be associated with the benefits of moderate alcohol intake on bone health.

Physical activity loading on the bone In accordance with Wolff's Law, it is generally believed that the architecture and mechanical behavior of skeletal tissue is dependent on the loading environment that it experiences.[85] The skeleton's response to a load depends on the strain magnitude, rate, distribution, and cycles in the target bone.[86, 87] Magnusson et al.[88] found higher densities in loaded regions of the bone as opposed to inactive regions, suggesting that unloaded and weight-loaded skeletal regions may respond differently to increased and decreased physical activity. Bone growth during modeling and remodeling, encountered by loading, varies in magnitude and distribution, and therefore, increases or decreases in direct relation to the applied force (See Figure 2.3).[30, 89] For example, a decrease in bone quality can be directly related to a change in lifestyle, such as a decrease in physical activity.[90-92]

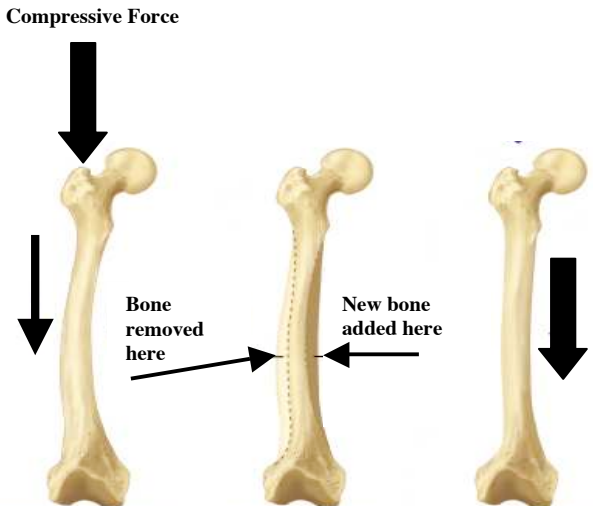

Figure 2.3. **Forces On The Bone**, Adapted from Einhorn, 1996.[3]

Bone cells control the amount of mineral present and how much tissue is organized. Loading from weight-bearing exercise can change the bone cells by affecting the cell size and shape or by affecting fluid flow or pressure.[93] The effect of loading on bone cells is very rapid and results in an increase in bone formation and a decrease in bone resorption activity.[87] It is believed that two areas within the bone respond to the loading strain. Osteocytes, including the osteoblasts that are next to them within the bone tissue, and the bone marrow cells are thought to be the two regulation sites.[94]

In the mid 1980s, Rubin and Lanyon published experiments using an avian ulna that helped define several aspects of the bone formation response to mechanical loading.[95-97] From their research, the following has been determined:

- Short sessions of variable loads increase bone formation in direct proportion to the strain imposed.[96]
- Initial response to increased loading is to increase woven bone formation, but if the strain is continued, bone is integrated.[96]
- Load that is not variable does not increase remodeling activity.[95]

- Short bursts of load bearing activity seem to have a much greater effect on the osteogenic response than longer periods of repetitive strains.[96]
- Distribution, intensity, and rate of strain are all important in the bone remodeling response.[96, 97]
- Torsion strain (twisting) is less osteogenic than longitudinal compression strain (force pushing down from both ends) in the avian ulna.[95]
- Applying a force to a bone causes the bone to bend until the structure of the bone keeps it from bending any further.[96]
- Bone cells detect the strain and respond by changing the bone structure to lessen the force placed on the bone.[95]

The practical importance of these findings by Rubin and Lanyon is the potential for further improvement of exercise regimens designed for bone building.

Generally, studies have demonstrated that physically active individuals of all ages have superior skeletal mass than those who are less active.[98-105] The magnitude of this difference in bone depends upon the mode and intensity of the activity, the age at which activity began, and the number of years spent training.[45] Weight-bearing exercise, particularly in childhood, adolescence, and young adulthood, may contribute to the prevention of osteoporosis by increasing the amount of bone accrued during growth.[88, 103, 106, 107] It has been suggested that during this period of growth, weight-bearing exercise produces its most beneficial effects because this is the time period in which peak bone mass is attained. [108-110] Peak bone mass has been defined as the highest level of bone mass achieved as a result of normal growth.[111] Studies have shown that peak bone mass usually occurs prior to age 20 at the proximal femoral sites, while total skeletal mass peaks 6-10 years later. [89, 112] By maximizing peak bone mass with

weight-bearing exercise in early development, one may be able lower the risk of osteoporotic related fractures (See Figure 2.4). However, before recommendations regarding the role of exercise can be made, more prospective research is needed to determine if the high bone mass attained in childhood, adolescence, and young adulthood is still present in late adulthood and more importantly, high enough to reduce incidences of fractures.

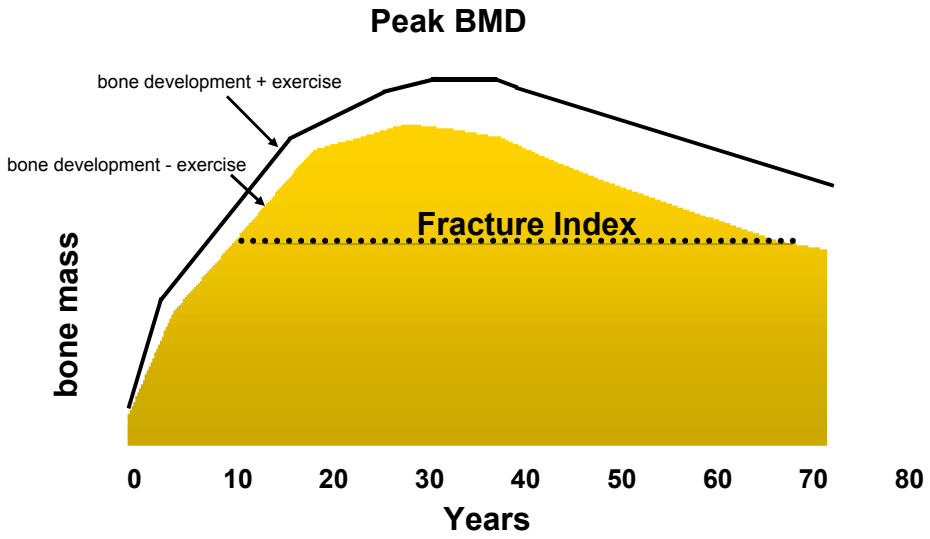

Figure 2.4. During bone development, the goal is to increase peak bone mass such that people enter adulthood with the greatest amount of bone possible. With early introduction of exercise, a higher peak bone mass can be achieved and therefore, reduces the possibility of fracture later in life.

Body composition

Total body mass (i.e., weight) can be divided into a number of relevant compartments and tissue masses. This can be done anatomically (bone, muscle, fat, residual) or chemically (water, lipid, protein). The relative proportion of each of the tissue types that comprise total body weight is uniquely different among individuals and can have an impact on bone health.

Most researchers attempt to analyze body composition with dual-energy X-ray absorptiometry (DXA). DXA measures bone mineral content (BMC) and area of the bone scanned. In adult bone studies, areal bone mineral density (aBMD) is often reported because the

quotient of BMC and area is a more useful measure because it partially corrects for the effect of size,[113] although it fails to assess the skeleton in three dimensions. Because DXA technology allows the researcher to measure total body bone and soft-tissue, it can compartmentalize bone mineral, fat, and lean tissue mass with reasonable accuracy. However, DXA soft-tissue measurements assume uniform hydration within each of the three compartments, yet lean mass in particular can vary in water content by up to 85%.[114, 115] This introduces the potential for error in DXA measurement of soft tissue. Error may also be introduced as DXA assumes that the soft tissue above and below the bone (where it cannot be measured) is equivalent to that on either side of the bone. If more fat is located above the spine than on either side of it, aBMD will be underestimated. Dual energy X-ray also assumes that bone marrow is included within these soft tissue calculations, although this is not true in the strictest sense, where a 50% change in bone marrow fat can change aBMD by 5-6%.[116] Aging causes a redistribution of body fat and an increase in yellow marrow.[116] Taken together, these changes could result in erroneous estimations by DXA in older populations. Despite these limitations, DXA has provided researchers with a better understanding of the role of soft tissue (fat and lean) in determining bone mass. The degree to which soft tissue mass influences bone remains an area of controversy since lean mass, fat mass, and total body weight are interrelated as well as being associated with lifestyle factors such as physical activity. Furthermore, the specific and relative contribution of each tissue to bone varies across life span and among diverse ethnic groups.[117]

Total body mass and aBMD

Studies in both children[118] and adults[119, 120] identified body weight as the primary predictor of bone mass at regional sites (hip, spine) and for the total body. Cross-sectional studies have demonstrated that body weight may be a more important predictor of aBMD in older than in

younger populations. Post-menopausal women who were at least 10% above their ideal body weight had significantly greater aBMD at the spine, hip, and radius than did post-menopausal women of normal weight.[121, 122] The correlation, however, was not found in pre-menopausal women, suggesting that the increased weight may be a larger factor in slowing post-menopausal bone loss than in maintaining bone mineral in the pre-menopausal adult skeleton.[121] In large cohort studies, such as the Framingham study, body weight explained up to 20% of the variance in aBMD for both weight-bearing (lumbar spine, femoral neck) and non-weight-bearing sites (radius).[123] The Rancho Bernado study found that body size explained a greater proportion of the variance of the weight-bearing hip and spine sites (17% and 12%, respectively) than of the non-weight-bearing radial sites (8% or less).[124] The four-year longitudinal Framingham Osteoporosis Study in women reported that lower baseline weight and weight-loss overtime were more strongly associated with aBMD loss than factors such as age, smoking, caffeine, alcohol use, or physical activity.[125]

Lean mass and aBMD

Among the specific tissues that DXA can distinguish between, lean (fat-free) mass proved to be the best predictor of aBMD in young,[39, 126-128] middle-aged,[103, 129] and elderly[119, 130, 131] women. In a cross-sectional study of young women (aged 10 to 26 years), each kilogram of lean mass, with all other factors held constant, was associated with approximately 1% greater proximal femoral BMD.[39]

Lean mass may have different effects on bone mass at various stages of life. Total lean mass may be the best soft tissue determinant of lumbar and total body aBMD in children[39, 118, 132] and pre-menopausal women,[133] whereas it may be less important during infancy[134] and following menopause.[129, 135] In elderly women (60 to 89 years) lean mass was independently associated with

aBMD at all sites when baseline data were analyzed cross-sectionally.[119] However, in the longitudinal follow-up, change in lean mass did not predict change in aBMD.[136] This may occur because fat mass accounts for a greater proportion of overall body mass in the very young and the elderly.[114] Moreover, the association between lean mass and aBMD may have been established the strongest during growth and development, so that the associations seen cross-sectionally in later life may reflect early bone development.[129]

Fat mass and aBMD

During childhood fat mass likely has less of an influence on aBMD compared to lean mass.[39] However, fat mass is an important determinant during the pre-menopausal years[137-139] and becomes even more significant after menopause.[140] Fat mass may more strongly predict changes in aBMD in postmenopausal women better compared to lean mass.[141] Potential mechanisms whereby fat may influence bone mass in addition to its loading effect are 1) aromatization of androgens, 2) alteration of the binding capacity of estrogen for sex hormone binding globulin, 3) acting as a storage place for steroid hormones, and 4) via development of obesity-related insulin resistance.[142]

Gymnastics Training and Bone

Due to concern regarding adverse effects of intense training on the physical health of gymnasts, growth and development have become major areas of study in women's gymnastics. Gymnasts on average are smaller than non-gymnasts, show a period of catch-up growth,[143, 144] and provide an ideal model for bone density studies investigating the interaction of exercise loading and bone.[143-148] Because female gymnasts typically begin training at such a young age, they also provide ideal models for studying the interaction of intense training and growth in female child athletes. Interestingly, in spite of questionable nutrition and menstrual irregularities,

college gymnasts commonly have higher aBMD than other college athletes or nongymnast controls.[42, 46, 47, 149] Gymnasts may provide an important insight into the development and maintenance of bone mass.

Young gymnasts

The importance of weight-bearing physical activity during childhood to maximize peak bone mass has become widely accepted. Before puberty and/or during puberty, both cross-sectional and longitudinal studies have demonstrated that gymnastics training is an effective means of promoting osteogenic effects.[105, 108, 150-154] The studies listed below examine the relationships between gymnastics and aBMD in children and adolescents.

Cross-sectional studies

Competitive gymnasts and swimmers, aged seven to nine years, who trained year-round (average 13.9 hours per week for gymnasts; 4.7 hours per week for swimmers) were compared to non-athletic controls.[150] Gymnasts had less fat mass and lower percent body fat than swimmers and controls, and weighed significantly less than the swimmers. After controlling for body weight, the gymnasts had significantly higher total body aBMD than swimmers and controls, 10% and 8% respectively.

Similarly, Dyson et al.[151] compared aBMD between16 female gymnasts with16 non-athletic controls between the ages of seven and eleven years. Areal BMD of the gymnasts was 16% higher at the trochanter, and 8% higher at the femoral neck compared to the controls. However, the controls were not matched for size with the gymnasts and were significantly taller with greater percent body fat.

Nickols-Richardson et al.[152] observed premenarcheal gymnasts (n = 16; aged eight to 13 years) who had been training for an average of six years and a group of age-, height- and weight-

matched controls (n = 16). The aBMD means of the gymnasts were significantly higher than controls at the total proximal femur (12%), femoral neck (14%), trochanter (12%), Ward's triangle (31%), and lumbar spine (13%).

In another study, peripubertal gymnasts (n = 65; mean age 13 years; prepubertal aged 12 years; pubertal aged 14 years) were compared to runners (n = 63) and non-athletes (n = 56).[153] Gymnasts were shorter and lighter than both control groups. The femoral neck aBMD in the prepubertal gymnasts was approximately 13% higher than the controls, whereas in the pubertal gymnasts the femoral neck aBMD was 20% higher than the controls. Although these results suggest that the gymnastics training had beneficial influences on aBMD, it seems to have the greatest effect on the skeleton of pubertal females, compared to females of prepubertal stages.

These cross-sectional studies in young gymnasts demonstrate that greater aBMD is seen in gymnasts, even at young ages. Only one of the above studies matched gymnasts and controls for age, height and weight, which could have affected results, possibly increasing the magnitude of differences seen. Due to the nature of the research design, it is difficult to rule out other environmental factors that could have affected aBMD in gymnasts compared to controls in these studies.

Longitudinal Studies

Bass et al.[108] followed 37 elite level gymnasts (mean age 10 years) for 12-months, who were bone-age-matched to 17 controls (mean age 9 years) with less than two hours of weight-bearing activity per week. The gymnasts had a 30 to 85% greater increase in aBMD than controls at the total body, spine, and legs. At baseline, the gymnasts' mean aBMD values were already 0.4 to 2.1 standard deviations higher than the predicted mean at the arm, spine, and hip sites. Volumetric bone density was also determined using a geometric formula. Volumetric BMD was

estimated in the gymnasts to be 12% higher at the lumbar spine and 16% higher at the femoral midshaft than the control group.

In another study, Courteix and colleagues[154] found similar results over 12 months. The experimental group included 14 gymnasts (mean age 12 years) who had trained 12 to 15 hours per week for three years before starting the study. The control group (mean age 12 years) consisted of 15 non-exercising children and six swimmers training for five to six hours per week. Lumbar spine, femoral neck, trochanter, Ward's triangle and radius aBMD were all significantly higher in gymnasts vs. controls at baseline (11%, 14%, 10%, 10%, and 13% higher, respectively). At follow-up, lumbar spine, femoral neck, trochanter, Ward's triangle and radius aBMD continued to remain significantly higher in the gymnasts compared to controls (12%, 15%, 14%, 16%, 17% higher, respectively). However, changes in aBMD over the one-year period were not significantly different between the two groups.

Laing et al.[105] examined changes in bone and body composition in peripubertal gymnasts (n = 7; mean age 11 years) and controls (n = 10; mean age 11 years) over three years. At baseline and year three, gymnasts were training a mean of 12 hours and 17 hours per week, respectively. The controls had never participated in gymnastics but were competitive in other activities such as basketball, soccer, softball, or tennis. At baseline, no initial differences in height or weight between the gymnasts and controls were observed; however, the gymnasts had significantly lower percent body fat and higher aBMD at all measured sites, except the total body. Over 36-months, the gymnasts increased up to 30% more than controls in the total body, trochanter and total proximal femur aBMD.

The prospective studies discussed provide evidence that gymnasts have higher aBMD than controls. However, the extent of the difference and gains achievable are not clear. The

differences in aBMD seen could be explained by training intensity, years of training, and length of follow-up. Since none of the aforementioned studies began with similar aBMD values in the two groups, the amount of bone gain achievable could be related to initial values or to baseline activity levels.

College-aged gymnasts

Over the past decade, several researchers have observed aBMD of college-aged gymnasts. From these cross-sectional and longitudinal studies, the gymnasts were found to have higher aBMD compared to other sports groups or sedentary controls.[42, 46, 155-157] These studies are reviewed in detail below.

Cross-sectional studies

Nichols et al.[155] compared college-aged women (n = 46) involved in four varsity sports (gymnastics, basketball, volleyball, and tennis) with non-athletic, non-matched controls (n = 12). When compared to the other sport groups and controls, the gymnasts were significantly younger, shorter, lighter, and had lower percent body fat. The sport groups had significantly greater aBMD than non-athlete controls at the lumbar spine (8.7%), femoral neck (10.4%), and total body (7.5%). However, it was found that the gymnasts, even after adjusting for weight, did not have significantly higher aBMD than any of the other sports.

Kirchner et al.[46] compared college gymnasts (n = 26) to age-, height-, and weight-matched controls (n = 26). The gymnasts were found to have higher aBMD measures at the lumbar spine (18%), total hip (21%), femoral neck (22%), Ward's triangle (25%), and whole body (10%). Interestingly, these higher aBMD values were observed despite the fact that gymnasts as a group had inadequate dietary calcium and a higher tendency to have an interruption of their menstrual cycle.

Robinson and colleagues[42] observed aBMD and menstrual status in competitive college gymnasts (n = 21) and runners (n = 20) with a group of non-exercising, non-matched controls (n = 19). The gymnasts were the shortest of the three groups, weighed less than the controls, and had the highest occurrence of menstrual disturbances (28% compared to 15% in runners). The gymnasts had significantly higher aBMD than the controls at the femoral neck (10.3%) and significantly higher aBMD at all sites compared to the runners (6%, 16%, and 19% higher at whole body, lumbar spine, and femoral neck, respectively).

Taaffe et al.[156] compared competitive college swimmers (n = 26) and gymnasts (n = 13) with a group of non-matched controls (n = 19). When compared to the gymnasts and controls, the swimmers had greater amounts of lean mass. However, when body size was statistically adjusted for, the differences between the groups in terms of lean mass no longer existed. The gymnasts had the highest measures of aBMD among the groups tested at the femoral neck (21.7%, 12.8% higher than swimmers and controls, respectively) and trochanter (16.7%, 12.7% higher than swimmers and controls, respectively), although no differences were seen at the whole body and lumbar spine. The aBMD of the swimmers and controls did not differ from each other at any of the sites measured. One possible explanation suggested by the author for this finding was that the amount of muscle pull required for competitive swimming may not be great enough to cause bone mineral accrual.

These cross-sectional studies in college-aged athletes all found higher aBMD in gymnasts compared to other sports groups and controls with exception of only one. Additionally, the magnitude of difference between gymnasts and other groups appears to be greatest at the femoral neck (10 to 22%). This may be due to the fact that most of the loading from gymnastics training

is in the legs and hips. Again, however, cross-sectional studies cannot account for differences prior to measurement, resulting in less conclusive findings.

Longitudinal studies

Nichols et al.[157] tracked college gymnasts (n = 11) and non-matched, sedentary controls (n = 11) over a 27-week training period, measuring aBMD at the spine, hip, and whole body. At baseline and at follow-up, body weight and percent body fat were lower in the gymnasts. At the start of the training period, the gymnasts had significantly higher aBMD at the lumbar spine (7.8%) and femoral neck (9.6%) than the controls. At the end of the season, the gymnasts increased significantly in lumbar spine aBMD (1.3%) but not in the femoral neck region. No aBMD changes occurred in the control group.

Over an 8- (cohort I) or 12-month (cohort II) period, Taaffe et al.[158] followed the two groups of college gymnasts (n = 26 for 8 months and n = 8 for 12 months) with college swimmers (n = 11 for 12 months) and a group college runners (n = 36 for 8 months) with two control groups (n = 14 for 8 months and n = 11 for 12 months). From baseline to follow-up in both cohorts, the gymnasts had significantly higher gains at the lumbar spine aBMD (2.3 to 2.8% higher) compared to runners, swimmers and controls. Areal BMD gains for the gymnasts were also higher at the femoral neck (2.5 to 5.5% higher) than the runners and swimmers, but not controls. The group of runners had a significant decrease in aBMD at the femoral neck (-1.5%) after eight months. The researchers concluded that the high impact loading (rather than selection bias) experienced by the gymnasts underlies high aBMD values characteristic of gymnastics training.

Although gains of 1 to 5% in aBMD over time (seven to 12 months) were seen in the only two known longitudinal studies of college gymnasts, it is unlikely that these gains from the

college training years will persist after detraining.[159] However, since most college gymnasts begin their training in childhood, it is possible that their earlier training may benefit them more than their college training. Although, due to the nature of each study's design, this hypothesis cannot be determined.

Retired gymnasts

A few cross-sectional studies have been conducted in retired female gymnasts as well as one longitudinal study. With regard to the effects of previous gymnastics training, female former gymnasts have been shown to have higher site-specific measures of aBMD relative to age-matched controls.[47, 104, 108, 160] However, an important point is that a proportion of these former gymnasts have continued various forms of physical activity that might have assisted with retaining bone mass. The following sections will discuss the studies of former gymnasts in more detail.

Approximately 10 years from cessation of training, Lindholm and colleagues[161] compared aBMD measures in 19 young women (mean age of 21 years) who had been in elite gymnastic training during their prepubertal and pubertal years with 21 age- and weight-matched controls. The mean age of menarche of the former gymnasts and the controls was 14 and 12 years, respectively. Fourteen of the gymnasts had been or were using oral contraceptives and most of the non-users had regular menstrual periods at the time of the investigation. During the years preceding the study, physical activity among the former gymnasts had gradually declined. Although 11 of the 19 gymnasts had delayed puberty compared to controls, no significant difference was found in total body or lumbar spine aBMD compared to controls. However, it was observed that aBMD of the arm was significantly higher (7%) than controls. The authors did note from the study that during the first five years after detraining, 16 of the former gymnasts mended

injuries related to their training. Additionally, at the time of the follow-up measures, 5 of those 16 gymnasts were still suffering from previous sport-related injuries.

Bass et al.[108] observed significantly higher aBMD measures in a group of former female elite gymnasts (n = 36; mean age of 25 years) when compared to a group of age- and weight-matched controls (n = 15) at the femoral neck (13.4%), Ward's triangle (11.8%), and total body (3.4%). The retired gymnasts reported to have started training at a mean age of eight years, trained for a mean of 10 years, and had been retired for an average of 16 years. This observation suggests that residual benefits of bone may have been attained during pubertal years and is still present in early adulthood.

Zanker and colleagues [47] found similar results in 18 female former gymnasts (mean age of 25) and 18 sedentary controls. The former gymnasts and controls were paired individually to match for age, height and weight and the groups did not differ in fat mass, lean tissue, or percent body fat. The gymnasts reported to have commenced training at least three years pre-menarche and had trained post-menarche for two or more years. They also had trained continuously for 5-12 years and had retired between age 14 and 22 years. Greater aBMD measures were found in the former gymnasts at the total body (5.8%), lumbar spine (9.0%), non-dominant total proximal femur (9.0%), and non-dominant femoral neck (8.0%) when compared to controls.

Kirchner et al.[104] reported that 18 retired female college gymnasts (mean age of 36 years) who had started training at a mean age of 12 years had higher aBMD values at the lumbar spine (16%), femoral neck (18%), Ward's triangle (22%), and total body (9%) when compared to age-, height-, and weight-matched control group. When past physical activity was assessed between the two groups for the previous 10 years, the retired gymnasts reported significantly more hours per week of exercising and at higher intensities than the controls.

Kudlac et al.[160] is the only group to date to observe prospective changes in aBMD in retired female college gymnasts. At baseline, the former gymnasts (n = 10; mean age 20 years at baseline and 24 years at follow-up) were matched with controls (n = 9; mean age 24 years at baseline and 27 years at follow-up) with respect to height and weight; however, the age between the two groups was significantly dissimilar. Initially, the former gymnasts had significantly lower fat mass and higher aBMD measures (at the total body, femoral neck, trochanter, Ward's triangle, but not the lumbar spine) compared to controls. At the follow-up, approximately 4-years later, fat mass was no longer different between groups but aBMD for the gymnasts remained significantly greater than the controls at the total body, femoral neck, trochanter, and Ward's triangle but not for the lumbar spine. Over time, significant declines in femoral neck, Ward's triangle, and trochanter aBMD were found in both gymnasts and controls (0.72% to 1.9% per year), but only gymnasts had a significant decline at the lumbar spine (0.87% per year).

In summary, the findings of both the former female gymnast cross-sectional studies and longitudinal study suggest that an elevated aBMD, associated with gymnastics training throughout childhood, adolescence, and early adulthood, is at least partially retained in early to middle adulthood. This apparent retention occurred in spite of the adoption of a sedentary lifestyle compared to the years spent in training. It is possible that a minimal level of physical activity may have helped to conserve early bone gains; however, this remains to be determined.

Long-term Effects of Athletics on Bone Health

With high impacts on the skeleton, gymnastics training may be the type of sports activity that needs to be performed during early bone development in order to provide greater osteogenic effects later in life. More importantly, gymnastics training may result in adequate bone reserves to offset bone loss in the aging years and possibly reduce fracture risk. However, no studies have

been conducted in retired gymnasts over a long-term period to see if the higher aBMDs are maintained. Cross-sectionally, it was observed in former male soccer players retired for over 25 years and over 60 years or age, no significant residual benefit on aBMD existed when compared to controls.[162] Similarly, Magnusson et al.[88] did not find differences in aBMD in a group of former male soccer players between the ages of 50 to 85 years when compared to controls. The cross-sectional design of these studies, however, limits the interpretability of their results. Neither of the studies reported level of training of the former soccer players nor age of initiation of soccer activity, which may have significant impact on peak bone mass development and bone loss later life.[108, 163-165]

Evidence has revealed that the ground reaction forces from gymnastics training can generate up to 10 to 12 times body weight,[166] whereas, sports that involve running, lifting, or hitting only generate loading forces two to five times body weight.[167-170] Although sports other than gymnastics may have benefits in other dimensions of health, the possibility exists that the smaller loading forces experienced from running, lifting weights, or swinging a racquet may not provide enough loading force to have beneficial effects on bone in later life, particularly at the lumbar spine, femur, or radius.

Cross-sectional studies of college gymnasts and former competitive gymnasts (retired 15 years or less) have found significantly higher aBMD in gymnasts compared to nonathletes.[42, 46, 47, 104, 108, 157] Additionally, cross-sectional studies of non-gymnast adult athletes still competing or retired from the sport less than 16 years have shown greater aBMD or BMC percent differences at sport-specific measured sites when compared to controls (See Table 2.3).[171-178] In contrast, other cross-sectional studies of non-gymnast adult athletes retired from their sport more than 20 years have shown no benefit of bone maintenance when compared to controls.[88, 109, 162, 179-181] The

possibility exists that after 20 years of retirement from the sport, previous competitive training may not override other important bone-related factors such as nutrition, hormone status, or lifestyle. Currently, no aBMD data exist for former gymnasts who have been retired from the sport for more than 20 years.

It is possible that a lower level of undefined activity may retain some aBMD benefits acquired during an active career. The findings in the male soccer study accord with this view by showing a correlation between current activity level and femoral neck aBMD.[162] Moreover, Kontulainen et al.[182] observed similar results in a four-year prospective study of 13 formerly competitive male tennis players (mean age of 26 years at baseline) when comparing dominant-to-nondominant playing arm. The players reported training a mean of eight hours per week at baseline and a mean of three hours per week at follow-up. No significant percent differences were observed in BMC at the humeral shaft (25% at baseline and 26% four years later) or proximal humerus (19% at baseline and 18% four years later). The same group of researchers observed similar results in a group of former competitive female tennis and squash players (n = 36; mean age of 22 years at baseline) five years after intensive training.[183] The athletes reported a mean of five days per week and 80 minutes per session of training at baseline, whereas at follow-up, the training decreased to a mean of one day per week and 60 minutes per session. Again, no significant percent differences were seen in BMC at the humeral shaft (19% at baseline and 17% at follow-up) or proximal humerus (21% at baseline and 22% at follow-up). In another

Table 2.3. Cross-sectional studies of adult nongymnast athletes and controls in which aBMD or BMC was measured.

Authors	Group Specification **(# in each group)**	**Gender and Age** **(years)**	**Competitive Training (years)**	**Athletes/Controls (%) Difference aBMD or BMC or Dominant/non-dominant limb (%) Difference aBMD or BMC**	**Measurement Device/Comments**
Nilsson et al. (1974)[184]	*Athletes from a variety of sports (n=88) *Controls (15)	Males (18-25)		+ 27-47% at distal femur in the various sporting groups	SPA
Aloia et al. (1978)[172]	*Marathon runners (n=30) *Controls (n=16)	Males (30-50)		+ 4% BMC at the wrist	SPA
Huddleston et al. (1980)[185]	*Tennis players (n=35) comparing playing and non-playing arm	Males (70-84)	Range of 25 to 72	+ 10% BMD in playing arm versus non-playing arm	SPA
Lane et al. (1986)[186]	*Long-distance runners (n=8) *Controls (n=8)	Males (58 mean)		+ 44% BMC at lumbar spine (L1)	QCT
Suominen (1988)[187]	*Long-distance runners (n=10) *Power athletes (n=8) *Controls (n=52)	Males (46-60)	mean=27 for distance athletes; mean=34 for power athletes	+43% BMC more calcaneus in distance athletes +35% BMC more calcaneus in power athletes	SPA
Orwoll et al. (1989)[188]	*Swimmers (n=58) *Controls (n=78)	Males (40-85)	mean=13	+ 4% BMC in proximal radius	SPA
Virvidakis et al (1990[173]	*Competitive weightlifters (n=59) *Controls (n=60)	Males (15-20)		+ 51% BMC in distal forearm + 41% BMC in proximal forearm	SPA
Suominen et al. (1991)[189]	*Olympic trained athletes (n=97) *Controls (n=42)	Males (71-80)		+ 19-28% BMC in calcaneus + 11-16% BMD in calcaneus	SPA
Heinonen et al.(1993)[174]	*Competitive weightlifters (n=18) *Controls (n=25)	Females (20-29)	mean=4	+ 13% aBMD at lumbar spine + 9% aBMD at femoral neck + 23% aBMD at distal radius	DXA
Karlsson et al. (1993)[175]	*Competitive weightlifters (n=19) *Controls (n=26)	Males (41 mean)		+ 8% aBMD in total body +11% aBMD at lumbar spine	DXA
Haapasalo et al. (1994)[163]	*Competitive squash (n=19) *Controls (n=19)	Females (19-33)	mean=6	+ 18% BMC in proximal humerus in side-to-side comparison within athletes + 12% BMC in distal radius in side-to-side comparison within athletes + 4% BMC in proximal humerus in side-to-side comparison within controls + 2% BMC in distal radius in side-to-side comparison within controls	DXA

SPA = single photon absorptiometry X-ray
QCT = quantitative computed tomography
DXA = dual-energy X-ray absorptiometry

Authors	Group Specification **(# in each group)**	**Gender and Age** **(years)**	**Competitive Training (years)**	**Athletes/Controls (%) Difference aBMD or BMC or Dominant/non-dominant limb (%) Difference aBMD or BMC**	**Measurement Device/Comments**
Fehling et al. (1995)[176]	*College volleyball (n=8) *Controls (n=17)	Females (20 mean)		+ 16% aBMD at lumbar spine + 18% aBMD at femoral neck + 14% aBMD in total body	DXA
Fehling et al. (1995)[176]	*College swimming (n=7) *Controls (n=17)	Females (20 mean)		+ 3% aBMD at lumbar spine + 1% aBMD at femoral neck + 3% aBMD in total body	DXA
Karlsson et al. (1995)[179]	*Competitive weightlifters (n=48) *Controls (n=66)	Males (65-79)	mean=13	+ 7% aBMD at lumbar spine - 2% aBMD at femoral neck	DXA Retired for a mean of 30 years
Kannus et al. (1995)[190]	*Competitive tennis (n=105) *Controls (n=50)	Females (16-50)	mean=10	+ 15% BMC in proximal humerus in side-to-side comparison within athletes + 13% BMC in distal radius in side-to-side comparison within athletes + 5% BMC in proximal humerus in side-to-side comparison within controls +4% BMC in distal radius in side-to-side comparison within controls	DXA
Taaffe et al. (1995)[156]	*College swimmers (n=26) *Controls (n=19)	Females (19 mean)	mean=12	+ 0.3% aBMD at lumbar spine - 11% aBMD at femoral neck - 2% aBMD in total body	DXA
Etherington et al. (1996)[102]	*Elite middle/long distance runners (n=67) *Controls (n=585)	Females (40-65)		+ 9% aBMD at lumbar spine + 12% aBMD at femoral neck	DXA Retired for mean of 16 years
Karlsson et al. (1996)[180]	*Competitive weightlifters (n=16) *Controls (n=133)	Males (35-49)		+ 7% aBMD in total body +10% aBMD at femoral neck	DXA
Karlsson et al. (1996)[180]	*Competitive weightlifters (n=16) *Controls (n=133)	Males (65-79)	mean=7	+3% aBMD in total body - 4% aBMD at femoral neck	DXA Retired for a mean of 25 years
Khan et al. (1996)[181]	*Competitive ballet (n=101) *Controls (n=101)	Females (51 mean)		+ 0.009 aBMD at total femur - 0.09 aBMD at total body + 0.014 aBMD at femoral neck	DXA Retired for mean of 25 years
Alfredson et al. (1997)[177]	*College volleyball (n=13) *Controls (n=13)	Females (17-27)		+ 13% aBMD at lumbar spine + 16% aBMD at femoral neck + 8.2% aBMD at total proximal femur	DXA
Calbet et al. (1999)[178]	*Competitive volleyball (n=15) *Controls (n=15)	Males (22-28)		+ 14% aBMD at lumbar spine + 24% aBMD at femoral neck	DXA

DXA = dual-energy X-ray absorptiometry

Authors	Group Specification **(# in each group)**	**Gender and Age** **(years)**	**Competitive Training (years)**	**Athletes/Controls (%) Difference aBMD or BMC or Dominant/non-dominant limb (%) Difference aBMD or BMC**	**Measurement Device/Comments**
Uusi-Rasi et al. (1999)[109]	*Folk dancing/recreational gymnastics (n=54) *Controls (n=54)	Females (56-69)	>20	- 0.1% aBMD at lumbar spine + 1% aBMD at trochanter	DXA
Karlsson et al. (2000)[162]	*Soccer (n=128) *Controls (n=138)	Males (19-85)		+ 10.3% aBMD in leg (retired for mean 5 years) + 5.1% aBMD in leg (retired for mean 16 years) + 2.8% aBMD in leg (retired for mean 25 years) 0% aBMD in leg (retired for over 35 years)	DXA
Ito et al. (2001)[191]	*Volleyball (n=7) *Controls (n=11)	Perimenopausal (50 mean)	mean=19	+ 8% aBMD at lumbar spine +12% aBMD in calcaneus	DXA
Ito et al. (2001) [191]	*Volleyball (n=20) *Controls (n=35)	Postmenopausal (55 mean)	mean=20	+ 24% aBMD at lumbar spine + 14% aBMD in calcaneus	DXA
Magnusson et al. (2001) [88]	*Soccer (n=29) *Controls (n=21)	Males (40-49)		+ 7% aBMD at femoral neck	DXA Retired for a mean of 16 years
Magnusson et al. (2001) [88]	*Soccer (n=23) *Controls (n=23)	Males (50-59)		- 4% aBMD at femoral neck	DXA Retired for a mean of 24 years
Magnusson et al. (2001) [88]	*Soccer (n=26) *Controls (n=24)	Males (60-69)		+ 2% aBMD at femoral neck	DXA Retired for a mean of 29 years
Magnusson et al. (2001) [88]	*Soccer (n=25) *Controls (n=26)	Males (70-85)		0% aBMD at femoral neck	DXA Retired for a mean of 41 years

DXA = dual-energy X-ray absorptiometry

longitudinal study of 4 years, Kudlac et al.[160] observed aBMD changes in a group of former college gymnasts (n = 10; mean age of 20 years at baseline) and controls (n = 9; mean age of 24 years at baseline). At baseline and follow-up, aBMD of the former gymnasts remained significantly higher at the femoral neck, trochanter, Ward's triangle, and total body when compared to controls. The higher aBMD levels were still observed despite a decrease in the

reported hours of exercise per week (20 vs. 3 hours per week at baseline and follow-up, respectively). However, over time the percent difference between the former gymnasts and controls declined at the total body (7% to 5%), lumbar spine (6% to 2%), femoral neck (16% to 14%), and trochanter (18% to 13%). It is important to follow these former gymnasts into their later years and observe if aBMD continues to remain significantly higher than controls.

In summary, competitive athletes have shown residual benefits of high bone acquisition from their respective sports well into adulthood. However, when these adults reach late adulthood, the benefits seem to diminish. In athletes such as gymnasts, residual benefits are maintained for many years, but these subjects are still in their young adult years.[47, 104, 108] The residual benefits are approximately 1 to 1.5 standard deviations (SD), lower than the 2 to 3 SD benefits seen in active athletes, although this may reflect secular changes in training intensity where the older athletes may have achieved a lower peak bone size and mass than the contemporary athlete. Nonetheless, the evidence regarding persisting skeletal benefits is based on studies in retired competitive athletes, not on studies of those who participated in moderate exercise. Currently, there are no data to support or dispute the notion that modest benefits achieved by moderate exercise during growth are sustained into adulthood, when the exercise has declined or stopped. Therefore, it is important that we continue to study retired athletes from a variety of sports well into late adulthood particularly when fractures are more likely to occur. Subsequently, if skeletal benefits from certain types of sports prove more beneficial than others, then exercise interventions for the public could be developed and followed in order to reduce the prevalence and public health burden of osteoporosis.

Gaps in Past Athletic Participation and Bone Health

Conducting prospective studies of bone health using exercise programs of sufficient duration and intensity is difficult, expensive, and in certain cases unethical. Furthermore, studies are limited in their ability to accurately assess every lifestyle factor associated with bone development throughout the lifespan. Consequently, evidence from human studies supporting an association between long-term intensive exercise and bone health stems largely from cross-sectional studies of athletes. Competitive sports including gymnastics, tennis, soccer, volleyball, track, weightlifting, and many others have been examined cross-sectionally in relation to bone health in children and adults. However, caution must be exercised when evaluating cross-sectional data among former athletes and controls due to limitations in study design.

Cross-sectional studies of athletes and bone health have been conducted in male and female athletes representing many different age groups and sports. Generally these studies have investigated active athletes who have been engaged in habitual training for several years. The studies vary, however, in subject selection, specification of study groups, measuring techniques, reporting of the results and defining the units of measurement as either BMC or aBMD. With so many diverse variables, it is increasingly difficult to compare studies and more importantly, to make definitive conclusions. Moreover, there is considerable variation in the magnitude and differences observed between athletes and nonathletes depending on the age, sex, type of sport and training, and bone site under investigation. Several studies of young and middle-aged men and women have shown aBMD differences extending above 30%,[46, 173, 187] while the results of studies of older male and female athletes have generally reported aBMD or BMC values lower than 5% higher than those of the controls.[102, 109, 162, 185] Although habitual training involving strengthening and muscle building activities is associated with high aBMD and BMC values, the

difference observed may be partly explained by confounding variables such as age, weight, differences in fat and fat-free mass, nutrition, hormone status, prior activity patterns, lifestyle factors (smoking, high alcohol intake, medications and/or diseases that can affect bone metabolism) and selection based on genetic influences.[110] Ultimately, bone status is multifactorial and collecting information pertaining to all factors associated with bone health is paramount.

To date, there are no prospective studies that have tracked womens'past competitive athletic participation from childhood, adolescence, or early adulthood into the perimenopausal and menopausal years. Kudlac et al.[160] observed prospective changes in a group of former female college gymnasts (n = 10; mean age of 20 years at baseline) four years after competition training with results showing similar rates of bone loss compared to controls (n = 9; mean age of 24 years at baseline). However, significant age differences between the two groups and differences in the duration of follow-up were found and could have contributed to the disparity between the former gymnasts and controls. Nonetheless, additional prospective studies following athletes and controls/nonathletes into the years associated with fragility are warranted.

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CHAPTER 3

RETIRED COLLEGIATE ARTISTIC GYMNASTS RETAIN HIGH BONE MASS[1]

[1]Pollock, N.K., Laing, E.M., and Lewis, R.D. 2004. To be submitted to The Journal of Bone and Mineral Research.

ABSTRACT

Fifteen years after cessation of the sport, we found that retired collegiate artistic gymnasts (GYM; n=18) had higher measures of areal bone mineral density (aBMD; g/cm^2) at all skeletal sites compared to nongymnast controls (CON; n=15) of similar age (years), height (cm) and body weight (BW; kg). It is unknown, however, if the aBMD differences in GYM and CON observed at that time are maintained into the years approaching menopause. A nine-year follow-up study was conducted to compare aBMD in GYM (n=16; age=45.3 ± 3.3 years) and CON (n=13; age=45.4 ± 3.8 years) and the changes over time. Total body fat mass (FM; kg), percent fat (%FAT), fat-free soft tissue (FFST; kg) and aBMD of the total body, lumbar spine, non-dominant proximal femur (PF), femoral neck and Ward's triangle were assessed using dual-energy X-ray absorptiometry (DXA; Hologic QDR-1000W). Past physical activity was estimated using a self-report, study-designed questionnaire. Independent samples *t*-tests were employed to compare aBMD in GYM and CON at baseline and at the nine-year follow-up. Analysis of covariance was used to compare the changes (Δ) in aBMD between GYM and CON and to quantify the magnitude of the effects (i.e., partial eta-squared; η^2; where 0.06 and 0.13 are medium and large effects, respectively). GYM had significantly lower BW, FM, and %FAT ($p<0.05$; $\eta^2>0.14$), and higher measures of FFST/BW and aBMD at all skeletal sites compared to CON ($p<0.05$; $\eta^2>0.14$) at both time points. Over time, changes in GYM and CON did not differ significantly with respect to BW ($p=0.12$; $\eta^2=0.09$), FM ($p=0.38$; $\eta^2=0.03$), %FAT ($p=0.92$; $\eta^2=0.00$), or aBMD at any skeletal site ($p>0.05$; $\eta^2<0.08$). CON had greater gains in FFST than GYM (8.68 ± 1.80% vs. 3.22 ± 0.92%; $p=0.01$; $\eta^2=0.23$); however, when FFST was corrected for BW, no significant difference was found between GYM and CON. Additionally, there were no significant differences in the total minutes of physical activity per week reported over the past

nine years between groups. In conclusion, the higher aBMD observed in GYM compared to CON fifteen years after the cessation of the sport, was maintained over the following nine years. While loading activity in GYM is clearly less than when competing, it is possible that a minimal defined level of physical activity is needed in the years following retirement to sustain the high aBMD. **Key Words**: GYMNASTICS, FORMER GYMNASTS, RETIRED GYMNASTS, AREAL BONE MINERAL DENSITY, PAST ATHLETIC PARTICIPATION AND BONE

INTRODUCTION

Participation in weight-bearing sports during the growing years has been shown to increase bone mineral accrual and improve the mechanical properties of bone.[1-4] In particular, high impact-load sports such as artistic gymnastics are thought to provide a greater osteogenic stimulus compared to other sports.[5-10] Two prospective studies in pre- and peri-pubertal artistic gymnasts of one and three years duration showed that bone mineral acquisition was greater (12-23%) than nongymnast controls.[8, 10] Other studies which involve jumping exercise interventions with prepubertal children have shown higher bone gains in the exercisers versus the controls.[11-13] It has been postulated that these higher bone gains observed in youth may lead to osteoporosis prevention in the later years, particularly when fracture risk is high.

Currently, however, there are no long-term prospective studies tracking bone mineral to determine if high bone mineral gains acquired from participation in youth sports persist into middle and late adulthood. Cross-sectional studies of active adult and former competitive athletes who started training in youth are suggestive, but equivocal, that bone gains are maintained into adulthood. Comparisons of active collegiate gymnasts[14-16] with nongymnast athletes or controls have demonstrated that the gymnasts have significantly higher aBMD values ranging from 5% to 36%. Competitive college-age soccer,[17, 18] tennis,[3] volleyball players,[19, 20] and weightlifters[21] have also been shown to have significantly higher aBMD compared to nonathletic controls ranging from 1% to 24%. Similar differences are also seen in retired athletes. Former artistic gymnasts,[5, 22, 23] soccer players,[17, 18] and weightlifters[21] retired from competitive training less than 20 years, were found to have significantly higher aBMD values compared to nonathlete controls with differences ranging from 5% to 22%. The aBMD differences observed in these studies of retired athletes would imply that potential bone gains from participation in high-

impact youth sports persist into adulthood, yet some studies suggest otherwise. Areal BMD of former soccer players[17, 18] and weightlifters,[24, 25] some 60 years of age and older and retired from their competitive training for more than 20 years, was not different when compared to nonathlete controls of the same age, leaving questionable the sustainability of skeletal benefits from earlier sports participation.

Only three studies have evaluated changes in bone longitudinally following cessation of intensive training in former competitive athletes.[26-28] In those studies, retired college gymnasts and tennis players continued to maintain significantly higher aBMD or BMC values when compared to controls or when observing side-to-side arm comparisons in racquet sports. The former athletes in these studies were still relatively young (mean age range 19 to 35 years), however, and were retired from their sport for five years or less. Whether this is the case in older former competitive athletes for longer periods of time since retirement is unclear.

The purpose of the present investigation was to determine if the higher aBMD of former college gymnasts compared to controls, previously reported by Kirchner et al.,[22] is still present in the same cohort of former gymnasts approaching menopause and approximately 25 years since the cessation of college gymnastics training and competition. The specific aim was to examine changes in aBMD and related factors including body composition, physical activity, and selected nutrient intakes in the former female college gymnasts and controls approximately nine years after baseline measurements. It was hypothesized that the higher bone mass observed in the former artistic gymnasts compared with controls will be maintained over nine years.

MATERIALS AND METHODS

Study Participants

In 1993, former college artistic gymnasts (n=22) and age-, height-, and weight-matched nongymnast controls (n=22) were recruited from the Southeastern United States and within a local community, respectively. Exclusion criteria, which included physician-diagnosed bone-disease or illness, medications known to affect bone development, and smoking, reduced the group sizes to 18 former gymnasts and 15 nongymnast controls. In the current study, every effort was made to locate and recruit the original participants. Three participants declined participation and one was not located resulting in smaller samples size to 16 former gymnasts (GYM) and 13 controls (CON).

All participants were apparently healthy and met the inclusion criteria from the original study. All participants were Caucasian (except one African-American control), non-smoking, and had no evidence of bone disease. All procedures were approved by the Institutional Review Board for Human Subjects at The University of Georgia. The participants signed a consent form prior to testing.

Procedures

Testing procedures were completed at baseline[22] and approximately nine years following the original measures. The same testing procedures used in 1993 were employed in the current project. Testing included anthropometric measures, bone scans and the completion of interviewer-administered questionnaires regarding medical, physical, and diet history.

Anthropometric Measures

A trained technician conducted weight and height measurements. Participants were weighed in light indoor clothing following the removal of shoes. Weight of each subject was measured to the nearest 0.25 lb by using a calibrated double-beam balance scale (Fairbanks Scales, Kansas City, MO) and then converted to the nearest 0.1 kg. Participants were weighed three times, and results were averaged. Three height measurements were measured to the nearest 0.1 cm by using a wall-mounted stadiometer (Novel Products Inc., Rockton, IL), and the values were averaged. Prior to testing, the Fairbanks double-beam balance scale was checked for accuracy using known weights. Recalibration of the scales was not required during the testing sessions.

Physical Activity Assessment

Information on physical activity for the past week was collected using the interviewer-administered seven-day recall questionnaire.[29] Participants reported the amount of time spent sleeping or performing moderate, hard, and very hard activities during the previous week. Light physical activity was recorded for the remaining time. From this questionnaire, each participant's average daily energy expenditure (kcal/day) was estimated.

Estimates of physical activity over the last 10 years was collected using a study-designed questionnaire developed from the original study.[22] A list of different types of activities was given to each participant with this interviewer-administered questionnaire in the recall process. Participants were asked about the frequency (days per week), duration (minutes each session), and intensity [1-7 (very, very easy to very, very hard)] of physical activity completed during these time frames.

Dietary Intake

The Block Food Frequency Questionnaire (version 98.2, Berkley, CA) was used to estimate usual dietary intakes over the past year. This questionnaire, which has evidence of proven validity[30] and reliability,[31] was administered in an interview format by a trained laboratory technician. In addition to the two-dimensional serving size pictures included with each questionnaire, food models along with plates and cups were used to help participants more accurately estimate portion sizes. Mean estimates of energy, protein, carbohydrate, fat, calcium, vitamin D, phosphorus, and iron intake were calculated (Block Dietary Data Systems; Berkley, CA) and presented.

Bone Densitometry and Body Composition

Areal bone mineral density (aBMD; g/cm^2) of the total body, lumbar spine, and nondominant total proximal femur, including femoral neck and Ward's triangle were determined by dual energy X-ray absorptiometry (DXA, QDR-1000, Hologic Inc, Waltham, MA). All DXA scans were performed and analyzed by the same trained technician. Hologic software, versions 4.57P and 4.76P, were used in analyzing lumbar spine and proximal femur scans at baseline and follow-up, respectively. Total body aBMD and body composition [fat mass (kg), fat-free mass (kg), and % body fat] were assessed at baseline and follow-up using Hologic Whole Body Analysis software, versions 5.55 and 5.73, respectively.

Quality assurance for DXA was carried out by daily calibration against the manufacturer's standard phantom (DPA/QDR-1, Hologic x-caliper spine phantom, Hologic, Inc). In our laboratory, a coefficient of variation of 0.27% was observed from 365 scans of the spine phantom over a five-year period. Fat mass and fat-free mass measures were calibrated by concurrently scanning (with each total body scan) an external three-step soft tissue wedge

(Hologic, Inc) composed of different thickness levels of aluminum and lucite, calibrated against stearic acid (100% fat) and water (8.6% fat). Coefficient of variations for aBMD of the total body (0.58%), lumbar spine (0.63%), proximal femur (0.89%), femoral neck (3.0%) and % body fat (0.79%) were determined in premenopausal women (n=10) measured three separate occasions within an eight-day period.

Statistical Analysis

For the purpose of the current study, data from those participants who came in for both baseline and follow-up testing (GYM; n=16 and CON; n = 13) were used. Statistical analyses were performed using the Statistical Package for the Social Sciences, version 11.0.2 for the Mac OS X (SPSS, Chicago, IL). Descriptive statistics were calculated to determine the range, mean, and standard deviation of all variables measured. Independent samples *t*-tests were used to determine differences between GYM and CON at baseline and nine years later. Two-way (group x time), analysis of covariance was performed to determine significant differences in aBMD and body composition within groups over time and to quantify the magnitude of the effects of these variables between groups at baseline and at nine years. Group differences are reported for physical activity and dietary intake at the follow-up only. Values are reported as means ± SD, unless otherwise noted. Statistically significant differences are reported if $P < 0.05$. Medium and large effects are designated by partial eta-squared (η^2) ≥ 0.06 and 0.13, respectively.[32]

RESULTS

Participants

Participant characteristics at baseline and follow-up are listed in Table 3.1. Follow-up measurements for the GYM and CON were, on average, 9.3 and 9.1 years after their baseline measurements, respectively. No significant differences were found between groups in age and

height at baseline and follow-up. The GYM reported to have started gymnastics training at an average age of 11.1 ± 0.9 years.

Table 3.1. ***Characteristics of former gymnasts (GYM) and controls (CON)***

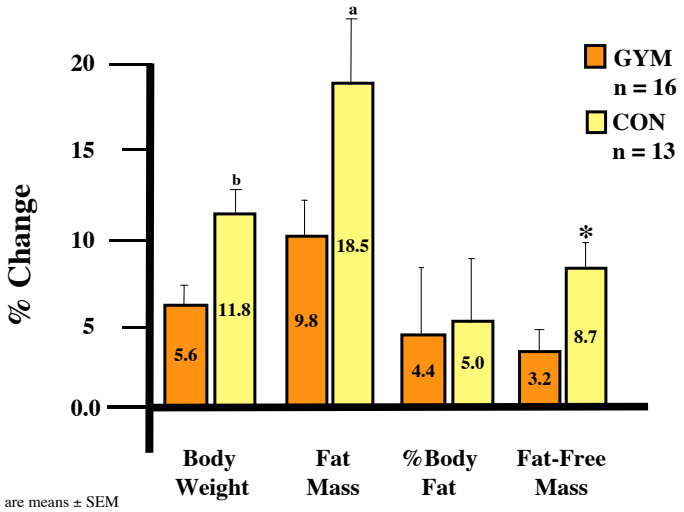
	1993-1994		2003-2004	
Characteristic	***GYM (n=16)***	***CON (n=13)***	***GYM (n=16)***	***CON (n=13)***
Age, yr	**36.1 ± 3.5**	**36.3 ± 3.8**	**45.3 ± 3.3**	**45.4 ± 3.8**
Height, cm	**162.4 ± 6.1**	**161.6 ± 5.6**	**162.4 ± 6.2**	**161.6 ± 5.4**
Weight, kg	**57.4 ± 4.5***	**62.1 ± 7.7**	**60.6 ± 5.9***	**69.1 ± 9.5**
Fat mass, kg	**13.1 ± 2.1***	**18.8 ± 6.4**	**14.3 ± 2.8***	**21.4 ± 6.1**
Body fat, %	**22.9 ± 2.7***	**29.8 ± 5.3**	**23.8 ± 3.3***	**30.9 ± 5.2**
Fat-free mass, kg	**41.7 ± 3.9**	**40.9 ± 3.2**	**43.1 ± 4.2**	**44.5 ± 4.4**
Fat-free mass/weight	**0.73 ± 0.03***	**0.66 ± 0.04**	**0.71 ± 0.03***	**0.65 ± 0.06**

Values are means ± SD
* $P < 0.05$; $\eta^2 > 0.14$ between GYM and CON

The former gymnasts had significantly lower body weight, fat mass, % body fat ($P < 0.05$; $\eta 2 \geq 0.14$) and higher measures of fat-free mass/body weight ($P < 0.05$; $\eta 2 = 0.44$) compared to CON at baseline (See Table 3.1). At the nine-year follow-up, similar results were observed as GYM continued to have lower body weight, fat mass, and % body fat ($P < 0.05$; $\eta 2 \geq 0.24$) and greater measures of fat-free mass/body weight ($P < 0.05$; $\eta 2 = 0.36$).

Over time, changes in GYM and CON did not differ significantly with respect to body weight, fat mass, and % body fat (See Figure 3.1). Controls had significantly greater gains in fat-free mass than GYM (8.7 ± 1.8% vs. 3.2 ± 0.9%); however, when fat-free mass was corrected for body weight, no significant difference was found between groups.

Figure 3.1. ***Body weight and composition changes from baseline to 9-years in former gymnasts (GYM) and controls (CON)***



Absolute values are means ± SEM
* Difference between groups, P=0.01; η^2= 0.23
[a] Difference between groups, P=0.38; η^2= 0.03
[b] Difference between groups, P=0.12; η^2= 0.09

Age of menarche was similar between GYM and CON (P = 0.28; 13.7 years ± 1.7 vs. 13.1 years ± 1.3, respectively). The majority of the participants reported having normal menstrual cycles over the past nine years. One GYM and one CON reported having menopausal symptoms (e.g., irregularity of menses, hot flashes, or night sweats). Fourteen of the 16 GYM reported birth control use, via oral contraceptive (OC), and the average duration of OC use was 12.6 ± 10.0 years. Eleven of the 13 CON reported OC usage and their average time on the OC was 12.2 ± 8.7 years. No differences were found in length of OC usage between GYM and CON.

At the follow-up, 12 of the GYM and 12 of the CON reported having given birth. The mean parity for the GYM and CON was 1.8 ± 1.2 vs. 1.7 ± 1.0, respectively. Of those participants reporting parity, 12 GYM and 10 CON reported breastfeeding. Three GYM and six CON reported breastfeeding less than six months, while nine GYM and four CON reported breastfeeding greater than six months.

Physical Activity Measures

Current physical activity data from the seven-day recall are reported in Table 3.2. No significant differences were found between GYM and CON for hours of sleep, light activity, hard activity, and very hard activity ($P > 0.12$; $\eta^2 < 0.04$). While not statistically significant, GYM had greater levels of moderate physical activity, as evidenced by a moderate effect size ($P = 0.12$; $\eta^2 = 0.09$). Overall, no differences in total daily energy expenditure were found between the two groups ($P = 0.17$; $\eta^2 = 0.07$), however a medium effect size was observed as CON expended more energy than GYM (2,703 ± 428 vs. 2,417 ± 656 kcal/day, respectively). Moreover, when energy expenditure was corrected for body weight, no differences were found between the groups ($P = 0.63$; $\eta^2 = 0.01$).

Table 3.2. ***Hours per day of activity reported from the Seven-Day Recall in former gymnasts (GYM) and control (CON)***

	GYM (n=16)	*CON (n=13)*
Sleep *(hours)*	**7.4 ± 0.8**	**7.6 ± 1.2**
Physical Activity *(hours)*		
Light	**14.5 ± 1.3**	**14.6 ± 1.7**
Moderate	**1.5 ± 1.2[a]**	**1.0 ± 0.6**
Hard	**0.4 ± 0.5**	**0.6 ± 0.6**
Very Hard	**0.2 ± 0.3**	**0.3 ± 0.3**

Values are means ± SD
[a] $P = 0.12$; $\eta^2 = 0.09$

Table 3.3 lists the questions asked in the study-designed past-physical activity questionnaire, which pertain specifically to activity performed in last 10 years. During the last 10-year period, the questionnaire did not capture any differences in activity between the GYM and CON with respect to frequency and intensity. A large effect size existed for duration of

exercise session as GYM reported more minutes of activity per session than CON. Walking was the physical activity that both groups engaged in most often over the last 10 years (Table 3.3).

Table 3.3. ***Self-reported physical activity of former gymnasts (GYM) and controls (CON) from 1993-1994 to 2003-2004***

	GYM (n=16)	*CON (n=13)*
On average, how frequently have you exercised over last 10 years? ***(days/week)***	**3.6 ± 1.9**	**3.9 ± 2.0**
On average, how long do you exercise during each session ***(minutes)?***	**61.3 ± 26.0** [a]	**44.6 ± 17.6**
What is your intensity level of a typical exercise bout over last 10 years? ***[Scale 1-7 (very,very easy - very,very hard, respectively)]***	**4.7 ± 0.9**	**4.8 ± 1.0**
In what specific physical activities have you regularly participated? ***(Self-reported activities)***	**n** **•Walking 12** **•Tennis 4** **•Wt train 1** **•Aerobics 3** **•Bike 1** **•Running 3**	**n** **•Walking 13** **•Tennis 1** **•Wt train 5** **•Aerobics 3** **•Bike 1** **•Running 4**

Values are means ± SD
[a] $P = 0.06$; $\eta^2 = 0.13$

Dietary Intake

Mean dietary intakes, which include dietary supplements, for GYM and CON are reported in Table 3.4. At follow-up, there were no significant differences between GYM and CON for any of the nutrients reported. Former gymnasts and CON met the recommended dietary allowances for macronutrients; however, neither group met the estimated energy requirement of 2200 kcal per day. Both GYM and CON reported low intakes of calcium and vitamin D. Sixty-three percent of GYM (10/16) and 69% of CON (9/13) consumed less than 2/3 of the AI for calcium; whereas, 69% of both GYM (11/16) and CON (9/13) consumed less than 2/3 of the AI for vitamin D.

Table 3.4. ***Mean daily intake and percentages of Recommended Dietary Allowances (RDA) in former gymnasts (GYM) and controls (CON)***

Variable	*GYM (n=16)*	*% RDA*	*CON (n=13)*	*% RDA*
Kilocalories	**1804 ± 559**	**82**	**1600 ± 458**	**73**
Protein, *g*	**63.6 ± 19.8**	**139**	**59.3 ± 20.7**	**128**
Carbohydrate, *g*	**207 ± 56**	**159**	**197 ± 52**	**152**
Fat, *g* †	**77.7 ± 32.4**	**105**	**66.4 ± 28.1**	**90**
Calcium, *mg* [a]	**722 ± 275**	**72**	**687 ± 253**	**69**
Vitamin D, *IU* [a]	**107 ± 66**	**54**	**105 ± 76**	**53**
Phosphorus, *mg*	**1089 ± 338**	**156**	**1044 ± 355**	**149**
Iron, *mg*	**12.6 ± 4.1**	**70**	**11.8 ± 2.9**	**66**

Values are means ± SD
[a] Percentage of Adequate Intake
† 30% of 2200 Kcal /day

Areal Bone Mineral Density

The aBMD values of GYM and CON are presented in Table 3.5. At baseline, aBMD of GYM was significantly higher than CON at all sites measured. Similarly, at the nine-year follow-up, aBMD of GYM remained significantly higher than CON at the same measured sites.

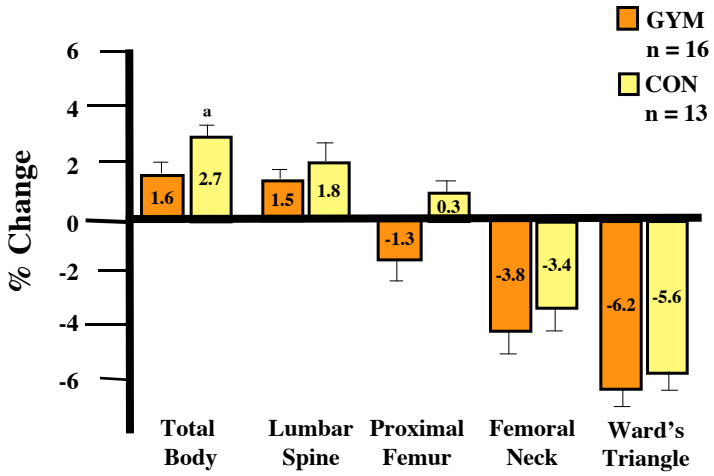
Table 3.5. ***Areal bone mineral density (aBMD) at baseline and 9-years in former gymnasts (GYM) and controls (CON)***

	1993-1994			**2003-2004**		
aBMD †	**GYM (n=16)**	**CON (n=13)**	*% Difference*	**GYM (n=16)**	**CON (n=13)**	*% Difference*
Total Body	**1.16 ± 0.07***	**1.08 ± 0.06**	*6.9*	**1.17 ± 0.08***	**1.11 ± 0.06**	*5.1*
Lumbar Spine	**1.17 ± 0.14***	**1.01 ± 0.08**	*13.6*	**1.18 ± 0.13***	**1.04 ± 0.12**	*11.9*
Proximal femur	**1.03 ± 0.12***	**0.92 ± 0.10**	*10.6*	**1.01 ± 0.12***	**0.93 ± 0.08**	*7.9*
Femoral neck	**0.98 ± 0.13***	**0.87 ± 0.15**	*11.2*	**0.95 ± 0.13***	**0.84 ± 0.11**	*11.6*
Ward's triangle	**0.85 ± 0.17***	**0.72 ± 0.13**	*15.3*	**0.80 ± 0.16***	**0.68 ± 0.13**	*15.0*

Values are means ± SD
* $P < 0.05$; $\eta^2 > 0.14$ between GYM and CON
† aBMD; g/cm^2

Figure 3.2 presents the aBMD changes over nine years between GYM and CON. The CON had greater change in total body aBMD, as evidenced by a moderate effect size ($P = 0.14$; $\eta^2 = 0.08$). However, over the nine years, aBMD changes in GYM and CON did not differ significantly at any of the measured sites.

Figure 3.2. ***Areal bone mineral density changes from baseline to 9-years in former gymnasts (GYM) and controls (CON)***



Absolute values are means ± SEM
[a] $P = 0.14$; $\eta^2 = 0.08$

DISCUSSION

The present study is the first prospective report showing that former competitive athletes maintained higher aBMD values compared to nonathletes over an extended time period and approximately 25 years following their retirement from the sport. The primary finding was that former artistic gymnasts maintained significantly higher total body (5.1%), lumbar spine (11.9%), proximal femur (7.9%), femoral neck (11.6%), and Ward's triangle aBMD (15.0%) over the past nine years compared to controls.

In contrast with our findings, both Karlsson et al.[17] and Magnusson et al.[18] observed in former male soccer players having been retired from competitive training for more than 25 years

that femoral neck aBMD was no different than controls. Former male weightlifters also had similar femoral neck aBMD values when compared to controls 25 years following cessation of the sport.[24, 25] Additionally, Khan et al.[33] examined weight-bearing skeletal sites in retired elite female ballet dancers (mean age of 51 years; retired an average of 25 years) and found that aBMD values did not differ from controls. These cross-sectional studies suggest that there is no evidence of aBMD benefits in former elite athletes 25 years following their retirement from their respective sport. Whether these former athletes possessed higher aBMD than nonathletic controls 25 years ago is unknown. We cannot exclude the possibility of secular changes in intensity and frequency of training. Those former competitive athletes of soccer, weightlifting, and ballet may have achieved a lower peak bone mass than would an athlete of those same sports in modern times. Furthermore, the quantity and quality of physical activity between retirement and the bone measurements 25 years later is unknown.

Over the nine years, former gymnasts and controls from the present study lost similar amounts of aBMD at the femoral neck (–3.8% vs. –3.4%) and Ward's triangle (–6.2% vs. –5.6%), while similar aBMD gains were observed at the lumbar spine (1.5% vs. 1.8%) and total body (1.6% vs. 2.7%). At the proximal femur, aBMD loss of –1.3% was found in the former gymnasts, whereas the controls gained 0.3% over the 10 years. Hence, the higher aBMD at baseline in former gymnasts was maintained even with reduced levels of physical activity since college gymnastics training and competition. Kudlac et al.[28] also found rates of aBMD loss in former college gymnasts (mean age, 24 years at baseline) compared to controls. However, aBMD of the former gymnasts remained significantly higher than controls at baseline and follow-up at the femoral neck (16% to 14%), trochanter (18% to 13%), and total body (7% to 5%) than the controls. Likewise, male tennis players (mean age, 26 at baseline) maintained bone

mineral content (BMC) benefits gained during adolescence despite reduced playing activity over four years.[26] Moreover, in a five-year follow-up, former female racquet sport athletes (mean age, 22 at baseline) who started their training before or at menarche maintained higher humeral shaft BMC in the dominant versus nondominant arm.[27] However, a significant change over time was observed at the proximal humerus and distal radius comparing the dominant and nondominant arms (2.7% and 2.1% decrease, respectively). While these studies are suggestive of maintenance of bone gain following retirement, the follow-up periods after retirement are relatively short and former athletes still young.

There are several possible explanations for the higher aBMD observed in our former gymnasts vs. controls in the fifth decade of life. One possibility is that the higher aBMD observed in the retired gymnasts could be the residual effect of childhood gymnastics training on bone mass preservation. It was reported by our former gymnasts that their gymnastics training began at average age of 11 years. It has been suggested that the greater accumulation of bone mineral seen in adult athletes is achieved during the pubertal years.[1, 5] The epoch of puberty reflects a two- to three-year period when 25 to 30 percent of total adult bone mass is gained.[34] During this time, bone may be more responsive to activity-induced loading, due to the influence of increasing hormones on bone gains.[35] MacKelvie et al.[12] found that early pubertal girls, compared to pre-pubertal girls, had greater gains in bone mass with physical activity. Similar results were found in humeral side-to-side BMC difference in female tennis and squash players, where bone mineral benefits were approximately two times greater in the women who started their careers at or before menarche than those who started playing 15 years after menarche, 17% to 24% compared to 8% to 14% respectively.[3] Additionally, Khan et al.[36] found a positive association between starting age of ballet training and bone mass at load-bearing sites in former

female dancers. In our study, former gymnasts were found to have started menarche after the initiation of gymnastics training. Thus, the higher bone mass observed in our retired gymnasts could be the result of a more responsive skeleton exposed to higher levels of exercise.

We initially predicted that the higher bone mass in the retired college gymnasts observed approximately nine years ago [22] would be sustained in the present study. In support of this hypothesis, we also expected to see higher levels of physical activity among former gymnasts compared to controls. Although both groups demonstrated similar rates of bone gain or loss over time, it appears that former gymnasts maintained higher aBMD than controls despite the reported decline in loading activity. It is possible that the lower level of activity performed by our former gymnasts was satisfactory to retain the high bone mineral acquired during their active career compared to controls.

Conserving bone mineral is of significant clinical benefit, but it is unclear whether this benefit persists into late adult years after cessation of athletic training or even after exercise intervention. Detraining studies suggest that gains in bone are lost with complete detraining[37-39] but not with reduced training.[40-42] Follow-up studies in former elite athletes have observed similar results.[26-28] Decreased training, not full detraining, maintained the significant bone mineral content differences between the dominant and nondominant arms of male tennis players over four years.[26] Likewise, female racquet sport players maintained skeletal benefits despite reducing their playing frequency from 4.7 ± 2.7 to 1.4 ± 1.3 times per week during a five-year follow-up period.[27] Furthermore, Kudlac and colleagues[28] found that retired gymnasts have maintained their greater proximal femur aBMD over controls despite decreasing their exercising hours per week from 20 to 4. Kirchner et al.[22] reported that retired gymnasts had been exercising 5.8 ± 1.2 hours per week the previous ten years, whereas in the next 10 years, we found them to have been

exercising slightly less at a rate of 3.6 ± 1.9 times per week at approximately 60 minutes per session. It is possible that continued activity, although at a lower level, preserved the exercise-induced, beneficial skeletal effects acquired during growth and adolescence in our sample of retired gymnasts. Perhaps a minimal activity level is required in adulthood to maintain aBMD, but currently there are no conclusive data on the quality and quantity of exercise needed to preserve exercise-induced skeletal benefits. Further research is needed to determine types and amounts of activity necessary to maintain bone mass after cessation of competitive sports and into later adulthood.

The retired gymnasts and controls were found to have similar mean dietary intakes at the nine-year follow-up. It was interesting to note that the mean calcium intakes in both former gymnasts and controls (722 mg and 687 mg, respectively), were considerably less than the recommended 1000 mg/day for this age group[43] and slightly less than the U.S. mean intake of 744 mg for this particular age group and gender.[44] In prospective double-blind, placebo-controlled trials, it has been observed in premenopausal women (over 40 years of age) that with calcium intakes of 1000 to 1500 mg, bone loss was attenuated.[45, 46] Hence, it is possible that both former gymnasts and controls limited their bone mass attainment by consuming inadequate calcium. Although intakes of vitamin D and iron were also low, the food frequency questionnaire used in this study has only shown published validity[47] and reliability[31] for assessing past calcium intakes.

At this stage in our study participant's life, declining estrogen levels can impact bone health. Studies of perimenopausal women have found rates of femoral neck bone loss ranging from –0.3% to –1.3% per year as a result of decreasing estrogen levels.[48-50] Two women in our study, one gymnast and one control, reported to have been experiencing menopausal symptoms.

Because of the rapid bone loss associated with the menopausal transition, future follow-up studies of the study participants should account for menopausal status by including assessment of estradiol and follicle-stimulating hormone.

In conclusion, our study provides greater insight into the effects of past athletic participation on skeletal health in women approaching menopause. Our primary finding from the present study was that the former female college artistic gymnasts maintained significantly higher aBMD values over the controls since our baseline measures of approximately nine-years ago, even with the absence of high-load gymnastics activity. Whether the former gymnasts will maintain these higher aBMD values into their menopausal years, particularly when bone loss is accelerated, remains to be determined.

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CHAPTER 4

SUMMARY AND CONCLUSIONS

The present study was conducted to determine if the higher aBMD seen in former female college artistic gymnasts compared to controls, previously reported by Kirchner et al.,[1] is still present nine years later and approximately 25 years since the cessation of college gymnastics training. Specific variables examined at both time points were age, height, weight, % body fat, fat-mass, fat-free mass, and total body, lumbar spine, proximal femur, femoral neck, and Ward's triangle aBMD. At the nine-year follow-up, information was collected regarding dietary intake, menstrual history, and current and past physical activity.

The primary finding from the current study was that the former gymnasts over the past nine years maintained significantly higher aBMD compared to controls at all measured skeletal sites. Additionally, former gymnasts had significantly lower body weight, fat-mass, % body fat, and higher measures of fat-free mass/body weight compared to controls at baseline and nine years later. Changes in body weight, fat mass, % body fat, and aBMD at all skeletal sites over nine years were not different from baseline between the two groups. Furthermore, at the nine-year follow-up, mean dietary intakes and physical activity levels were not different between the former gymnasts and controls.

Other prospective studies in retired competitive athletes have observed similar results to our study; however the athletes in those studies were still in their 20s and 30s and only retired for five years or less.[2-4] Previous cross-sectional studies in former competitive athletes[5-8], retired for 25 years or more, have observed no aBMD benefits from earlier participation in sports during

adolescence and young adulthood. Our study was the first to report significantly higher aBMD values in former competitive athletes compared to controls retired from their sport for 25 years.

The results presented here are important with respect to artistic gymnastics and bone. Although our baseline data was collected approximately 15 years after cessation from college gymnastics, we speculate that the higher aBMD values observed were a reflection of the training during adolescence and college, since the average start age of gymnastics training was 11 years of age. Approximately nine years after baseline, we still observe those higher aBMD values. It is possible that continued physical activity, although at a lower level, preserved the skeletal benefits from earlier intense gymnastics training. Perhaps a minimal activity level is required in adulthood to maintain bone gains achieved from youth, but currently there are no conclusive data on the amount or type of activity necessary. Thus, further research is needed to clarify the quantity and quality of activity necessary to maintain bone mass after cessation of competitive sports and into later adulthood.

At this stage in our study participant's life, other factors such as declining estrogen levels or low calcium intakes can impact the bone response to exercise. Studies of perimenopausal women have found rates of femoral neck bone loss ranging from –0.3% to –1.3% per year as a result of decreasing estrogen levels.[9-11] Two women in our study, one gymnast and one control, reported to have been experiencing menopausal symptoms. Because of the rapid bone loss associated with menopausal transition, future follow-up studies of the study participants should account for menopausal status by including assessment of estradiol and follicle-stimulating hormone.

The women in the current study, both gymnasts and controls, had calcium intakes much lower than the AI for calcium. Calcium supplementation of 1000mg above regular dietary

intakes in perimenopausal women, has been shown to prevent bone loss.[12, 13] It is possible that the skeletal benefits of physical activity engaged in by participants over the past nine years was attenuated as a result of low calcium intakes. This potential calcium-exercise interaction should be explored more thoroughly in future follow-up studies, including more detailed measures of calcium intakes and supplementation.

In conclusion, our study provides greater insight into the effects of past athletic participation on skeletal health in women approaching menopause. Our primary finding from the present study was that the former female college artistic gymnasts maintained significantly higher aBMD values over the controls since our baseline measures of approximately nine-years ago, even with absence of high-load gymnastics activity. Whether the former gymnasts will maintain these higher aBMD values into their menopausal years, particularly when bone loss is accelerated, remains to be determined.

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APPENDICES

APPENDIX A

Consent Form

Consent Form

I _____ agree to participate in a research study titled "The long term effect of gymnastics participation on bone health," a follow-up to a study I formerly participated. Dr. Richard Lewis and Mr. Norman Pollock of the Department of Foods and Nutrition of the University of Georgia are conducting the study. Dr. Lewis and Mr. Pollock may be reached in room 279 Dawson Hall at (706) 542-4901 or (706) 542-4918. I understand that my participation is voluntary. I can withdraw consent at any time without penalty and have the results of the participation, to the extent that it can be identified as my own, returned to me, removed from the research records, or destroyed.

The following points have been explained to me:

1) The purpose of the study is to determine how participation in competitive gymnastics during college may affect bone mineral density (BMD) later in adulthood. The benefits I can expect from participation are the assessment of bone health (bone mineral density), body composition (percentage of body fat and nonfat tissue), diet, and physical activity patterns. All measurements are being used for research purposes only, not medical purposes. However, if abnormalities are found in any measure, I will be notified and referred to my health care professional.
1) The procedures are as follows:
    a. On the day of testing, I will arrive in Sports Nutrition Lab in Dawson Hall at the scheduled time. Prior to any testing or participation, an investigator will read a consent form to me, after which the researcher and I will sign the consent form. During the reading of the consent form, I will be familiarized with the testing procedures that will be used during the study. Each aspect of the study will be explained to me during testing and I can withdraw from the study at any time. A copy of the consent form will be given to me and I will be reminded that I am able to withdraw from the study at any time.
    a. I will then be asked by the researcher to fill out 6 questionnaires and take home a 3-day food diary. The questionnaires will include: health history questionnaire, food frequency questionnaire, eating disorders inventory symptom checklist, eating disorders inventory II questionnaire, past physical activity questionnaire, and a interviewer- administered 7-day physical activity recall. The approximate total time to complete all 6 questionnaires will be 45 minutes. In addition, I will take home a 3-day food diary to complete and mail back to the Sports Nutrition Lab.
    a. After completion of all 6 questionnaires, I will then give measurements for height and weight. Finally, I will complete the bone density scans of the total body, lumbar spine, and femur on the Hologic DXA machine. The total time for completion of all 3 scans will be approximately 45 minutes. The researcher will provide me with a copy of the scan information along with a brief interpretation of their meaning.
1) The discomforts or stresses that I may face during this research include psychological discomfort from the disclosure of information concerning diet, physical activity, and history of menstruation status. In addition, I may be asked sensitive questions about body image and eating disorders. However, I may skip any question that may be distressing. If undue discomfort or stress occurs, I have the right to discontinue the rest at any time.
1) I understand that the only foreseen risk is exposure to a small amount of radiation during the bone scan. The 3 scans will expose me to a maximum effective radiation dose of approximately 5 mR, which is minimal given the consideration that background exposure is 3.5 mR per week and that chest x-ray films are about 25-40 mR for two standard films. Thus the exposure is only 13-20% of standard chest x-ray. In the event that information from any scan is lost or unstable, no additional scans will be performed.
1) The results of my participation will be confidential and will not be released in any identifiable form without my prior consent unless required by law. My signature on this form authorized the use of my data in-group analyses, which may be prepared for public dissemination, without breaching my confidentiality. To accomplish this, I will be assigned a four digit subject participation code which will be used on all data collected during my participation in this research. A master list with my name and corresponding code number will be kept separated from testing data and locked at all times.
1) The investigator will answer any further questions that I may have about this research, either now or during the course of the project.

I understand that I am agreeing by my signature on this form to take part in this research project and understand that I will receive a signed copy of this consent form for my records.

_____	_____	_____
Name of Researcher	**Signature**	**Date**

Telephone: _____
Email: _____

_____	_____	_____
Name of Participant	**Signature**	**Date**

Please sign both copies, keep one and return one to the researcher.

Additional questions or problems regarding your rights as a research participant should be addressed to Chris A. Joseph, Ph.D. Human Subjects Office, University of Georgia, 606A Boyd Graduate Studies Research Center, Athens, Georgia 30602-7411; Telephone (706) 542-3199; E-Mail Address IRB@uga.edu

APPENDIX B

Height and weight recording sheet

Former Gymnasts 10-year Follow-up

Height and Weight

I.D. _____ **Date:_____**

Height:

1. _____
1. _____
1. _____

Average _____

Weight:

1. _____
2. _____
2. _____

Average _____

APPENDIX C

Health History Questionnaire

Health History Questionnaire

Name: _____ Age: _____ Date Of Birth: _____

Address: _____ Home Phone: _____ Work Phone: _____

_____ Occupation: _____ Race/Ethnic Background:

Child Bearing History

1. How many children have you given birth to? _____

1. What are their ages? _____

1. Did you breast feed any of your children? ___ Yes _____ no. If yes, how long? _____

1. Did you have any C-sections?_____yes _____no. If yes, how many? _____

Menopausal History

1. Have you gone through menopause (12 months without a period)? _____yes, ___ no. If yes, how old were you when it occurred? _____

2. Are you presently going through menopause? _____ yes, _____ no. If yes, how long ago did you start going through it? _____

2. Are you using any medications relating to your menopause? ___ yes, _____ no. If yes, which medications?

_____

2. When did you start using these medications? _____

Surgery/Medication History

1. Please list major medical procedures, surgeries and/or injuries in your lifetime and related medications? Give the time of the procedure or injury and/or the frequency and duration of medication.

1. Have you ever gone through an extended period of time where you were bedridden or immobilized? _____ yes, _____ no. If yes, how old were you and how long did this immobilization last? Briefly explain the circumstances.

Other History

1. Do you smoke cigarettes now? _____ yes, _____ no. If yes, on the average, about how many cigarettes a day do you smoke now? ____1-5, ____6-14, ____15-24, ____25-35, ____35 or more
1. If you used to smoke but do not smoke now, how long did you smoke? _____years. On the average, about how many cigarettes a day did you smoke? ____1-5, ____6-14, ____15-24, ____25-35, ____35-more
1. How old were you when you began using birth control pills (if ever used)? _____
   How long have you been using them?

1. What periods of time did you stop using birth control pills? (Please give dates, if applicable)

1. How would you rate your present health? ____Poor____Good____Fair____Excellent

1. Any History of Bone Diseases? ____ yes, _____ no. If yes, explain?

1. Are your menstrual cycles regular? _ yes, _____ no. If not, how long have they been irregular? And when was your most recent period?

1. Any significant weight changes in the last ten years?

1. Are you on any nutritional supplements?

1. Are you currently dieting, or on a special type of weight loss program (Weight Watchers, Atkins, etc…)

1. Has any member of your family been diagnosed with osteoporosis?

1. Do you have any health problems that limit your physical activity?

1. How many hours, on average, do you spend watching TV, or on the computer?

APPENDIX D

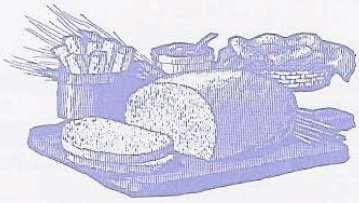
Food Frequency Questionnaire

RESPONDENT ID NUMBER

TODAY'S DATE

	DAY	YEAR
○ Jan		
○ Feb		
○ Mar	0 0	1998 ○
○ Apr	1 1	1999 ○
○ May	2 2	2000 ○
○ Jun	3 3	2001 ○
○ Jul	4	2002 ○
○ Aug	5	2003 ○
○ Sep	6	2004 ○
○ Oct	7	2005 ○
○ Nov	8	2006 ○
○ Dec	9	2007 ○

FOOD QUESTIONNAIRE



This form is about the foods you usually eat. It will take about 30 - 40 minutes to complete.

- **Please answer each question as best you can. Estimate if you aren't sure.**
- **Use only a No. 2 pencil.**
- **Fill in the circles completely, and erase completely if you make any changes.**

Please print your name in this box.

SEX
○ Male
○ Female

If female, are you pregnant or breast feeding?
○ No
○ Yes
○ Not female

AGE

WEIGHT pounds

HEIGHT ft. in.

First, a few general questions about what you eat.	AVERAGE USE IN THE PAST YEAR								
	LESS THAN ONCE per WEEK	1-2 per WEEK	3-4 per WEEK	5-6 per WEEK	1 per DAY	1 1/2 per DAY	2 per DAY	3 per DAY	4+ per DAY
About how many servings of vegetables do you eat, per day or per week, not counting salad or potatoes?	○	○	○	○	○	○	○	○	○
About how many servings of fruit do you eat, not counting juices?	○	○	○	○	○	○	○	○	○
How often do you eat cold cereal?	○	○	○	○	○	○	○	○	○
How often do you use fat or oil in cooking?	○	○	○	○	○	○	○	○	○

What kinds of fat or oil do you usually use in cooking? MARK ONLY ONE OR TWO

- ○ Don't know, or Pam
- ○ Stick margarine
- ○ Soft tub margarine
- ○ Butter
- ○ Butter/margarine blend
- ○ Low-fat margarine
- ○ Corn oil, vegetable oil
- ○ Olive oil or canola oil
- ○ Lard, fatback, bacon fat
- ○ Crisco

PLEASE DO NOT WRITE IN THIS AREA

21710

During the past year, have you taken any vitamins or minerals regularly, at least once a month?

○ No, not regularly ○ Yes, fairly regularly →

(IF YES) WHAT DID YOU TAKE FAIRLY REGULARLY?

VITAMIN TYPE	HOW OFTEN					FOR HOW MANY YEARS?					
	DIDN'T TAKE	*A FEW DAYS per MONTH*	*1-3 DAYS per WEEK*	*4-6 DAYS per WEEK*	*EVERY DAY*	*LESS THAN 1 YR.*	*1 YEAR*	*2 YEARS*	*3-4 YEARS*	*5-9 YEARS*	*10+ YEARS*
Multiple Vitamins. Did you take…											
Regular Once-A-Day, Centrum, or Thera type	○	○	○	○	○	○	○	○	○	○	○
Stress-tabs or B-Complex type	○	○	○	○	○	○	○	○	○	○	○
Antioxidant combination type	○	○	○	○	○	○	○	○	○	○	○
Single Vitamins (not part of multiple vitamins)											
Vitamin A (not beta-carotene)	○	○	○	○	○	○	○	○	○	○	○
Beta-carotene	○	○	○	○	○	○	○	○	○	○	○
Vitamin C	○	○	○	○	○	○	○	○	○	○	○
Vitamin E	○	○	○	○	○	○	○	○	○	○	○
Folic acid, folate	○	○	○	○	○	○	○	○	○	○	○
Calcium, alone or combined with something else	○	○	○	○	○	○	○	○	○	○	○
Zinc, alone or combined with something else	○	○	○	○	○	○	○	○	○	○	○
Iron	○	○	○	○	○	○	○	○	○	○	○
Selenium	○	○	○	○	○	○	○	○	○	○	○

If you took Once-a-day, Centrum or Thera-type multiple vitamins, did you usually take types that ○ contain minerals, iron, zinc, etc. ○ do not contain minerals ○ don't know

If you took vitamin C or vitamin E:

How many milligrams of **vitamin C** did you usually take, on the days you took it?
○ 100 ○ 250 ○ 500 ○ 750 ○ 1000 ○ 1500 ○ 2000 ○ 3000+ ○ Don't know

How many IUs of **vitamin E** did you usually take, on the days you took it?
○ *100* ○ *200* ○ *300* ○ *400* ○ *600* ○ *800* ○ *1000* ○ *2000+* ○ *Don't know*

Did you take any of these supplements at least once a month?

○ Ginkgo ○ Ginseng ○ St. John's Wort ○ Kava Kava ○ Echinacea ○ Melatonin ○ DHEA
○ Glucosamine/Chondroitin ○ Something else ○ Didn't take these

The next section is about your <u>usual</u> eating habits in the past year or so. This includes all meals or snacks, at home or in a restaurant or carry-out. There are two kinds of questions to answer for each food:

HOW OFTEN, on average, did you eat the food during the past year?
*Please DO NOT SKIP any foods. Mark "Never" if you didn't eat it.

HOW MUCH did you usually eat of the food?

*Sometimes we ask how <u>many</u> you eat, such as 1 egg, 2 eggs, etc., ON THE DAYS YOU EAT IT.
*Sometimes we ask "how much" as A, B, C or D. LOOK AT THE ENCLOSED PICTURES. For each food, pick the picture (bowls or plates) that looks the most like the serving size you usually eat.
(If you don't have pictures: A=1/4 cup, B=1/2 cup, C=1 cup, D=2 cups.)
*Sometimes we made the "D" column a darker color. This is just to remind you to make sure you really eat that large a serving.

EXAMPLE: This person drank apple juice twice a week, and had one glass each time. Once a week he ate a "C" sized serving of rice (about 1 cup).

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MON.	2-3 TIMES per MON.	ONCE per WEEK	TWICE per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH <u>EACH TIME</u> SEE PORTION SIZE PICTURES FOR A-B-C-D				
Apple juice	○	○	○	○	○	●	○	○	○	How many glasses each time	● 1	○ 2	○ 3	○ 4
Rice	○	○	○	○	●	○	○	○	○	How much each time	○ A	○ B	● C	○ D

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME How many glasses on the days you drink it?				
How often do you drink the following beverages?														
Tomato juice or V-8 juice	○	○	○	○	○	○	○	○	○	How many glasses **each time**	○ 1	○ 2	○ 3	○ 4
Real 100% orange juice or grapefruit juice, including fresh, frozen or bottled	○	○	○	○	○	○	○	○	○	How many glasses **each time**	○ 1	○ 2	○ 3	○ 4
When you drink orange juice, how often do you drink a calcium-fortified brand?	○ Usually calcium-fortified ○ Sometimes calcium-fortified ○ Hardly ever calcium-fortified									○ I don't know ○ I don't drink orange juice				
Other real fruit juices like apple juice, prune juice, lemonade	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3	○ 4
Kool-Aid, Hi-C, or other drinks with added vitamin C	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3	○ 4
Drinks with some juice in them, like Sunny Delight, Juice Squeeze	○	○	○	○	○	○	○	○	○	How many bottles	○ 1	○ 2	○ 3	○ 4
Instant breakfast milkshakes like Carnation, diet shakes like SlimFast, or liquid supplements like Ensure	○	○	○	○	○	○	○	○	○	How many glasses or cans	○ 1	○ 2	○ 3	○ 4
Glasses of milk (any kind)	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3	○ 4

When you drink glasses of milk, what kind do you usually drink? **MARK ONLY ONE:**

○ Whole milk ○ Reduced-fat 2% milk ○ Low-fat 1% milk ○ Non-fat milk
○ Rice milk ○ Soy milk ○ I don't drink milk or soy milk

HOW OFTEN	NEVER	FEW/ YEAR	ONCE/ MONTH	2-3 TIMES/ MONTH	ONCE/ WEEK	TWICE/ WEEK	3-4 TIMES/ WEEK	5-6 TIMES/ WEEK	EVERY DAY	HOW MUCH EACH TIME				
Regular soft drinks, or bottled drinks like Snapple (not diet drinks)	○	○	○	○	○	○	○	○	○	How many bottles or cans	○ 1	○ 2	○ 3-4	○ 5+
Beer or non-alcoholic beer	○	○	○	○	○	○	○	○	○	How many bottles or cans	○ 1	○ 2	○ 3-4	○ 5+
What kind? **MARK ONLY ONE:**	○ Regular beer	○ Light beer	○ Non-alcoholic beer	○ I don't drink beer										
Wine or wine coolers	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3-4	○ 5+
Liquor or mixed drinks	○	○	○	○	○	○	○	○	○	How many drinks	○ 1	○ 2	○ 3-4	○ 5+
Glasses of water, tap or bottled	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3-4	○ 5+
Coffee, regular or decaf	○	○	○	○	○	○	○	○	○	How many cups	○ 1	○ 2	○ 3-4	○ 5+
Tea or iced tea (not herb teas)	○	○	○	○	○	○	○	○	○	How many cups	○ 1	○ 2	○ 3-4	○ 5+

What do you usually add to coffee? **MARK ONLY ONE:** ○ Cream or half & half ○ Nondairy creamer ○ Milk ○ None of these

What do you usually add to tea? **MARK ONLY ONE:** ○ Cream or half & half ○ Nondairy creamer ○ Milk ○ None of these

Do you usually add sugar (or honey) to coffee? ○ No ○ Yes IF YES, how many teaspoons each cup? ① ② ③-④ ⑤+

Do you usually add sugar (or honey) to tea? ○ No ○ Yes IF YES, how many teaspoons each cup? ① ② ③-④ ⑤+

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HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME (SEE PORTION SIZE PICTURES FOR A-B-C-D)				
How often do you eat each of the following fruits, just during the 2-3 months when they are in season?														
Raw peaches, apricots, nectarines, while they are in season	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Cantaloupe, in season	○	○	○	○	○	○	○	○	○	How much	○ 1/8	○ 1/4	○ 1/2	○ 1
Strawberries, in season	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Watermelon, in season	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Any other fruit in season, like grapes, honeydew, pineapple, kiwi	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
How often do you eat the following foods all year round? Estimate your average for the whole year.														
Bananas	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Apples or pears	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Oranges or tangerines	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Grapefruit	○	○	○	○	○	○	○	○	○	How much	○ 1/2	○ 1	○ 2	○ 3
Canned fruit like applesauce, fruit cocktail, or dried fruit like raisins	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

HOW OFTEN	NEVER	FEW/ YEAR	ONCE/ MONTH	2-3 TIMES/ MONTH	ONCE/ WEEK	TWICE/ WEEK	3-4 TIMES/ WEEK	5-6 TIMES/ WEEK	EVERY DAY	HOW MUCH EACH TIME				
Eggs, including egg biscuits or Egg McMuffins (Not egg substitutes)	○	○	○	○	○	○	○	○	○	How many eggs each time	○ 1	○ 2	○ 3	○ 4
Bacon	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Breakfast sausage, including sausage biscuits	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Pancakes, waffles, French toast, Pop Tarts	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Breakfast bars, granola bars, Power bars	○	○	○	○	○	○	○	○	○	How many	○ 1	○ 2	○ 3	○ 4
Cooked cereals like oatmeal, cream of wheat or grits	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D
High-fiber cereals like All Bran, Raisin Bran, Fruit-n-Fiber	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D

Which high-fiber cereal do you eat most often? **MARK ONLY ONE:** ○ All Bran or Bran Buds ○ Raisin Bran ○ Fiber One, Fruit-n-Fiber, etc. ○ Something else ○ I don't know ○ I don't eat it

Product 19, Just Right or Total cereal	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D
Any other cold cereal, like Corn Flakes, Cheerios, Special K	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D
Milk or milk substitutes on cereal	○	○	○	○	○	○	○	○	○	How many oz. on cereal	○ 3 oz.	○ 4-5 oz.	○ 6-7 oz.	○ 8+ oz.
Yogurt or frozen yogurt	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Cheese, sliced cheese or cheese spread, including on sandwiches	○	○	○	○	○	○	○	○	○	How many slices	○ 1	○ 2	○ 3	○ 4

When you eat cheese, is it ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat

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HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
How often do you eat the following vegetables, including fresh, frozen, canned or in stir-fry, at home or in a restaurant?														
Broccoli	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Carrots, or mixed vegetables or stews containing carrots	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Corn	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Green beans or green peas	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Spinach	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Mustard greens, turnip greens, collards	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
French fries, fried potatoes or hash browns	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
White potatoes not fried, incl. boiled, baked, mashed & potato salad	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Sweet potatoes, yams (Not in pie)	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Cole slaw, cabbage	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Green salad	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Raw tomatoes, including in salad	○	○	○	○	○	○	○	○	○	How much	○ 1/4	○ 1/2	○ 1	○ 2
Salad dressing	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4

Is your salad dressing ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't use

HOW OFTEN	NEVER	FEW/ YEAR	ONCE/ MONTH	2-3 TIMES/ MONTH	ONCE/ WEEK	TWICE/ WEEK	3-4 TIMES/ WEEK	5-6 TIMES/ WEEK	EVERY DAY	HOW MUCH EACH TIME				
Any other vegetable, like okra, squash, cooked green peppers	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Refried beans or bean burritos	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Chili with beans (with or without meat)	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Baked beans, black-eye peas, pintos, any other dried beans	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Vegetable stew	○	○	○	○	○	○	○	○	○	Which Bowl		○ B	○ C	○ D
Vegetable soup, vegetable beef, chicken vegetable, or tomato soup	○	○	○	○	○	○	○	○	○	Which Bowl		○ B	○ C	○ D
Split pea, bean or lentil soup	○	○	○	○	○	○	○	○	○	Which Bowl		○ B	○ C	○ D
Any other soup, like chicken noodle, chowder, mushroom, instant soups	○	○	○	○	○	○	○	○	○	Which Bowl		○ B	○ C	○ D
Spaghetti, lasagna or other pasta with tomato sauce	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Cheese dishes without tomato sauce, like macaroni and cheese	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Pizza, including carry-out	○	○	○	○	○	○	○	○	○	How many slices	○ 1	○ 2	○ 3	○ 4

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
Do you ever eat chicken, meat or fish? ○ Yes ○ No **IF NO, SKIP TO NEXT PAGE**														
Hamburgers, cheeseburgers, meat loaf, at home or in a restaurant	○	○	○	○	○	○	○	○	○	How much meat	○ 1/8 lb.	○ 1/4 lb.	○ 1/2 lb.	○ 3/4 lb.
Tacos, burritos, enchiladas, tamales, etc. with meat or chicken	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Beef steaks, roasts, pot roast, or in frozen dinners or sandwiches	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
How do you like beef cooked? ○ Rare ○ Medium ○ Well done ○ I don't eat beef														
Pork chops, pork roasts, or dinner ham	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
When you eat meat, do you ○ Avoid eating the fat ○ Sometimes eat the fat ○ Often eat the fat ○ I don't eat meat														
Veal, lamb or deer meat	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Ribs, spareribs	○	○	○	○	○	○	○	○	○	How many ribs	○ 3-4	○ 5-6	○ 7-8	○ 9+
Liver, including chicken livers or liverwurst	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Gizzard, pork neckbones, chitlins, pigs feet, etc.	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Mixed dishes with beef or pork, like *stew, corned beef hash, stuffed* cabbage, meat dish with noodles	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Mixed dishes with chicken, like chicken casserole, chicken & noodles, pot pie or in stir-fry	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Fried chicken, at home or in a restaurant	○	○	○	○	○	○	○	○	○	# medium pieces	○ 1	○ 2	○ 3	○ 4
Chicken or turkey not fried, such as baked, grilled, or on sandwiches	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
When you eat chicken, do you ○ Avoid eating the skin ○ Sometimes eat the skin ○ Often eat the skin														

HOW OFTEN	NEVER	FEW/ YEAR	ONCE/ MONTH	2-3 TIMES/ MONTH	ONCE/ WEEK	TWICE/ WEEK	3-4 TIMES/ WEEK	5-6 TIMES/ WEEK	EVERY DAY	HOW MUCH EACH TIME				
Oysters	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Other shellfish like shrimp, scallops, crabs	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Tuna, tuna salad, tuna casserole	○	○	○	○	○	○	○	○	○	How much of the tuna	○ A	○ B	○ C	○ D
Fried fish or fish sandwich, at home or in a restaurant	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Other fish, not fried	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Hot dogs, or sausage like Polish, Italian or chorizos	○	○	○	○	○	○	○	○	○	How many	○ 1	○ 2	○ 3	○ 4
Are your hot dogs ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat them														
Boloney, sliced ham, turkey lunch meat, other lunch meat	○	○	○	○	○	○	○	○	○	How many slices	○ 1	○ 2	○ 3	○ 4
Are your lunch meats ○ Usually low-fat or turkey ○ Sometimes low-fat ○ Hardly ever low-fat														

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME — SEE PORTION SIZE PICTURES FOR A-B-C-D				
Noodles, macaroni, pasta salad	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Tofu, bean curd	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Meat substitutes, such as veggie burgers, Gardenburgers	○	○	○	○	○	○	○	○	○	How many patties	○ 1	○ 2	○ 3	○ 4
Chinese food, Thai or other Asian *food, not counted above*	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Snacks like potato chips, corn chips, popcorn (not pretzels)	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

Are these snacks ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat

HOW OFTEN	NEVER	FEW/ YEAR	ONCE/ MONTH	2-3 TIMES/ MONTH	ONCE/ WEEK	TWICE/ WEEK	3-4 TIMES/ WEEK	5-6 TIMES/ WEEK	EVERY DAY	HOW MUCH EACH TIME				
Peanuts, other nuts or seeds	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Crackers	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Doughnuts, Danish pastry	○	○	○	○	○	○	○	○	○	How many	○ 1	○ 2	○ 3	○ 4
Cake, sweet rolls, coffee cake	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

Are they ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat

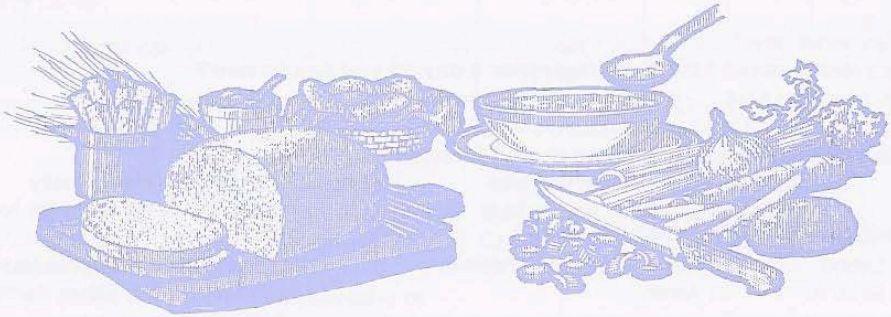
Cookies	○	○	○	○	○	○	○	○	○	How many	○ 1-2	○ 3-5	○ 6-7	○ 8+

Are your cookies ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ I don't know/don't eat

Ice cream, ice milk, ice cream bars	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

Is your ice cream ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ I don't know/don't eat

Pumpkin pie, sweet potato pie	○	○	○	○	○	○	○	○	○	How many slices	○ 1/2	○ 1	○ 2	○ 3
Any other pie or cobbler	○	○	○	○	○	○	○	○	○	How many slices	○ 1/2	○ 1	○ 2	○ 3
Chocolate candy, candy bars	○	○	○	○	○	○	○	○	○	How many bars	○ 1 small	○ 1 medium	○ 1 large	○ 2 large
Other candy, not chocolate, like hard candy, caramel, jelly beans	○	○	○	○	○	○	○	○	○	How many pieces	○ 1-2	○ 3-5	○ 6-7	○ 8+



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HOW OFTEN	NEVER OR A FEW TIMES PER YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	2+ TIMES per DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
Biscuits or muffins	○	○	○	○	○	○	○	○	○	How many **each time**	○ 1	○ 2	○ 3	○ 4
Rolls, hamburger buns, English muffins, bagels	○	○	○	○	○	○	○	○	○	How many **each time**	○ 1/2	○ 1	○ 2	○ 3
Dark bread like rye or whole wheat, including in sandwiches	○	○	○	○	○	○	○	○	○	How many slices **each time**	○ 1	○ 2	○ 3	○ 4
White bread or toast, including French, Italian, or in sandwiches	○	○	○	○	○	○	○	○	○	How many slices **each time**	○ 1	○ 2	○ 3	○ 4
Corn bread, corn muffins	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Tortillas	○	○	○	○	○	○	○	○	○	How many **each time**	○ 1	○ 2	○ 3	○ 4
Rice, or dishes made with rice	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Margarine (not butter) on bread or on potatoes or vegetables, etc.	○	○	○	○	○	○	○	○	○	How many pats (tsp.)	○ 1	○ 2	○ 3	○ 4
Butter (not margarine) on bread or on potatoes or vegetables, etc.	○	○	○	○	○	○	○	○	○	How many pats (tsp.)	○ 1	○ 2	○ 3	○ 4
Gravy	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Peanut butter	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Jelly, jam, or syrup	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Mayonnaise, sandwich spreads	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Catsup, salsa or chile peppers	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Mustard, soy sauce, steak sauce, barbecue sauce, other sauces	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4

Did you use the pictures to choose your serving size on this form? ○ Yes ○ No ○ I didn't have any pictures.

Would you say your health is ○ Excellent ○ Very good ○ Good ○ Fair ○ Poor

How many times have you gone on a diet? ○ Never ○ 1-2 ○ 3-5 ○ 6-8 ○ 9 or more

Did you ever drink more beer, wine or liquor than you do now? ○ Yes ○ No

How many hours do you watch television or video, per day or per week on average?
○ None ○ 1-6 hours/week ○ 1 hour/day ○ 2 hours/day ○ 3 hours/day ○ 4+ hours/day

Do you smoke cigarettes now? ○ No ○ Yes
IF YES, On the average about how many cigarettes a day do you smoke now?
○ 1-5 ○ 6-14 ○ 15-24 ○ 25-34 ○ 35 or more

What language do you usually speak at home or with friends?
○ English ○ Spanish ○ Something else ○ English & something else equally

What is your ethnic group? (MARK ONE OR MORE)
○ Hispanic or Latino ○ Black or African American ○ American Indian or Alaska Native
○ White, not Hispanic ○ Asian ○ Native Hawaiian or Other Pacific Islander

Thank you very much for filling out this questionnaire. Please take a minute to go back and fill in anything you may have skipped.

PLEASE DO NOT WRITE IN THIS AREA

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APPENDIX E

Seven-day Recall

Physical Activity Questionnaire

PHYSICAL ACTIVITY LIST

Moderate Activities

Occupational Tasks:

1. Delivering mail or paroling on foot
2. House painting
3. Truck driving (making deliveries- lifting and carrying light objects)

Household Activities:

1. Raking the lawn
2. Sweeping and mopping
3. Mowing the lawn with a power mower
4. Cleaning windows

Sports Activities: (Actual playing time)

1. Volleyball
2. Ping Pong
3. Brisk walking for pleasure or to work
4. Golf-walking, pulling, or carrying clubs
5. Calisthenics

Hard Activities

Occupational Tasks:

1. Heavy carpentry
2. Construction work- doing physical labor

Household Tasks:

1. Scrubbing floors

Sports Activities (Actual playing time):

1. Doubles tennis
2. Disco, Square, or Folk dancing

Very Hard Activities

Occupational Tasks:

1. Very hard physical labor- digging or chopping with heavy tools
2. Carrying heavy loads, such as bricks or lumber

Sports Activities (Actual playing time):

1. Jogging or swimming (Actual playing time)
2. Singles tennis
3. Racquetball
4. Soccer
5. Aerobics
6. Stair climbing
7. Weight training
8. Gymnastics

ID#_____

7-Day Physical Activity Recall Questionnaire

1. On the average, how many hours did you sleep each night during the last 5 weekday nights (Sunday-Thursday)? Record to the nearest quarter-hour.

   Hours:_____ Minutes:_____

2. On the average, how many hours did you sleep each night last Friday and Saturday nights?
   Hours:_____ Minutes:_____

3. First let's consider moderate activities. What activities did you do and how many total hours did you spend during the last 5 weekdays doing these moderate activities or others like them? Please tell me to the nearest half-hour.

   Hours:_____ Minutes:_____

4. Last Saturday and Sunday, how many hours did you spend on moderate activities and what did you do? (Can you think of any other sport, job, or household activities that would fit in this category?)

   Hours:_____ Minutes:_____

5. Now let's look at hard activities. What activities did you do and how many total hours did you spend during the last 5 weekdays doing these hard activities or others like them? Please tell me to the nearest half-hour.

   Hours:_____ Minutes:_____

6. Last Saturday and Sunday, how many hours did you spend on hard activities and what did you do? (Can you think of any other sport, job, or household activities that would fit in this category?)

   Hours:_____ Minutes:_____

7. Now let's look at very hard activities. What activities did you do and how many total hours did you spend during the last 5 weekdays doing these very hard activities or others like them? Please tell me to the nearest half-hour.

   Hours:_____ Minutes:_____

8. Last Saturday and Sunday, how many hours did you spend on very hard activities and what did you do? (Can you think of any other sport, job, or household activities that would fit in this category?)

   Hours:_____ Minutes:_____

9. Compared with your physical activity over the past 3 months, was last week's physical activity more, less, or about the same? (Circle One)

   More
   Less
   About the same

APPENDIX F

Past Physical Activity Questionnaire

I.D._____

Physical Activity Questionnaire

1. In the last 10 years, on average, how frequently have you exercised (including going on walks, riding a bicycle, dancing, etc.;)? Report the total number of times that you have exercised in a typical week.
   _____
   _____

2. On average, how long do you exercise each time? _____

3. Circle the number that best represents the intensity of your typical exercise bout:

   1. Very, very easy
   2. Very easy
   3. Easy
   4. Average
   5. Hard
   6. Very hard
   7. Very, very hard

4. What specific physical activities have made up your exercise routine? How long have you spent doing each activity?
   _____
   _____

5. During the ages of 20-30 years, how frequently did you exercise (including going on walks, riding a bicycle, dancing, etc.;) Report the total number of times that you exercised in a typical week.
   _____
   _____

6. On average, how long did you exercise each time? _____

7. Circle the number that best represents the intensity of your typical exercise bout:

   1. Very, very easy
   2. Very easy
   3. Easy
   4. Average
   5. Hard
   6. Very hard
   7. Very, very hard

For former gymnasts:

8. At what age did you begin your gymnastics training?