

Does Exercise During Growth Have a Long-Term Effect on Bone Health?

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MODLESKY, C.M., and R.D. LEWIS. Does exercise during growth have a long-term effect on bone health? *Exerc. Sport Sci. Rev.*, Vol. 30, No. 4, pp. 171–176, 2002. *There is increasing evidence that growth is a critical time for altering body tissue composition and fostering either the development or the prevention of disease. The focus of this review is to examine the effect of regular exercise during growth on long-term bone health.* **Keywords:** physical activity, mechanical loading, osteoporosis, fracture risk, childhood, adolescence, puberty

INTRODUCTION

Tissue composition strongly reflects the health and fitness status of an individual. A large quantity of body fat is associated with increased risk of metabolic and cardiovascular disease, small muscle mass is associated with limited physical function, and low bone mass is associated with increased risk of skeletal fracture. Because a less than optimal tissue profile and its connection to disease are not necessarily apparent until the adult years, research in these areas has frequently centered on adult population groups. However, there is increasing evidence that adverse tissue composition and related disease processes begin during childhood and track into adulthood. Accordingly, many scientists and clinicians have embraced the idea that childhood and adolescence are critical times for fostering the development or the prevention of disease through early lifestyle choices, such as regular exercise.

Exercise scientists involved in the study of bone have developed a particular appreciation for the potential influence of regular exercise during growth on body tissue development and disease prevention. The interest stems in part from observations that adult athletes involved in high-load activities have very high bone mass, yet the adult skeleton typically demonstrates a limited response to exercise intervention. The inconsistency in these observations suggests

that the enhanced bone mass in adult athletes is the result of genetic factors and/or early initiation of training. Considering that approximately 90% of total bone mineral content (BMC) is accumulated by the end of adolescence, coupled with the continual change in the size and shape of the immature skeleton, the growth period may be an optimal time for altering the mass, geometry, and microarchitecture of bone. The focus of this brief review is to evaluate evidence that regular exercise participation during growth can optimize bone mass and structure and inevitably reduce fracture incidence throughout the life cycle.

BONE AND GROWTH

Bone is a dynamic tissue that is under constant construction during the growing years, maintenance throughout the early and middle adulthood years, and an inevitable deterioration during the final stages of life. Growth and maintenance of the skeleton are dictated primarily by the modeling and remodeling processes. Modeling promotes the continuous increases in the size and changes in the shape of the skeleton during childhood and adolescence, whereas remodeling is involved in the resorption and replacement of established skeletal tissue. Skeletal growth and maturation are regulated by a network of growth factors, gonadal hormones, and pituitary hormones. As bones grow, there is a substantial increase in the width and cross-sectional area (CSA) of the total bone and the cortical regions, increases in the thickness of trabeculae, but no change in relative trabecular number. Figure 1 depicts the different structural aspects of a long bone.

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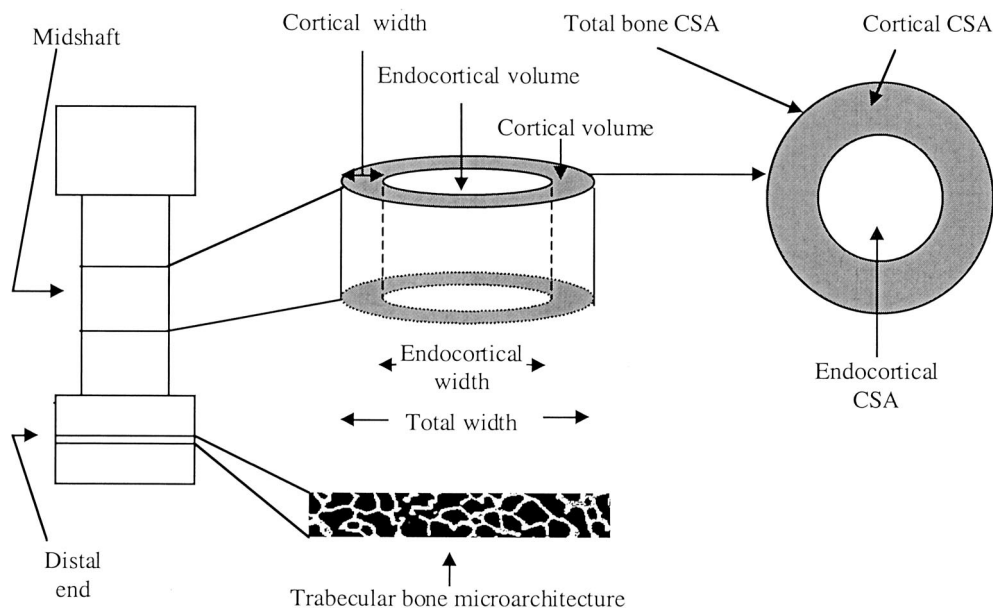


Figure 1. Structural components and different regions of a long bone that may contribute to its strength and resistance to fracture.

EFFECTS OF EXERCISE ON BONE DURING GROWTH

The idea that regular exercise participation during the growing years can optimize tissue growth and have long-term residual effects is not new. Investigations conducted during the 1960s and 1970s suggested that aerobic training during growth could optimize the development of the oxygen transport system, namely the heart and the lungs (2). However, because the evidence supporting the importance of exercise participation in the optimal development of oxygen-carrying tissues extends little beyond studies of athletes, the hypothesis remains insufficiently tested. The effect of exercise on the growing skeleton has undergone a more thorough examination.

Critical Years for Exercise: Two Major Assumptions

The notion that the growing years are a critical time for altering the skeleton's composition is based on two major assumptions: 1) peak bone mass is increased and structure is enhanced, or more specifically, the total bone and its cortical walls become wider and/or its trabeculae become more connected (Fig. 1), leading to increased bone strength, and 2) exercise-induced increases in bone mass and improvements in bone structure during growth are maintained, at least in part, throughout the life cycle. Figure 2 depicts the theorized increase in peak bone mass induced by regular exercise during growth and two potential responses if exercise participation is reduced or ceased after peak bone mass is reached. If peak bone mass and structure are improved and the gains are sustained over time, then a reduction in fracture risk should result. Conversely, if bone gains are lost over time and no longer present during the final stages of the life cycle, then exercise during growth should not be viewed as an effective remedy for future fracture risk. Although a longitudinal trial in which subjects were randomly assigned to an exercise intervention or control group and followed throughout the life cycle would be the preferred research design, no such study exists. Hence, evidence based on available studies must be pieced together to assess the accuracy of these assumptions.

Are peak bone mass, structure, and strength optimized by regular exercise during growth?

A growing number of studies support the first assumption that exercise during growth can increase peak bone mass. For instance, approximately 1/2 of the elevation in areal bone mineral density (aBMD) observed in adult gymnasts is already present in 10-yr-old premenarcheal gymnasts (12). Moreover, the greater dominant versus nondominant arm BMC discrepancy observed in racquet sport athletes than

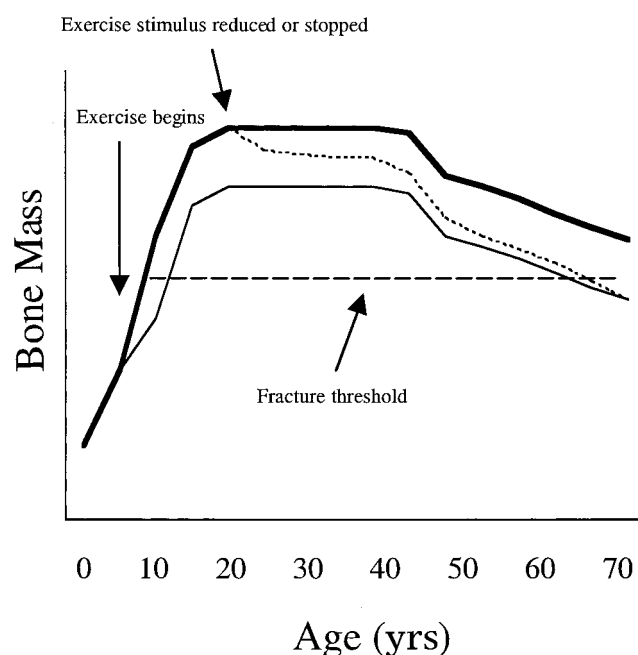


Figure 2. Theoretical changes in bone mass with age if there is: 1) no regular exercise participation during growth (—), 2) regular participation in exercise during growth and the gains are maintained throughout adulthood following a reduction or cessation of regular exercise (—), and 3) regular participation in exercise during growth but gains are gradually lost during adulthood if the level of exercise is reduced or ceased (---).

controls (6), particularly in players who initiated training before menarche (6) (Fig. 3), is compelling evidence that exercise during growth can increase peak bone mass at specific bone sites. The unique aspect of this unilateral model is that the effect of regular exercise on bone can be examined without a prospective research design and without the influence of genetic, hormonal, or nutritional factors.

Although studies of athletes are insightful, investigations of nonathletes are needed to determine whether the potential benefits of exercise can be reaped with reasonable levels of exercise that can be prescribed to the general population. The handful of observational and intervention studies conducted with nonathlete children suggests that the potential benefits of regular bone-loading exercise are not limited to athletes (3,13). A 20% greater gain in BMC has been observed in children in the highest versus the lowest tertile for physical activity during the 2 yrs surrounding peak BMC accrual (1). In particular, aBMD and BMC are increased in children who participate in activities that load the hip and spine, such as jumping (3,13). Although these studies and others suggest bone mass can be increased at a greater rate during growth if bone-loading exercises are conducted regularly, additional studies are needed to determine whether these gains actually lead to a higher peak bone mass.

It is clear that aBMD and BMC assessed using dual-energy x-ray absorptiometry (DXA) are indicators of bone strength and bone mineral mass, respectively; however, focus on these measures has oversimplified the complexity of bone, especially in the growing skeleton. A more complete understanding of the biology of bone and its response to different environmental factors can be gained if structural parameters, such as size, shape, cortical thickness, volume, CSA, and trabecular microarchitecture (Fig. 1), are assessed. Moreover, assessment of these structural components in conjunction with aBMD and BMC, may give a better indication of bone strength and fracture risk. For instance, knowledge of total and endocortical bone width can be used to determine cross-sectional moment of inertia (CSMI; Fig. 4), a measure of bone's resistance to bending and torsion. The potential importance of measuring structural aspects of bone is demonstrated by the significant overlap in aBMD and BMC in those

who do and do not experience skeletal fracture (11). Hence, if regular exercise during childhood can indeed permanently alter the skeleton, the effects may not be limited to its mass.

Studies using peripheral quantitative computed tomography (pQCT) on racquet sport athletes suggest that the higher BMC in the dominant versus nondominant humerus and radius is due to an increase in the width, rather than an increase in the material density, of the cortical bone layer (4). Because total CSA is elevated and endocortical CSA is either increased or not different depending upon the site measured along the two bones, the increase in cortical width can be attributed to an increased apposition of bone on the periosteal surface (4). Such a finding is not entirely surprising considering a hollow bone (*i.e.*, increased endocortical width and CSA) with a large diameter (*i.e.*, increased total bone width and CSA) has a higher CSMI and can withstand bending and torsional loads better than a thin bone of the same mass and length. Similarly, estimates of bone structure and strength using DXA suggest that activities resulting in significant compression of the proximal femur increase the cortical width of the femoral neck and intertrochanter in girls during early puberty. However, the increased thickness is due to smaller increases in endocortical width than normally occur with growth (13). The different response than that observed in tennis players is likely attributable to the greater compressive loading at the hip associated with jumping than occurs in the arm playing tennis. It is unclear whether the blunted expansion is due to increased bone formation or less bone resorption on the endocortical surface. Because the structural changes in the tennis players and in the children involved in the jump training are not the same, the two forms of training likely have different effects on bone strength. For instance, indices of resistance to bending and torsion were elevated at all sites in the dominant arm of the tennis players (4), but only elevated in the femoral neck of the child jumpers (13).

These findings suggest that the skeleton's response to exercise is specific to the site and type of load applied. Studies that further explore the location of bone apposition in re-

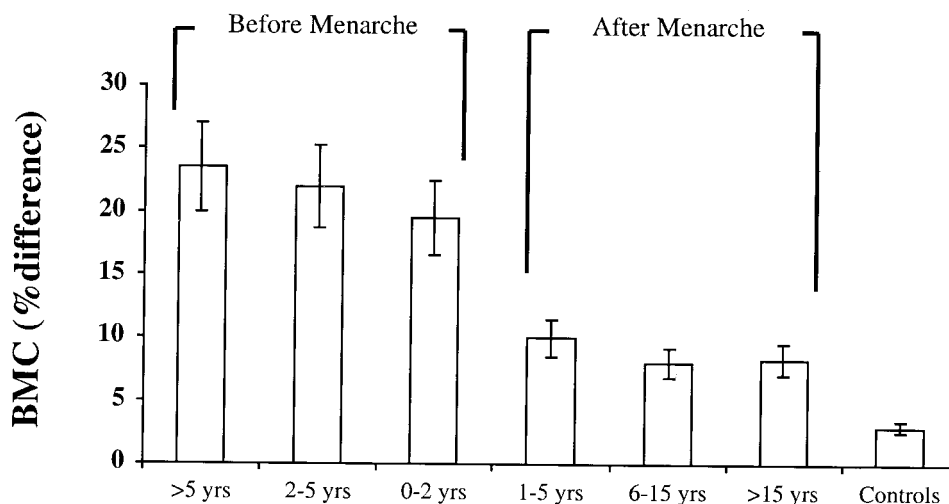
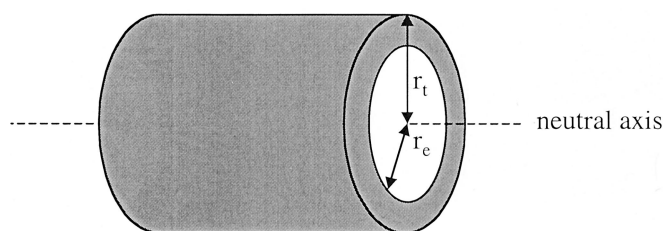


Figure 3. The degree of difference in the dominant vs nondominant humerus bone mineral content (BMC) in tennis players who began training before and after menarche. The figure suggests especially training initiated before menarche is more beneficial to bone than training after menarche. *Intersecting lines represent 95% confidence intervals.* (Adapted from Kannus, P., H. Haapasalo, M. Sankelo, H. Sievanen, M. Pasanen, A. Heinonen, P. Oja, and I. Vuori. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. *Ann. Intern. Med.* 123:27–31, 1995.)



$$CSMI = \pi/4 \times (r_t^4 - r_e^4)$$

Figure 4. The cross-sectional moment of inertia (CSMI), a measure of a bone's resistance to bending and torsional loading, can be determined if the total bone radius (r_t) and endocortical radius (r_e) are known.

sponse to different types of exercise and the effect on bone strength in the growing human skeleton are needed, especially studies that use methodologies that can provide more direct measures of the structural aspects of bone, such as pQCT and magnetic resonance imaging. Moreover, studies using these methodologies to assess the effects of exercise on other structural components, such as trabecular microarchitecture, are also needed.

Are exercise-induced bone gains during growth permanent?

Even if progressive skeletal loading exercise during the growing years does lead to a higher peak bone mass, optimal structure, and increased bone strength, the importance of these improvements hinges on the second assumption that the skeleton can preserve these gains and fracture risk is subsequently reduced. Our current understanding of the preservation of exercise-induced bone gains achieved during growth is limited to studies of former athletes. Observations that women retired from collegiate gymnastics for more than 10 yrs have higher hip, lumbar spine, and total body aBMD (9–22%) than age-, height-, and weight-matched controls (9) suggest there is at least partial maintenance of exercise-induced bone gains. However, whether these elevations in bone are sustained throughout the rest of the life cycle is uncertain. When compared with current collegiate gymnasts, the retired gymnasts had lower aBMD at the femoral neck, whereas aBMD at this hip site was not different in the younger versus older controls (9). There are two potential explanations for the discrepancy. First, it is possible that the retired gymnasts never achieved the level of aBMD reported in the current gymnasts. This is plausible considering that the retired gymnasts began training at an older age than the current gymnasts (11.9 yrs compared with 6.2 yrs, respectively). As demonstrated in Figure 3, earlier initiation of training is associated with greater gains in bone mass. Second, the retired gymnasts may have lost some of the gains originally achieved.

The second explanation is consistent with a recent cross-sectional study of active and retired soccer players (7). Leg aBMD was 11.6% higher in the active soccer players compared with controls, but no difference in arm

aBMD was detected. A similar pattern was observed in the retired soccer players and controls, but the extent of the differences in leg aBMD became progressively smaller and no longer existed in those soccer players retired for more than 35 yrs. Moreover, the number of fractures in former soccer players and controls was not different. The authors concluded that the retired soccer players lost progressively more leg aBMD with age (0.33% per yr vs 0.22% per yr) and did not experience a lower rate of fracture. Although the scenario proposed is certainly possible, the design of the study limits the interpretability of the results. There is no indication that the level of training and the age of initiation of soccer activity were the same in the active players and each group of retired players. Therefore, the reduction in the leg aBMD discrepancy between soccer players and controls may instead reflect a trend toward a progressively higher intensity and earlier onset of soccer participation during the past 70 yrs.

Longitudinal studies tracking changes in retired athletes may provide some insight into the permanence of bone gains likely achieved, in large part, during growth.

Attainment of Bone Gains and Link to Maturity Level

Consistent with the observations in tennis players that girls who initiate exercise training before menarche have greater differences in dominant versus nondominant arm BMC (Fig. 3), and the higher aBMD observed in the earlier-starting current gymnasts than retired, the degree of bone changes attributed to regular exercise may be linked to the level of skeletal maturity when exercise is initiated. Some studies suggest the skeleton is most sensitive to mechanical loading during the early pubertal yrs (13). For instance, it was recently reported that activities providing enough stimulus to accelerate the increase in aBMD and total bone CSA associated with growth, to blunt the expansion of the medullary cavity (*i.e.*, endocortical width and CSA) and to increase measures of bone strength in regions within the femur of early pubertal girls, did not alter bone in prepubertal girls. It is suspected that the hypothesized increase in sensitivity of the skeleton to mechanical loading during early puberty is due to the simultaneous elevation in growth hormone, insulin-like growth factor-1, androstenedione, and estradiol (8). However, the connection between elevated hormonal levels and the sensitivity of the skeleton to loading requires further investigation.

Permanence of Bone Gains and Required Stimulus

If gains in bone mass and improvements in structure are indeed attained in response to regular exercise during growth, it would be of interest to determine the stimulus required, if any, to maintain the benefit. A recent study of competitive tennis players who maintained substantially higher BMC in the dominant versus nondominant arm despite a reduction in

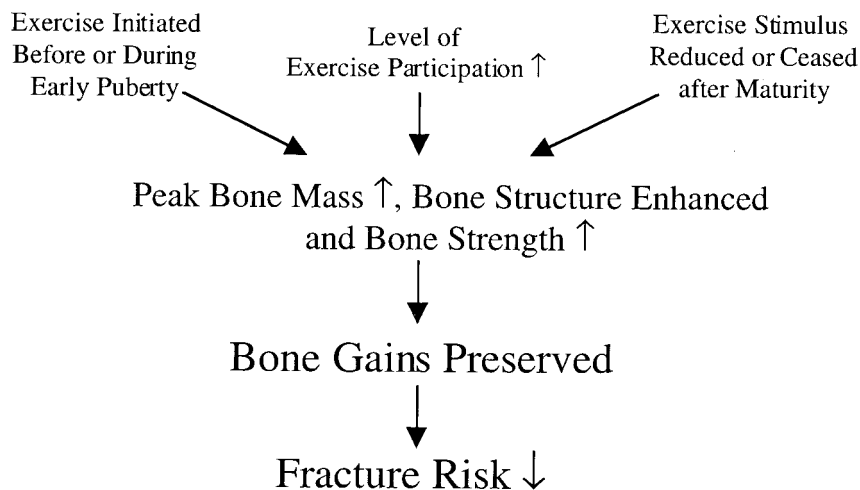


Figure 5. A model describing the potential role of maturity and the level of exercise participation during growth in the attainment of optimal peak bone mass, structure, and strength, and the subsequent effect on fracture risk.

training time, suggests the benefits of exercise reaped by the skeleton can be maintained, at least in part, with a shorter stimulus period (10). A study in lower mammals suggests the exercised-induced bone gains achieved during growth are maintained throughout the life cycle, even when the stimulus is removed entirely (14). Rats run-trained on a treadmill for 10.5 months experienced greater increases in vertebral weight, BMC, trabecular bone volume, bone protein content, bone calcium content, and bone alkaline phosphatase activity compared with controls (14), which remained systematically higher during the period of age-related bone loss. The difference between the latter study and studies that observed loss of exercised-induced increases in bone mass with detraining in growing animals (5) is that the duration of training extended though the entire maturation of the skeleton. It is possible that the ability of the skeleton to preserve benefits from childhood activity is tied to the length of training during growth, the timing of the initiation of training, and/or the maturity of the bones when exercise training is reduced (or ceased).

SUMMARY

The idea that the growing years are an opportune time to optimize bone mass, structure, and strength certainly has merit. The evidence supporting such a theory is based primarily on studies of athletes and short-term studies of jumping exercise in school-age children. Even if bone mass and structure are optimized by exercise during the growing years, the importance of these gains depends largely on their permanence. As depicted in the model presented in Figure 5, the preservation of bone gains throughout the lifecycle may be closely tied to the degree of skeletal maturity when exercise is initiated and reduced (or ceased).

Long-term prospective studies that test whether exercise suitable for the general population can enhance and preserve bone mass and structure are sorely needed. Moreover, studies utilizing techniques that provide insight into the structural adaptations of immature bone to exercise will further our understanding of the potential role of

exercise initiated during the growing years in the prevention of osteoporotic fractures.

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