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Perspective Article

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The skeleton in a long-term perspective – Are exercise induced benefits eroded by time?

M.K. Karlsson

Department of Orthopaedics, Malmo University Hospital, Malmo, Sweden

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The fragility fracture problem

About 30-50% of women, and 15-30% of men will suffer a fracture related to osteoporosis in their lifetime[1]. Prevention of fractures is important because fractures are associated with increased morbidity, mortality and impose a financial burden on the community[2,3]. During the last few years, several studies have shown that drugs reduce the fracture risk by about 50% in women with osteoporosis, a bone mineral density (BMD) 2.5 standard deviations (SD) below the young normal mean, the definition of osteoporosis advocated by the World Health Organisation[4,5]. This risk reduction is only confined to the most vulnerable high risk group, whereas most fractures are derived from the much larger population of individuals at more modest risk, individuals with osteopenia (BMD between - 1 to - 2.5 SD below the young normal mean)[5]. These subjects should not be given drugs because drug trials have not shown a fracture reduction in this group. Lifelong treatment of all women and men from the age of 50 years, irrespective of their BMD, can not be recommended because this approach is not evidence based, it is not feasible, or cost effective[6].

Thus, the public health burden of fractures cannot be solved with drugs. Instead, community-based interventions are needed that are safe, widely accessible, inexpensive to implement and that do reduce the number of individuals with fractures. Exercise has the first three features and so could be an attractive approach to reducing the burden of fractures. But, what evidence exists that exercise does reduce the number of fractures? Most individuals who are subjected to exercise during adolescence reduce their exercise level in the middle aged period, the period in life when work and family increase the demands of the individual. If exercise has a preventive effect of fragility fractures, exercise induced skeletal benefits must not be eroded by time. The purpose of this review is to critically examine the evidence that exercise induced benefits of the skeleton are retained into old age and if there exists an association with exercise during growth and young years and reduced risk to sustain fragility fractures.

Exercise increases bone strength

In the following sections, all presented differences are significant unless otherwise stated but for brevity these are presented without confidence intervals or p-values. Exercise seems to increase bone mass, with the most obvious benefits to the skeleton reached during growth[7-12]. Also, bone size and skeletal architecture, both independently providing bone strength, may be affected by exercise during this period[13-18]. This is of advantage for skeletal strength, but at the age when individuals usually are subjected to an intense level of physical activity, few individuals are at risk for low energy related fractures even if they have at that age a relatively low BMD. The fragility fractures become a problem in magnitude much later in life, and if exercise during growth should be recommended as a prevention strategy for future fragility fractures, exercise induced skeletal benefits must be retained even after a reduction in exercise level, into old age.

The Achilles heel of exercise – its cessation

Currently, there exists only short-term prospective data following former athletes with retirement from exercise. Kontulainen et al. reported that the differences observed in bone mineral content (BMC) between the playing and non-playing arm in racket players remained after detraining, suggesting bone mass benefits are maintained also after retirement from exercise[19]. However, the two years of detraining in the cited study may be too short to detect a higher bone

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Corresponding author: Magnus K. Karlsson, M.D., Ph.D., Department of Orthopaedics, University Hospital UMAS, Malmo, SE 20502, Sweden
E-mail: magnus.karlsson@orto.mas.lu.se

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loss in the formerly loaded arms in comparison with the minor loaded arms. Additionally, the former tennis players were still training 3 hours per week, a level that may be enough to preserve the bone mass benefits achieved during a higher level of training. The correlation found between current training and BMD in retired male soccer players ($r = \sim 0.25$)[20], support the notion that a lower level of exercise may preserve previously achieved exercise induced benefits. Similar conclusions have also been reported by Huddlestone et al. when reporting a difference of 4-7% remaining when comparing the dominant and non-dominant arm in lifelong tennis players[21]. Cited studies support the notion that residual benefits are found in retired athletes and that continued activity may contribute to these benefits.

In contrast, Michel et al. reported in a prospective study an increased bone loss in runners who ceased to run, whereas there was no loss in the individuals who continued with the running[22]. Similarly data was presented by Vuori et al. when reporting that unilateral leg presses four times a week for 12 months increased bone mass but that the BMD returned to pre-training levels with only 3 months of retirement from exercise[23]. A larger prospective study supports this, when reporting that 55 former athletes, retired for a mean of 4 years, decreased the exercise induced benefits in BMD by 0.3 standard deviations (SD) at the femoral neck in comparison with controls[24]. Additionally, the retired athletes had a higher BMD loss in comparison with changes in the 42 athletes who continued with exercise during the same period. Thus, prospective data from independent studies following former athletes for several years suggest that exercise induced bone mass benefits seem to be eroded by time.

So far no prospective long-term bone mass data exist in retired athletes, forcing us to base our inferences on cross-sectional data. Retired male soccer players had a residual higher BMD during the first two decades after retirement, but when looking at four to five decades of retirement, no BMD benefits were found in the former soccer players[20]. Leg BMD was 10% higher than age-matched controls in players retired for 5 years, 5% higher in players retired for 16 years, but no higher in players retired for 42 years. The diminution in leg BMD was 0.33%/year in the former soccer players, ~50% higher than the 0.21%/year diminution in the controls. No significant residual benefits were seen in the over 70-year-old former soccer players[20]. Similar data were found in female soccer players, with a BMD benefit found after a decade of retirement, however less than that found in active soccer players[25]. Former male weight lifters had 8% total body BMD when they were 35-49 years old, 6% when they were 50-64 years old but no higher when they were 65-79 years[26-28]. Also male and female ballet dancers had a higher BMD loss across the ages than the age- and gender-matched controls[29]. Benefits of about 1-1.5 SD are found in retired gymnasts retired for 1-2 decades, around half that found in active gymnasts (2-2.5 SD)[13]. Thus, three to five decades after cessation of an active career most exercise induced skeletal benefits seem to be lost[13,20,26-32].

Are exercise induced structural changes of the skeleton preserved with cessation of exercise?

Exercise may also affect the structural characteristics of the skeleton. Haapasalo et al. reported that exercise caused enlargement in bone size (humerus, radius shafts, and distal humerus) without a change in volumetric bone density[17]. The structural characteristics were continually maintained after retirement. Twelve former male tennis players retired for 1 to 3 years had higher, side-to-side difference when comparing dominant and non-dominant are in bone mineral content (BMC) (20%), total cross-sectional area of bone (18%), cortical area (22%), bone strength index (30%) and cortical wall thickness (15%). The side-to-side cross-sectional area of the marrow cavity difference was higher at the proximal humerus (19%) and radial shaft (29%). Volumetric density of cortical bone was lower at the distal humerus and trabecular bone side-to-side difference was not significant. The marrow cavity was larger, not smaller, in the playing arm suggesting that the players had greater endocortical expansion during activity or that bone loss occurred on the endocortical surface after retirement[17]. These observations fit in with the notion that exercise produces enlargement of bone size which is permanent after retirement but any endocortical thickening due to endocortical apposition may be lost or partly lost with retirement. Bone size was also evaluated in 90 former male soccer players and weight lifters, all above the age of 50 years, and retired from exercise for 3-65 years. In this cohort, both femoral neck area and lumbar spine width were larger in the former athletes than in the 77 previously sedentary age- and gender-matched controls[33]. Additionally, quantitative ultrasound, usually regarded as an evaluation of skeletal architecture, was higher in the former athletes than the controls indicating that exercise induced structural changes of the skeleton may be maintained in former athletes into old age. If so, this would increase bone strength and reduce the number of fragility fractures in the former athletes.

Fracture risk after cessation of exercise

A reduced fracture risk has also been reported in retired athletes. The prevalence of fractures in 663 former athletes above the age of 50 years, and retired from sport for up to 65 years were lower than in 943 age- and gender-matched controls after an active exercise career[24,33]. The proportion of subjects with fractures was no lower in former athletes than controls overall (25.9% versus 25.2%), but lower than in controls after retirement from exercise (8.9% versus 12.1%). Additionally, the proportion of subjects with low energy related fragility fractures sustained after the age of 50 years was lower in the former athletes (2.3% versus 4.2 %) as well as the proportion of individuals with distal radius fractures (0.8% versus 2.3%) in comparison with the controls[24,33]. In contrast, Wyshak et al. reported in 2,622 former female college athletes and 2,776 controls, now aged 20-80 years, that the former college athletes had a higher lifetime occurrence

of fractures compared to controls (40.5% versus 31.9%) but no fewer former athletes with fractures after retirement (29% versus 32%)[34]. However, as this study included individuals from the age of 20 years, maybe there were too few elderly individuals to evaluate the risk to sustain fragility related fractures. Similar data have also been presented in the Leisure World Study, when they reported that an activity level of > 1 hour/day had reduced the risk of hip fracture but the beneficial effect was lost if the activity level was reduced[35]. However, the level of exercise in this study may have been too low and conducted only during adulthood, as to lead to skeletal benefits at a magnitude to reduce the number of fragility fractures.

Summary

The most compelling evidence for a beneficial effect of exercise on bone mineral density (BMD) is during growth. If physical activity could prevent age-related bone loss or restore already lost BMD in adults and specifically in individuals above the age of 65 years, the age when fragility fractures exponentially rise, is unclear and at least of a questionable biological significance. Most prospective data following athletes in retirement indicate a higher bone loss in retired athletes in comparison with both athletes who continue with exercise and controls. Additional, cross-sectional long-term data suggest the benefits in BMD are lost in former athletes. In contrast, residual benefits in bone size or shape may be permanent. When looking at the clinical relevant end point, fractures, it seems that there are fewer former athletes with fragility fractures than among individuals who were never subjected to exercise, but the quality of the evidence supporting this notion is weak.

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