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ORIGINAL ARTICLE

Exercise and bone

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Cellular responses to mechanical strain

Osteoclasts. Very little is known about osteoclastic responses to mechanical stimuli, but mechanical strain reduces osteoclast recruitment (Rubin et al., 1999).

Osteoblasts. Mechanical stimulation of bone tissue by physical activity stimulates bone formation in normal bone and may attenuate bone loss in osteoporotic patients. Normal bone cells seem to increase proliferation and TGF- β secretion in response to mechanical strain, while osteoporotic cells do not (Neidlinger-Wilke et al., 1995). Osteoblastic cells from different locations in the skeleton react differently to mechanical strain in animal studies.

Osteocytes seem to be the primary mechanosensory cells of bone, and the lacunocanalicular network constitutes the structure that mediates mechanosensing. Strain-derived flow of interstitial fluid through this network seems to mechanically activate the osteocytes, as well as ensuring transport of cell signalling molecules and nutrients and waste products. This concept allows an explanation of local bone gain and loss, as well as remodelling in response to fatigue damage.

It has been shown that mechanical loading stimulates the transformation of lining cells to osteoblasts (Boppart et al., 1998; Pead et al., 1988).

Effects of mechanical strain on bone turnover and bone remodelling

Bone remodelling is affected by mechanical strain. The general trend is decreased bone degradation, possibly caused by reduced osteoclast recruitment (Etherington et al., 1999). The impact of physical

activity on bone turnover may, however, depend on the kind of exercise performed (Layon, 1993; Zorbas et al., 1994). There are inconsistencies in the effects of physical exercise on biomarkers of bone turnover. An increased bone resorption has been observed in acute (Welsh et al., 1997) or chronic exercise (Jaffre et al., 2001). On the contrary it has been demonstrated that treadmill running had inhibited the increase in urinary deoxypyridinoline excretion observed in castrated rats (Horcajada et al., 1997).

Physical activity and bone mass in the young

There exist evidence from observational studies and prospective randomised controlled trials, that support the notion that that exercise increase bone mass in the young (Bradney et al., 1998; Haapasalo et al., 1996; Heinonen et al., 1999; MacDougall et al., 1992; Margulies et al., 1986; Morris et al., 1997; Nilsson & Westlin, 1971; Sinaki et al., 1996). Studies have reported that both girls and boys in a high impact group on a moderate level gained significantly more bone than controls over a 7–10 month period (Morris et al., 1997). In examining the effect of loading activities in premenopausal women, longitudinal studies often demonstrate rather small increases in bone mass, 1–6% over an 8–24 month period (Sinaki et al., 1996). In young males, it has been shown that intensive exercise can accelerate bone accretion to a very high extent in a short period as 14 weeks, even if more than 40% of the subjects discontinued the training program due to stress fractures of the tibia (Margulies et al., 1986). In children, activities characterized by impact have been shown to be more osteogenic than non-impact

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loading activities (Courteix et al., 1998). The duration of the load seems to be of minor importance, while high strain, multidirectional, impact-loaded, high frequency training are of significant importance (Lanyon & Rubin, 1984; Rubin & Lanyon, 1985, 1984a, b).

Peak bone mass and physical activity

Peak Bone Mass is defined as the amount of bone tissue present at the end of the skeletal maturation. Among the factors that play a major role to improve the peak at adolescence, nutrition and physical activities are those on which one can act. A lot of studies give evidence that the best period to improve the bone mass is during the pre- and peripubertal years, a stage of growth when the skeleton is most responsive to exercise. Exercise may enhance bone formation in a synergistic action in the presence of growth hormone or sex steroid (Bass, 2000).

Physical activity and bone mass in adults

Limited data are available on the effect of exercise on bone in men, but the few studies show significant increases in BMD compared to controls in short-time and long-time studies (Michel et al., 1989; Williams et al., 1984).

Quite a few prospective studies have investigated physical training and BMD in pre- and post-menopausal women. Most studies of pre-menopausal women show some positive influence. It appears that higher loads, such as those produced by greater impact, lead to greater bone mass. The activities through which these loads can be achieved need to be identified. The data also suggest that it is important to distinguish young adult women from older premenopausal women, since bone may respond better to increased mechanical loads in the earlier years. Berard et al. performed a meta-analysis on the effect of exercise on bone, based on the literature from 1966 to 1996, and found no significant BMD benefit of training, but a trend. However, this study includes the literature from the early years of the DXA-scanning method. Studies from 1995 seem to show BMD benefit to exercise. More specific methods for bone mass and bone strength measurements in prospective, randomised studies at all ages are needed in order to evaluate the possible positive effect of training on BMD.

Physical activity and skeletal structure

There is evidence that supports the notion that exercises during growth influence not only bone mineral density (BMD) but also the skeletal structure. This is of clinical relevance as not only BMD

but also bone structure is of importance for skeletal strength. The earliest studies that evaluated the structural effects of exercise compared radiographs of the arms in tennis players. After this dual energy x-ray absorptiometry, computerised tomography (CT) and magnetic resonance imaging (MRI) has supported that exercise does influence the cortical thickness, the skeletal size and the skeletal structure with a magnitude that could be of biological significance for fracture reduction (Haapasalo et al., 1996; Huddleston et al., 1980; Jones et al., 1977; Kannus et al., 1996). The response in the skeletal structure seems to be related to pubertal development, as pre- and peri-pubertal tennis players produce a periosteal expansion (Bass et al., 1999), while endocortical contraction (Bass et al., 1999; Margulies et al., 1986) and trabecular thickening (Ashizawa et al., 1999) is the dominant response in post-pubertal athletes. The response to loading seems also to be site specific, as young tennis players have an endocortical contraction distally at the humerus and endocortical expansion proximally (Bass et al., 1999). But, the surface specific answer is also different in the proximal, mid-diaphyseal and distal region as well as in the antero-posterior and lateral medial direction (Bass et al., 1999; Bass et al., 2002; Haapasalo et al., 2000). Furthermore, the type of activity undertaken may also influence the skeletal structural response.

Physical activity and the skeleton in a long-term perspective

Exercise increases BMD during adolescence but what happens when the activity level is reduced, as in most individuals in the third decade of life? There exist only cross-sectional studies that evaluate BMD in former exercising individuals in a long-term perspective, after age 65 the period when the fragility fractures exponentially rise. These studies consistently infer that no benefits in BMD of a biological significance remain in old former athletes (Karlsson et al., 2000). It seems as if a training-induced anabolic skeletal effect achieved during adolescence has disappeared 4–5 decades after cessation of active career (Bass et al., 1998; Duppe et al., 1996; Karlsson et al., 1993; Karlsson et al., 1996; Karlsson et al., 2000). There exist few prospective long-term studies that support the notion, when reporting a higher BMD loss in retired athletes compared to both still active athletes and controls. In contrast, there exist some reports that there could be some residual benefits in bone structure found in old former athletes (Kontulainen et al., 1999), benefits that hypothetically could influence the risk to sustain fragility fractures.

Physical activity and fractures

Several studies report that women with low physical activity, now or previously, have an increased risk of sustaining a hip fracture (Coupland et al., 1993; Gregg et al., 1998). One study including close to 10 000 elderly women reported that the quintile of women with the lowest activity has a 42% higher hip fracture risk compared to the women in the high quintile (Gregg et al., 1998). Moderate physical activity was associated with a 30% reduction in hip fracture risk compared to physically inactive women, in a study including 8 600 middle-aged and elderly women (Paganini-Hill et al., 1991). When, additionally, a dose–response relation seems to exist, that is an increasing activity level confers a diminishing risk to sustain a hip fracture, this strengthens the view that physical activity does protect against hip fractures. There are considerably fewer studies in women, which investigate the association between physical activity and the risk to sustain a vertebral fracture or other fragility fractures. Several studies support that physical activity is associated with reduced vertebral fracture incidence, when reporting that moderate physical activity is associated with around 30% reduction in the number of vertebral fractures in active compared to inactive women (Gregg et al., 1998; Silman et al., 1997). It is more difficult to draw conclusion as regards physical activity and the fracture risk in men, as there exist fewer studies, most following small cohorts during a short follow-up period. However, the larger studies that do exist support the notion that physical activity, also in men, is associated with reduced risk to sustain hip fractures (Joakimsen et al., 1998; Kujala et al., 2000; Meyer et al., 1993; Paganini-Hill et al., 1991). The conclusion as regards physical activity and vertebral fractures and other fragility fractures in men is even more unclear and there even exist reports that infer that those men with the highest activity level also have the highest prevalence of vertebral fractures (Silman et al., 1997).

If individuals, formerly physically active but now sedentary, have a reduced fracture risk is debated, former athletes seems to have a lower fracture risk compared to controls, 9% compared to 12% and the proportion of individuals with fragility fractures are only half than among the controls, 2% compared to 4% (Karlsson et al., 2002). The reason for the lower fracture risk is unclear, but both exercise-reduced residual benefits in bone structure and neuro-muscular function as well as a genetic selection bias among the athletes may explain the results. Whether former activity on a low level reduces the fracture incidence in old ages is unclear.

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