Exercise in the Prevention of Osteoporosis

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Osteoporosis is a weakening of the skeleton due to a decrease in mineral content and a change in bone architecture. It continues to be an incurable, debilitating, and painful condition of the elderly, especially women. The cost to the United States is $10–20 billion dollars a year, and is likely to increase as more persons live to advanced old age.

Bone loss occurs throughout adult life. Bones are continually being remodeled, and the slight inefficiencies of bone turnover result in a gradual loss of mineral, especially from trabecular bone. Persons who begin their adult life with a large bone mass may never suffer from osteoporosis because their bone density will not fall below the fracture threshold. The first step in preventing osteoporosis is ensuring that children and adolescents acquire the largest possible bone mass consistent with their genetic possibilities. High peak bone mass is mainly an inherited trait, but the intake of calcium and other nutrients, endocrine status, and exercise habits also affect bone acquisition. Some 60% of peak bone mass is acquired during adolescence, making this a period of vulnerability to low calcium intake and endocrine changes that impair adequate development. As physical activity and diet can usually be modified at will, these interventions during adolescence, the adult years, and old age have been the subject of much research.

Physical activity covers a very broad range. Persons can be almost completely inactive (e.g., patients with severe arthritis, or with neuromuscular diseases) or so athletic that they require more than 5,000 kcal/day to maintain body weight. Clearly, bone density is different for these two extremes. Most persons in developed countries lead quite sedentary lives, spending nearly all of their time lying down, sitting, standing, or walking slowly. As they age, their physical activity declines further. What advice should they get about exercise that will prevent osteoporosis? From a public health point of view, we need to address the type and intensity of physical exercise that is appropriate for gaining and maintaining optimum bone mass throughout life. More recently, scientists are inquiring about the interactions between exercise and other treatments, such as increased calcium intake or hormone treatment, to determine their combined effect on bone health.
In the last century, Wolff observed that bone was shaped according to the forces it sustained (1). If you compress or pull a bone to apply stress (force/cross-section), the bone undergoes strain (deformation/length). The trabecular density of a bone is the result of the sum of all loading events over time. Studies in experimental animals show that stress increases bone mineral, and that both the rate and total load have an effect. Intermittent stress is more effective than constant stress, but the total load applied is more important than the frequency of stress. The molecular basis for changes in bone architecture due to exercise is not known, nor is it possible to examine bone architecture in vivo and non-invasively. Studies in humans are based on measurements of bone mineral density, which predicts most but not all of the variability in bone strength.

The mechanism for translating stress/strain into increased bone mass is not well understood. One mechanism could be a local resistance to the resorbing effects of circulating parathyroid hormone, but the mediator is not known. As stress/strain acts locally, it has been suggested that the deformation of the elastic molecules in the matrix surrounding osteoblasts and osteoclasts imparts a message. Lanyon and his colleagues have suggested that proteoglycans in the bone may act as a messenger. These are highly charged molecules that can alter the electrochemical environment as they become more aligned. Loading applied as compression or torsion increases the alignment of proteoglycans in bone, to an extent that depends on the amount of applied strain (2). The increased orientation persists for some 48 hours, providing a local memory of loading to the system.

A fundamental cause of stress/strain on the skeleton is the weight of the body while standing, walking, or carrying out more vigorous activities. Obesity, with all its disadvantages, is actually protective of bone health. Hip fractures are more frequent among persons with lower body mass index (weight/height²), lower body fat, and lower muscle mass (3). Persons who are overweight due to a large muscle mass or to obesity have denser and larger bones (4). In adolescent girls, body mass index is the best predictor of bone mineral density of the whole body (5). Taking away the stimulus of weight, such as during bed rest or microgravity, rapidly produces bone loss, in addition to other deconditioning effects (6). Weight-bearing, even if it is sporadic and not associated with exercise, can prevent the massive bone loss produced by bed rest (7). An hour of quiet standing every day in men otherwise confined to bed proved sufficient to conserve bone mineral (7). During convalescence from illness, or in severely deconditioned frail elderly people, rehabilitation activities that promote sitting, standing, and some walking may prevent accelerated bone loss.

Activities that provide intermittent but powerful compressive loads have a marked effect. This can be seen in the difference in radius width and mineral density between the dominant and non-dominant arm of tennis players (8). Activities that are not weight-bearing may also increase bone density by increasing the size, strength, and activity of muscles attached to bones. An analysis of cadavers found a linear relationship between the weight of the psoas muscles and the ash content of the vertebrae.
Similarly, there is a linear relationship between isometric strength of the back extensors and mineral density of the lumbar spine in older women (10). Although some studies have found increased bone mineral density of the spine in swimmers, the activities that most benefit bone health involve the load of body weight or extraneous weights.

**EXERCISE AND THE ACCUMULATION OF PEAK BONE MASS**

Exercise promotes linear growth in well-fed children (11), and therefore stimulates bone growth. This could be due to hormonal as well as paracrine or electrochemical effects on the skeleton.

Cross-sectional comparisons of sedentary or active young adults show differences of 10% to 40% in the bone density of various sites. Exercises that emphasize load may provide greater stress to the bones. A comparison of runners (high frequency, moderate load on the skeleton), weight-trained men (low frequency, very high load on the skeleton), and circuit-trained men (moderate frequency, high load on the skeleton) showed the highest values for the circuit-trained men, followed by the weight-trained group, and last the aerobically trained group (12). Exercise that promotes muscle growth and strength may favor increased bone density. Muscle strength is an independent predictor of bone mineral density, accounting for 15% to 20% of the variance in bone density of young women (13).

In college-age women, there is a linear relationship between the energy expended in exercise and vertebral bone mineral density in young normally menstruating women (14).

Exercise can affect bone health indirectly, through its effects on food intake and endocrine status. Young women gymnasts, dancers, runners, and ice skaters are encouraged to maintain a very lean physique. There are two consequences of this desire to lose weight. First, food intake is reduced, thus lowering the intake of calcium and other nutrients at a time of rapid skeletal growth. Second, the young woman's endocrine status may adapt, with a decline in reproductive hormones. There may be a delay in menarche, or a subsequent loss of menstrual cycles (amenorrhea). In women who begin a rigorous training program, amenorrhea is more likely if the diet is curtailed (15). Not all athletic women experience amenorrhea: it is more likely in those who are younger, vegetarian, thin, consume insufficient energy, and who have some sort of eating disorder (16). The loss of estrogen has dramatic effects on bone. Amenorrheic athletes have lower mineral density of the spine and the whole skeleton, compared to normally menstruating athletes or sedentary controls (Table 1). Bone strength is reduced, as shown by the greater incidence of stress fractures (20) and scoliosis (21). Studies of amenorrheic athletes in their twenties have shown that with a reduction in training intensity, menstrual cycles resume and there is an increase in vertebral bone mineral density (22). However, in young athletes, skeletal health is affected by poor bone accretion as well as by bone loss. In anorectic young girls, recovery of bone mass is a function of weight gain as well as recovery of normal
TABLE 1. Lumbar bone mineral density in active young women: effects of amenorrhea

<table>
<thead>
<tr>
<th>Reference</th>
<th>% of value for sedentary, normally menstruating women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Amenorrheic</td>
</tr>
<tr>
<td>(17)</td>
<td>89</td>
</tr>
<tr>
<td>(18)</td>
<td>91</td>
</tr>
<tr>
<td>(19)</td>
<td>85</td>
</tr>
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menstrual status (23). Young women training for competition, who want to maintain low weight, may not accept a reduction in exercise activities or an increase in energy intake, in order to improve bone health. Increasing calcium intake alone will not improve their bone health (5), and endocrine treatments may be undesirable in young girls who have not yet reached adult height and body composition. The effects of strenuous training on endocrine status and bone health have not been as well studied in men.

Habitual physical exercise combined with a good diet is likely to favor bone health. When both calcium intake and exercise habits were examined in young women with normal menstrual cycles, active women consuming more than 800 mg/day of calcium had the highest lumbar bone mineral density, whereas sedentary women consuming less than 800 mg/day had the lowest values (14). These relationships may be less important in men, whose calcium intake is substantially higher than for women.

The problem with cross-sectional studies is that persons who already have superior muscles, bones, and motor coordination are more likely to become athletes. Intervention studies show less dramatic effects of exercise and often illustrate some of the disadvantages of training, such as injuries. In a 14-week study of a group of Israeli men undergoing military service, examined before and after extremely rigorous training, 40% of the men failed to complete the study, mostly because of injuries (24). They had to march, jog, and run, with and without heavy backpacks, for 8 hours a day, 6 days a week. Those completing the program showed an 11% increase in the bone mineral density of the left tibia, which is a remarkably high response for such a brief intervention. However, had these men been volunteers in an exercise program rather than army conscripts, another effect would have been a substantial dropout rate from the program.

EXERCISE AND THE PRESERVATION OF BONE

Many attempts have been made to slow the loss of bone that occurs in adult life, especially during and immediately after menopause. Estrogen replacement therapy has proved to be an effective treatment not only for delaying osteoporosis in women but also for preventing cardiovascular disease. Changes in diet and exercise also help preserve bone mass, and are worth pursuing because they improve health and well-being in a variety of ways.
TABLE 2. Effects of weight-bearing exercise on the change in vertebral mineral density of women: longitudinal studies of 9 to 12 months

<table>
<thead>
<tr>
<th>Women</th>
<th>Reference</th>
<th>Exercise (%)</th>
<th>No exercise (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postmenopausal</td>
<td>(28)</td>
<td>+2.5</td>
<td>-2.4</td>
</tr>
<tr>
<td></td>
<td>(29)</td>
<td>+3.5</td>
<td>-2.7</td>
</tr>
<tr>
<td></td>
<td>(30)</td>
<td>+4.9</td>
<td>-0.8</td>
</tr>
<tr>
<td></td>
<td>(31)</td>
<td>+1.2</td>
<td>-6.0</td>
</tr>
<tr>
<td></td>
<td>(32)</td>
<td>-5.6</td>
<td>-4.0</td>
</tr>
<tr>
<td>Premenopausal</td>
<td>(33)*</td>
<td>+0.8</td>
<td>-0.5</td>
</tr>
</tbody>
</table>

*Heavy resistance training.

Middle-aged men who have been active for 25 years or more have a higher bone mineral density than sedentary men (25). Women who take up running after menopause may provide divergent stimuli to their bones: increased stress/strain while running, but a decreased effect of mass if they lose body weight, and a lower capacity for producing bone-conserving steroidal hormones in their adipose tissue. Lumbar bone mineral density in lean middle-aged women runners was not different from the values for sedentary and heavier women (26).

Exercise intervention studies are effective. In 63-year-old women who had been losing 2% to 3% of bone mineral per year from the radius, a year of loading exercises for the arms produced a 3.8% gain in bone mineral (27). Weight-bearing aerobic exercise used in several studies has been shown, with few exceptions, to increase lumbar bone mineral density and the mineral density of other stressed sites. Some of these interventions in women are summarized in Table 2. The single study that showed no change with exercise involved less vigorous and prolonged activity (i.e., walking for a total of 120 minutes per week), no calcium supplements, and no estrogen treatment (32). In the study showing the most marked response, the women combined walking with some weight training, some were on estrogen therapy, and all were provided with a substantial calcium supplement of 1,500 mg/day (26). The benefits of exercise training are not permanent. After resuming sedentary habits, bone mineral density returns to baseline values (30).

The indirect effects of beginning an exercise program in older persons can help prevent some of the consequences of bone loss. Falling is usually the cause of bone fracture. Older persons who walk more than 3 miles (4.8 km) per week are less likely to suffer fractures (34). A review of six studies showed that muscle weakness, especially of the hip extensors, was a major risk factor for falling (35). Habitual exercise increases endurance, strength, coordination, and balance and thus improves the way persons negotiate hazards and take corrective action when they stumble or trip. In the elderly, the effort of walking, climbing stairs, and other everyday activities that help maintain bone mass becomes less strenuous after a certain amount of training. Physical activity, even if it is very mild, takes time away from hours of bone-losing activities such as lying down, lounging, or sitting. Physical activity demands energy,
and active persons are likely to eat more food, making it easier for them to satisfy their needs for calcium, vitamin D, zinc, magnesium, and other nutrients that are important for the health of their musculoskeletal system.

CONCLUSION

Physical activity favors increased bone mineral density, especially if the diet provides adequate calcium and other nutrients. During growth, the most important way to improve bone health may be to increase the intake of dairy products. During and after menopause, estrogen treatment may be the most effective way to prevent bone loss. However, physical activity also has a role throughout life. In older women, exercise can prevent bone loss from certain sites. The changes are modest but significant. They disappear if the activity is not sustained. Excessive physical activity can be harmful; it can cause stress fractures and, in susceptible young women, lead to amenorrhea. Without normal menstrual cycles, thin and active young women develop osteopenia, increasing their risk of osteoporosis in old age. Habitual exercise is useful for ameliorating many conditions and diseases of older persons, such as diabetes mellitus or cardiovascular disease. It should also be advocated for improving bone health.

REFERENCES

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DISCUSSION

This chapter was part of the Round-Table Conference on prevention of osteoporosis. Please refer to the round-table discussion, page 187.