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The peak bone mass concept

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SUMMARY *Peak bone mass (PBM), i.e. the bone mass developed after growth in early adult life, is a major determinant for bone mass in the senescence, resp. for the risk of osteoporosis. Individual differences among young adults are so important that a person with a high PBM has a relatively low risk for osteoporosis, even if its annual bone loss is fast. PBM is conditioned by genetic, hormonal, and nutritional factors, and by physical activity, the latter two offering possible impact for preventive measures. Preservation of PBM through the early, resp. premenopausal life, can be favoured by adequate nutrition and physical activity, both together being capable of postponing the appearance of osteopororis by several years. But various parts of the skeleton decrease constantly through life, starting already in the twenties; even when absorptiometry shows no loss of bone density until menopause, resp. during early adult life, there is a constant decrease of the mechanical properties of the bone with age. Therefore, the development of an optimal PBM is probably more effective in preventing osteoporosis than the measures for preservation of bone.*

Key words: Peak Bone Mass, Influencing Factors, Preservation.

Introduction

Bone mass increases during growth, especially during the pubertal sprint of growth, and somewhat during early adolescent life. Thereafter, bone mass remains in the average constant for one to two decades, and finally declines progressively. Patterns of age-related bone gain and diminution differ between sexes and measuring sites (1). The peak bone mass, i.e. the highest value of quantity and of density of bone reached in life, seems to be obtained in males at about 25 years, while women still gain peripheral bone later on (1). For the lumbar spine, bone loss might already start at about age 25 (2, 1). Although there is some controversy about the age at which bone mass starts to decrease, bone mass obtained in the early adult life represents the peak value. This notion is of great importance, because at any age bone density in any given elderly individual is a function of his or her peak bone mass in young adult life and of the amount of bone loss since that time (3). From that it can be concluded, that the risk of fracture is at any age a function of the peak bone mass, because bone density, resp. bone mineral content, is indeed inversely correlated with the incidence of fractures of the spine (4,5), or of the forearm (6). For instance, in a study of 557 postmenopausal women, it has been shown that the mean forearm bone density was significantly lower in the subjects who had experienced a forearm fracture, than in those who had not experienced previous forearm fractures (6). A correlation was found between the incidence of vertebral fractures and vertebral density,

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as measured in a study on 189 postmenopausal women (7). In another study, the incidence of vertebral and nonvertebral fractures was correlated with bone mineral content of the forearm (5).

The variance of the peak bone mass in young adults is very high and might be of ± 20 % in the vertebrae (8). Compared to this, the subsequent rate of bone loss of 1-2 % per year is small, as pointed out by Nordin (3). It follows that for many years after the midlife, resp. menopausal onset of bone loss, the initial value i.e. the peak bone mass in the main determinant of bone density. A woman presenting a relative fast bone loss in the postmenopausal period will reach the osteoporotic, clinical threshold very late in life, if ever, if her peak bone mass was high. On the other hand, even a small loss of bone will soon lead to fractures, if the initial value, i.e. the peak bone mass, was already low. Remembering the small increments of bone density that can be obtained by the various treatments of osteoporosis currently available, development of a relatively high peak bone mass in early adult life becomes an important goal for the prevention of osteoporosis, as far as it can be voluntarily influenced.

Factors influencing the peak bone mass

Several factors condition peak bone mass: genetic, hormonal and nutritional factors, and physical activity during growth and early adult life.

Hormonal factors

In children from 6 to 14 years old, bone mineral content increases by about 8.5% each year (10). Bone density remains constant (9). Skeletal age, as evaluated by the classical radiological method of Greulich-Pyle, is a poor predictor of bone mineral content; height and weight are better predictors (10). Bone density increases markedly during puberty (11,9). In the age groups up to 15 years and above 21 years, men exhibit a much larger increase in the total area of the radius than women. It cannot be determined whether this relative increase of area in the male takes place during growth or after the longitudinal growth has ceased. The increase of bone density between puberty and adulthood is attributed to the increased cortical area (9).

The gonadal hormones are the true initiators of the short-lived growth spurt, and of the prolonged acceleration of bone mineralization (12). Hypogonadism is accompanied by low skeletal mass (12). Androgens are of special importance for the constitution of a normal bone mass. There is a positive relation between plasma testosterone levels and the percent of cortical area in patients who suffer from Klinefelter syndrome, traumatic castration and hypogonadotropic hypogonadism (13). In these patients, androgen therapy increases the relative osteoid volume, total osteoid surface and bone mineralization. The positive histological response to hormonal replacement therapy confirms the importance of androgens, for bone modeling and remodeling (14). The sex difference does not concern exclusively bone mass, but also muscle mass which in turn favors development and preservation of bone mass. When bone mass is corrected by lean body mass, as it was done by Thomsen et al. in 574 healthy subjects (15), the difference of bone mineral content between male and female up to age 50, which was almost 30%, is reduced to less than 10%. Other hormonal determinants which can influence peak bone mass are late menarche and early menopause, which in some studies are associated with a lower average of bone mineral content (16). Intercurrent diseases and treatment with corticosteroids during childhood interfere with normal growth (17), and might have irreversibly negative effect on the final peak bone mass.

Furthermore, a normal development is necessary for constitution of a normal peak bone mass. Measurement of bone mineral content by quantitative computed spinal tomography in children with developmental de-

lay, e.g. because of hydrocephalus, showed values which were about 30% lower than in normal children (18).

Physical activity

Physical activity is another distinct factor influencing peak bone mass, although its importance is difficult to prove. At least, in young men engaged in regular and vigorous exercise programs, it could be demonstrated that bone mineral content is greater than in controls (19,20). There was even a significant difference in spinal trabecular bone density based on the type of physical activity, the combination of weight bearing and aerobic form of exercise being associated with the highest values (20).

Nutrition

It is common knowledge that nutritional factors during growth and puberty play an important role for the development of the skeleton, particularly the calcium intake, although direct evidence derived from longitudinal studies in children is missing. Matkovic's study (21) on bone density of two populations differing in their calcium intake, has suggested such a role for calcium intake on maximal cortical bone mass. In another study, bone mineral content was higher in subjects whose calcium intake was over 1000 mg per day than in subjects who took only 500 mg per day or less (22). While such relationships are questioned by several authors, it seems that the elimination of the effects of physical activity might disclose a significant role of the calcium intake (23). Kanders et al even suggest that the average age of onset of osteoporotic fractures would be expected to be delayed approximately 10 years by modification of diet and activity (23).

These observations mainly concern the importance of the calcium intake in prevention of bone loss, and not in achieving a high peak mass. On this behalf, the study of Sandler et al (24) seems of special interest; it shows relatively high bone density in postmenopausal women whose self reported milk consumption during childhood (and in a lesser degree during adolescence) was regular.

In addition to nutritional and hormonal factors during growth and pubertal development, nutritional factors during early adult life still can determine a modification of bone mass. For example, the combination of high calcium intake and physical activity was estimated for being responsible for an additional 2-4% of bone mass (25). It was also found that in a group of women with longterm lactation, bone mass was lower than in a comparable group of women with short term lactation, even if the latter consumed the recommended dietary allowance of calcium (26). On the other side, regular intake of calcium during lactation in adolescent mothers prevented the bone loss observed in controls (36). These studies demonstrate the importance of nutritional adequacy for maintaining peak bone mass through early adult life. The opposite situation, the crack down of the nutritional and hormonal status, as observed in anorexia nervosa, is inevitably accompanied by low bone mass (27,28,29).

Genetic factors

Although the importance of the nutritional factors is well recognized, perhaps because they offer an impact for preventive measures, it must be admitted that genetic factors are essential, perhaps even predominant. First, it should be remembered that the racial differences are quantitatively important, black people having for this reason, less osteoporosis (30). The larger muscle mass is in part responsible for this difference (30). Second, it was already mentioned, that within the same cohort differences of about 40% were found in the peak bone mass of young adults (8) which could hardly be explained by differences in nutritional conditions only. Measurements of forearm bone density in 71 juvenile

and 80 adult twinpairs revealed significantly smaller variances of intrapair differences in monzygotic twins than in dizygotic twins indicating that these traits have significant genetic determinants. These intrapair differences increased with age, suggesting that genetic environmental interaction also contributes to the observed variation in bone mass (31). Studies of metacarpal cortical thickness in elderly twins confirmed the role of inheritance (32). In another study, which included spinal measurements, a significant genetic determinant was found for the bone mass of the radius in adults, and for the spinal bone mass in the age group younger than 25 years (33). The absence of a conclusive demonstration of a genetic determinant in adult twins for the spines in this study suggests that environmental factors may play a more dominant role in the diminution of axial bone during adult life. A study of the relationship of bone mass between premenopausal mothers and adolescent daughters again suggests strong influences of both familial environment such as diet, and genetic inheritance (34). It should be mentioned that the daughters had only 91-95% of their mothers' peak bone mass, which lets us presume that an additional amount of 5% or more may be added after the age of 18 years.

Preservation of the peak bone mass

Peak bone mass is conditioned by genetic and environmental factors, and represents probably the predominant determinant for bone mass in adult life. This predominance is explained by the fact that the differences in the rate of bone loss are in general smaller than the differences in the initial bone mass. During early adult life, resp. during premenopausal life, the peak bone mass seems to be mainly conserved, although there is conflicting data on this matter. For instance, while some studies showed progressive bone loss during the early adult and premenopausal life in the spine (1,35,2), the forearm (4) and the femoral neck (37) others do not show a decline with age in young adults and premenopausal women (38,39,8), at least not in the spine (37). For vertebral bone density, separated regression lines for pre-, peri-, and postmenopausal groups were found (8,39). In addition, not all the sites have the same rate of decline. The femoral shaft, for example, is nearly unaltered until the 6th decade (37). In the male, the diminution of the radial bone mineral content is gradual (2% to 3.4% per decade) with age, but vertebral trabecular content falls more rapidly (12% per decade) (41). Therefore, the process of bone loss is not homogeneous and the trabecular and cortical bone are independently modulated. K. Thomsen et al (15) have calculated a lean body mass index in 574 healthy women and men. When bone mineral content was corrected by this index, there was almost no bone loss in men with age, and only after the menopause in women. Mazess et al even found in 280 women between 20 and 39 years that density values at all bone sites did not differ more than 2% for any single factor such as age, oestrogens, calcium intake or physical activity (25).

But even if the bone mass remains constant during early adult life, the biomechanical competence of vertebral trabecular bone declines with age, since it depends not only on the mass but also on the continuity of the trabecular lattice. The maximum stress, the maximum stiffness, the energy absorption capacity decrease and compressibility increases with age, independently of the bone quantity as measured by absorptiometry (40,42).

Conclusion

In conclusion, peak bone mass is a quantitatively predominant determinant for bone mass in advanced age, because the higher the bone mineral content is in early life, the later begins the osteoporotic disease for the same bone loss. Peak bone mass is an essential concept for evaluation of the risk of osteoporo-

sis, of preventive measures and of the clinical effectiveness of therapeutical interventions. It depends on genetic and hormonal factors, on nutrition and physical activity, the two latter offering major impact for prevention of osteoporosis.

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