The effects of nutrition and physical activity on bone development in male adolescents

Muhsin Doğan, Orhan Derman, Nuray Öksüz-Kanbur, Sinem Akgül, Tezer Kutluk
Unit of Adolescent Medicine, Department of Pediatrics, Hacettepe University Faculty of Medicine, Ankara, Turkey

SUMMARY: Doğan M, Derman O, Öksüz-Kanbur N, Akgül S, Kutluk T. The effects of nutrition and physical activity on bone development in male adolescents. Turk J Pediatr 2009; 51: 545-550.

Peak bone mass (PBM) is defined as the highest bone mineral content (BMC) reached in any period of a person's life. The bone mass once gained at the peak begins to decline and continues to do so until the end of life. The aim of this study was to evaluate the relationship of nutrition and physical activity on bone mineralization during the adolescent period. The study took place at Hacettepe University İhsan Doğramacı Children's Hospital Adolescent Unit. One hundred fourteen healthy male adolescents applying for different reasons, with ages ranging between 11.1 and 16.5 years, participated in the study. When all adolescents were evaluated, no statistical relationship between the daily calcium intake, BMC and bone mineral density (BMD) was obtained. However, a positive statistical relationship was found for those participants in Tanner stage I. This result is in support of previous studies stating the importance of calcium intake and bone mineralization in the prepubertal stage, suggested by our findings, which yielded a positive correlation only in the prepubertal stage. One of the reasons for the same effect not being observed in puberty is thought to be due to the hormonal changes and active role of sex steroids. This shows how critical the prepubertal period is for future bone health. During this critical period of prepuberty, the significance of nutritions and physical activity is evident.

Key words: nutrition, physical activity, bone development, male adolescents.

In a short period of time, the anthropometric measurements of adolescents reach those of adults, and there exists a distinct increase in bone, fat and muscle mass. Peak bone mass (PBM) is defined as the highest bone mineral content (BMC) reached in any period of a person's life[1]. The bone mass once gained at the peak begins to decline and continues to do so until the end of life[2]. Therefore, for lifelong bone health, the development of PBM, bone mass protection and bone loss prevention should all be ensured. The time PBM is reached is not exactly known; however, studies show that at least 90% of it is reached in the second decade of life and 25% of this occurs in the two-year period wherein the growth spurt occurs[3]. This again shows that childhood, particularly adolescence, is an important period for bone health. It has been shown that a significant part of the risk of osteoporosis occurs due to insufficient bone mineral development during this period and that senile osteoporosis is actually a pediatric disease[4]. One of the most important factors affecting PBM is genetic potential, but it has been shown that in addition to the genetic potential, certain environmental factors such as nourishment and physical activity can also affect bone mineralization[5]. The aim of this study was to evaluate the association of nutrition and physical activity on bone mineralization during the adolescent period.

Material and Methods

The study took place at Hacettepe University İhsan Doğramacı Children's Hospital Adolescent Unit between May 2005-November 2005. One hundred fourteen healthy male adolescents applying for different reasons, with ages ranging between 11.1 and 16.5 years, participated in

the study. Medical information was gained from the adolescents and parents including history of illness, medications taken, and smoking or alcohol habits.

Those participants with a history of chronic illness and continuous use of medication, alcohol or cigarettes were not included in the study. Using the height and weight of participants at application, weight and height percentiles, ideal weight, ideal weight ratios, body mass index (BMI), and weight and height standard deviations were calculated. A full physical examination was administered to all adolescents and the pubertal staging was evaluated according to the Tanner method[6]. Based on the information collected from the daily nutrition consumption of the adolescents, using the nutrition information system program (BeBIS 4), the daily calcium consumption was calculated. Every adolescent's sports activities and weekly metabolic equivalence (MET) consumed was calculated[7]. MET (kcal/kg/hr) is defined as the kilocalories per kilogram of body weight burned per hour[8]. The weekly MET value (kcal/kg/week) was calculated according to the (frequency x time x intensity) formula[7]. Bone mineral density (BMD) was measured in all participants between the L1-4 vertebrae using the dual energy X-ray absorptiometer (DEXA-Hologic QDR 4500) method. BMC value as "gram" and BMD as "g/cm^2" were obtained by being divided into the BMC measurement value. Biochemically, serum calcium, inorganic phosphorus, alkaline phosphatase, and osteocalcin were measured.

The parathormone and $25(OH)D_3$ vitamin levels were also screened for adolescents found to have osteoporosis. Only those participants with normal levels of these two hormones were included in the study. To determine bone age, left wrist direct radiographic imaging was obtained, and bone age was determined using bone age atlases (Greulich-Pyle Atlas) arranged according to age and sex[9]. All adolescents and their families were given information regarding the study and both verbal and written consent was obtained.

Statistical analyses were conducted using the Statistical Package for Social Science (SPSS) 11.5 for Windows XP package program, in which descriptive data was obtained. In addition, Spearman correlational analyses were conducted to analyze the linear relationship between the ordinal variables, and Mann-Whitney U test was used to compare the means of two independent groups.

Results

The mean age of the 114 male adolescents was 13.5±1.2 years (11.1-16.5). We found a significant correlation between age, BMC and BMD ($p<0.001$). The demographic characteristics of participants, their anthropometric measurements, laboratory findings, daily calcium intake, weekly MET and bone mineral measurement values are shown in Table I. When we evaluated the relationship between pubertal stage, BMC and BMD, we found a positive correlation, similar to the relationship between age, height, and weight ($p<0.001$). Changes in BMC and BMD according to pubertal stage are shown in Table II. The mean bone age of adolescents was found to be 13.34±1.97 years (10-18 years). A positive correlation was again obtained between bone age, BMC and BMD ($p<0.001$). When we evaluated the factors affecting BMC and BMD, we found that age, bone age, weight, height, and pubertal stage all had a distinctive positive correlation ($p<0.001$ for all variables).

The daily calcium intake consumed by adolescents was 735.30±337.64 mg/day (162.18-1824.89). The recommended daily intake for this age group is 1200-1500 mg, and only 12 (10.5%) adolescents in this study received this amount. Two (1.7%) adolescents were found to be receiving over 1500 mg. The remaining 100 (87.8%) were receiving less than the recommended daily allowance. After all participants in this study were tested and evaluated, we could calculate the relationship and found no significant statistical correlation between the daily calcium intake (calculated from the nutrition consumption information given) and BMC and BMD values obtained (For BMC $p=0.397$, for BMD $p=0.496$). However, when the adolescents were divided according to pubertal stages, the relationship between daily calcium intake and BMC and BMD was re-evaluated and found to yield a positive statistical relationship for those participants in Tanner stage I (BMC, $p=0.040$; BMD, $p=0.006$). A similar relationship, however, was not obtained for other pubertal stages. The MET

Table I. Demographic Characteristics, Anthropometric Measurements, Laboratory Findings, Daily Calcium Intake, Weekly MET and Bone Mineral Measurement Values of Adolescents

Characteristic	Mean ± SD	Distribution
Age (years)	13.52 ± 1.25	11.16-16.50
Bone age (years)	13.34 ± 1.97	10-18
Weight (kg)	50.70 ± 12.85	30.5-88
Height (cm)	159.11 ± 11.65	136-183
Serum Ca (mg/dl)	10.32 ± 0.32	9.3-11.1
Serum P (mg/dl)	4.99 ± 0.60	3.3-6.4
ALP (U/L)	263.21 ± 81.65	92-521
Osteocalcin (ng/ml)	20.49 ± 7.38	2-35.6
BMC (g)	37.70 ± 11.60	21.36-69.97
BMD (g/cm^2)	0.70 ± 0.12	0.49-1.07
BMD z-score	(-1.61) ± 0.98	(-3.65)-(+1.04)
Daily Ca (mg/day)	735.30 ± 337.64	162.18 ± 1824.89
Weekly MET (kcal/kg/week)	64.26 ± 52.17	0-196

MET: Metabolic equivalence consumed. Ca: Calcium. P: Phosphorus. ALP: Alkaline phosphatase. BMC: Bone mineral content. BMD: Bone mineral density.

Table II. Changes in Bone Mineral Content (BMC) (g) and Bone Mineral Density (BMD) (g/cm^2) According to Pubertal Stage

Pubertal Stage	n (%)	BMC Mean ± SD	BMD Mean ± SD
Tanner stage I	21 (18.4)	28.02 ± 4.71	0.60 ± 0.07
Tanner stage II	20 (17.5)	28.80 ± 4.09	0.61 ± 0.06
Tanner stage III	22 (19.3)	32.16 ± 5.53	0.64 ± 0.07
Tanner stage IV	25 (21.9)	42.83 ± 7.43	0.76 ± 0.10
Tanner stage V	26 (22.8)	52.10 ± 9.39	0.84 ± 0.10

value, which shows the amount of calories used after physical activity per week and aims to show the physical activity levels of adolescents, and BMC and BMD were evaluated; when all adolescents were considered, no statistical relationship was found (for BMC, p=0.144; for BMD, p=0.194). When the adolescents were divided according to pubertal stages and the relationship between MET, BMC and BMD was re-evaluated, similarly, a positive statistical relationship was found for those participants in Tanner stage I; however, this time between MET and BMC alone (BMC, p=0.036; BMD, p=0.963). A similar relationship was not found for any of the other pubertal stages.

Discussion

Osteoporosis is the most common disease to affect bone metabolism and it continues to be an important public health concern, particularly for adults. The most important attribute of the disease is the increased risk of bone fractures. The physical, psychosocial and financial burdens make it a destructive illness; however, with early precautions it can be prevented. The critical period is childhood and adolescence when bone mass is stored[10]. Many studies show that risk of osteoporosis can be explained by insufficient bone mineral development in childhood and that senile osteoporosis is really a pediatric illness[4]. The most important factor affecting bone health is thought to be PBM, the majority of which is gained throughout childhood, particularly adolescence. As stated earlier, PBM is the highest BMC reached at any period during a person's life[1]. Many factors have been shown to affect the PBM, the most important of which is genetic potential[11-15]. However, it has also been shown that full genetic capacity can only be reached with sufficient nutrition, physical activity, endocrine functioning, and other factors affecting lifestyle[3,5]. Nutrition, lifestyle and physical activity have been shown to be the most important environmental factors affecting PBM[5,16]. Our study examined the effects of nutrition and physical activity on bone mineralization. Since sex is a known

factor that affects bone mineralization, in order to minimize the effective factors and thus obtain more accurate results of the effects of nutrition and physical activity, only male adolescents were included into the study. Hasanoglu's study[17] is the first reporting normal values of BMD measured by DEXA in Turkish children. According to their study, BMD increased with age in children of both sexes. The increase was steeper at the time of puberty. There were no significant differences between boys and girls until the age of 10. After the age of 10, lumbar BMD was higher in girls than in boys, probably because of the earlier onset of puberty in females. Nutrition is one of the most important factors affecting bone mineralization during childhood and adolescence. Calcium is the most important nutritional factor affecting bone density in this age group. During adolescence, bone storage of calcium increases, thus calcium necessity increases. For sufficient bone mineralization and an optimal peak of bone mass to occur, it is important to receive the recommended daily allowance of calcium. Studies show that when additional calcium supplementation is received in the prepubertal period, the PBM development enhances and osteoporosis is likely to be prevented. A study performed in subjects aged between 6-14 years with a three-year follow-up showed that prepubertal calcium supplementation increased BMD; however, the same effect was not seen when given during puberty[18]. Another study on 94 adolescent girls with a mean age of 11.9±0.5 years showed that an increase in BMD in the whole body and lumbar vertebrae occurred with an additional 18-month calcium supplementation, and it was stated that calcium supplementation in this age group can protect against fractures caused due to osteoporosis[19]. Another research stressing the importance of prepubertal calcium supplementation showed that prepubertal calcium supplementation improved bone mass and density, calling for the continuation of high bone density calcium supplementation[20]. Our study examined the relationship between calcium intake, calculated from the daily nutrition consumption, and bone mineralization. When all adolescents were evaluated, no statistical relationship between the daily calcium intake, BMC and BMD was obtained. However, a positive statistical relationship was found for those participants in Tanner stage I. This result is in support of previous studies stating the importance of calcium intake and bone mineralization in the prepubertal stage, as suggested by our findings yielding a positive correlation only in the prepubertal stage. One of the reasons for the same effect not being observed in puberty is thought to be due to the hormonal changes and the active role of sex steroids. The significant effects of sex steroids during the growth spurt in puberty are well documented[21]. In both males and females, androgen receptors are found on the growth plaques of osteoblasts and are responsible for the anabolic effects of testosterone on bones. However, in terms of skeleton development and mineralization, estrogen plays a more significant role. Despite sufficient androgen levels, aromatase enzyme deficiency or estrogen receptor defects results in osteoporosis[1]. Both estrogen and androgens have been shown to stimulate bone cells through the structuring and proliferation of osteoblastic function signifiers such as alkaline phosphatase (ALP), type 1 collagen, interleukin (IL)-6 and transforming growth factor (TGF)-β[20]. When no other risk factors have been defined, it has been shown that osteopenia is seen in adolescents with hypogonadism[22]. Osteopenia has been reported with diseases causing sex steroid deficiency such as a chromosome disorders like Turner syndrome or anorexia nervosa or other genetic illnesses[22-24]. The effect of sex steroids and other hormones on bone mineralization during puberty is more dominant when compared to calcium intake, and we believe this is why a positive correlation between bone mineralization and calcium intake was not seen in our study. Another reason for this finding may be due to the fact that the amount of calcium intake was calculated via the surveys given to participants, and personal mistakes may be a cause for error. For these calculations, it was very difficult to obtain full objectivity. Additional daily nutritional intakes of the adolescents varied, and this may have also affected the results. Another factor examined in this study for its effects on bone mineralization was physical activity. Many studies have shown a positive relationship between appropriate and regular physical activity and the development of BMI. Recent research stresses the importance of physical activity on

bone and muscle development in addition to sufficient calcium intake[25]. Many studies have shown a positive relationship between physical activity and bone mineralization during puberty and prepubertal stages. However, some of these studies show that this relationship is more evident in the prepubertal stage. A three-year longitudinal study with 90 children between the ages of 6-14 evaluated physical activity by using a questionnaire administered every six months. At the end of the three-year observation, a positive relationship between physical activity and bone mineralization in prepubertal children was shown; the same relationship, however, was not obtained in pubertal children[26]. On the contrary, in a seven-month study, the simple physical activity levels of 87 children and 90 control patients ranging between the ages of 8.7 and 11.7 were evaluated. Results showed an increase in BMC on some areas of the skeleton, particularly in the early pubertal stage, yet not in the prepubertal stage, showing that in females, the best period to obtain the positive effects on bone health is in the early pubertal stage[27]. Another study performed on prepubertal and early pubertal children showed that simple jumping activity causes an increase in BMC in the trochanteric region of the femur[28]. The relationship between the MET value and BMC and BMD was evaluated; when all adolescents were considered, no statistical relationship was found. Similarly, only in prepubertal adolescents was a positive relationship with calcium intake observed; however, this relationship was only found between weekly MET values and BMC. In prepubertal adolescents, a nonsignificant relationship between weekly MET values and BMD was found. This result could be explained with similar reasoning of calcium intake. Weekly MET values could be influenced by personal mistakes, seasonal changes in physical activity, and weekly or daily changes. In line with previous research, the fact that a positive relationship between physical activity and mineralization in the prepubertal stage was found signifies the importance of physical activity in this period. With another perspective in line, the physical and hormonal changes specific to the pubertal period may direct bone mineralization. Based on the results of this study, we propose that sufficient calcium intake in children begins before puberty and continues throughout it. We also recommend that during the prepubertal stage, children should be encouraged to do sports and to make this a habit, toward positive bone development. Furthermore, developing awareness regarding bone health during puberty and closely monitoring child and adolescent entry to puberty and pubertal development are of great importance.

To conclude, the increased bone mineral content and density during puberty is more related to nutrition and physical activity levels than pubertal development. This shows how critical the adolescent period is for future bone health. During such a critical period, the significance of nutrition and physical activity is evident. However, in order for these factors to show their effects, independent of pubertal development, we believe it is necessary to research a wider range of cases throughout puberty.

REFERENCES

1. Soyka LA, Fairfield WP, Klibanski A. Clinical review 117: hormonal determinants and disorders of peak bone mass in children. J Clin Endocrinol Metab 2000; 85: 3951-3963.
2. Ilich JZ, Kerstetter JE. Nutrition in bone health revisited: a story beyond calcium. J Am Coll Nutr 2000; 19: 715-737.
3. Leonard MB, Zemel BS. Current concepts in pediatric bone disease. Pediatr Clin North Am 2002; 49: 143-173.
4. Dent C. Problems in metabolic bone disease. In: Frame B, Parfitt MA, Duncan H, (eds). Clinical Aspects of Metabolic Bone Disease. Amsterdam: Exerpta Medica; 1973: 1-7.
5. Heaney RP, Abrams S, Dawson-Hughes B, et al. Peak bone mass. Osteoporosis Int 2000; 11: 985-1009.
6. Tanner JM. Growth at adolescence. Oxford: Blackwell; 1962: 1-54.
7. Karaca A, Ergen E, Koruç Z. Fiziksel aktivite değerlendirme anketi (FADA) güvenilirlik ve geçerlilik çalışması. Spor Bilimleri Dergisi 2000; 11: 17-28.
8. Ainsworth BE, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc 2000; 32: 498-504.
9. Greulich WW, Pyle SI. Radiographic Atlas of Skeletal Development of the Hand Wrist (2nd ed). Standford: Standford University Press; 1959.
10. Baroncelli GI, Bertelloni S, Sodini F, Saggese G. Osteoporosis in children and adolescents: etiology and management. Paediatr Drugs 2005; 7: 295-323.
11. Rubin K, Schirduan V, Gendreau P, Mendola R, Dalsky G. Predictors of axial and peripheral bone mineral density in healthy children and adolescents, with special attention to the role of puberty. J Pediatr 1993; 123: 863-870.

12. Slemenda CW, Christian JC, Williams CJ, Norton JA, Johnston CC Jr. Genetic determinants of bone mass in adult women: a reevaluation of the twin model and the potential importance of gene interaction on heritability estimates. J Bone Miner Res 1991; 6: 561-567.
13. Pollitzer WS, Anderson JJ. Ethnic and genetic differences in bone mass: a review with a hereditary vs environmental perspective. J Am Coll Nutr 1989; 50: 1244-1259.
14. Hopper JL, Green RM, Nowson CA, et al. Genetic, common environment, and individual specific components of variance for bone mineral density in 10- to 26-year-old females: a twin study. Am J Epidemiol 1998; 147: 17-29.
15. Kelly PJ, Nguyen T, Hopper J, Pocock N, Sambrook P, Eisman J. Changes in axial bone density with age: a twin study. J Bone Miner Res 1993; 8: 11-17.
16. Eastell R, Lambert H. Diet and healthy bones. Calcif Tissue Int 2002; 70: 400-404.
17. Hasanoğlu A, Tümer L, Ezgü FS. Vertebra and femur neck bone mineral density values in healthy Turkish children. Turk J Pediatr 2004; 46: 298-302.
18. Johnston CC, Miller JZ, Slemenda CW, et al. Calcium supplementation and increases in bone mineral density in children. New Engl J Med 1992; 327: 82-87.
19. Lloyd T, Andon MB, Rollings N, et al. Calcium supplementation and bone mineral density in adolescent girls. JAMA 1993; 270: 841-844.
20. Lee WT, Leung SS, Leung DM, et al. Bone mineral acquisition in low calcium intake children following the withdrawal of calcium supplement. Acta Paediatr 1997; 86: 570-576.
21. Heath DA, Shaw NJ. Calcium and bone metabolism. In: Brook CG, Hindmarsch PC, (eds). Clinical Pediatric Endocrinology (4th ed). Oxford: Blackwell Science Press; 2001: 377-389.
22. Bachrach LK. Malnutrition, endocrinopathies and deficits in bone mass acquisition. In: Bonjour JP, Tsang RC (eds). Nutrition and Bone Development. Philadelphia: Lippincott Raven; 1999: 262-277.
23. Soyka LA, Misra M, Frenchman A, et al. Abnormal bone mineral accrual in adolescent girls with anorexia nervosa. J Clin Endocrinol Metab 2002; 87: 4177-4185.
24. Seeman E, Karlsson MK, Duan Y. On exposure to anorexia nervosa, the temporal variation in axial and appendicular skeletal development predisposes to site-specific deficits in bone size and density: a cross-sectional study. J Bone Miner Res 2000; 15: 2259-2265.
25. Bachrach LK. Making an impact on pediatric bone health. J Pediatr 2000; 136: 137-139.
26. Slemenda CW, Reister TK, Hui SL, Miller JZ, Christian JC, Johnston CC Jr. Influences on skeletal mineralization in children and adolescents: evidence for varying effects of sexual maturation and physical activity. J Pediatr 1994; 125: 201-207.
27. Mackelvie KJ, McKay HA, Khan KM, Crocker PR. A school-based exercise intervention augments bone mineral accrual in early pubertal girls. J Pediatr 2001; 139: 501-508.
28. McKay HA, Petit MA, Schutz RW, Prior JC, Barr SI, Khan KM. Augmented trochanteric bone mineral density after modified physical education classes: a randomized school-based exercise intervention study in prepubescent and early pubescent children. J Pediatr 2000; 136: 156-162.