report: Kathryn Bistany

bone health in adolescent athletes: the importance of nutrition

Approximately 60% of adult bone mass is gained during the adolescent years, with the most rapid bone accretion occurring during late childhood and the pubertal growth spurts.[1,2] Peak bone mass (PBM), which is to a great extent genetically determined – although hormonal factors and the age of onset of puberty are important determinants – occurs at the conclusion of growth.[1,3] The key determinants of bone mineral density (BMD) in later life are the amount of peak bone mass achieved and the subsequent rate of bone loss.[3] Together with exercise and hormonal action, nutritional factors play a crucial role in the timely acquisition and maintenance of body bone.[4]

composition of bones

Bone is a living tissue which is constantly undergoing change. It consists of an interwoven meshwork of collagen fibres (the matrix) which are bound together and hardened by calcium phosphate and small traces of magnesium and fluoride. Regulation of the bone matrix is the responsibility of osteoblasts, osteocytes and osteoclasts. Osteoblasts build up new bone and are particularly active during childhood. Osteocytes are mature bone cells that become imbedded into the bone matrix as they mature from osteoblasts. These cells serve as a communication network within the bone. Osteoclasts are responsible for bone resorption. They work in conjunction with the osteoblasts through a "coupling" action. As such, when bone resorption increases, the rate of bone formation also increases.[3]

Two primary types of bone are cortical and trabecular bone. Cortical bone primarily functions to provide structure and protection, whereas trabecular bone has an active metabolic function resulting from its contact with bone marrow, blood vessels, and connective tissue.[3]

nutritional factors that are determinants of normal bone mass

Vitamin D is obtained from sunlight and dietary consumption in the form of Vitamin D3 (cholecalciferol). Vitamin D2 (ergocalciferol), which is produced by irradiating fungi, is much less efficient as a precursor to the biologically active 1.25-dihydroxyvitamin D (calcitriol).[5] Calcitriol is the most biologically active form of vitamin D and increases calcium and phosphorus absorption from the intestine, induces osteoclast maturation for bone remodelling, and promotes calcium deposition in bone and a reduction in parathyroid hormone (PTH).[5,6] The clinical importance of lower levels of PTH in promoting health is inferred as higher levels correlate with increased risk of myocardial infarction, stroke, and hypertension.[5]

Recent studies suggest that hypovitaminosis D is more common in children and adolescents than previously believed, even in notoriously sunny countries.[4,6,7] The consequences of hypovitaminosis D is impaired calcium absorption, increased calcium resorption from bone and a contributory factor in a wide variety of chronic diseases as well as common clinical disorders such as low back pain and generalised musculoskeletal pain.[8,9] The benefit of early supplementation administered in the first year of life is associated with significantly increased BMD.[1]

Vitamin K appears to be involved in several mechanisms essential for bone metabolism, including its positive influence on calcium absorption, its synergistic work with vitamin D on bone metabolism and its inverse correlation with fracture risk.[10,11,12] Supplementing with vitamin K for just one month resulted in increased bone formation markers and decreased bone resorption markers in female elite athletes, of whom four were amenorrhoeic.[12]

There is accumulating evidence that the current intake of vitamin K is too low for optimal bone health, although the optimal requirement is still unknown.[14] Clinical intervention trials have demonstrated the wide safety range of this nutrient although further studies are needed to clarify its mechanism of action in bone health.[11]

Calcium is the provider of resistance and rigidity to bone mass and teeth.[15] The reduction of mineral bioavailability by phytic acid, found in most grains, seeds and beans, is widely recognised. Phytates form insoluble complexes with calcium rendering it unavailable for absorption.[15,16]

Dairy foods have often been cited as the most important source of calcium yet most studies of dairy food intake and bone health provided inconclusive results.[16,17] The group that appears to have the greatest beneficial effect from the ingestion of dairy products is women under 30 years.[17]

The suggestion that protein intake reduces calcium absorption is strongly disputed, particularly as most studies coming to this conclusion appear to have ignored the positive correlation between calcium intake and calcium balance; that is protein exerts a negative impact only under conditions of low calcium intake.[18,19] Overall, protein tends to have a positive effect on bone health.[18]

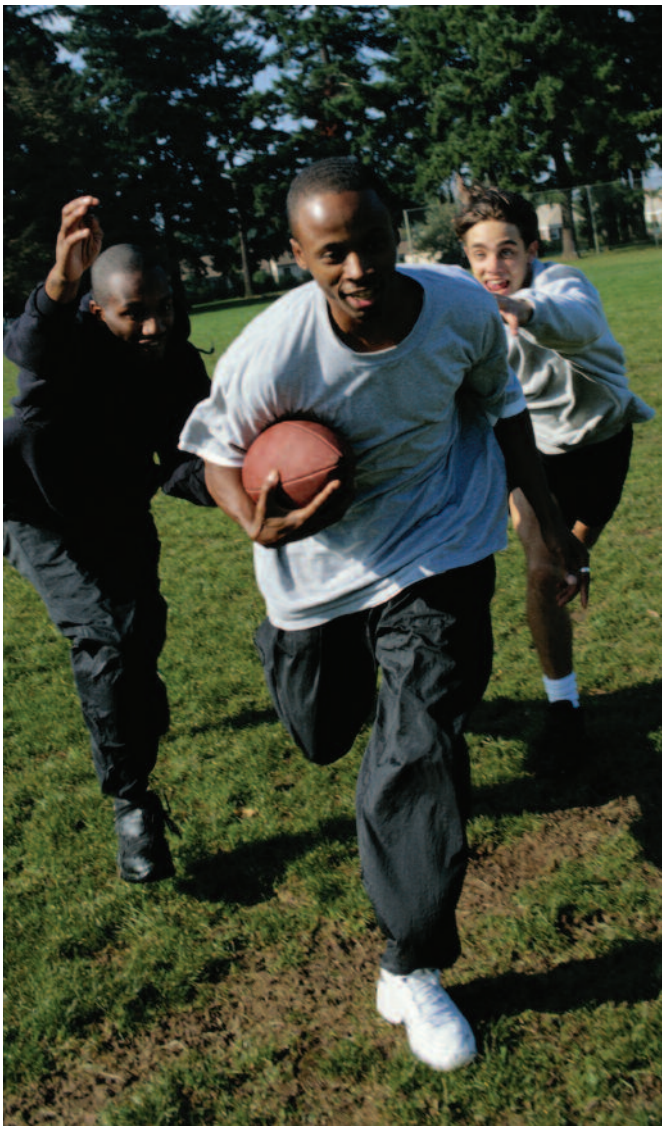
Metabolic acidosis, caused by the ingestion of acid ash foods, appears to increase calcium excretion. This is discussed in greater detail below.

Magnesium is an important mineral for bone metabolism but is often ignored. Magnesium levels are decreased with excessive intakes of phosphoric acid found in carbonated drinks, coffee intake and excessive menstruation.[20] As a magnesium deficiency can lead to calcium deficiency, the two minerals should be supplemented together, although good quality supplements will also include vitamins A, D3 and K. Johnson[20] states that even though magnesium is readily available in food, absorption and retention is a complex issue and deficiencies are common.

The acid-base equilibrium of blood and extracellular fluid is controlled to a significant extent by bone tissue. Food components represent the most important source of acid stresses liable to promote bone resorption in otherwise healthy individuals.[21]

It is important to understand that the acidity of food components is not linked with their acidity in the native state but to the pH of the ashes obtained from their incineration in the laboratory to simulate metabolic oxidation.[21] Most fruits and vegetables have an alkaline ash while meat, fish, eggs, grains and legumes have an acidic ash.

Metabolic acidosis, however minor, represents a strong stimulus for bone resorption through increased osteoclastic activity and an inhibition of osteoblastic activity.[21] The converse reaction exists in metabolic alkalosis which suppresses osteoclastic activity. This has been shown by the administration of bicarbonate to volunteers to improve calcium balance.[21,22]



The typical Western diet is high in omega-6 polyunsaturated fatty acid (PUFA). It is thought that our present intake of omega-6 to omega-3 is 20-30:1 while the optimal suggested ratio of these two essential fatty acids (EFAs) is 1-4:1.[23]

Part of the problem is due to our high intake of grains. Grains contain an imbalanced ratio of EFAs.[16] Although the total fat content and the amount of omega-6 fatty acids is not significant in terms of total calories, the ratio of EFAs determines the production of pro or anti-inflammatory eicosanoids. For example, omega-6, via the eicosanoids metabolic products from arachidonic acid, induces the production of pro-inflammatory prostaglandin E2 (PGE2) and omega-3 stimulates anti-inflammatory PGE3. PGE2 is known to reduce the bone-forming activity of osteoblasts and to increase the bone-resorbing activity of osteoclasts.[16]

Modern agriculture is thought to be responsible for a decrease in omega-3 fatty acid content in many foods including meats, vegetables, eggs and even cultured fish.[23] In addition, our intake of omega-3 fatty acid found in cold water fish has decreased. As such, our diet has become highly pro-inflammatory with negative consequences on bone health.

other considerations influencing bone mass

Any significant **decrease in calorific intake** can have a profound effect on bone health of male and female athletes, both from a lack of nutrients and from a possible disruption of hormonal balance. Some athletes are under pressure to maintain a particular level of body fat composition and appearance to perform in certain sports such as gymnastics, track and field and figure skating. Extreme dietary restrictions can lead to endocrine dysfunction, decreasing oestrogen and elevating glucocorticoid levels resulting in increased bone resorption[2,3] (see The Female Athlete Triad in the April/May issue).

Another consideration is the use of **glucocorticoids**. These are commonly used to treat allergic and inflammatory conditions. However, they do increase calcium secretion and suppress osteoblastic activity.[3] Increasing particular nutrient intake may have to be considered if use of these drugs must be continued.

Finally, over recent years the use of **oral contraceptives** (OC) and its effect on peak bone mass has caused some concern.[24] Although the data are controversial, it appears that OC use may be negatively associated with the accrual of peak bone mass in young women.[24]

In conclusion, the key to preventing stress injuries in athletes is to maximise PBM in the paediatric, adolescent and young adult age group.[3] The key to maximising PBM is to consume a diet containing a variety of foods at each meal, something that is often lacking in young adolescents. Clinically, we have found that fruit intake is often optimal but vegetable intake, particularly the green leafy kind, is well below recommended levels. As a result, we have often diagnosed magnesium deficiencies. Increasing fruit intake is not, and must not, be a substitute for a lack of vegetable intake. Vegetables, of which there is a huge variety, must form a large part of every adolescent athlete's daily food intake; as should the reduction or elimination of carbonated beverages. Supplementation of certain nutrients may need to be seriously considered.

One thing is certain: without strong bones, our young athletes will never make it to the top of their sport. fn

references

1. Eastell R, Lambert H (2002) Diet and healthy bones. *Calcified Tissue International* 70(5): 400-404.

2. Gordon CM (2003) Normal bone accretion and effects of nutritional disorders in childhood. *Journal of Women's Health* 12(2): 137-143.

3. Peer KS (2004) Bone health in athletes. *Orthopaedic Nursing* 23(3): 174-181.

4. Seibel MJ (2002) Nutrition and molecular markers of bone remodelling. Current Opinion in *Clinical Nutrition and Metabolic Care* 5(5): 525-531.

5. Vasquez A (2004) The clinical importance of vitamin D (cholecalciferol): a paradigm shift with implications for all healthcare providers. *Alternative Therapies* 10(5): 28-36.

6. New SA (2001) Nutrition and skeletal health – a review of current thinking. The 1st Joint Meeting of the International Bone and Mineral Society and the European Calcified Tissue Society, June 5-10, Madrid, Spain (www.medscape.com/viewarticle/420853).

7. Gannagé-Yared MH, Chemali R, Yaacoub N, Halaby G (2000) Hypovitaminosis D in a sunny country: relation to lifestyle and bone markers. *Journal of Bone and Mineral Research* 15(9): 1856-1862.

8. Vasquez A (2005) Health care for our bones: a practical nutritional approach to preventing osteoporosis (letter). *Journal of Manipulative and Physiological Therapeutics* 28(3): 213.

9. Holick MF (2004) Vitamin D: importance in the prevention of cancers, type 1 diabetes, heart disease and osteoporosis. *The American Journal of Clinical Nutrition* 79:362-71.

10. Vermeer C, Hamulyák (2004) Vitamin K: lessons from the past. *Journal of Thombosis and Haemostasis* 2(12): 2115-2117.

11. Weber P (2001) Vitamin K and bone health. *Nutrition* 17(10): 880-887.

12. Booth SL, Broe KE, Gagnon DR, Tucker KL, Hannan MT, McLean RR, Dawson-Hughes B, Wilson PWF, Cupples LA, Kiel DP (2003) Vitamin K intake and bone mineral density in women and men. *The American Journal of Clinical Nutrition* 77(2): 512-16.

13. Craciun AM, Wolf J, Knapen HJ, Brouns F, Vermeer C (1998) Improved bone metabolism in female elite athletes after vitamin K supplementation. *International Journal of Sports Medicine* 19(7): 479-484.

14. Bügel S (2003) Vitamin K and bone health. Proceedings of the Nutrition Society 62(4): 839-843.

15. Cámara-Martos F, Amaro-Lfipez AM (2002) Influence of dietary factors on calcium bioavailability. Biological Trace Element Research 89(1): 43-52.

16. Seaman D (2004) Health care for our bones: a practical nutritional approach to preventing osteoporosis. *Journal of Manipulative and Physiological Therapeutics* 27(9): 591-595.

17. Weinsier RL, Krumdieck CL (2000) Dairy foods and bone health: examination of the evidence. *The American Journal of Clinical Nutrition* 72(3): 681-689.

18. Heany RP (2001) Protein intake and bone health: the influence of belief systems on the conduct of nutritional science. *The American Journal of Clinical Nutrition* 73(1): 5-6.

19. Heany RP (2000) Dietary protein and phosphorus do not affect calcium absorption. *American Journal of Clinical Nutrition* 72(3): 758-761.

20. Johnson S (2001) The multifaceted and widespread pathology of magnesium deficiency. *Medical Hypotheses* 56(2): 163-170.

21. Buclin T, Cosma M, Appenzeller M, Jacquet AF, Décosterd LA, Biollaz J, Burckhardt P (2001) Diet acids and alkalis influence calcium retention in bone. *Osteoporosis International* 12(6): 493-499.

22. Maurer M, Riesen W, Muser J, Hulter HN, Krapf R (2003) Neutralization of Western diet inhibits bone resorption independently of K intake and reduces cortisol secretion in humans. *American Journal of Physiology* 284(1) Part 2: F32-F40.

23. Simopoulos AP (1999) Essential fatty acids in health and chronic disease. *The American Journal of Clinical Nutrition* 70(3S): 560S-569S.

24. Hartard M, Kleinmond C, Kirchbichler A, Jeschke D, Wiseman M, Weissenbacher ER, Felsenberg D, Erben RG (2004) Age at first oral contraceptive use as a major determinant of vertebral bone mass in female endurance athletes. Bone 35(4): 836-841.

Kathryn Bistany, Founder and Managing Director of Corpotential Limited, is based in London. Individual nutrition consultations, of which the first three hours, cost £250. For further information about Corpotential, please visit www.corpotential.com or call +44 (0) 20 8994 3701.