

The Influence of Vitamin D on Bone Health Across the Life Cycle

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Vitamin D is often looked upon only as that fat-soluble vitamin found in cod liver oil, fatty fish (salmon, mackerel), fortified milk, and fortified breads and cereals, and which is associated with sun exposure and preventing rickets in children. However, in the Nutrition and Bone Health Working Group of the American Society for Bone and Mineral Research, we learned that vitamin D has important ramifications for health, not only throughout life, but even during fetal development. Dr. Cooper reviewed the important factors that are related to normal patterns of skeletal growth. He noted that many factors influence the accumulation of bone mineral during childhood and adolescence including heredity, gender, diet, physical activity, endocrine status, and a wide variety of risk factors. He also emphasized that maternal vitamin D deficiency, as well as other factors, modulated bone mineral acquisition during intrauterine life and was associated with low birth size and poor childhood growth that were directly linked to increased risk of hip fracture later in life. Children born to mothers who had suboptimal vitamin D status had significantly reduced whole body bone mineral content at age 9 y. This deficit in skeletal growth remained significant even after adjustment for childhood weight and bone area.

Dr. Tylavsky reviewed evidence identifying factors related to vitamin D status in adolescents. Serum 25(OH)D levels in adolescents are greatly affected by ethnicity, gender, puberty stage, parathyroid hormone level, dietary vitamin D intake, and sun exposure. She noted that there was no consensus on the concentration of serum 25(OH)D that would yield the most benefit for bone health for adolescents. She also remarked that vitamin D deficiency is also not well defined in this young population but that the majority consensus in the past has been a 25(OH)D level below 10 ng/mL. Using

this as the marker for vitamin D deficiency, the prevalence of vitamin D deficiency in adolescents ranged from 0 to 32%, depending on the season measured and the latitude of the population under study. However, if higher cut-points for 25(OH)D are used, the prevalence rates increased upward to 75% of some populations. She reviewed the studies on vitamin D demands during adolescence being a function of dietary intake and growth velocity. Some studies have reported a negative association between serum 25(OH)D and fractional absorption of calcium in young girls ages 9–17 y. However, in Caucasian, Mexican-American, and African-American children receiving an average calcium intake of between 821 to 1110 mg a day, there was no significant correlation between serum 25(OH)D levels and fractional calcium absorption. These results may be explained by the fact that 25(OH)D was more efficiently metabolized to 1,25-dihydroxycholecalciferol during the growth spurt, which increases the efficiency of intestinal calcium absorption even when vitamin D stores are not maximal. Studies do support the fact that 25(OH)D levels are associated with bone health during puberty. Young women with 25(OH)D concentrations above 40 nmol/L (16 ng/mL) had greater radial and ulna bone mineral density (BMD). Furthermore, there was progressive increase in cortical BMD with increasing serum 25(OH)D. However, there were no statistically significant differences in total femur, lumbar spine, or whole body BMD when related to 25(OH)D levels. She concluded that the paucity of research relating to vitamin D nutrition in bone accrual in adolescents begs the need for more research in this important area of human nutrition.

Dr. Holick followed, reminding the audience that vitamin D deficiency is epidemic, not only in the elderly, but is also commonly seen in prepubertal children, adolescents, and young and middle-aged adults. Vitamin D deficiency has insidious consequences for the skeleton. It prevents the accrual of the maximum amount of calcium that is genetically preprogrammed for the skeleton. Once peak bone mass is attained, both young and middle-aged adults who are vitamin D deficient will lose, on average, 0.25 to 0.5% of their skeletal mass per year if they do not have adequate calcium and vitamin D. Vitamin D deficiency results in secondary hyperparathyroidism, which increases the production of osteoclasts, which, in turn, dissolve bone releasing calcium into the blood stream. This results in osteopenia and can progress to osteoporosis. In addition, vitamin D deficiency causes a mineralization defect of the collagen matrix resulting in osteomalacia. Unlike os-

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teoporosis, which is a painless disease until fracture occurs, osteomalacia is often associated with aching, throbbing bone pain. Dr. Holick noted that this is often misdiagnosed as fibromyalgia. He also noted that vitamin D appeared to play an important role in the prevention of many chronic diseases, including common cancers, such as breast, colon, and prostate; cardiovascular heart disease; and common autoimmune diseases, including multiple sclerosis and type I diabetes. He noted that most children and adults obtain their vitamin D requirement from casual, sensible exposure to sunlight. In the

absence of sunlight, 1000 IU of cholecalciferol per day is needed to satisfy the body's requirement and to maintain a 25(OH)D in a healthy range of above 30 ng/mL.

In conclusion, this workshop discussed the importance of vigilance for vitamin D status [i.e., serum 25(OH)D levels] to ensure maximal skeletal health from birth until death. Measuring 25(OH)D yearly and providing everyone with guidelines for sensible sun exposure and/or vitamin D from food/supplements is a prudent strategy for preventing fractures and improving overall health and well-being.