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Vitamin D in adolescents: Are current recommendations enough?☆

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ABSTRACT

Vitamin D is essential for bone development during adolescence and low vitamin D status during this critical period of growth may impact bone mineralization, potentially reducing peak bone mass and consequently increasing the risk of osteoporosis in adulthood. Therefore, the high prevalence of vitamin D inadequacy and deficiency in adolescent populations is of great concern. However, there is currently a lack of consensus on the 25-hydroxyvitamin D [25(OH)D] concentration, the widely accepted biomarker of vitamin D status, that defines adequacy, and the vitamin D intake requirements to maintain various 25(OH)D thresholds are not well established. While the current intake recommendations of 10–15 μg/day may be sufficient to prevent vitamin D deficiency (25(OH)D < 25–30 nmol/l), greater intakes may be needed to achieve the higher threshold levels proposed to represent adequacy (25(OH)D > 50 nmol/l). This review will address these concerns and consider if the current dietary recommendations for vitamin D in adolescents are sufficient.

1. Introduction

Vitamin D insufficiency is widely recognised as a global public health concern, and can affect all population groups irrespective of gender, age or ethnicity. More severe and prolonged vitamin D deficiency can result in rickets in children and osteomalacia in adults [1,2]. Adolescence is a critical period of growth and bone development, and less severe vitamin D insufficiency may prevent adolescents achieving peak bone mass [3,4]. Given the important and well-recognized role of vitamin D in calcium absorption and bone health, it is therefore concerning that low vitamin D status is prevalent among adolescents worldwide [5]. Despite this, adolescents appear to be a somewhat neglected population group with a limited evidence base for setting vitamin D intake requirements, with those that exist extrapolated from adult data, which may not always be appropriate. This review will consider firstly the definitions of optimal vitamin D status in adolescents, and secondly whether the current dietary intake recommendations are adequate to achieve this, given the high prevalence of low vitamin D status in this vulnerable population group.

2. Sources of vitamin D

Vitamin D is a unique nutrient in that the main source is sun exposure, rather than diet. The majority of vitamin D is synthesized within the human body via the action of ultraviolet B (UVB) radiation on the skin, which mediates the conversion of 7-dehydrocholesterol, a cholesterol precursor present within the skin, to vitamin D_3. Vitamin D_3 then undergoes two hydroxylation steps, first in the liver to the biologically inactive form, 25-hydroxyvitamin D [25(OH)D], and secondly in the kidneys to the active 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] [6].

The amount of vitamin D_3 synthesized within the skin is a function of the amount of UVB radiation reaching the skin and the availability of 7-dehydrocholesterol within the epidermis. Consequently the level of vitamin D_3 synthesis is dependent on a number of environmental and individual factors, including latitude, season, ethnicity, clothing that completely covers the skin for cultural or religious beliefs, use of sunscreen, time spent outdoors, obesity and aging [7]. During the winter-time at northerly latitudes, the increasing zenith angle of the sun results in little, if any, cutaneous synthesis of vitamin D_3. Therefore, during times of insufficient sun exposure, dietary sources of vitamin D become important in maintaining vitamin D status. However, this proves to be challenging as there are few foods that are a naturally rich source

Abbreviations: BMD, bone mineral density; DBP, vitamin D binding protein; IOM, institute of Medicine; PTH, parathyroid hormone; UVB, ultraviolet B; $1,25(OH)_2D$, 1,25-dihydroxyvitamin D; 25(OH)D, 25-hydroxyvitamin D.

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of vitamin D [6] and there is increasing evidence to suggest that current vitamin D intakes are inadequate to compensate for the seasonal deficit in sunlight during the winter-time. While food fortification and supplement use can be effective in increasing dietary intakes of vitamin D, fortification policies currently vary dramatically between countries and often occur on a voluntary basis, while uptake and adherence to supplement use varies by gender and age group [8].

Several studies have reported inadequate dietary vitamin D intakes among adolescents, although this varies by country due to dietary habits, fortification policies and supplement use. In the European Nutrition and Health Report, vitamin D intakes ranged from 1.5–7.5 μg/day in 13–24 year old males and females [9]. The lowest intakes were seen among Spanish adolescents (1.8 and 1.5 μg/day in male and female 15–18 year olds respectively), closely followed by Austrian males and British females at 2.0 μg/day. Intakes were below 5 μg/day in all sub-groups except for Polish males (5.5 μg/day) and Norwegian males and females (15–18 years), who had the highest intakes at 7.5 and 7.1 μg/day respectively. This could be attributed to high consumption of fish and supplement use, as Norway, like much of Europe, has limited vitamin D fortification of foods [8,10]. The exception to this is Finland, who since 2003 have fortified fluid milks, margarines and spreads. While this policy increased dietary vitamin D intakes in 4 year old children [11], supporting the assertion that fortified milk is a major determinant of 25(OH)D status in young children [12], it had little impact on the vitamin D intakes of 12–18 year old adolescent females, with no change in 25(OH)D concentrations [13]. This is likely due to low consumption of dairy products in adolescent females, demonstrating the importance of giving consideration to food consumption patterns when developing fortification policies. Furthermore, this may also be influenced by ethnic differences in dietary patterns, with African American adolescent females reported to consume more vitamin D from meat and bean food sources compared to white females who consumed more from milk [14]. Fortification is also more commonly practiced in the United States compared with Europe. Using data from the National Health Nutrition and Examination Survey (NHANES) 2003–2006, Fulgoni et al. reported intakes of 1.7 μg/day in 2–18 year olds from naturally occurring food sources of vitamin D, which increased to 6.1 μg/day when fortified foods were also included [15]. When supplement use was also considered, intakes increased to 8.3 μg/day. While supplements can increase intakes of vitamin D, uptake tends to be lower in adolescents compared with younger children [16], so perhaps may not be the most appropriate strategy for improving vitamin D intakes in the adolescent population.

3. Defining vitamin D deficiency and adequacy and selected outcome measures

3.1. Vitamin D deficiency and rickets

Circulating 25(OH)D concentration serves as the best indicator of vitamin D status as it reflects both dietary intakes and cutaneous synthesis of vitamin D and has a long half-life of 2–3 weeks. Measurement of 1,25$(OH)_2$D is not considered useful for assessment of vitamin D due to its short half-life (4–7 h) and its tight homeostatic regulation [17].

There is generally good agreement that populations should not have 25(OH)D concentrations of less than 25–30 nmol/l based on an increased risk of rickets and impaired bone growth [18]. Vitamin D deficiency rickets remains a public health concern among infants particularly in communities in Asia, Africa and the Middle East, due to a high prevalence of low vitamin D status during pregnancy, breast feeding and breast milk that is low in vitamin D, darker skin pigmentation and cultural dress [1]. However it is important to recognise that rickets can also occur during childhood and adolescence, which is attributable to insufficient dietary calcium intakes. Children presenting with rickets in South Africa and Nigeria were found to have low intakes of dairy products and diets high in unrefined cereals and phytate, which impairs calcium absorption [19,20]. In a 24-week randomized trial of 4 year old Nigerian children with rickets and low habitual dietary calcium intakes (~200 mg/day), supplementation with calcium alone (1000 mg/day) and calcium in combination with vitamin D (1000 mg/day calcium and 1500 μg vitamin D administered at baseline and 12 weeks) were found to be equally effective in treating rickets and more so than vitamin D treatment alone [21]. The mean serum 25(OH)D concentration of these children at baseline was 35 ± 25 nmol/l, with 37% having concentrations below 30 nmol/l. At the end of the intervention serum 25(OH)D concentrations were 52, 87 and 102 nmol/l in the calcium alone, vitamin D alone and calcium plus vitamin D groups [21].

3.2. Defining vitamin D adequacy and measures of bone health

There is much debate and controversy surrounding which levels of circulating 25(OH)D should be considered as adequate or 'optimal'. At present, the Institute of Medicine (IOM) [18], European Food Safety Authority (EFSA) [22], American Academy of Pediatrics [23] and the European Society for Paediatric Gastroenterology, Hepatology and Nutrition [24], among others, suggest a serum 25(OH)D concentration of >50 nmol/l as being adequate. However others have proposed much greater 25(OH)D sufficiency thresholds. The Endocrine Society [25] and The Society for Adolescent Health and Medicine [26] for example, consider sufficiency at 25(OH)D concentrations >75 nmol/l, and deficiency below 50 nmol/l. These cut-off thresholds are often based on data from adult studies that use various biochemical and/or functional outcome criteria to define vitamin D adequacy, such as suppression of parathyroid hormone (PTH), maximal calcium absorption and bone mineral density. However it is important to note that it may not always be appropriate to use such criteria for the adolescent population. A good example of this is the relationship between vitamin D and PTH. While it is well established that vitamin D deficiency in elderly subjects leads to increases in PTH and consequently increased bone turnover and bone loss [27], the elevated PTH concentrations observed in adolescents may not necessarily be detrimental to bone health when the rate of bone modelling, skeletal growth and bone consolidation are at a peak [28,29]. Adolescent studies have failed to identify a consistent inflection point for maximal suppression of PTH and this therefore brings into question the use of such a criteria in defining vitamin D adequacy in adolescents [3,30–33]. Furthermore, it has been suggested that increasing 25(OH)D concentrations in adults may enhance calcium absorption, although studies in adolescents have shown that 25(OH)D concentrations above ~30–50 nmol/l are not associated with any increased benefits with respect to calcium absorption [34–36].

A review of data linking vitamin D status to various health outcomes indicated that 25(OH)D concentrations of 90–100 nmol/l were optimal in relation to bone mineral density, fracture prevention, lower extremity function and colorectal cancer in adult populations [37]. Studies evaluating vitamin D status and bone mineralization in the adolescent age group have reported unfavourable effects of circulating 25(OH)D concentrations of less than 40 nmol/l on bone mineral density (BMD) of various skeletal sites. In particular, studies in Finnish females reported that those with 25(OH)D concentrations <40 nmol/l had lower BMD of the radius and ulnar [3], whilst reduced lumbar spine BMD was