e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com doi: 10.25258/ijpqa.16.6.69

International Journal of Pharmaceutical Quality Assurance 2025; 16(6); 414-420

Original Research Article

Prevalence and Health Impacts of Vitamin D Deficiency in Pediatric Populations

Anjali Singh

SMO, Department of Pediatrics, Sheikh Bhikhari Medical College and Hospital, Hazaribagh, Jharkhand, India

Received: 09-04-2025 / Revised: 16-05-2025 / Accepted: 23-06-2025
Corresponding Author: Dr. Anjali Singh
Conflict of interest: Nil

Abstract:

Background: Vitamin D is key for bone mineralization, growth, and overall health. It is increasingly recognized that deficiency occurs among children as their exposure to sunlight has decreased and dietary intake is inadequate. Deficiency may affect the skeletal system as well as extra-skeletal systems.

Objective: The primary objective of the study was to identify the prevalence and the effects of vitamin D deficiency on health conditions in children aged 6-12 years living near Sheikh Bhikhari Medical College and Hospital, Hazaribagh, Jharkhand, India.

Methods: A cross-sectional study of 250 children was conducted, collecting anthropometric, clinical, and biochemical data. Serum 25(OH)D was measured using electrochemiluminescence immunoassay, with deficiency defined as <20 ng/mL. All analysis was performed using SPSS 26.0 for Windows.

Results: Vitamin D deficiency was found to be present in 56.8% of participants. Deficient children had significantly lower serum calcium ($p = 0.00045$) and higher alkaline phosphatase ($p = 0.016$). Vitamin D Deficiency increased during the spring (81.0%) and winter (69.1%) season respectively ($p < 0.001$). Although overweight children were at a higher risk of deficiency, this was not statistically significant (OR = 1.9; $p = 0.097$).

Conclusion: Vitamin D deficiency appears to be present in a high prevalence among children and is ascertainable from not only their season, but also their body weight. Preventative measures such as promoting outdoor activity, dietary fortification, and supplementation during non-sunny months could assist in preventing vitamin D deficiency.

Keywords: Bone Metabolism, Pediatric Health, Prevalence, Seasonal Variation, Sunlight Exposure, Vitamin D Deficiency.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Vitamin D is a crucial micronutrient that plays a role in the preservation of bone health, skeletal development, and overall physiological functioning in humans. It plays a role in the metabolism of calcium and phosphorus, which are essential for bone/teeth development and mineralization during growth and development (especially during childhood, a time of rapid growth and metabolic demand) [1]. Adequate levels of vitamin D are necessary for adequate absorption of these minerals in the gut and assistance in facilitating normal serum calcium and phosphate levels to help ensure appropriate mineralization of bone and neuromuscular function. Therefore, a vitamin D deficiency can have physiological health implications across multiple organ systems.

Even though vitamin D is essential, the natural dietary sources of vitamin D are very limited. In fact, only a small number of foods contain larger amounts of vitamin D. The most well-known vitamin D sources are fatty fish, liver, egg yolks, and fortified dairy products. Therefore, for most individuals, sun-induced synthesis in the skin, stimulated by ultraviolet B (UVB) rays from sunlight, represents the most significant source of vitamin D. However, it is important to note that the skin synthesis of vitamin D varies significantly based on geographic location, season, and lifestyle. In particular, we have seen a demographic shift toward modern lifestyles that involve more time being indoors, the use of sunscreens, and urbanization, which all contribute to decreased sun exposure. Collectively, decreased exposure to sunlight has contributed to a global decline in sunlight exposure, thus resulting in decreased synthesis of vitamin D and a greater prevalence of deficiency.

Vitamin D deficiency is a well-established factor in several disorders of the skeleton. In children, such deficiency leads to rickets (abnormal bone mineralization), skeletal deformity, and growth failure) [3]. In adults, vitamin D deficiency can lead to

osteomalacia, which results in soft, fragile bones. However, beyond these traditional skeletal manifestations, there is increasing evidence to support that vitamin D has many extra-skeletal functions. Vitamin D receptors are found throughout many tissue types, including endocrine tissues and vascular tissues, and they regulate differentiation of cells and cytokine and interleukin production) [4]. This suggests that Vitamin D is involved in multiple biological processes, including immune regulation, insulin secretion, and proper function of cardiovascular tissues. Given this, vitamin D deficiency has been related to increased risks of metabolic syndrome, type 2 diabetes, autoimmune diseases, and cardiovascular diseases) [5]. Our findings indicate the systemic importance of vitamin D and support the notion that vitamin D deficiency may have far greater health implications beyond bone health.

The lack of vitamin D has been associated, in the past few years, with several non-skeletal conditions in pediatric populations. Therefore, low levels of vitamin D have been linked to a higher incidence of respiratory infections, food allergies, and asthma [6]. There is also evidence that vitamin D status potentially impacts endocrine function, including onset of puberty; earlier menarche may occur in girls with insufficient vitamin D status [7]. These findings suggest the multifactorial impact of vitamin D in child health and development; its roles are not limited to concerns of bone health but also include regulation of immune and endocrine function".

There are several risk factors for vitamin D deficiency in children. These can be categorized into intrinsic factors (skin pigmentation, which reduces UVB absorption) and extrinsic factors (less sunlight exposure, less usage of vitamin D-rich food and supplementation, higher body mass index (BMI), and older age) [8]. There are also certain factors displayed in children that can elevate risk, such as darker skin tone, living at a northern latitude where sunlight levels are generally low for much of the year, and spending little time outdoors. Additionally, specific clothing that limits sun exposure, as well as urban lifestyles with little outside activity, can increase risk even more. Another important risk factor is nutritional deficiency, particularly in areas where food is not fortified.

Recent estimates suggest that vitamin D deficiency has reached epidemic levels around the world in adults and children) [9]. This is due in part to the reduced sun exposure caused by changes to lifestyle and environmental factors that absorb UVB, such as air pollution) [9]. The criteria for defining vitamin D deficiency is still debated and threshold values for serum 25-hydroxyvitamin D [25(OH)D] concentration used by health organizations and researchers to define deficient, insufficient and sufficient, are not agreed upon [9]. Due to this lack of uniformity, data regarding the prevalence of vitamin D deficiency varies greatly across and within countries. Prevalence is reported as ranging between 15% and 60% in studies conducted around the world depending on population characteristics, geographical locale, time of year, and definitions of deficiency) [10]. These aspects also pose challenges for reaching consensus on recommendations for the prevention, diagnosis, and treatment of vitamin D Deficiency in children and youth.

Vitamin D deficiency in children has emerged to be a significant public health challenge, which can be associated with both immediate and long-term health consequences. Vitamin D is essential during childhood for healthy skeletal growth, as well as biologic function related to the maturation of both the immune and endocrine systems. Persistence of deficiency during this crucial stage of life may lead to chronic disease in adulthood (e.g., osteoporosis, metabolic syndrome, cardiovascular disease, etc.). The complexity of simultaneous multiple nutritional deficiencies, socio-economic inequalities, and changes in social lifestyle factors have further complicated pediatric vitamin D health.

Given this added complexity, resolving these issues for influencing children's health leads us to consider the burdens of vitamin D deficiency in children to develop effective prevention and treatment strategies. Effective estimation of prevalence, identification of risk factors, and evaluation of epidemiological health outcomes related to vitamin D deficiency are the first steps to addressing chronic disease burden. The purpose of this paper is to assess the prevalence of vitamin D deficiency in children aged 6 to 12 years, examine associated risk factors of vitamin D deficiency, and provide evidence-based recommendations to support clinical and public health practices to improve vitamin D health, and children's health overall.

Methodology

Study Design: This was a hospital-based, observational cross-sectional study investigating the prevalence and health effects of vitamin D deficiency in children. Data on the participant's demographics, anthropometrics, biochemistry, and clinical assessment were collected and analyzed in an attempt to establish any relationship between serum vitamin D and health measures.

Study Area: The study was carried out in the Department of Pediatrics, Sheikh Bhikhari Medical College and Hospital, Hazaribagh, Jharkhand, India.

Study Duration: The study was carried out over a period of nine months.

Study Population: The sample included children between the ages of 6 and 12 years who presented to the pediatric outpatient clinic for routine health check-ups or to evaluate growth and development.

Only those who were free from acute illness during the time of examination were enrolled.

Sample Size: The total number of pediatric patients in the study was 250. Sample size was determined based on outpatient attendance records and feasibility of data collection during the period of the study.

Inclusion Criteria

- Children aged 6 to 12 years.
- Attending the Department of Pediatrics, Sheikh Bhikhari Medical College and Hospital during the study period.
- Children presenting for routine examination, growth evaluation, or general health assessment.
- Children whose parents/guardians provided written informed consent.

Exclusion Criteria

- Children with known chronic illnesses such as epilepsy, hypothyroidism, malabsorption syndromes, renal or hepatic disorders.
- Children on vitamin D supplementation or medications affecting calcium/vitamin D metabolism.
- Children with acute illness or infection at the time of sampling.

Data Collection: Data were collected from both medical records and direct clinical assessments conducted in the Department of Pediatrics, Sheikh Bhikhari Medical College and Hospital. Each participant was assessed for a set of anthropometric, clinical, and biochemical parameters. Anthropometric data like height and weight were measured by standardized techniques. Body mass index was calculated. Percentiles for all BMI measurements were graded according to the IAP growth charts: between the 3rd and 84th percentile, normal; between the 85th and 94th percentile, overweight; and at or above the 95th percentile, obese. Pubertal status was assessed by Tanner staging to identify the stage of sexual maturation. Bone age assessment by the Greulich and Pyle method was done based on left-hand and wrist radiographs. Clinical and anthropometric data along with the history of sun exposure, dietary habits, and physical activity were assessed through a structured pro forma. Parents or guardians were interviewed to obtain the above information. All information obtained was entered on a predesigned proforma.

Laboratory Analysis: With informed consent from the guardians, aseptic venous blood samples were collected from all the participants. The serum, separated from the blood samples, was assayed for 25-hydroxyvitamin D [25(OH)D], calcium, phosphorus, and alkaline phosphatase (ALP) levels. Serum 25(OH)D concentration, as an indicator of vitamin D status, was determined by an electrochemiluminescence immunoassay method of Roche Diagnostics, Indianapolis, IN, USA. Vitamin D status was classified in accordance with the recommendations of the U.S. Institute of Medicine, where serum 25(OH)D below 20 ng/mL was defined as deficient and equal to/greater than 20 ng/mL as sufficient. Serum calcium, phosphorus, and alkaline phosphatase (ALP) was assessed on the hospital's main laboratory on an automated biochemical analyzer following standard operating protocols and with precision and accuracy. Laboratory analyses were performed under strict quality control by trained laboratory technicians.

Procedure: After informed consent, physical examination and anthropometric measurements were taken of all eligible participants. Blood samples were taken after aseptic precautions for biochemical analyses. All relevant clinical data were recorded on a predesigned proforma. Laboratory and radiological results were reviewed and grouped as per the predefined criteria.

Statistical Analysis: All analyses were performed with SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The data are presented as mean ± SD. Comparisons of continuous and categorical variables between the vitamin D-deficient and sufficient groups were performed using the independent t-test and chi-square test, respectively. Multivariate logistic regression analysis was conducted to identify the independent predictors of vitamin D deficiency. A p-value of less than 0.05 was considered significant."

Result

Table 1 displays the characteristics of the 250 participants, including general demographic, seasonal, and clinical characteristics applicable to the cohort. There were more females than males in the sample, as 92 (36.8%) were males and 158 (63.2%) were females. In regards to body weight status, the majority of children were categorized as normal (68.8%), 18.4% were categorized as overweight, and 12.8% were categorized as obese. There was significant burden of excess body weight observed in the sample. When segregating the sample by season of sampling, most participants were sampled in the winter (44.0%), followed by autumn (39.2%), and the least participants were sampled in the spring (16.8%). Variations in sun exposure can influence vitamin D status. Of the total sample, 56.8% of the participants were classified as vitamin D deficient (<20 ng/mL) and 43.2% as sufficient (≥20 ng/ml), reflecting a large prevalence of hypovitaminosis D. Of pubertal maturation, 219 (87.6%) were classified as normal and 31 (12.4%) were classified as precocious, suggesting that vitamin D status, adiposity, and early maturation may need further inquiry.

Table 1: General characteristics of the study participants (N = 250)	
Characteristic	**No. (%)**
Sex	
Male	92 (36.8)
Female	158 (63.2)
Body weight category	
Normal (<85th percentile)	172 (68.8)
Overweight (85th–95th percentile)	46 (18.4)
Obese (≥95th percentile)	32 (12.8)
Season of sample collection	
Spring (Mar–May)	42 (16.8)
Autumn (Sep–Nov)	98 (39.2)
Winter (Dec–Feb)	110 (44.0)
Vitamin D status (25[OH]D)	
Deficiency (<20 ng/mL)	142 (56.8)
Sufficiency (≥20 ng/mL)	108 (43.2)
Pubertal status	
Normal	219 (87.6)
Precocious puberty	31 (12.4)

Table 2 illustrates the characteristics of the 250 participants according to their vitamin D status. Results indicate that the prevalence of vitamin D deficiency was 56.8%, while 43.2% of participants had sufficient vitamin D levels. The mean serum 25(OH)D concentration of the deficient group was 14.9 ± 3.4ng/mL, which was significantly lower than the mean serum 25(OH)D concentration for the sufficiently classified group, which was approximately 27.6 ± 5.8 ng/mL ($p < 0.001$). There were no significant differences in sex distribution, age or pubertal status between groups, but seasonality did have a strong association with 81.0% of individuals who qualified as vitamin D deficient in the spring, and 69.1% in the winter months, while 67.3% qualified as sufficient in the autumn ($p < 0.001$). Regarding weight status, most participants had normal weight status, albeit overweight children tended to have higher deficiency prevalence rates. Biochemical analysis revealed that the deficient group had lower calcium levels (9.61 ± 0.38 vs. 9.77 ± 0.33 mg/dL, $p = 0.00045$) and higher alkaline phosphatase (ALP) levels (266.7 ± 67.8 vs. 247.2 ± 59.3 IU/L, $p = 0.016$), which suggests elevated bone turnover. Participants within the vitamin D deficient group also had a significantly higher weight and BMI standard deviation score (weight SDS, $p = 0.012$; BMI SDS, $p = 0.004$), which may suggest increased adiposity is related to lower availability of active vitamin D.

Table 2: Characteristics of participants according to vitamin D status				
Characteristic	**Total (n = 250)**	**Vitamin D deficiency (n = 142)**	**Vitamin D sufficiency (n = 108)**	**P-value[a]**
No. of participants	250 (100)	142 (56.8)	108 (43.2)	—
Serum 25(OH)D (ng/mL)	20.39 ± 7.79	14.9 ± 3.4	27.6 ± 5.8	<0.001
Sex				0.512
Male	92 (36.8)	56 (39.4)	36 (33.3)	
Female	158 (63.2)	86 (60.6)	72 (66.7)	
Age (yr)	8.9 ± 1.3	9.0 ± 1.2	8.7 ± 1.4	0.143
Season				<0.001
Spring (Mar–May)	42 (16.8)	34 (81.0)	8 (19.0)	
Autumn (Sep–Nov)	98 (39.2)	32 (32.7)	66 (67.3)	
Winter (Dec–Feb)	110 (44.0)	76 (69.1)	34 (30.9)	
Body weight				0.083
Normal (<85th percentile)	172 (68.8)	92 (64.8)	80 (74.1)	
Overweight (85th–95th percentile)	46 (18.4)	32 (22.5)	14 (13.0)	
Obese (≥95th percentile)	32 (12.8)	18 (12.7)	14 (13.0)	
Puberty				0.871
Normal	219 (87.6)	125 (88.0)	94 (87.0)	
Precocious puberty	31 (12.4)	17 (12.0)	14 (13.0)	
Calcium (mg/dL)	9.679 ± 0.367	9.61 ± 0.38	9.77 ± 0.33	0.00045

Phosphorus (mg/dL)	4.79 ± 0.54	4.74 ± 0.56	4.86 ± 0.51	0.112
ALP (IU/L)	258.4 ± 64.1	266.7 ± 67.8	247.2 ± 59.3	0.016
Weight SDS	0.49 ± 1.03	0.63 ± 1.01	0.30 ± 1.02	0.012
Height SDS	0.44 ± 1.02	0.52 ± 1.04	0.34 ± 0.99	0.083
BMI SDS	0.40 ± 1.04	0.58 ± 1.01	0.19 ± 1.06	0.004

Table 3 displays the logistic regression analysis revealing factors associated with vitamin D deficiency among the 250 participants in the study. Season was identified to be the most important indicator, with children assessed in the spring to have an 8.8 times significant increased risk (95% CI: 3.6-21.1, $p < 0.001$) and those in the winter having 4.6 times significant increased risk (95% CI: 2.6–8.3, $p < 0.001$), in comparison to children in autumn used as the referent. This pattern suggests a significant seasonal effect, likely driven by seasonal changes in outdoor activity and UV light exposure. Other factors including sex, body weight, and pubertal state, were not statistically significant after adjustment. Overweight children showed a trend toward increased odds of vitamin D deficiency (OR: 1.9; 95% CI: 1.0-3.8; $p = 0.097$), however this was not statistically significant. Female sex (OR: 1.1; 95% CI: 0.6-2.0; $p = 0.823$) and precocious puberty (OR: 0.9; 95% CI: 0.4-2.0; $p = 0.874$) did not significant association with vitamin D deficiency. These results suggest that lifestyle and environmental reasons, particularly season variance in sunlight, is a more important factor than biological reasons to achieve vitamin D status in children.

Table 3: Factors associated with vitamin D deficiency in logistic regression analysis

Variable	OR (95% CI)	Adjusted P-value[a]
Sex		0.823
Male	1 (reference)	
Female	1.1 (0.6 – 2.0)	
Season		<0.001
Spring (Mar–May)	8.8 (3.6 – 21.1)	
Autumn (Sep–Nov)	1 (reference)	
Winter (Dec–Feb)	4.6 (2.6 – 8.3)	
Body weight		0.097
Normal (<85th percentile)	1 (reference)	
Overweight (85th–95th percentile)	1.9 (1.0 – 3.8)	
Obese (≥95th percentile)	1.2 (0.6 – 2.4)	
Puberty		0.874
Normal	1 (reference)	
Precocious puberty	0.9 (0.4 – 2.0)	

Discussion

In the current investigation, we found a very high prevalence of vitamin D deficiency in children aged 6–12 years, where 56.8% of subjects had a mean serum 25(OH)D concentration of 20.39 ± 7.79 ng/mL which is consistent with recent studies among Asian children. For example. Lee et al. (2015) showed that Korean children and adolescents had a mean serum 25(OH)D concentration of 17.42 ± 8.95 ng/mL. Chung et al. (2014) found in a national sample of Korean children aged 4 – 15 years that 58.6% had a vitamin D deficiency. Other studies in other Asia locations such as China and Mongolia also show a high prevalence of deficiency that is exacerbated by the winter months due to low sunlight (See Foo et al., 2009; Fraser, 2004). These observations indicate that vitamin D deficiency is a common public health concern for many pediatric populations within similar latitudes."

In our study, the seasonal variation was very interesting as the highest prevalence of vitamin D deficiency was in spring (81.0%) and winter (69.1%) and only 32.7% in autumn. These findings are consistent with another study (Öhlund et al. (2013) [15]) that showed serum 25(OH)D levels in preschool children residing in Sweden's northern parts dropped dramatically in winter despite sufficient sunlight exposure during the summer. Similarly, other studies from France and the United States have shown similar seasonal drops (Guillemant et al., 1999; Weng et al., 2007) [16,17]. In our study, the association of vitamin D deficiency and season was strong (OR = 8.8 in spring, OR = 4.6 in winter), pointing to the importance of sun exposure in order to maintain adequate levels of vitamin D. As noted by other studies, in South Korea as well as other latitudes, effective UVB exposure generally occurs between approximately April and October, thus the high rate of deficiency in colder months is not surprising (DeLuca, 2004) [18]. This pattern could be exacerbated by decreased outdoor activity because of modern lifestyles and academic pressures. Lee et al. (2013) [11] previously reported that

Korean children spent less than four hours per week outdoors in winter, thus resulting in lower vitamin D synthesis.

We did not find gender differences in vitamin D status, although females had a slightly higher deficiency; this finding agrees with the study by Gordon et al. (2004) [19], which reported no significant difference in rates of deficiency between boys and girls. However, Foo et al. (2009) [13] found that adolescent girls in Beijing have one of the lowest levels in the world (12–14 ng/mL), perhaps due to cultural and behavioral factors affecting females' outdoor activities. Thus, as biochemical processes are the same for both sexes, the cultural and behavioral factors of a particular gender influence differences in vitamin D levels.

Body weight showed a marginal association with vitamin D deficiency in our analysis ($p = 0.097$), with overweight children being nearly twice as likely to be deficient (OR = 1.9) compared with those of normal weight. These findings support the hypothesis that adiposity has a negative impact on vitamin D bioavailability. Vitamin D is fat-soluble, and it can get sequestered in fat tissue, resulting in lower circulating 25(OH)D concentrations (Wortsman et al., 2000) [20]. Similarly, Bell et al. (1985) [21] demonstrated some changes in the vitamin D endocrine system in obese individuals. Aligning with the findings of Lee et al. (2013) [11], both weight SDS and BMI SDS were significantly higher for vitamin D–deficient participants in our data. Lee et al. (2013) [11] also reported that higher adiposity is inversely correlated with vitamin D levels. However, consistent with the logistic regression analysis of our study, obesity does not always remain a statistically significant independent risk factor for low vitamin D after adjustment for potential confounders such as season or sunlight exposure, as has been reported by another study (Chung et al., 2014 [12]).

Interestingly, despite the consistent association of vitamin D deficiency with negative skeletal and growth-related consequences, height SDS showed no significant difference between groups in our study, despite the fact that children who were deficient tended to have higher BMI SDS and ALP. The deficient participants actually had a higher level of ALP (266.7 ± 67.8 IU/L) than the sufficient participants (247.2 ± 59.3 IU/L), which demonstrates an increase in bone turnover, which is aligned with the direct effect of vitamin D on calcium homeostasis and mineralization of the bone. Moreover, significantly lower serum calcium in the deficient participants ($p=0.00045$) adds support to these physiological tenets. Our biochemical trends that reflect those of Akhtar (2016) also draw attention to the fact that children with low vitamin D levels may have higher levels of ALP and parathyroid hormone to compensate for the low state, all of which would indicate an abnormal response as a consequence of distressed bone metabolism.

The function of vitamin D in puberty remains unclear. In our analysis, we found no significant difference in pubertal status between groups classified as vitamin D deficient and sufficient, consistent with Carel and Léger (2008) [23] and Neely et al. (1995) [24] who reported that vitamin D status alone may not determine the onset of puberty. On the other hand, some studies indicate a potential association in an indirect manner. Vitamin D deficiency was noted to contribute to early menarche through associations with obesity in turn may affect hormone metabolism (Villamor et al., 2011) [25]. In consideration that we found 12.4% of our at participants with precocious puberty, future longitudinal studies may be able clarify if chronic deficiency is contributory.

Overall, the present results support the existence of vitamin D deficiency in children, especially at higher latitudes and during the months when sunlight is at a lower level. The 56.8% prevalence found in this study is consistent with previous studies conducted in Korea (Lee et al., 2015; Chung et al., 2014) [11,12] and relative to some reports from other temperate regions of the world (Öhlund et al., 2013; Gordon et al., 2004) [15,19]. Seasonal effect became the most important variable, while there was a small but biologically plausible effect for body weight. The differences in biochemistry between the deficient and sufficient groups demonstrate the role of vitamin D in skeletal health and metabolic health equilibrium. Public health strategies to promote vitamin D supplementation, safe sun exposure and dietary education are important to mitigate possible consequences of vitamin D deficiency in children for the long-term, especially in light of continuing to report levels adequate vitamin D below the recommended intake in many countries (Spiro & Buttriss, 2014) [26].

Conclusion

The high rates of vitamin D deficiency found in this study represent a practical issue for children's growth and overall health. The season of this study contributed quite a bit to the rates of vitamin D deficiency, which were highest in the winter and spring likely due to reduced exposure to sunlight and reduced synthesis of vitamin D. Sex, age, and pubertal stage were not strongly related to higher rates of vitamin D deficiency, but there was a trend that higher BMI children had reduced levels of vitamin D suggesting some form of relationship of body composition to vitamin D metabolism. Biochemical data suggested deficient children had lower calcium and higher alkaline phosphatase suggesting less effectiveness of bone mineralization and a higher turnover of bone / higher demand on the skeleton. This data reinforces the importance of preventing vitamin D deficiency and promoting sufficient vitamin D

status for optimal skeletal and metabolic health during development. Public health initiatives should promote outdoor activity, nutritional education, and supplementation during times of lower sunlight opportunities to improve vitamin D status and diminish negative health impact.

References

1. Wacker M, Holick MF. Vitamin D - effects on skeletal and extraskeletal health and the need for supplementation. Nutrients 2013; 5:111-48.
2. Holick MF. Vitamin D deficiency. N Engl J Med 2007;357: 266-81
3. Pettifor J, Daniels E, Feldman D. Vitamin D deficiency and nutritional rickets in children. In: Feldman D, Glorieux FH, Pike JW, editors. Vitamin D. San Diego: Academic Press. 1997:663-78
4. Verstuyf A, Carmeliet G, Bouillon R, Mathieu C. Vitamin D: a pleiotropic hormone. Kidney Int 2010; 78:140-5.
5. Temmerman JC. Vitamin D and cardiovascular disease. J Am Coll Nutr 2011; 30:167-70.
6. Searing DA, Leung DY. Vitamin D in atopic dermatitis, asthma and allergic diseases. Immunol Allergy Clin North Am 2010; 30:397-409.
7. Chew A, Harris SS. Does vitamin D affect timing of menarche? Nutr Rev 2013; 71:189-93.
8. Absoud M, Cummins C, Lim MJ, Wassmer E, Shaw N. Prevalence and predictors of vitamin D insufficiency in children: a Great Britain population-based study. PLoS One 2011;6: e22179
9. Munns CF, Simm PJ, Rodda CP, Garnett SP, Zacharin MR, Ward LM, et al. Incidence of vitamin D deficiency rickets among Australian children: an Australian Paediatric Surveillance Unit study. Med J Aust 2012; 196:466-8.
10. Ross AC, Manson JE, Abrams SA, Aloia JF, Brannon PM, Clinton SK, et al. The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: what clinicians need to know. J Clin Endocrinol Metab 2011; 96:53-8.
11. Lee DY, Kwon AR, Ahn JM, Kim YJ, Chae HW, Kim DH, et al. Relationship between serum 25-hydroxyvitamin D concentration and risks of metabolic syndrome in children and adolescents from Korean National Health and Nutrition Examination survey 2008-2010. Ann Pediatr Endocrinol Metab 2015; 20:46-52.
12. Chung IH, Kim HJ, Chung S, Yoo EG. Vitamin D deficiency in Korean children: prevalence, risk factors, and the relationship with parathyroid hormone levels. Ann Pediatr Endocrinol Metab 2014;19:86-90.
13. Foo LH, Zhang Q, Zhu K, Ma G, Trube A, Greenfield H, et al. Relationship between vitamin D status, body composition and physical exercise of adolescent girls in Beijing. Osteoporos Int 2009; 20:417-25.
14. Fraser DR. Vitamin D-deficiency in Asia. J Steroid Biochem Mol Biol 2004;89-90:491-5.
15. Öhlund I, Silfverdal SA, Hernell O, Lind T. Serum 25-hydroxyvitamin D levels in preschool-age children in northern Sweden are inadequate after summer and diminish further during winter. J Pediatr Gastroenterol Nutr 2013; 56:551-5
16. Guillemant J, Taupin P, Le HT, Tarıght N, Allemandou A, Pérès G, et al. Vitamin D status during puberty in French healthy male adolescents. Osteoporos Int 1999; 10:222-5.
17. Weng FL, Shults J, Leonard MB, Stallings VA, Zemel BS. Risk factors for low serum 25-hydroxyvitamin D concentrations in otherwise healthy children and adolescents. Am J Clin Nutr 2007; 86:150-8.
18. DeLuca HF. Overview of general physiologic features and functions of vitamin D. Am J Clin Nutr 2004;80(6 Suppl):1689S-1696S.
19. Gordon CM, DePeter KC, Feldman HA, Grace E, Emans SJ. Prevalence of vitamin D deficiency among healthy adolescents. Arch Pediatr Adolesc Med 2004; 158:531-7.
20. Wortsman J, Matsuoka LY, Chen TC, Lu Z, Holick MF. Decreased bioavailability of vitamin D in obesity. Am J Clin Nutr 2000; 72:690-3.
21. Bell NH, Epstein S, Greene A, Shary J, Oexmann MJ, Shaw S. Evidence for alteration of the vitamin D-endocrine system in obese subjects. J Clin Invest 1985; 76:370-3
22. Akhtar S. Vitamin D status in South Asian populations: risks and opp or tunit ies. Crit R e v Fo o d S ci Nut r 2016;56:1925-40.
23. Carel JC, Léger J. Clinical practice: precocious puberty. N Engl J Med 2008; 358:2366-77
24. Neely EK, Wilson DM, Lee PA, Stene M, Hintz RL. Spontaneous serum gonadotropin concentrations in the evaluation of precocious puberty. J Pediatr 1995; 127:47-52.
25. Villamor E, Marin C, Mora-Plazas M, Baylin A. Vitamin D deficiency and age at menarche: a prospective study. Am J Clin Nutr 2011; 94:1020-5.
26. Spiro A, Buttriss JL. Vitamin D: an overview of vitamin D status and intake in Europe. Nutr Bull 2014; 39:322-50.