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REVIEW ARTICLE

A reviewed approach to vitamin D supplementation in pediatric age in Portugal

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Abstract

Vitamin D deficiency is a well-established cause of rickets in children and has been reported as associated to impacts on other systems and organs. Vitamin D deficiency and insufficiency are pediatric worldwide global health problems. Although data on the prevalence of vitamin D deficiency and insufficiency in the Portuguese pediatric population are scarce, data for Portuguese subpopulations and equivalent regions allows us to speculate that the prevalence of vitamin D deficiency in Portugal should be also relevant. The current article focuses on vitamin D supplementation at pediatric age in Portugal reviewing the prevalence data of vitamin D insufficiency/deficiency for Portugal, the main groups and the risk factors for vitamin D deficiency and educational measures and vitamin D supplementation recommendations for different risk factors/groups.

Keywords: Adolescent. Child. Dietary supplements. Vitamin D deficiency.

Revisão à abordagem de suplementação de Vitamina D em idade pediátrica, em Portugal

Resumo

A deficiência de vitamina D é uma causa bem estabelecida de raquitismo em crianças e tem sido associada a compromisso funcional de outros órgãos e sistemas. A deficiência e insuficiência de vitamina D são um problema de saúde global, muito comum em crianças de todo o mundo. Embora os dados sobre a prevalência da deficiência/insuficiência de vitamina D na população pediátrica portuguesa sejam escassos, os dados relativos às subpopulações portuguesas e regiões equivalentes permitem-nos especular que a prevalência da deficiência de vitamina D em Portugal deverá ser relevante. O presente artigo centra-se na suplementação com vitamina D em idade pediátrica em Portugal, revendo os dados de prevalência da insuficiência/deficiência em vitamina D em Portugal, os principais grupos e os factores de risco de deficiência de vitamina D, bem como as medidas educativas e recomendações de suplementação de vitamina D para os diferentes factores/grupos de risco.

Palavras-chave: Adolescentes. Crianças. Deficiência de vitamina D. Suplementação alimentar.

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Introduction

Vitamin D plays a key role in bone and muscle health due to its interference in calcium metabolism and consequent role in bone formation. Vitamin D deficiency is a well-established cause of rickets in children and of osteomalacia in adults. Other effects associated with vitamin D deficiency on other organs and systems have also been reported. However, the available literature refers mostly to adult populations and pediatric data are often contradictory or inconclusive[1].

Clinical research on vitamin D has been stimulated by the discovery that many human cell types carry the vitamin D receptor. This receptor may play a role in the regulation of cell proliferation and differentiation, for example, in cells of the immune system as T cells, macrophages or monocytes. In children and adolescents, additional health effects of vitamin D have been proposed due the plethora of vitamin D functions, as well as data from observational studies on the association between vitamin D status and diseases. These additional effects of vitamin D may include prevention of immune-related diseases (asthma, type 1 diabetes mellitus, multiple sclerosis), prevention of infectious diseases (e.g., respiratory infections) and prevention of cardiovascular diseases and cancer. Infants and children with severe vitamin D deficiency and rickets may also present delayed motor development, muscle hypotonia, and weakness[1-3].

Vitamin D deficiency and insufficiency are a global health problem, very common in infants and children worldwide[4,5]. Data on the prevalence of vitamin D deficiency and insufficiency in the Portuguese pediatric population are scarce. However, regional data available for pediatric populations of Portugal[6,7] and data for other Mediterranean countries[2,8], allows us to speculate that the prevalence of vitamin D deficiency in Portugal can be higher.

In this scenario, this review article will focus on vitamin D supplementation in pediatric age in Portugal and will, therefore: 1) review the prevalence data of vitamin D insufficiency/deficiency available for Portuguese pediatric populations and/or other Mediterranean countries; 2) review and document the main groups and the risk factors for vitamin D deficiency and the mechanism responsible for the deficit in each case; 3) review educational measures and vitamin D supplementation recommendations according to the age of the child/adolescent and the presence (or absence) of risk factors/groups.

Vitamin D biomarkers, reference values and prevalence data of vitamin D deficiency/insufficiency in pediatrics in Portugal

Vitamin D, also designated as calciferol, is a pre-hormone obtained by dietary sources but mainly formed by sun exposure of the skin (almost 90% of our needs). In the liver, vitamin D suffers metabolization to an inactive form designated as 25-hydroxyvitamin D [25(OH)D] or calcidiol. This inactive form is activated at the renal level into 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] or calcitriol, which plays a critical role in calcium and mineral homeostasis. 25(OH)D, of the overall circulating vitamin D metabolites, is the established biomarker for assessing vitamin D status, because it is the most abundant form, it has the longest half-life, and it reflects both skin synthesis and dietary intake. Furthermore, it shows greater sensitivity to variations in vitamin D status of individuals, serving not only as a status indicator of this micronutrient but also as the main storage form in the body[6,9].

Definition of vitamin D status and reference values are not consensual between different committees or societies. For children, the Direção-Geral da Saúde (DGS - the Portuguese General Directorate for Health) establishes vitamin D deficiency for plasmatic concentration of 25(OH)D lower than 12 ng/mL (30 nmol/L) and insufficiency when plasmatic concentration lies between 12-20 ng/mL (30-50 nmol/L). For adult age, established values are different and vitamin D deficiency was defined for plasmatic concentration of 25(OH)D lower than 20 ng/mL (50 nmol/L) and insufficiency if it lies between 20-30 ng/mL (50-75 nmol/L)[10].

On the other hand, the European Society for Pediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) does not use the concept of insufficiency and define deficiency and severe deficiency of vitamin D for plasmatic concentration of 25(OH)D between 10-20 ng/mL (25-50 nmol/L) and lower than 10 ng/mL (25 nmol/L), respectively[1].

Considering the reference values of DGS and ESPGHAN, there is no consensus on the characterization of adequate vitamin D levels, particularly with regard to cut-off points for defining insufficiency/deficiency. However, it is agreed that serum levels below 20 ng/mL (50 nmol/L) indicate children at risk. While describing the prevalence of vitamin D deficiency in the following paragraphs, plasmatic values of 25(OH)D are used to avoid ambiguous classification.

As previously mentioned, data on the prevalence of vitamin D deficiency in Portuguese pediatric populations are very limited. A study conducted in a northern region of Portugal (Porto region) (n = 67; 5-18 years), showed that 41.8% presented serum values of 25(OH)D between 10 ng/mL and 20 ng/mL, and 6% below 10 ng/mL, indicating that almost half of the sample (47.8%) suffered from vitamin D deficiency/insufficiency[5]. A study done to assess vitamin D levels and cardiometabolic risk factors

in the same region showed mean serum vitamin D levels below the recommendations (16.52 +/–5.72 ng/mL) (514 subjects) for adolescents (13 years old)[7].

In adults, a study conducted to give an overall epidemiological picture of vitamin D status in the Portuguese population (n = 3092) estimated 25(OH)D levels < 20 ng/ml and < 30 ng/ml to affect 66.6%, and 96.4% of the population, respectively, with strong geographical and seasonal variation[3].

A recent systematic review on pediatric populations covering over 107 studies (representing 630,093 individuals) showed that in the Southern Europe and Eastern Mediterranean regions, more than one-third of the studies reported mean 25(OH)D levels < 20 ng/mL across all population subgroups, but especially among neonates/infants and adolescents[8]. A study conducted in Spain (a neighbor country of Portugal) in the sunny region of Valencia (n = 169) with children aged under two, found 8.3% with plasmatic levels of 25(OH)D under 20 ng/mL[2].

Risk groups and factors for vitamin D deficiency in the pediatric populations

This section identifies groups of children/adolescents with a higher predisposition or risk of developing vitamin D deficiency/insufficiency, also identifying the mechanism that leads to the possible deficiency. All the information is summarized in table 1.

Endogenous production of vitamin D after exposure to natural sunlight is the most important source of vitamin D. Thus, lack of natural sunlight exposure is one of the most relevant risk factors for vitamin D deficiency, with the geographic location (latitude) having an important role[11]. Lack of sun exposure is associated with lifestyle (clothing habits, cultural and religious habits, sedentarism and long indoor permanence) but may also be associated with conditions or pathologies that impair sun exposure. For example, dark-skinned individuals (photo-type V) require about 3 to 5 times more time of sun exposure to synthesize the same amount of vitamin D, in comparison to a person with a photo-type II[12]. Conditions such solar urticaria, vitiligo or chemical photosensitivity (for example, acne under isotretinoin) have strong indication to avoid sun exposure. Last, but not least, the use of high protection factor sunscreen (above 30) significantly reduces the capacity for vitamin D synthesis (~ 95%, when used in the recommended amount and frequency)[13].

Vitamin D deficiency is also a consequence of reduced dietary intake of vitamin D or of variabilities in absorption/distribution/metabolization/excretion of the vitamin, in a variety of different situations in children and adolescents.

Preterm newborns have a high risk of vitamin D deficiency due to maternal vitamin D supply deprivation (early birth inhibits the fetus to store adequate levels of vitamin D obtained essentially via maternal source) and potential exposure to additional risk factors such as long-term parenteral nutrition use, intolerance to human milk fortifiers and formulas and neonatal cholestasis[14]. A study showed that about 65% of preterm infants with less than 32 weeks of gestation had vitamin D deficiency and associated with this deficiency, an increased risk of prematurity-related bone disease (which affects up to 55% of preterm infants weighing < 1000 g)[15].

Children who did not comply with the universal supplementation of 400 IU per day of vitamin D in the first year of life are also considered a risk group for vitamin D deficiency. The importance of universal, early and regular supplementation is related to: 1) increased vitamin deficiency in the general population and in pregnant women; 2) often unknown status of vitamin D deficiency in pregnant women; 3) reported vitamin D deficiency in the first month of life (both for mother and child), particularly in breastfeeding (around 50% in the woman [insufficiency plus deficiency] and 80% in the newborn [deficiency])[16]; 4) medical recommendation to avoid direct sun exposure of children in the first year of life (due to higher risk of skin cancer and premature skin aging)[17]. As this avoidance of direct sun exposure is recommended at least until 2 years old, some scientific societies recommend the universal supplementation until 18-24 months of age[18].

A study that interviewed caretakers of approximately 1% of the Portuguese children with ages comprised between 12-36 months, showed lack of compliance of vitamin D supplementation in the first year of life in about one-third of the cases (31.7%)[15], although universal supplementation of vitamin D is recommended by DGS and ESPGHAN for this period[9]. In the United States of America prevalence of lack of compliance of recommendations is almost the same, with nearly one third of infants not meeting American Academy of Pediatrics (AAP) recommendations[19].

Breastfeeding infants are also at risk of vitamin D deficiency since breast milk contains very low amounts of vitamin D. Considering an intake of 150 ml/kg/day, the amount of vitamin D obtained by a breastfeeding an infant through breast milk is only 30 IU/day[20].

Obese children and adolescents are also at a higher risk of vitamin D deficiency because vitamin D, a fat-soluble vitamin, is sequestered by the higher amount of adipose tissue[11] and because body and serum volume are higher in these individuals, causing a dilution effect[21].

Table 1. Summary of risk groups for vitamin D deficiency, associated mechanism and recommendations

Risk groups	Mechanism		Recommendation
Children/adolescents living in lifestyle contexts with limited sun exposure: – Clothing habits – Cultural/religious habits – Sedentarism – Long indoor permanence	Limited sun exposure reduces the endogenous production of vitamin D (which is the most important source of vitamin D).	Low endogeneous production	Educational measures. If educational measures are not successful/possible, supplement with 600 IU/day of vitamin D (autumn-spring). Routine 25(OH)D determination not recommended.
Children/adolescents with conditions or pathologies which do not allow for sun exposure, e.g: – Children/adolescents with high phototypes – Children/adolescents with solar urticaria, vitiligo and chemical photosensitivity	Dark-skinned individuals require about 3 to 5 times more time of sun exposure to synthesize the same amount of vitamin D, in comparison to person with a lower phototype. Limited sun exposure reduces the endogenous production of vitamin D.	Low endogeneous production	
Children/adolescents with use of high protection factor sunscreen (above 30)	Sunscreen (above 30) significantly reduces the capacity for vitamin D synthesis (~ 95%, when used in the recommended amount and frequency).	Low endogeneous production	
Pre-term newborns	Maternal vitamin D supply deprivation. Other factors such as: – Long-term parenteral nutrition use – Intolerance to human milk fortifiers and formulas – neonatal cholestasis	Low intake Low reserve Low absorption	Supplementation with 800-1000 IU/day of vitamin D.
Infants in the 1st year of life Children who did not comply with the universal supplementation in 1st year of life	Maternal vitamin D deficiency. Unknown status of vitamin D in pregnant women. Reported vitamin D deficiency in the first month of life (both for mother and child). Recommendations to avoid direct sun exposure of children in the first year of life.	Low intake Absence of cutaneous synthesis	Universal supplementation with 400 IU/day of vitamin D.
Breastfeeding infants	Breast milk contains very low amounts of vitamin D.	Low intake	If in the 1st year of life, universal supplementation with 400 IU/day of vitamin D.
Obese children/adolescents	Vitamin D is sequestered by the higher amounts of adipose tissue. Dilution effect to higher body and serum volume.	Distribution variability	Consider supplementing with 600 IU/day of vitamin D at least from autumn to spring.
Children/adolescents following certain diets (such as veganism or macrobiotics). Nutritionally inadequate diets	Low intake due to dietary restrictions or inadequate diet.	Low intake	
Children/adolescents with a known history of congenital or acquired metabolic disorders of calcium and vitamin D metabolism	Deficiencies in enzymes responsible for metabolization to the active for of vitamin D (p.e. 1α-hydroxylase deficiency). Vitamin D resistance (receptor defect).	Impaired metabolism Receptor deficiency	Determine 25(OH) D basal levels. If levels are deficient supplement with 600 IU/day (vitamin D deficiency) 1000-2000 IU/day (severe vitamin D deficiency)

(Continues)

Table 1. Summary of risk groups for vitamin D deficiency, associated mechanism and recommendations *(continued)*

Risk groups	Mechanism		Recommendation
Children/adolescents with chronic diseases, such as:			Always respect the maximum allowed daily doses for vitamin D: – 1000 IU/day < 6 months – 1400 IU/day: 6-12 months – 2000 IU/day: 12 months-12 years – 4000 IU/day: > 12 years Conduct a new 25(OH)D determination after 3-6 months and adjust supplementation if necessary. Adapt supplementation to specific situations: – In hepatic cholestasis: 1300-2200 IU/day – In liver failure: supplement with 25(OH)D – In chronic kidney disease: supplement with 25(OH)2D (as in congenital or acquired metabolic disorders or chronic diseases)
Malabsorption syndromes	Cause reduction the intestinal absorption of vitamin D.	Low absorption	
Inflammatory bowel disease	Might be associated to reduced absorption and increased excretion of vitamin D.	Low absorption Increased excretion	
Hepatic cholestasis	Causes absorption disturbances of vitamin D.	Low absorption	
Liver failure	Compromises the first hydroxylation mechanism, which converts vitamin D into 25(OH)D.	Impaired metabolism	
Chronic kidney disease	Causes increased urinary excretion of 25(OH)D, before it is converted into the active form of vitamin D.	Increased excretion	
Adolescent/children under chronic/prolonged treatments with:			
Anticonvulsants – Antiretrovirals	These drugs increase the metabolism of 25(OH)D and 1,25(OH)2D.	Increased metabolization	
Systemic corticoids	Antagonize the effect of vitamin D and can lead to hypocalcemia.	Antagonization mechanism	
Anti-fungals	Inhibit the enzymes responsible for metabolization to the active for of vitamin (hydroxylases).	Impaired metabolization	
Children/adolescents health conditions or tests suggestive of vitamin D deficiency: – Bone disease (rickets) – Non-traumatic fractures – Known history of vitamin D deficiency – Low 25(OH)D levels – Low urinary calcium excretion in 24h urine – Elevated parathyroid hormone or bone alkaline phosphatase levels – Bone mass compromise for age (z score for bone mineral density (BMD) < –2)	-	-	Nutritional rickets in children 12 months-10 years: 2000 IU/day for 3 months. Low 25(OH) D levels: Vitamin D deficiency: 600 IU/day Severe vitamin D deficiency: 1000-2000 IU/day

Another risk groups for vitamin D deficiency are children and adolescents following certain diets (such as veganism or macrobiotics or inadequate diets), which include limited sources of vitamin D (see list of foods with high vitamin D content in Table 2)[11,22,23].

Children/adolescents with a known history of congenital or acquired metabolic disorders of calcium and vitamin D metabolism are also a risk group. Examples include changes that compromise the availability of the active form of vitamin D (1.25$(OH)_2$D or calcitriol) such as deficiency in 1α-hydroxylase, the enzyme responsible for the second hydroxylation of 25(OH)D (calcidiol) into 25$(OH)_2$D (calcitriol). Another risk group includes individuals with hereditary resistance to vitamin D, which is characterized by a defect in vitamin D receptors. In these individuals, vitamin D levels are normal, calcitriol levels are high, yet there is resistance to vitamin D action due to unavailability/deficiency of its receptors[24].

Several chronic pathologies may be linked to vitamin deficiency due to disrupted absorption, metabolization

or excretion mechanisms. Examples include: 1) malabsorption syndromes, in which the intestinal absorption of vitamin D is reduced; 2) inflammatory bowel disease, which might be associated to reduced absorption and increased excretion of vitamin D; 3) hepatic cholestasis, which impairs vitamin D absorption; 4) liver failure, which might compromise the first hydroxylation mechanism, and which converts vitamin D into 25(OH)D; 5) chronic kidney disease which causes increased urinary excretion of 25(OH)D before it is converted into the active form of vitamin D[11,25].

Chronic or prolonged treatments with specific drugs might also be a risk factor for vitamin deficiency in children or adolescents. These include: 1) anticonvulsants and antiretrovirals, since these drugs increase the metabolism of 25(OH)D and 1,25(OH)2D; 2) systemic corticoids, which antagonize the effect of vitamin D and can lead to hypocalcemia; 3) anti-fungals, which inhibit the enzymes responsible for metabolization to the active for of vitamin (hydroxylases)[11,26]. Additionally, children/adolescents with health conditions or tests suggestive of vitamin D deficiency should also be considered at risk. These include children/adolescents with bone disease, non-traumatic fractures or with a known history of vitamin D deficiency, and indicators, such as low urinary calcium excretion in 24h urine, elevated parathyroid hormone or bone alkaline phosphatase levels and bone mass compromise for age (z score for bone mineral density (BMD) < –2)[10].

Table 2. Vitamin D content in food[11,22,23]

Food	Amount of food	Vitamin D content/IU*
Salmon (wild)	100 g	600-1000
Salmon (farmed)	100 g	100-250
Sardines (tinned)	100 g	300
Sardines (fresh)	100 g	280-320
Mackerel (wild)	100 g	104
Mackerel (tinned)	100 g	250
Herring (fresh)	100 g	215
Tuna (fresh)	100 g	292
Tuna (tinned)	100 g	236
Codfish	100 g	44
Eggs† (chicken bred with sun exposure)	1 egg	148
Eggs† (aviary chicken)	1 egg	32
Shitake mushrooms (dried)	100 g	1660
Cow milk	100 g	4
Yogurt	100 g	2,4
Cheese (varies according to type of cheese)	100 g	6,8-80

*The amount of vitamin D reported for each type of food may vary in the literature.
†Vitamin D exists in egg yolk.

Educational measures for the prevention of vitamin D deficiency in children and adolescents

Health education directed at pediatric age and their caregivers will be essential to prevent vitamin D deficiency/insufficiency and is an attempt to avoid pharmacological measures. Educational measures include recommendations about diet and sun exposure.

Nutritional advice should include the recommendation to ingest foods rich in vitamin D, such as: 1) oily fish (e.g. salmon, sardines, mackerel, herring, tuna, codfish); 2) eggs (yolk); 3) mushrooms; 4) dairy products (e.g. milk, yogurt, cheese), preferably fortified. However, the practice of food fortification in Portugal is not very common and, when available, fortified products contain relatively low amounts of vitamin D. Table 2 lists vitamin D-rich foods and their respective content of vitamin D[11, 22,23].

Dermatologic recommendations about sun exposure should include advice about moderate and regular exposure to natural sunlight for short periods, taking into consideration the dermatology recommendations for the prevention of neoplasms. Infants (< 12 months) and young children at least until 2 years old must avoid direct sun exposure[27]. An adequate exposure for vitamin D production seems to correspond to exposing face, hands and part of the arms, without the use of sunscreen, for 10 to 15 minutes a day (before the skin turns red), two or three days a week, in spring, summer and autumn[22].

Vitamin D supplementation recommendations according to the age of the child/adolescent and the presence (or absence) of risk factors/groups

The recommendations described in this section are summarized in table 1. For children/adolescents with low sun exposure, educational measures should be implemented (diet and sun exposure) and the levels of 25(OH)D should not be determined regularly. Supplementation should be considered from autumn to spring, especially if educational measures are not successful. Supplementation should be conducted according to established daily doses, namely 600 IU/day from

the 2nd year of life, always respecting the maximum allowed daily doses of 2000 IU/day for children aged from 12 months to 10 years and 4000 IU/day for children older than 10 years[1,28,29].

Vitamin D supplementation is already recommended in certain groups or clinical framings. In preterm newborns the recommended supplementation is 800-1000 IU/day. In preterm newborns with rickets or with high alkaline phosphatase levels, higher doses of vitamin D are recommended, with a maximum of 1000 IU/day[20]. In term infants a universal prophylactic supplementation is recommended during the first year of life with 400 IU/day of vitamin D[10, 20]. Those with dark skin or living in a high latitude (> 40º) may need a higher dose (800 IU/day)[30]. Children aged from 12 months to 10 years with confirmed diagnosis of nutritional rickets (based on a clinical history, physical examination, biochemical tests and confirmed by radiological study) should be supplemented with 2000 IU/day during 3 months. When vitamin D deficiency is confirmed by determination of 25(OH)D levels, children/adolescents should be supplemented with 600 IU/day. In severe deficiency supplementation of 1000-2000 IU/day should be implemented[10].

Other groups should also be considered for supplementation. For obese children/adolescents and children/adolescents following low vitamin D diets (veganism, macrobiotics or nutritionally inadequate diets) a supplementation of 600 IU/day, at least from autumn to spring, may be considered. In the case of obese children/adolescents, higher doses may be needed due to the sequestration of vitamin D in the adipose tissue[11]. In the case of weight loss, vitamin D liberation from the adipose tissue should be taken into account for dose adjustment.

For children/adolescents with: 1) a known history of metabolic disorders of calcium and vitamin D; 2) chronic diseases that disturb vitamin D absorption, metabolism and excretion; 3) under chronic or prolonged treatment with drugs that interfere in vitamin D availability; 4) known history of vitamin D deficiency and with other health conditions or tests indicative of vitamin D deficiency[10], the clinician should start by evaluating 25(OH)D basal levels. The initial supplementation should be defined according to the basal level of 25(OH)D. Children/adolescents should be supplemented with 600 IU/day (for vitamin D deficiency) and 1000-2000 IU/day (for severe vitamin D deficiency) always considering the maximum allowed daily dose: 1000 IU/day for children < 6 months; 1400 IU/day for children 6-12 months; 2000 IU/day for children aged from 12 months to 12 years; 4000 IU/day for children > 12 years[28,29]. The doses should be readjusted, if necessary, after a new evaluation of 25(OH)D levels and comparison with the basal levels, 3-6 months after the initial evaluation. In certain diseases, the supplementation should be done by resorting to alternative ways. In the case of renal failure, the supplementation should be conducted with $25(OH)_2D$ or calcitriol, due to the accelerated excretion mechanism of 25(OH)D under these conditions. In the case of liver failure, the supplementation should be done directly with 25(OH)D or calcidiol, since as described above, the hydroxylation mechanism from vitamin D to 25(OH)D is impaired. Micellar or hydro-soluble formulations should be preferred. In hepatic cholestasis, vitamin D absorption is disturbed and, therefore, supplementation with higher doses is recommended (1300-2200 IU/day)[20].

Conclusions

Vitamin D plays a key role in bone and muscle health. Other effects associated with vitamin D deficiency on other systems and organs have also been reported. There is no consensus on the characterization of vitamin D status compromise, particularly with regard to cut-off points for defining insufficiency/deficiency. However, it is agreed that serum levels below 20 ng/mL (50 nmol/L) indicate individuals at risk.

Data on the prevalence of vitamin D deficiency and insufficiency in the Portuguese pediatric population are non-existent. However, regional data and data for other Mediterranean countries allows us to speculate that the prevalence of vitamin D deficiency is a relevant challenge in our country.

Vitamin D supplementation should be universal in the first year of life, and there is epidemiological and scientific support for extending this recommendation to the end of the second year. It is mandatory to check this accomplishment during all clinical interviews. In the child health consultation, particular attention should be given to the early identification of children/adolescents at risk, in order to diagnose early and prevent vitamin D deficiency/insufficiency. Health education directed at pediatric age and their caregivers will be essential to prevent vitamin D deficiency/insufficiency and is an attempt to avoid pharmacological measures. Educational measures include recommendations about diet and sun exposure. There are risk groups in which prophylactic supplementation should be considered, in the autumn, winter and spring months. Plasmatic 25(OH)D determination and supplementation is recommended only in well-defined pathological situations.

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Conflicts of interest

None.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

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Right to privacy and informed consent. The authors declare that no patient data appear in this article.

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