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EDITORIAL

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Dear Readers,
In this issue we are returning to the subject of childhood vitamin D deficiency, the prevalence of which has begun to rise again in some countries, as has been documented even in the UK. According to the article by the author whom we asked to prepare an update on this topic, certain risk factors such as ethnic composition and overweight have changed and become more frequent.
Recently, in Europe, there has been an increase in populations with darker skin and different cultural habits such as wearing full-body garments for religious reasons. Both of these factors are known to hinder endogenous vitamin D production by reducing exposure to sunlight. Moreover, the prevalence of overweight has risen to include 30% of the paediatric population. This condition is also a significant risk factor for vitamin D deficiency since it is known that persons with obesity require supplementation with higher doses. Therefore, to provide for adequate bone development, but also plausible extra-skeletal benefits, it should be remembered that it important to ensure adequate vitamin D status in children during all stages of growth. This requires supplementation, especially in the first years of life, and particularly if the mother was vitamin D deficient during pregnancy.
The author has further emphasised that in a paediatric age it is likely that high bolus doses can induce the expression of catabolic enzymes that inactivate vitamin D, which we also hypothesised based on the results of our recent pharmacokinetics study [1]. Thus, vitamin D supplements should be taken daily rather than in monthly or weekly bolus doses. In an earlier issue of this journal [2], we also provided pharmacokinetic and pharmacodynamic justification in support of the exclusive benefits, and especially extra-skeletal advantages, conferred by daily administration of vitamin D [3].
For the other article, I asked the authors to focus on a possible correlation between vitamin D deficiency and complex regional pain syndrome (CRPS), also termed algodystrophy. This is because it was recently shown that patients with distal radius fractures complicated by CRPS had significantly lower plasma concentrations of vitamin D than those who had not experienced this complication. Based on the available evidence, as you will see, the authors acknowledge that vitamin D deficiency may lead to an increased risk of CRPS for essentially two reasons. The first is because deficiency may lead to an increase in fracture events, especially intra-articular fractures, which tend to induce the syndrome, and which can also be due to the associated risk of falling. The second is likely related to the fact that vitamin D deficiency is a predisposing condition for neuroinflammation and proinflammatory immunological status, both of which are involved in the pathogenesis of CRPS.
As evidence of a possible causal link between vitamin D levels and proinflammatory cytokines, some investigations have demonstrated that vitamin D supplementation is able to reduce serum concentrations of TNF-α and IL-6, as well as IL-17 [3]. Thus, there arises the

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hypothesis that vitamin D supplementation may contribute to an additional or faster benefit than the recognised approach to treating algodystrophy with neridronate. It is likely that vitamin D supplementation also helps to reduce the side effects of aminobisphosphonates in the acute phase, since these would be modulated by serum levels of 25(OH)D [4].

What are your thoughts?

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Vitamin D deficiency and Complex Regional Pain Syndrome

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To consider the possible relationships between vitamin D and algodystrophy syndrome, i.e. complex regional pain syndrome (CRPS), one must understand that bone tissue is a key player in the pathogenetic dynamics of the syndrome [1]. In addition to the results obtained by treatment with drugs whose mechanism of action involves bone tissue as their main target, there is also much evidence supporting the fundamental role that bone has in the onset and maintenance of the disease. Aside from findings arising from diagnostic testing (osteoporosis on standard radiology, hypercaptation on scintigraphy scans, bone oedema on magnetic resonance imaging), epidemiological studies have reported that fracture is the most frequent predisposing event. Consequently, all diseases that lead to an increase in bone fragility and therefore to the incidence of fractures (postmenopausal and senile osteoporosis, osteogenesis imperfecta) are often exacerbated by an increase in the incidence of algodystrophy. As further proof of this pathogenetic link, there are reports showing that osteoporosis is present in patients with CRPS at a significantly higher prevalence than in the general population [2]. Additionally, an animal model that closely reproduces human disease can been obtained by inducing a distal fracture of the tibia. Lastly, it is worth mentioning that an increase in osteoprotegerin (OPG), the molecule involved in the regulation of the RANK/RANKL system, has been implicated in the early stages of the disease.

As previously reported, in most cases, it has been ascertained that a traumatic fracture event is the most frequent predisposing factor for CRPS. In addition, the most reliable epidemiological findings [3] have shown that the peak incidence of distal radius fractures, i.e. the fracture event that is most often complicated by CRPS [4], among females and in the decade following menopause, is likely to be reflective of a similar trend within the general population. Data on the incidence following distal radius fractures in the literature vary widely (ranging from 1% to 37%). This variability can undoubtedly be attributed to the different diagnostic criteria used to document these events. The most recent studies using the diagnostic criteria adopted by the International Association for the Study of Pain (IASP), i.e. Budapest criteria, which have been recognised to be the best in terms of sensitivity and specificity, report that CRPS is present in 14% of patients who have suffered distal radius fractures [4].

It has been widely acknowledged that this type of fracture is the earliest clinical event related to osteoporosis, in that it occurs, on average, 15 years before proximal femur fractures, and that it is also a predictive event for other fragility fractures, namely vertebral fractures and proximal femur fractures [5]. Among the many clinical variables identified as being predictive of distal radius fractures is vitamin D deficiency [6]. Therefore, beginning from the premise that adequate vitamin D levels are essential for good bone health, researchers have investigated whether vitamin D deficiency might be why deficient subjects, who would clearly be more prone to fragility fractures, are more likely to be affected by CRPS.

Another aspect under investigation is whether vitamin D deficiency can, in the presence of a fracture event and independently of other variables, favour the onset of CRPS. Distal radius fracture (Colles' fracture) has been the most extensively investigated fracture event. In a retrospective orthopaedic study in 2020 of more than 100 postmenopausal women, those who experienced the onset of CRPS after a distal radius fracture had significantly lower levels of plasma vitamin D than those without CRPS (Fig. 1) [7]. It is compelling to point out that biochemical markers for bone turnover (i.e., osteocalcin and alkaline phosphatase), as well as bone density assessments carried out on both the lumbar

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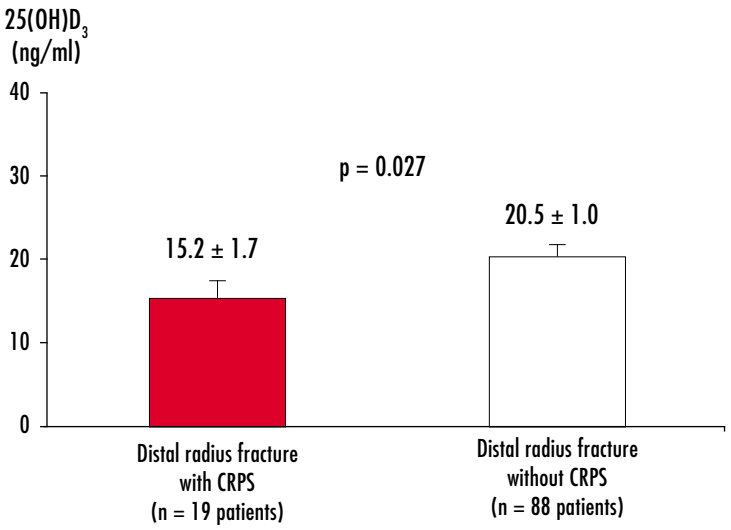

FIGURE 1.
Comparison of levels of $25(OH)D_3$ in 19 patients who developed CRPS after a distal radius fracture and 88 patients who did not develop CRPS.

spine and proximal femur, showed no significant differences between patients who developed CRPS and those who did not, even if they had the same fractures and the same surgical treatments.

From a theoretical standpoint, the results of the study have opened the door to a number of likely pathogenetic possibilities that link low levels of vitamin D to CRPS. First, bone density investigations showed no significant differences in the occurrence or non-occurrence of CRPS. This makes it possible to hypothesise that osteoporosis defined by bone density alone does not represent a risk factor for CRPS. On the other hand, the above epidemiological considerations are consistent with an indirect role of osteoporosis: the presence of low bone mass values might be considered the reason for which subjects with osteoporosis more frequently experience a predisposing event such as fracture of the distal radius. Similar considerations could also be made for metabolic biomarkers of bone. The fact that findings in subjects with CRPS and those without it are similar would tend to exclude that the levels of bone turnover represent a risk factor for the onset of the condition. Notwithstanding, other reports in the literature reveal a possible key to the interpretation of these results.

In a recent study of subjects who presented with distal radius fractures, it was shown that at the time of the fracture those with intra-articular fractures (with involvement of the distal cortical bone of the radius) had significantly lower serum levels of $25(OH)D_3$ than those with extra-articular metaphyseal fractures [8]. To further investigate this aspect, an observational study of approximately 600 patients with fracture explored predictive factors for CRPS [4]. It was found that those who developed CRPS more frequently had intra-articular and multifragmentary fractures than those who did not. Accordingly, vitamin D deficiency could play an indirect role and could be predictive of intra-articular fractures, which would in turn correlate with an increased likelihood of developing CRPS.

The same could easily apply to rheumatoid arthritis. Indeed, the high prevalence of vitamin D deficiency in patients with rheumatoid arthritis is well known [9], and its presence is a risk factor for development of CRPS following a fracture event [4]. Last, an additional causal link considering CRPS, fractures and vitamin D deficiency must include the propensity for falls among the elderly with inadequate vitamin D levels [10].

CRPS is characterised by intense pain, along with sensory and vasomotor changes, local oedema and functional deficits. Significant insights into the pathogenetic mechanisms of CRPS have been made in recent years (Fig. 2). A local increase in proinflammatory cytokines and the release of neuropeptide mediators by nociceptive afferents that interfere with the regulation of local microcirculation helps to trigger and maintain the condition. These events lead to hyperalgesia, i.e. a painful perception disproportionate to the intensity of the stimulus, together with and allodynia, i.e. a painful perception following a stimulus that is not normally capable of evoking pain. Subsequently, altered capillary permeability, interstitial oedema and hypoxia, as well as local acidosis, comprise the subsequent pathogenetic events that sustain the typical clinical manifestations, namely intense pain, oedema, alterations in palpable heat and local discolouration (Fig. 3) [11].

The use of highly sensitive biochemical methods and animal models have made it possible to identify neuroinflammatory events that are pathogenetically connected with the initial clinical manifestations of the condition. In mouse models of CRPS-1, high local concentrations of nerve growth factor, which is potentially implicated in the onset and transmission of pain and in inducing the production of local cytokines, has been observed. It is well-known that high local concentrations of proinflammatory cytokines, such as tumour necrosis factor-α (TNF-α), interleukin-1 (IL-1) and interleukin-6 (IL-6), are present during the early stages of the CRPS. Other studies have reported that high systemic concentrations of these cytokines are also present. Furthermore, there is evidence that the local release of certain proinflammatory cytokines is also mediated by the involvement of keratinocytes in skin, easily justifying the intense inflammation that is typical at the onset of CRPS.

To consolidate the epidemiological and clinical findings mentioned above, consideration must also be given to other pathogenetic factors correlating CRPS with vitamin D levels. One must also link the primary role of vitamin D in regulation of mineral metabolism with its deficiency and an increase in bone fragility and risk of fracture. The likelihood of developing neurological diseases characterised by inflammation/neuroinflammation also appears to be influenced by inadequate levels of vitamin D [12].

Further evidence of a pathogenetic role in the development of CRPS is the influence that vitamin D has on the endogenous production of proinflammatory cytokines. In an observational study published in 2014, which included 957 subjects over

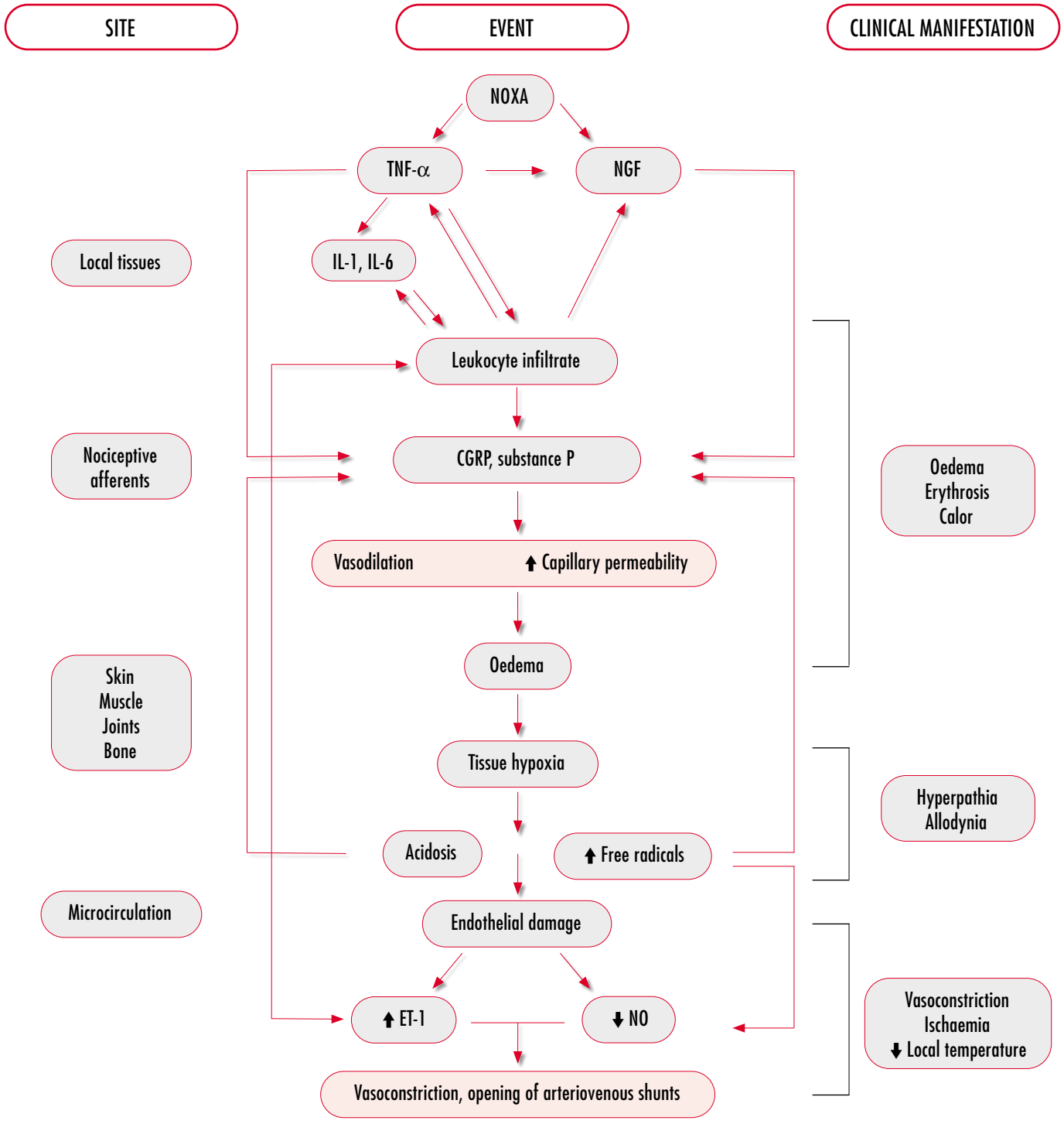

FIGURE 2.
Pathogenetic mechanisms involved in the onset, maintenance and clinical manifestations of CRPS-1.

the age of 60 years, plasma levels of selected cytokines were assessed along with $25(OH)D_3$ [13]. Plasma levels of IL-6 were found to be significantly higher in those with vitamin D levels < 25 nmol/L compared to those with normal values (> 75 nmol/L). It should be pointed out that IL-6 is among the cytokines whose levels are increased both locally and systemically during CRPS.

A similar finding was also observed for another proinflammatory cytokine involved in the pathogenesis of CRPS, namely TNF-α. In 69 healthy women between 25 and 82 years of age, the

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FIGURE 3.
Images of CRPS with involvement of the hand and the foot. The clearly intense inflammatory profile is evident in the early stages of the disease.

plasma levels of 25(OH)D_3 were inversely correlated with the levels of TNF-α [14]. As further corroboration of a plausible causal link between levels of vitamin D levels and proinflammatory cytokines, some studies have reported that vitamin D supplementation can reduce the plasma concentrations of TNF-α, IL-1 and IL-6 [15].

At present, there is no definitive evidence that vitamin D supplementation is a valid strategy to prevent CRPS. Nonetheless, it has been shown that vitamin D deficiency can promote algodystrophy, and the rationale for this correlation is based on the below considerations:

- Vitamin D deficiency leads to an increased risk of fracture events, which are the most typical predisposing event for CRPS;
- Low levels of vitamin D favour the occurrence of intra-articular fractures, which more frequently correlate with the development of CRPS;
- Vitamin D deficiency correlates with an increased risk of falls, thus favouring fractures, which are a trigger for the development of CRPS.

Since vitamin D deficiency promotes predisposition to CRPS following a fracture, the most likely pathogenetic pathway seems to be an immunological status characterised by increased serum levels of pro-inflammatory cytokines, which promote the inflammatory phase in CRPS. Accordingly, the rationale for vitamin D supplementation in those with deficiency is that normalisation of plasma 25(OH)D_3 levels should lead to reduced production of the inflammatory mediators of CRPS.
A point worthy of clinical investigation is the possible therapeutic role of vitamin D administration in patients with CRPS. In this respect, there are now therapeutic strategies that have profoundly improved patient outcomes. The efficacy of bisphosphonates should be considered a definitive finding, which has been shown in randomised placebo-controlled trials and meta-analyses, i.e. the tools with the highest levels of evidence. Among the different bisphosphonates, neridronate, which has been shown to have the highest efficacy, induces rapid remission of CRPS that is maintained in the long term [16,17]. In fact, neridronate is the only bisphosphonate approved by the Italian Medicines Agency (AIFA) for this indication.

Since the fundamental assumption is that high doses of the drug are required, which can only be obtained with intravenous administration, this implies treatment in a hospital setting, leading to logistic issues. From this consideration, attempts have been made to use older drugs via intramuscular administration, with the possibility of more manageable home-based treatments. Unfortunately, clodronate has not been demonstrated to be effective when administered intramuscularly, which is likely due to its pharmacokinetic and pharmacodynamic profile. However, some studies have recently reported that intravenous and intramuscular administration of neridronate have similar efficacy [17,18]. In this regard, AIFA has approved neridronate for treatment of CPRS when administered intramuscularly [19].
Lastly, another interesting aspect to investigate would be involve the potential benefits of vitamin D supplementation in combination with a bisphosphonate. However, it must be kept in mind that despite advances in therapy, early treatment, at a stage when the levels of proinflammatory cytokines trigger and maintain the condition, is essential.

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Vitamin D deficiency in children: a distant but re-emerging problem

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BACKGROUND, WHY THERE IS RISK

Vitamin D deficiency in children is a health problem with social implications that must be considered unresolved. Indeed, since vitamin D is a fundamental factor in development of the musculoskeletal system, it is at the core of children's growth. The primary action of vitamin D in childhood is to promote the proper formation of bone mass. Of note, 90-95% of vitamin D production is generated by exposure to sunlight, while only 5-10% comes from diet [1,2]. Thus, deficiencies can occur if exposure to sunlight is inadequate.

In this regard, a British study evaluating data on the prevalence of rachitis caused by vitamin D deficiency found that cases of rickets in the UK have risen, especially during the last decade [3]. This situation has been attributed to the fact that the ethnic component of the population living in the UK has probably undergone profound changes. Emergence of populations with darker skin tones has increased significantly, in which supplementation, even in the first year of life, has not been strongly recommended, which has brought about a greater risk of deficiency. In fact, darker phototypes do not allow complete absorption through exposure to sun and tend to require supplementation [3].

Vitamin D deficiency, however, is also possible in other countries such as Italy, where, although sun exposure is far more prevalent, the increase in the number of dark-skinned children has also been observed in the last decade. While some northern European countries often apply a policy of supplementing food with vitamin D (food fortification), resulting in a significant and widespread decrease in risk, lack of supplementation can increase the incidence of vitamin D deficiency. Therefore, in Italy as in other Mediterranean countries, lack of supplementation can also be a significant risk factor for vitamin D deficiency [4]. Considering this, several scientific societies have established recommended dosages and timing for vitamin D administration in children that allows for adequate bone growth [2]. However, during the first year of life even if administration is strongly recommended, there may be poor compliance, causing a marked increase in risk. Such risk may also be present at other stages of growth [2].

VITAMIN D DEFICIENCY: RISK FACTORS

The risk of vitamin D deficiency in children can be related to several factors:

1. Latitude: the further from the equator, the less sunlight there is to promote natural vitamin D production;
2. Ethnicity: darker skin is an obstacle to the formation of vitamin D by sunlight;
3. Cultural factors: for example, the extreme coverage of a mother's body with clothing for religious reasons during pregnancy is a risk factor for severe vitamin D deficiency;
4. Diet: this can play a role if intake or absorption of foods containing vitamin D is restricted.

During the first year of life, prophylaxis with 400 IU/day should be given, since supplementation is necessary for the prevention of rickets. An infant's vitamin D stores will be directly proportional to the mother's vitamin D status, which is often low: the newborn and nursing baby are exposed to minimal sunlight, while the infant's growth is expected to be high. Furthermore, breast milk and formula milk, often contain insufficient amounts of vitamin D. Although supplementation during the first year is highly recommended by paediatricians, mothers may interrupt it or be inconsistent in its administration. An study in the US showed that the reasons for discontinuation were often due to the fact that it was believed that vitamin D was also present in formula milk, that it would also be found in other foods introduced at weaning and that the child was considered to be old enough to not need it [4].

Situations of risk of vitamin D deficiency have also been reported for children over the age of 12 months [1], which is often due to inad-

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equate dietary regimens. For example, a diet rich in phytates (mainly found in seeds, cereals, beans and legumes, and are considered an anti-nutrient since they reduce the absorption of calcium, magnesium and zinc during digestion, thus decreasing their assimilation by the human body) can hinder the absorption of vitamin D [2]. Increased risk can also be caused by chronic disorders such as liver disease or kidney disease, as well as obesity, which is another significant social and health issue. Since vitamin D is sequestered by fat tissue, it becomes unavailable, which significantly lowers serum concentrations in overweight individuals. This set of circumstances, which has been estimated to be present in 30% of the paediatric population, may constitute important risk factors for vitamin D deficiency. In addition, malabsorption disorders, such as cystic fibrosis, chronic inflammatory bowel disease, and undiagnosed coeliac disease, also hinder vitamin D absorption. Some drugs taken for chronic diseases such as antiepileptics, phenobarbital, phenytoin, systemic corticosteroids, antiretroviral drugs, and systemic antifungal agents can also represent risk factors for vitamin D deficiency. Finally, a number of other conditions such as prolonged immobilisation due to cerebral palsy or neuromuscular/neurodegenerative disorders, for example, are significant risk factors [2].

VITAMIN D AND EXTRASKELETAL ACTIONS

Vitamin D, especially in children, has also been attributed a number of extraskeletal functions, which are the subject of ongoing discussion. Many authors view vitamin D as a pleiotropic hormone. This means that the presence of vitamin D and the activation of vitamin D receptors in different cells has homeostatic effects on different organs and systems. Vitamin D receptors are present in various cells that make up the immune system, which influences both its innate and adaptive components [5].

Although vitamin D is able to interact with monocytes, macrophages, and dendritic cells (innate immune system), it will also interact with T lymphocytes (adaptive immune system), thus modulating a child's immunological response. Vitamin D deficiency at different and successive times can expose a child, who may also have other risk factors, to the development of allergic sensitisation and bronchial asthma. Moreover, data from the literature has confirmed that there is a relationship between respiratory function and serum levels of vitamin D, which can be present during pregnancy. Mothers with low serum levels of vitamin D during gestation often give birth to children with reduced lung function. This represents an important risk factor for developing wheezing and bronchial obstruction in the first few months of life [6]. Vitamin D is also able to significantly modify the effect of unfavourable prognostic factors affecting lung function in newborns, such as exposure to cigarette smoke. If a mother smokes during pregnancy, adequate levels of vitamin D will act as a protective factor that appears to neutralise the negative effects of smoke exposure on lung development [7].

SUPPLEMENTATION AND EXTRASKELETAL EFFECTS

Conflicting data on the health effects of vitamin D supplementation has fuelled controversy over its extraskeletal role. A recently published review by endocrinologists concluded that vitamin D supplementation offers very few health benefits. No data has emerged in favour of supplementation for a number of diseases such as diabetes, cancer, autoimmune diseases, multiple sclerosis or asthma [8]. However, other meta-analyses on the effects of vitamin D supplementation on asthma during childhood have been published with more positive results. While vitamin D supplementation cannot reduce the number of exacerbations of asthma in all asthmatic children, the risk of having asthma may be reduced in children who habitually have particularly low levels of vitamin D, i.e. < 10 ng/mL [9]. In another very recent review that assessed the use of vitamin D for management of asthma, the authors found little evidence that the risk of exacerbations was reduced by vitamin D supplementation or its metabolites. However, it must be noted that, the patients most at risk, i.e. those with severe asthma and particularly low vitamin D levels, were poorly represented in the studies in that review.

From a practical standpoint, a child with asthma at higher risk (with moderate to severe asthma and risk factors for vitamin D deficiency) may benefit from vitamin D supplementation [10]. Supplementation during gestation does not appear to prevent asthma in school-age children: notwithstanding, evidence of a trend towards its efficacy has been seen in the prevention of wheezing and bronchospasms in pre-scholastic children. At the age of three years, a trend for greater protection from episodes of wheezing and bronchospasms was seen in the supplemented cohort. This finding was recently confirmed in an analysis of data from the same cohort, attributing it to the effect of vitamin D on lung function and the immune system as early as pregnancy [11].

VITAMIN D AND OBESITY

A clear relationship has been found between vitamin D and metabolic syndrome. In Italy, 30% of children are overweight and many of these are obese. Vitamin D deficiency appears to influence the development of metabolic syndrome and obesity. Serum levels acquired through vitamin D supplementation in children are influenced by body mass index (BMI). While supplementing a child of normal weight with a conventional dose may lead to adequate levels of vitamin D, the same may not be true when providing the same supplementation to a child who is overweight/obese. One study reported that BMI appeared to be associated with a reduced response to vitamin D supplementation [12]. In particular, children with obesity had greater resistance, in terms of non-response, to vitamin D supplementation. Furthermore, another study has shown that there is a conditioning effect determined by the association between BMI and vitamin D levels on respiratory mechanics in patients with mild asthma. Indeed, in patients whose weight is normal, adequate levels of vitamin D were associated with significantly better respiratory function, which was not evident in overweight patients [13].

HOW SHOULD VITAMIN D BE SUPPLEMENTED IN CHILDREN?

Proposed dosages vary widely. Therefore, it is important to refer to the doses recommended by national and international guidelines [2,14]. Only supplementation or food fortification can achieve and maintain adequate levels of vitamin D, especially in children at risk. Exposure to sunlight alone has often been found to not be sufficient at all paediatric ages. A study in 2018 found that only supplementation significantly and effectively increased vitamin D levels in the paediatric population and in pregnant women [15]. A significant aspect that has now been clarified is that dose should be taken daily, and not as a monthly or weekly bolus. A biologically plausible explanation for this is the fact that

TABLE I.
Recommended daily vitamin D requirements at ages between 1-18 years (from Peroni, 2022) [14].

Age	IOM 2011 and AAP 2012			LARN 2012			Endocrine Society 2011	
	EAR, UI/day	RDA, UI/day	UL, UI/day	EAR, UI/day	RDA, UI/day	UL, UI/day	Daily Requirements, UI/day	UL, UI/day
1-3 years	400	600	2,500	400	600	2,000	600-1,000	4,000
4-8 years	400	600	3,000	400	600	2,000 (4-10 years)	600-1,000	4,000
9-18 years	400	600	4,000	400	600	4,000 (11-18 years)	600-1,000	4,000

EAR: Estimate Average Requirement (estimated intake to cover the needs of 50% of the population); RDA: Recommended Dietary Allowances (recommended intake for the population: intake estimated to cover the needs of more than 97.5% of the population); UL: Tolerable Upper Intake (intake above which adverse events may occur). * Recommended requirements for individuals at risk of vitamin D deficiency.

high single bolus doses may induce the long-term expression of enzymes involved in the catabolism of vitamin D, which inactivate the vitamin when it is administered in large quantities [16]. Thus, it is important to supplement daily. This was highlighted in a review of the literature published in 2013, where the risk for development of respiratory tract disease in children was more significantly reduced in subjects receiving vitamin D as a daily dose and not as a bolus [17,18].

WHAT DOSES CAN BE USED FOR SUPPLEMENTATION?

One must first consider that supplementation is important to achieve adequate serum levels (Tab. I). Vitamin D is relevant to the development of appropriate bone mass, also because osteoporosis is a disease that many believe begins in childhood. Therefore, it is essential to ensure adequate intake of vitamin D and calcium early in life, which will build up bone mass. Nevertheless, to achieve levels of vitamin D levels that are effective on extraskeletal functions, it will probably be necessary to achieve higher serum levels than that what are considered beneficial for bone health. All children should receive adequate supplementation during the first 12 months, and probably for 24 months: 400 and 600 units of vitamin D, respectively. In middle and late childhood, it is also important to give vitamin D to children and pre-adolescents who have risk factors for vitamin D deficiency. Lifestyle also makes a difference. In children and adolescents who are frequently outdoors and eat a broad variety of foods, exposure to sunlight and diet should ensure good vitamin D absorption. During winter months, vitamin D supplementation is worthwhile, while considering the presence of risk factors such as lack of exposure to sunlight and overweight/obesity. Daily intake should be 400 units/day during the first year of life, which should then vary between 600 and 1000 units/day. A child who presents with very low levels of vitamin D, and with a clinically evident deficiency, will need higher levels of supplementation 2.

CONCLUSIONS

Levels of vitamin D may be low at all paediatric ages, especially in the presence of certain risk factors. Food fortification, a situation that, at the level of the entire population in countries where vitamin D levels have traditionally been low, for example due to inadequate exposure to sunlight, has helped overcome health problems such as rickets [17,18]. However, the problem is not yet solved. Food fortification, together with daily supplementation, must be considered because, as risk factors have changed, with more dark-skinned children, and as lifestyles have evolved, the likelihood of vitamin D deficiency has tended to increase. In Italy, the number of at-risk and vulnerable children has also increased notably in recent years. Therefore, the benefits of vitamin D supplementation should be acknowledged and thus recommended.

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