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EDITORIAL

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Dear Readers,
As you know, vitamin D is a subject of great interest; we are daily bombarded with news on the topic in different fields of specialization, some swaying us in one direction and some in another... You may have noticed the growing scientific interest on this subject, as attested by the increasing number of publications on PubMed since 2000, which even today stands at a high level, even if the number has begun to plateau (Fig. 1).
It comes as no surprise that in addition to its noted impact on human skeleton and phosphocalcic metabolism, vitamin D can also have extraskeletal effects. There are at least five good reasons in support of this: vitamin D receptors are present in numerous cells – I would say they are nearly ubiquitous; vitamin D controls the transcription of numerous genes; it has endocrine effects, and not only calciotropic ones; the activation and catabolism of vitamin D take place in several organs and tissues; and it has intracrine and paracrine effects in numerous cells of various natures.
Most available studies on the effects of vitamin D are preclinical or observational (Fig. 2). The latter often describe associations between a lack of vitamin D and the incidence, activity or outcomes of many illnesses, but they have an intrinsic limit of not being able to document a sure causal relationship. On the other hand, randomized, double-blind, placebo controlled clinical trials relative to supplementation – the only ones able to scientifically verify the effects of vitamin D – are few in number, sometimes for ethical reasons, and often suffer from bias (Fig. 3).
The most frequent bias is the treatment of subjects without deficiency, forgetting that as a nutrient vitamin D can only have effects when it is lacking. Recently we have seen, for example, a publication of a study [1] in which subjects, who were for the most part not deficient, were given supplements: the conclusion of the ineffectiveness, in terms of preventing fractures and falls, of vitamin D supplements for adults living in senior communities created confusion among both doctors and patients. Instead, the researchers should have first verified – by means of an epidemiologic study – the prevalence of vitamin D deficiency to understand whether the administration of a supplement would be at best useless, if not harmful, in that kind of community and in that population group. When studies are carried out which make little sense – like the one we have just described – which are of poor quality, which are conducted with extremely variable doses and administration modes of vitamin D, and which adopt different or unknown protocols in completely different clinical conditions – in these cases, meta-analyses may produce misleading results and conclusions.
For example, the recent meta-analysis conducted by Zhao et al., published in JAMA [2], mixed together studies using D2 or D3, with doses that ranged from 400 IU/day of vitamin D to 500000 IU/year ± variable doses of calcium, in subjects with completely different or unknown vitamin D profiles and calcium intake, and with extremely variable – or worse, unknown – fracture risk conditions. It should not surprise us that the results are not statistically significant. Likewise, the attempt in this meta-analysis to rationalize the analysis by having recourse to the evaluation of a subgroup with baseline serum levels of 25(OH)D < 20 ng/mL is tainted by the fact that this fundamental datum is only available in very few studies; it was therefore mostly estimated on the basis of the dosage in a small subgroup of subjects, who were not necessarily representative of the entire population under examination.
Furthermore, available studies almost always lack verification of the 25(OH)D serum level at the end of the study in the untreatedplacebo group; as has been observed in some studies [3], the control group does not turn out to be mostly deficient, probably as a result of the widespread and common tendency today of self-managed vitamin D supplementation.

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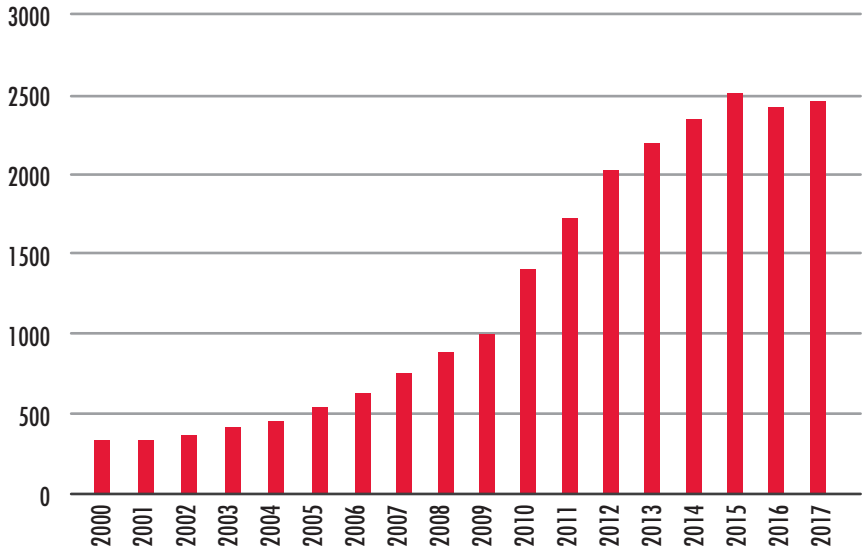

FIGURE 1.
Number of publications on PubMed with vitamin D mentioned in the title.

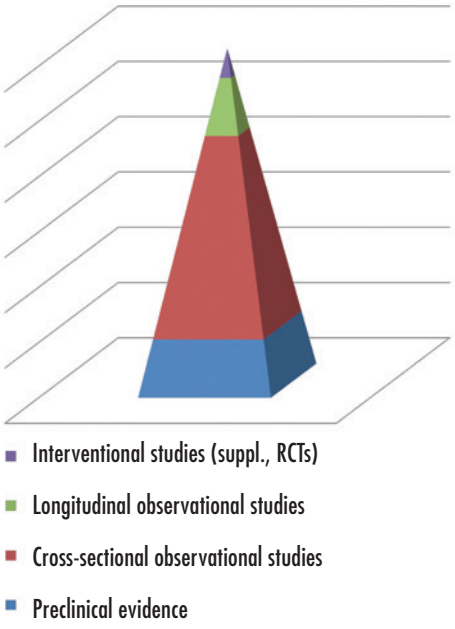

FIGURE 2.
Characteristics of available vitamin D studies.

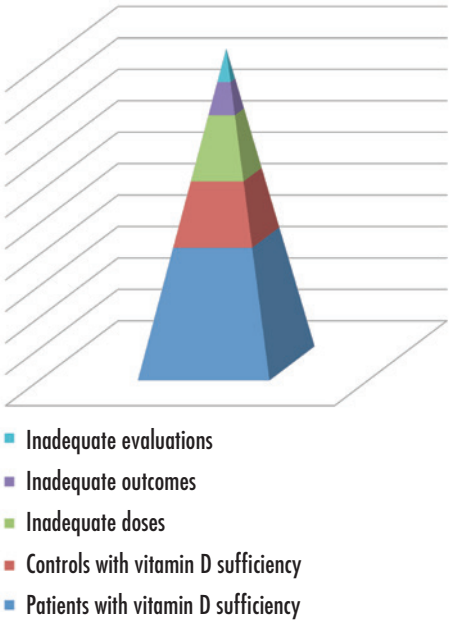

FIGURE 3.
Biases in vitamin D RTCs.

Such considerations are not possible if – whether for a lack of competence or simply of time – it is not feasible to access the enormous literature that is becoming progressively available, even if we limit ourselves to reading abstracts.
This new journal emerges from the need – which is, I believe, widely felt – to have access to a publication which provides updates and guidance on this topic. It therefore aims to collect by field of specialization articles published on PubMed over the last several months (over 200 in the month of January alone!), in the hope is that this will enable readers to stay in touch with important developments and to make sense of the nearly daily barrage of news in this field. The journal will also present comments, in-depth analyses and reviews on the part of some of the major experts in the various main specializations to help readers keep abreast of the certainties and uncertainties regarding vitamin D.
I hope you enjoy reading the journal.

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VITAMIN D AND ISCHEMIC STROKE: Promise for prevention and improved outcomes

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Ischemic stroke produces irreversible damage in the brain and is one of the main causes of disability and mortality. In recent years, scientific research has progressively documented the role of vitamin D (VitD) in a wide range of physiological functions, beyond its classic role of regulating the homeostasis of calcium and phosphorus. In particular, it has been shown that VitD deficiency is associated with numerous chronic diseases, including cardiovascular, musculoskeletal, infective and autoimmune diseases as well as tumors. Low vitamin D levels are a common symptom in patients with cardiovascular pathologies such as ischemic stroke, myocardial infarction and hypertension; they are further linked to a greater risk of future cardio- and cerebrovascular events. Epidemiologic studies have demonstrated that VitD deficiency is a risk factor for stroke. Patients who have suffered stroke show a high incidence of VitD deficiency, which may be attributed to reduced mobility and diminished exposure to sunlight, on one hand, and to an inadequate dietary regime, on the other. Reduced vitamin D levels can increase the risk of a future cerebrovascular event and contribute to functional deficits subsequent to a stroke. It is further necessary to note a seasonal variation in the incidence of ischemic stroke, with lower percentages during summer when exposure to sunlight allows for increased synthesis of active vitamin D metabolites.

This evidence may have important clinical implications, as high vitamin D levels can be useful for controlling cerebrovascular risk factors, such as high blood pressure, diabetes mellitus and metabolic syndrome; they may also produce antithrombotic and neuroprotective effects, such as a stimulation of neurotrophic factors, a reduction of oxidative stress, autoimmune response of the nervous system and regulation of the apoptosis, thereby reducing the risk of future stroke (Fig. 1).

Currently available data indicate that vitamin D supplementation could represent a promising approach for preventing and treating stroke. Nonetheless, clinical trials that show a true association between vitamin D deficiency and stroke are still required; furthermore, trials are needed to establish whether vitamin D administration can reduce the incidence of stroke and the morbidity and mortality associated with it.

The aim of this review is to analyze currently available data on the correlation between VitD deficiency and cerebrovascular events; it further considers possible etiopathogenetic mechanisms and the use of vitamin D in stroke prevention and therapy.

CORRELATION BETWEEN HYPOVITAMINOSIS D AND CEREBROVASCULAR EVENTS

The Ludwigshafen Risk and Cardiovascular Health study (LURIC) [1], which involved over 3000 patients who underwent an angiography as well as a follow-up after an average of 8 years, showed that low vitamin D levels were a predictive factor for fatal stroke. In particular, after adjusting for possible confounding factors, the odds ratios remained significant for 25(OH)VitD at 0.67 (0.46, 0.97; p = 0.032), and for 1,25(OH)VitD at 0.72 (0.52, 0.99; p = 0.047). The authors suggest that vitamin D could have a protective effect against stroke, as the data indicate a negative association with hypertension, diabetes and atherosclerosis.

Data from a population study have shown

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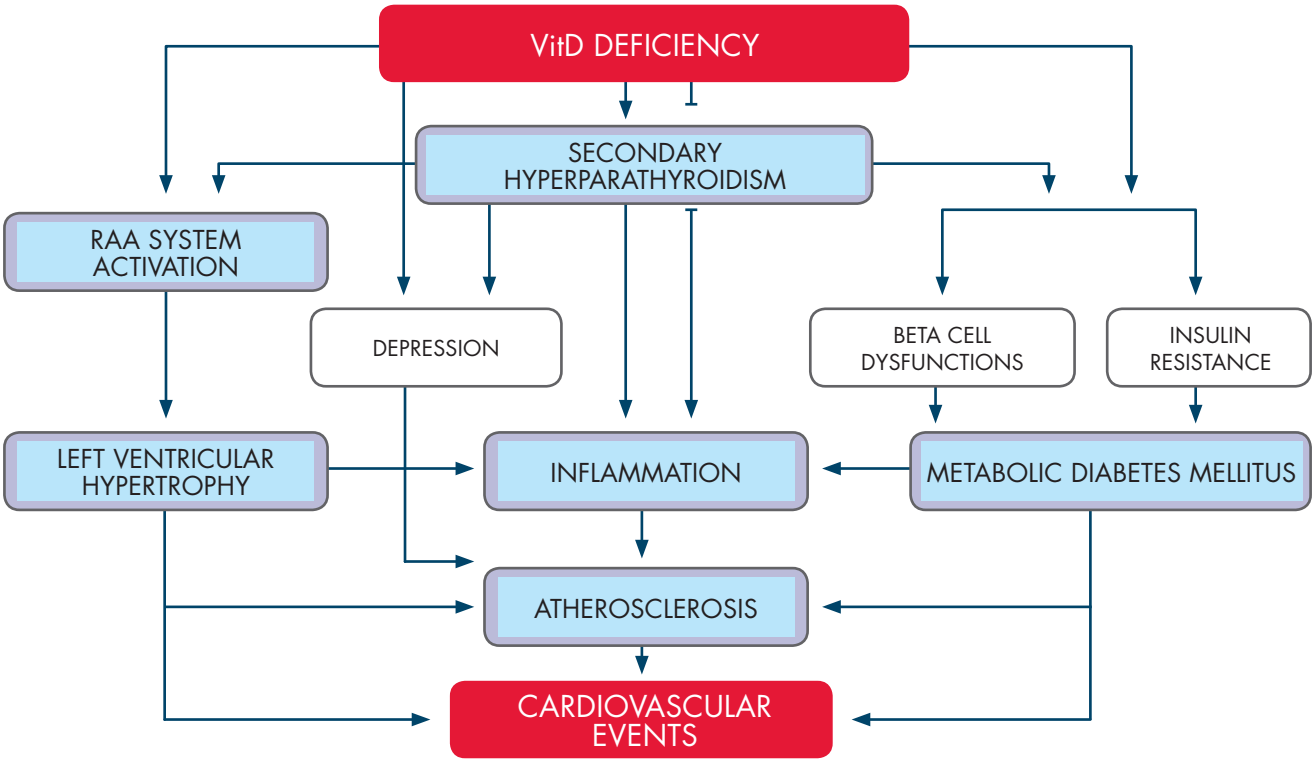

FIGURE 1.
Role of vitamin D in the pathogenesis of cardiovascular events.

that elderly individuals with VitD deficiency are at an increased risk of future stroke, even after adjusting for age, gender, smoker status and functional capacity [2]. The risk turns out to be significantly lower for individuals in the highest and middle thirds of vitamin D intake compared to subjects in the lowest third (risk ratio 0.47, p = 0.011, and risk ratio 0.46, p = 0.024, respectively). In addition, vitamin D serum levels seemed to be predictive for stroke (risk ratio 0.41, p = 0.0053 in the highest third). For the authors, these findings indicate a real causal association between low vitamin D intake and future stroke; they consider supplementation as a promising approach for prevention.

The REGARDS study (Reasons for Geographic and Racial Differences in Stroke) [3], which was conducted on over 16,000 white and black patients, showed that those who lived in areas with less exposure to sunlight presented an increased stroke risk of roughly 56%. In addition, other researchers studied ca. 21,400 participants from the REGARDS study every 6 months over a 5-year period; they found that individuals with higher vitamin D levels in their diets had an 11% reduction in stroke risk and 24% lesser risk for cognitive decline. Such reductions held even after adjusting for factors for cardiovascular risk.

In any case, it is necessary to point out that the association between vitamin D, on one hand, and reduced risks for stroke and cognitive decline, on the other, could also be correlated to unmeasurable confounding factors, such as the fact that those with higher vitamin D levels potentially followed healthier diets.

Another study, based on data extrapolated from a medical database of 41,504 subjects [4], found that the prevalence of VitD deficiency (< 30 ng/mL) was 63.6%. VitD deficiency turned out to be closely associated (p < 0.0001) with an increased prevalence of hypertension, hyperlipidemia, diabetes and peripheral arterial disease. Levels of 25(OH)VitD were furthermore strongly linked to coronary disease, myocardial infarction, heart failure and stroke (p > 0.0001). Of particular interest were the increases in the prevalence of heart failure (90% relative and 9% absolute), myocardial infarction (81% relative and 2.6% absolute) and stroke (51% relative and 2% absolute), as these individuals showed very low vitamin D levels with respect to controls (p trend < 0.0001 for all categories). In addition, very low vitamin D serum concentrations produced a higher composite relative risk for death, coronary disease, myocardial infarction, heart failure or cerebrovascular accidents (Hazard Ratio = 2.13, 95% CI, 1.75, 2.58, p < 0.0001).

A systematic review and meta-analysis has indicated that there was no significant reduction in mortality and cardiovascular risk associated with vitamin D levels (25(OH) VitD > 20 ng/mL) [5]. In particular, the review found no evidence for a significant link with outcomes for mortality, myocardial infarction and stroke, and no proof for the surrogate outcomes of hypertension, lipid fraction or glycaemia. Nonetheless, the authors acknowledged that the quality of the available evidence, in the best of cases, was low or moderate.

By contrast, another meta-analysis has provided evidence for an overall association between basal levels of 25(OH)VitD in the lowest categories (< 20 or 15 ng/mL) – compared with those in the highest (> 30 or 20 or 15 ng/mL) – and cardiovascu-

lar diseases (overall Hazard Ratio = 1.54, 95% CI, 1.22, 1.95) [6].
Evidence extrapolated from a review of the literature indicates that further clinical trials are necessary to verify an association between VitD deficiency and cerebral stroke.

POSSIBLE ETIOPATHOGENETIC MECHANISMS

Laboratory data suggest a potential causal association of VitD deficiency as a risk factor for stroke by means of a mechanism of systematic and vascular inflammation which determines – either directly or indirectly – atherogenesis (Table I). It has been shown that the activation of the nuclear receptor of vitamin D (VDR) can elicit *in vivo* antithrombotic effects, which suggests that the VDR system may play a physiological role in maintaining antithrombotic homeostasis. In an experimental study, the platelet aggregation induced by ADP showed a significant increase in VDR-knockout (VDR KO) mice with normal calcium levels. In addition, the genetic expression of antithrombin in the liver and that of thrombomodulin in the aorta, liver and kidney was down-regulated, while the expression of mRNA for the tissue factor in the liver and kidney was upregulated in VDR KO mice, independently of calcium plasma levels. Therefore, the vitamin D/VDR system increases the expression of antithrombotic factors and inhibits the expression of one of the thrombogenic factors, such as the tissue factor [7].
Another study, in which female rats were fed a diet with low levels of vitamin D (VDD) for 8 weeks, showed that the animals presented significantly greater cortical infarction volumes compared with the control group; they further had a more severe post-stroke behavioral deficit [8]. These findings were in part attributed to lower levels of Insulin Growth Factor-1 (IGF-1) in the cerebral tissue of the VDD rats, which normally plays a neuroprotective and bioregulatory role in ischemia; and in part to the involvement of an inflammatory response, with subsequent interleukin 6 (IL-6) upregulation induced by ischemia after a VDD diet.
One of the hypothesized actions of vitamin D in the central nervous system, mediated through the influence of the active form of vitamin D, is that it modifies the production and release of neurotrophic factors, such as the nerve growth factor (NGF), essential for neuronal differentiation; vitamin D is further believed to be responsible for the increase in levels of the glial cell line-derived neurotrophic factor (GDNF) [9].
In this context, researchers using an experimental model of cortical infarction in rats reported a significant reduction of the infarcted area (which was obtained through the ligament of the middle cerebral artery) in animals which had received an intraperitoneal injection of 1 μg/kg/day of 1,25(OH2)D for 8 consecutive days, resulting in a significant reduction of the volume and scope of infarction. Consequently, vitamin D pretreatment significantly increased the levels of cortical GDNF, generating the hypothesis that vitamin D reduces cerebral ischemia by means of GDNF [10].
Calcium homeostasis is essential for neuronal physiology, as an excess of calcium in neurons can contribute to neurotoxicity. Vitamin D has also been considered a modulator of the opening of type-L calcium channels through non genomic effects by means of various kinases and enzymatic activity in the cerebral cortex [11]. Previous studies demonstrated that the glutamate receptor type N-methyl-D-aspartate (NMDAR) can promote neural survival [12]. In a previous model of ischemia damage in the brain of rats, intraperitoneal treatment with calcitriol for 6 consecutive days (with doses of 2 μg/kg) produced a significantly reduced infarcted area and volume compared to controls ($p < 0.01$). This effect was correlated to a significant increase of the NMDA subunit and of the NR3A-MEK/ERK-CREB pathway [13].
The integrity of the hematoencephalic barrier is of vital importance in reducing the neuronal damage following ischemic stroke. In an *in vitro* model of hypoxia utilizing bEnd. 3 cells, treatment with 1,25(OH2)D preserved the function of the hematoencephalic barrier (BEE) by means of activation of vitamin D receptors (VDRs) through NF-Kb, proving a protective effect, mediated by the VDRs, of vitamin D against the BEE dysregulation induced by the ischemia [14].

TABLE I.
Association between vitamin D deficiency and cerebrovascular risk factors.

INDIRECT
• Hypertension
• Diabetes Mellitus
• Metabolic Syndrome
• Atherosclerosis
DIRECT
Limitation of antithrombotic effects
• Platelet aggregation increase
• Tissue factor gene upregulation
• Antithrombin gene downregulation
• Thrombomodulin gene downregulation
Limitation of neuroprotective effects
• Neurotrophic factor biosynthesis:
– Nerve growth factor (NGF)
– Glial-derived neurotrophic factor (GDNF)
• Neurotransmitter biosynthesis
• Modes of brain detoxification:
– Expression of inducible nitric oxide synthase (iNOS)
– Intracellular levels of glutathione
– Gamma-GT levels
• Modulation of neuron death:
– Regulation of L-type voltage sensitive calcium channels (L-VSCCs) in hippocampal neurons

THE USE OF VITAMIN D IN STROKE PREVENTION AND THERAPY

Currently, very few trials are available that were specifically designed to study the effect of the administration of vitamin D for preventing stroke. A study which tested the possibility that vitamin D supplementation could improve some vascular markers (hypertension, cholesterol, B-type natriuretic peptide, heart rhythm disorder) in patients with previous ischemic stroke did not reveal statistically significant effects [15]. Nonetheless, the endothelial function, measured as flow mediated dilatation (FMD), showed significant improvement after 8 weeks of vitamin D supplementation compared with the placebo group. This finding turns out to be quite important, as endothelial dysfunction paves the way for atherosclerosis; it is also an independent risk factor for future cerebrovascular events.
In the Women's Health Initiative study, 36,282 menopausal women were divided into two groups: the first took 1000 mg of calcium and 400 IU of vitamin D a day, while the other took a placebo [16]. During the 7-year follow-up, 739 strokes occurred: the Hazard Ratio (HR) of the treated group with respect to the placebo was

0.95 (0.82, 1.10). With regard to the analysis of fatal cerebrovascular events (n = 114), the HR was 0.89 (0.62, 1.29) in the treated group compared to the placebo. The primary limitation of the study was the extremely low daily dose of 400 IU of vitamin D.

Recently, the findings of the ViDA trial have been published, a study conducted on 5,108 individuals who were given an initial dose of vitamin D3 of 200000 IU, followed – beginning a month later – by a monthly dose of 100000 IU or of placebo, for an average of 3.3 years (range 2.5-4.2 years) [17]. The primary outcome (cardiovascular diseases) was found in 303 participants (11.8%) of the group who took vitamin D, and in 293 (11.5%) of the placebo group, with an adjusted hazard ratio of 1.02 (CI 95%: 0.87, 1.20). The same results were observed in participants who had baseline VitD deficiency and for secondary outcomes. The authors conclude that monthly supplementation of high doses of vitamin D does not prevent cardiovascular diseases, but that further studies are necessary to ascertain the effects of daily or weekly vitamin D administration on cardiovascular risk.

With the aim of verifying the use of vitamin D supplementation in preventing cerebrovascular events, several large-scale clinical trials have recently begun [18]. The American study VITamin D and OmegA-3 TriaL (VITAL), which is currently underway, involves 25,871 patients of both sexes (men > 50 years of age and women > 55). It aims to investigate whether daily intake of 2000 IU of vitamin D or of omega-3 fatty acids reduces the risk of developing cancer, cardiovascular disease or stroke in patients with negative anamneses for these pathologies. The first results are expected sometime this year.

The Finnish Vitamin D Trial (FIND), another 5-year study, is being conducted on 2,500 participants (men > 59 years of age and women > 64) divided into three groups: 1) 1600 IU of vitamin D3/day; 2) 3200 IU/day; 3) placebo. The main outcomes of the study regard the prevention of cancer, cardiovascular diseases and diabetes. The results should be available in 2020.

CONCLUSIONS

Observational studies indicate that vitamin D might play a protective role against stroke. Nonetheless, only a few interventional studies are currently available, and each of these has methodological limitations. To the Aim of identifying the potential benefits of vitamin D supplementation on stroke incidence and outcomes, further studies are necessary; scientists will be able to extrapolate useful information when data from trials currently in progress are available. Available data seem to indicate that vitamin D can represent a safe and cost-effective preventive and therapeutic approach for patients with VitD deficiency (< 30 ng/mL) associated with other cerebrovascular risk factors or who have had ischemic stroke [19].

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VITAMIN D AND RECURRENT INFECTIONS: Risk of hypovitaminosis and treatment effects

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IMMUNOLOGICAL BASES

Vitamin D plays an ever clearer role in regulating physiological processes concerning systems and organs which are involved in calcium homeostasis, such as bones, intestines and kidneys. Indeed the vitamin D receptor (VDR) – the receptor at the level of the vitamin nucleus which mediates many, if not all, of the functions of its preferred ligand (1,25-dihydroxyvitamin D [$1,25(OH)_2D$], or calcitriol) – is present in many body tissues. Many of these tissues contain the enzyme CYP27B1, which converts the most important circulating metabolite of vitamin D, 25-hydroxyvitamin D (25OHD), or calcifediol, into the active form $1,25(OH)_2D$. Vitamin D is able to influence the susceptibility and severity of infections through multiple mechanisms which involve the immune system in both its innate and adaptive forms [1]. As a pleiotropic agent, vitamin D is able to activate memory T cells (Tregs), to modulate the action of the toll-like receptors (TLRs) present on the dendritic cells, to regulate the production of cytokines (decrease of inflammatory cytokines, increase of IL-10), and to activate factors of innate immunity such as cathelicidins and defensins.

ASSOCIATION BETWEEN HYPOVITAMINOSIS D AND RECURRENT INFECTIONS

Data from an American study on a vast population of individuals over 12 years of age clearly showed that having deficient or insufficient vitamin D serum levels constituted a risk factor in developing a greater number of infections of the upper respiratory tract in the days prior to the evaluation. The association between infections of this type and hypovitaminosis was particularly significant in individuals with asthma or chronic obstructive pulmonary disease (COPD) [2]. This finding was then confirmed by various other studies carried out above all on pediatric subjects, taking other recurrent pathologies into account, such as gastroenteritis, otitis media and infections of the lower respiratory tract. In the clinical follow-up, the lowest vitamin D serum levels were accompanied by a heightened risk of this type of infection.

It is been emphasized that this correlation is particularly significant in cases of greater clinical severity. A study of pediatric patients under 5 years of age, who had been hospitalized for infections of the lower respiratory tract, showed a series of clinical conditions that were decidedly more complicated in patients that had low vitamin D levels, sometimes in association with low vitamin A levels. The clinical outcome, interpreted as a need for intensive therapy and/or mechanical ventilation, was particularly trying for children who showed hypovitaminosis correlated to the isolation of respiratory syncytial virus in cell culture or metapneumovirus [3]. The association between low vitamin D levels, failed response to treatment and duration of the pathology was once again demonstrated in a recent study on severe pneumonia in children [4], leading the authors to propose that vitamin D levels in children, especially in those at risk of recurrent infections, should be monitored and perhaps supplemented. It is clear that maternal serum levels can also influence the clinical prospects of the newborn: high vitamin D levels in the mother reduce the risk by half that the child will develop bronchospasm or persistent asthma.

Conversely, low levels in the mother or in the cord blood are linked to a more frequent and serious risk of bronchospasm, to reduced pulmonary function and to a greater risk of res-

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piratory infections in the first six month of life [5]. In this regard, some authors have shown that hypovitaminosis D can constitute one of the main factors of risk in developing asthma during the first 10 years of life. During the natural course of their lives, vitamin D can protect children from asthma by preventing the development of sensitivity to allergies, favoring the growth of intestinal and respiratory tract microbiome, developing normal pulmonary function and regulating the development and response of the immune system [6].

Indeed, a longitudinal study which evaluated vitamin D levels at six months, 1, 2, 3, 4, 5 and 10 years of age showed that individuals with repeated episodes of hypovitaminosis in the first decade of their lives are at a significantly higher risk for asthma, eczema and proneness to allergy, conditions which then persist past 10 years of age [7].

Prenatal vitamin D supplementation can be useful in preventing recurrent infections in children. A recent meta-analysis has demonstrated how the intake of vitamins and microelements, in particular vitamin D, may be useful in preventing infantile wheezing. The same does not, however, hold true for bronchial asthma, where other risk factors may play a role in the course of an individual's life. The two most recent studies on prenatal vitamin D supplementation compared the effects of a therapy of 400 IU/day, commonly used in Anglophone countries during pregnancy, with supplementation of 2400 IU/day or 4000 IU/day. In the first study, the risk of persistent wheezing was reduced, though not significantly, perhaps in part because of a wide confidence interval in the obtained results [8].

In the second study, by contrast, which used the higher supplementation dosage, a nearly significant trend was found for a reduction in the treated group for asthma or recurrent wheezing during the first 3 years of life [9].

In a third study, meanwhile, generous supplementation was effective in reducing the incidence of wheezing in newborns of African-American mothers who had good vitamin D serum levels from the first trimester of pregnancy [10].

Supplementation during pregnancy and which continues into the first years of infancy has proved to be effective in attaining normal vitamin D levels in mother and child from birth; it has also resulted in a later onset of the first viral infection and a decreased risk of proneness to allergies in the child [11].

Vitamin D supplementation in children can be useful in preventing recurrent infections.

Two landmark studies evaluated the effects of vitamin D supplementation in preventing respiratory infections in particular regional situations. In Mongolia, the administration of milk fortified with vitamin D (300 IU/day) produced significant effects in protecting against acute respiratory infections, thereby reducing this risk [12].

On the other hand, in another study conducted in Afghanistan, the administration of 100000 IU every 3 months did not produce protective effects against the incidence of pneumonia [13]. A more recent study compared two different supplementation regimes: one with 2000 IU/day, the other with 400 IU/day. The findings showed that the different doses did not have an effect in the prevention of the incidence of respiratory diseases [14].

In children with asthma, supplementing the basic therapy with vitamin D has led to a significant reduction of bronchial exacerbations caused by infective agents.

A 2013 meta-analysis compared the effects of supplementation on infections of the respiratory tract, finding a statistically relevant positive effect, especially when giving daily vitamin D doses as opposed to bolus administrations [15]. A more recent literature review and meta-analysis, which examined data gathered on nearly 11,000 patients from 25 randomized studies, found an overall protective effect in vitamin D supplementation against acute respiratory infections, though with a "number needed to treat" (NNT: the number of patients that need to be treated to have one who is protected) of 33, a rather poor ratio [16].

It is evident that systematic reviews take into account studies which are quite different in terms of doses, timing and modes of administration: in this case, the benefit was greater for patients who received daily or weekly supplementation compared to bolus doses (NNT = 20) and was particularly significant in those with serious vitamin D deficiencies (NNT = 4).

There is a clear need to further investigate clinical advantages through randomized clinical trials for supplementation, even if the results cited above are important for implementing public health measures, given the frequency with which values of hypovitaminosis D are found in our population. These review data may not change our clinical practices, but the observation that for the general population increased serum levels of 25-hydroxyvitamin D can reduce the risk of respiratory infections, and of influenza in particular, has led some authors to point to the savings in health care costs that could be achieved with vitamin D supplementation. These Canadian authors have in fact analyzed the costs of recurrent respiratory infections in terms of utilization of health care resources, absences from school, absence from work for parents and the use of medications [17]. As these pathologies are quite frequent, they have a notable economic impact; in this context, fortified foods or vitamin D supplements play an efficient and beneficial role, in part because they lead to economic savings.

CONCLUSIONS

Vitamin D therefore offers concrete prospects in terms of preventing and curing recurrent infections of the respiratory system. Aspects of the question, which until now have not been sufficiently understood, concern vitamin D supplementation for both pregnant mothers and children. The data at our disposal are very heterogeneous, such that the evidence is of low quality and ambiguous. Further large-scale studies on supplementation are needed to clarify such important questions as the timing, duration and dosage of the therapy. One of the most interesting supplementation strategies regards the period of prenatal life and the first stages after birth: adequate levels of vitamin D in these phases may constitute a window of opportunity during which the responses of the immune system can be programed in a stable and lasting manner.

Normal serum values of vitamin D are probably crucial in obtaining clinical efficacy, beyond that which is required for bone metabolism. Although one third of the population of western countries, including Italy, shows deficient vitamin D levels (serum levels < 20 ng/mL – 50 nmol/L), it has been suggested that levels that effectively maintain an adequate response of the immune system must be higher (at least 30-40 ng/mL – 75-100 nmol/L).

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