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Editorial

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Vitamin D is still surprising. Notice how in this issue we go from a possible role for vitamin D in psychiatric disorders to application to Oncology. We know that vitamin D can have pleiotropic effects, but what might the common biological mechanism that determines them be? Could it be the ubiquitous nature of its receptors? Perhaps its effects on the immune system? Maybe its ability to modulate certain enzyme activities? Or its genomic effects?
It will be interesting to see what you think in view of the authors' contributions in this issue.
The stated aim of the article on vitamin D and psychiatric disorders is to identify its causal relationship, because this would make it possible to understand whether, and to what extent, vitamin D supplementation could prevent the onset of mental disorders or reduce their symptoms. Initially, we begin by describing vitamin D's possible mechanisms of action at a neurological level, before describing the current findings from observational or interventional studies in this field. Specifically, it is noted that vitamin D is involved in the region-specific expression of vitamin D receptors (VDR) in areas such as the cingulate cortex, thalamus, cerebellum, substantia nigra, amygdala and in the hippocampus. Most of these regions express 1α-hydroxylase enzymes capable of metabolising 25(OH)D in $1,25(OH)_2D_3$. This means that vitamin D also has both an autocrine and a paracrine function in the human brain, both of which could play a relevant role in neuroimmune modulation or protection and normal brain development and function. Given the involvement of vitamin D in the control of the inflammatory response, in the case of depression, it is hypothesised that it acts as a modulating mechanism by regulating the over-expression of pro-inflammatory cytokines associated with depression. However, it has also been pointed out that vitamin D is involved in regulating the activity of enzymes such as tyrosine-hydroxylase and the rate-limiting enzyme in the biosynthesis of dopamine, norepinephrine and epinephrine, which are all mechanisms that may justify a positive association between vitamin D deficiency and depression.
Our oncologist colleague, who has also followed the same path, describes the possible biological mechanisms of action, before summarising current clinical findings. He points out that a first level of interaction between vitamin D and neoplastic transformation or progression may refer to the local biosynthetic capacity of the enzyme CYP27B1, whose expression is reduced in some tumours depending on the stage and degree of differentiation. In this context, variations in VDR expression at the intra-tumour level may also influence the biological aggressiveness of the neoplasm by modulating the autocrine, paracrine and intracrine action of vitamin D.
Therefore, I would like to point out that only cholecalciferol and not the different vitamin D metabolites can ensure complete physiological local effects at the level of the various organs and tissues. In addition, the vitamin's potential anti-tumour action can also be

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expressed mainly through genomic mechanisms as well as through non-genomic mechanisms.
With the usual caution that characterises leading experts' assertions, including our authors, the conclusions regarding the clinical effects of vitamin D supplementation in psychiatry and oncology are similar. The results are still conflicting, probably also due to the multifactorial nature of the pathogenesis, the different evaluations of the outcomes (e.g., impact on the incidence of cancer, compared with the more convincing impact on cancer mortality) and the uncertainties about dosages.

However, to me, it already seems advisable to also include the assessment of vitamin D levels in the diagnostic pathway of these diseases as well as to avoid vitamin deficiency among these patients.

Happy reading!

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Vitamin D and psychiatric disorders: analysis of possible causal relationships

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INTRODUCTION

According to the World Health Organisation, more than one billion people suffer from a mental or behavioural disorder. It has been estimated that more than 300 million people in the world, i.e., 4.4% of the global population, suffer from depression. Schizophrenia has a prevalence of 4 to 7 per 1000 people, thus affecting about 20 million people. It follows that not only does treating such a large number of people represent a significant economic effort for the entire healthcare system it also constitutes a challenge for the entire medical sector, given the impact of these illnesses on several clinical fields.

Many scientific findings have clearly shown an association between vitamin D deficiency and depression or schizophrenia. Vitamin D, which is found in the human brain, has been identified as one of the key factors in the regulation of many neurotransmission pathways, including those of dopamine, serotonin, noradrenaline and glutamine. Recent studies have shown that vitamin D deficiency is associated with dysfunction of the hippocampus, a region involved in the pathogenesis of mental disorders, whilst it has also been positively correlated with grey matter volume.

Nevertheless, the causal relationship between vitamin D and mental disorders is still unclear. Even though it has been observed [1].

The aim of this study is to summarise the main scientific findings on the association between vitamin D deficiency and mental disorders, in order to increase the level of knowledge available to clinicians in all fields of medicine as well as to stimulate scientific production and experimental observation on this subject. Indeed, the establishment of a causal relationship would make it possible to understand whether and to what extent vitamin D supplementation might prevent the onset of mental disorders or reduce their symptoms.

VITAMIN D: MECHANISM OF ACTION IN MENTAL DISORDERS

Vitamin D is a steroid hormone that plays key roles in the body's mineral balance, in the proper functioning of the immune system and in the pathogenesis of various disorders, such as cancer and autoimmune diseases.

In psychiatric disorders, vitamin D itself is involved in the region-specific expression of vitamin D receptors (VDRs) in areas such as the cingulate cortex, thalamus, cerebellum, substantia nigra, amygdala and the hippocampus [2]. Most of these regions express 1α-hydroxylase enzymes, which are capable of metabolising 25(OH)D in 1,25$(OH)_2$ D_3, which means that vitamin D has both an autocrine and a paracrine function in the human brain.

The presence of vitamin D, VDRs and related enzymes (CYP27B1 and CYP24A1) in different regions of the brain has made it possible to understand vitamin D's fundamental role as a neuroactive neurosteroid hormone in neuro-immunomodulation, neuroprotection, brain development and normal brain function. Vitamin D deficiency in early life negatively affects neuronal differentiation, axonal connectivity and brain structure and function. All these mechanisms explain the association between vitamin D deficiency in infancy and increased risk of schizophrenia [3]. Analogously, alterations in dopamine transporter expression, neonatal catechol-O-methyltransferase expression and dopamine metabolism have also been reported, which are findings that link vitamin D and its deficiency to schizophrenia.

Given vitamin D's involvement in the control of the inflammatory response, it has been hypothesised that, in the case of depression it acts as a modulating mechanism, regulating the overexpression of depression-associated pro-inflammatory cytokines and the same inflammatory response [4]. Vitamin D is also in-

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Andrea Fagiolini has been a consultant, speaker and/or received research grants from Allergan, Angelini, Apsen, Boheringer Ingelheim, Daiichi Sankyo Brasil Farmacêutica, Doc Generici, FB-Health, Italfarmaco, Janssen, Lundbeck, Mylan, Otsuka, Pfizer, Recordati, Sanofi Aventis, Sunovion and Vifor.
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volved in regulating the activity of enzymes such as tyrosine-hydroxylase and the rate-limiting enzyme for the biosynthesis of dopamine, norepinephrine and epinephrine. Again, all these mechanisms may justify a positive association between vitamin D deficiency and depression.

VITAMIN D AND DEPRESSION

Depression is the most commonly debilitating psychiatric disorder. The etiopathogenetic mechanisms of depression are pathological and related to multiple aspects of neuronal function [1]. Among the elderly, depression mainly affects those with chronic conditions and cognitive impairment, as the processes associated with ageing and chronic illness and associated endocrine and immune inflammatory changes compromise the integrity of frontostriatal, amygdala and hippocampal circuits, thus increasing vulnerability to depression. The association between meagre sun exposure and mood changes has been acknowledged for over 2000 years [5]. Recent studies have shown a correlation between low blood levels of vitamin D and an 8% to 14% increased risk of depression, with a 30% risk of suicide [6-8], with no significant difference between the populations of young adults and the elderly [7].

Comparably, Sherchand et al. (2018), in analysing the relationship between low vitamin D levels and the risk of depression, showed that individuals with vitamin D deficiency have a 3.8-fold increased likelihood of developing depression compared to those with normal vitamin D levels [9]. The Third National Health and Nutrition Examination Survey, studying a sample of 7,970 individuals aged 15-39, identified a higher risk of developing depression in patients with vitamin D levels < 50 nmol/L compared to those with vitamin D levels > 75 nmol/L [6].

Furthermore, hypovitaminosis D appears to be linked to the development of postpartum depression [10,11]. Recent studies have shown a negative correlation between low vitamin D levels during the first trimester of pregnancy and the development of symptoms of depression during the second trimester [12,13]. Similarly, a correlation has been shown between hypovitaminosis D in the second trimester and a higher risk of developing symptoms of depression six months postpartum [14].

Another important observation concerns the association between vitamin D deficiency and the development of depression in later life and in northern populations [15]. One study of 1,282 Dutch patients (aged 65 to 95 years) observed vitamin D deficiency in 14% of depressed patients. Significantly lower rates of vitamin D deficiency were found in the control group [7].

Vitamin D deficiency was found in approximately 58% of the sample in a cross-sectional study of 80 elderly patients (aged 60-92 years), including 40 with Alzheimer's disease and 40 without any form of dementia (Wilkins, 2006).

Finally, patients with chronic diseases such as fibromyalgia or those affected by comorbid depression have a higher incidence of vitamin D deficiency or insufficiency (Hospital and Anxiety Depression Scale = 31) [16].

VITAMIN D AND SCHIZOPHRENIA

Schizophrenia is a chronic mental disorder, characterised by the affected individual presenting abnormal social behaviour and severely impaired thought content and conduct. Schizophrenia has an acknowledged multifactorial pathogenesis. The significant risk factors include genetic vulnerability, neurodevelopmental disorders, viral infections, smoking, IQ, cannabis use and childhood trauma. According to a recent meta-analysis, the risk of developing schizophrenia is 2.14 times higher in individuals with vitamin D deficiency compared to individuals with normal blood levels [17]. Environmental risk factors such as the season of birth, place of birth, latitude and migration have been acknowledged as linking vitamin D deficiency to schizophrenia [3,18].

Specifically, according to a review of 86 studies involving a total of 437,710 individuals, there was a 5-8% higher risk of schizophrenia in individuals born between December and May, with a peak of increased vulnerability in those born between January and February, due to the lower presence of UVB rays capable of stimulating vitamin D production [19].

In a study of 424 schizophrenia patients and an equal number of controls selected by age, gender and date of birth, the patients were divided into quintiles according to their vitamin D levels measured within one year of birth. Those who were in the lowest two quintiles and the highest quintile were found to be at twice the risk of developing schizophrenia compared to individuals in the other quintiles [3]. Correspondingly, a study conducted on a cohort of 12,058 Finnish children showed that vitamin D supplementation during the first year of life was associated with a significant reduction in the risk of developing schizophrenia in adulthood, especially among the males [20].

To support these hypotheses, in a recent study by Okasha et al. (2020) on 20 schizophrenic patients and 20 control patients, vitamin D levels were observed to be statistically lower in patients with schizophrenia (55%) than in non-schizophrenia patients [21].

Moreover, vitamin D deficiency appears to be correlated with a higher risk of isolated psychotic symptoms. In a Swedish study conducted on 33,623 women, a significant association was found between low levels of vitamin D and the development of isolated psychotic symptoms, which suggests that hypovitaminosis D is a possible risk factor for the development of psychosis in adulthood [22].

VITAMIN D SUPPLEMENTATION

Vitamin D supplementation may be a valid approach to improving symptoms of depression and psychosis. In a randomised, double-blind clinical trial of 441 subjects (aged 21-70 years) divided into three groups and treated for one year with 20,000 IU (first group) and 40,000 IU (second group) of vitamin D or with placebo (third group), an improvement in the subjects' Back Depression Inventory Scale (BDI) values was observed after one year of treatment [23]. In an 8-week randomised clinical trial of 42 patients with major depressive disorder, the concomitant use of fluoxetine (20 mg) and vitamin D (1500 IU) led to a significant improvement in symptoms of depression compared with the fluoxetine-placebo group, after just four weeks of treatment [24].

However, there are still conflicting results on the efficacy of supplementation. According to the meta-analyses conducted by Gowda et al. (2015) and Shaffer (2014), vitamin D supplementation was shown to not generally produce any improvement of depression symptoms, except for a moderate effect in patients with clinically significant symptoms of depression [25,26].

In the case of schizophrenia, a randomised open-label clinical trial conducted by Sheikhmoonesi (2016) in a sample of schizophrenic patients treated with vitamin D supplements in addition to standard basic therapy showed no change in symptoms [27].

CONCLUSIONS

This brief narrative review provides numerous insights. Although many studies have highlighted the relationship between hypovitaminosis D during embryonic development and childhood and the onset of schizophrenia in adulthood, this correlation has not yet been accepted definitively. The relationship to depression, which is multifactorial, is still unclear. In addition to the mechanisms of action linking vitamin D deficiency to the biological mechanisms typical of depression, other behavioural factors make understanding the relationship even more complex. On the one hand, depression is associated with reduced outdoor physical activity, with a resulting reduction in the absorption of sunlight. On the other hand, the symptoms of vitamin D deficiency, such as feelings of fatigue and pain, are in and of themselves capable of generating lower mood and thus a state of depression.

Further research is needed to establish appropriate protocols for vitamin D testing and supplementation in clinical practice and to determine whether, when and to what extent vitamin D supplementation may improve the course of depression or reduce the incidence of schizophrenia. It is advisable that clinicians include a vitamin D level assessment in their diagnostic decision-making process. By analysing these levels, the possible psychophysical consequences related to low vitamin D levels can be predicted with a good margin of probability and thus prevented.

Healthy lifestyles that include eating foods high in vitamin D, taking food supplements to reduce vitamin D deficiency or insufficiency, and increasing exposure to sunlight are essential indications for improving mental wellbeing, which should be part of every patient's routine education, regardless of the specific pathological condition.

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The role of vitamin D in oncology: where are we?

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INTRODUCTION

Preclinical studies, using in vitro and in vivo models, show that vitamin D (vitD) is capable of inhibiting neoplastic transformation and progression by inducing cell differentiation, inhibiting proliferation of the neoplastic clone, and performing multiple other biological activities of an anti-inflammatory, immunomodulatory, pro-apoptotic and anti-angiogenic nature.
From a clinical point of view, circulating levels of vitD and its active metabolites have been linked to improved survival of cancer patients. Multiple randomised trials have been conducted, albeit with conflicting results, on the possible impact of vitD supplementation on human cancer incidence, mortality and survival.
This short review of the literature is intended to take stock of the latest preclinical and clinical data and the possible role of vitD in oncology.

BIOLOGICAL MECHANISMS OF ACTION

VitD, produced by the conversion of 7-dehydrocholesterol by UV radiation in the skin, is the precursor of the potent multifunctional hormone calcitriol [1,25-dihydroxy-vitamin D_3 (1,25$(OH)_2D_3$)], which is produced by dihydroxylation in the liver and kidney by cytochrome P450 [1-4]. Through binding to its receptor (VDR), calcitriol regulates, directly or indirectly, 3-5% of the human genome.
An initial level of interaction between vitD and neoplastic transformation and progression relates to the local biosynthetic capacity of the enzyme CYP27B1, whose expression is reduced in some tumours in a stage- and differentiation-dependent manner. In this context, variations in VDR expression at the intra-tumour level may also influence the biological aggressiveness of the neoplasm by modulating the autocrine, paracrine and intracrine action of vitD [1-4].
VitD's potential anti-cancer action is expressed through predominantly genomic mechanisms, but also through non-genomic mechanisms involving, for example, the VDR and endoplasmic reticulum stress protein 57 (ERP57) [5].
The genomic actions of vitD involve the modulation of a wide range of mediators, which regulate pathways of proliferation, apoptosis, and differentiation of tumour cells. For instance, in the three malignancies with the most evidence, including clinical findings, of potential sensitivity to the anti-neoplastic effects of vitD/VDR (breast, prostate and colorectal cancer), this action is expressed through the modulation of proliferative pathways regulated by oestrogen, androgen and the WNT/β-catenin system, both in partially differentiated tumour cell populations and in neoplastic stem cell populations (CSCs), respectively. In addition, signalling through the vitD/VDR axis may also influence the interaction between cancer cells and the tumour microenvironment (TME) in an anti-tumour direction, through modulation of invasive and metastatic capacity and the inhibition of pro-inflammatory and pro-angiogenic pathways [1-4]. Molecular mechanisms involved in the regulation of the anti-tumour activities of vitD include a bi-directional role of a large panel of micro-RNAs (miRNAs), which on the one hand are regulated by the vitD/VDR system, mediating its downstream anti-tumour effects, and on the other hand can regulate the expression of VDR and CYP24A1, modulating the sensitivity of tumour cells to the action of vitD [1-4].
In most, although not all, studies using animal models, dietary vitD supplementation and/or administration of calcitriol and its analogues delay transformation and inhibit neoplastic progression. These models include those of pre-neoplastic lesion progression, human tumour xenografts, models of spontaneous or diet-induced carcinogenesis, models of chemical or known carcinogen-induced carcinogenesis and transgenic models of tumour development [1-4].

CLINICAL FINDINGS

Although the findings from epidemiological studies and randomised clinical trials have not conclusively documented clinically relevant

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effects of vitD levels on the most significant cancer outcomes, overall, the data available to date indicate a greater effect on cancer mortality than on cancer incidence. This suggests possible biological effects on progression or promotion mechanisms rather than on those linked to neoplastic transformation or initiation. These findings (summarised briefly below) would place interventions based on dietary supplementation or pharmacological administration of vitD, calcitriol and related molecules within the conceptual framework of chemoprevention.

Impact on cancer incidence

In three recent systematic reviews of the literature with meta-analysis of pooled data [6-8] (Table I) the relative risk (RR) of developing malignant neoplastic disease in the vitD-supplemented group ranged from 0.98 to 1.03, without significant heterogeneity. These figures, along with the results of the three largest single studies (RECORD, ViDA and VITAL) [9-11], do not support a significant association between vitD supplementation and cancer incidence. In general, in the studies analysed, there is no evidence of a differential effect in particular subgroups.

Impact on cancer mortality

Although the reductions in cancer mortality have not always reached statistical significance in individual studies, in three of the four main studies [9,11,12], there was surprising uniformity in the estimated reduction in the risk of death from cancer, ranging from 14 to 18%, with the exception of the ViDA study [10], where the reduction was minimal (7%). Consequently, the four available meta-analyses [6-8,13] have documented a RR of cancer mortality ranging from 0.85 to 0.88 in favour of the vitD supplementation-based intervention, without significant heterogeneity, reaching statistical significance in three meta-analysis studies (see Table I) [6,7,13].

Some subgroup analyses have indicated a greater likelihood of benefit in terms of mortality reduction, for studies that included subjects of both sexes and with no previous history of cancer, for studies that used daily vitD administration, and for studies with relatively low doses of vitD and that achieved circulating 25(OH)D levels < 100 nmol/L [6-8]. An additional subgroup analysis has suggested that the reduction in cancer mortality is restricted to interventions using $vitD_3$, but is not evident for interventions using $vitD_2$ [13].

INTERPRETATION OF AVAILABLE DATA AND LINES OF FUTURE DEVELOPMENT

The clinical findings cited above suggest, as already mentioned, a prevailing effect of vitD on progression or promotion mechanisms rather than on those linked to neoplastic transformation or initiation. This is also supported by the results of a sub-analysis of the VITAL study, which indicate a significant reduction in the incidence of advanced cancers (metastatic or fatal, hazard ratio - HR -0.83, 95% CI 0.69-0.99, P = 0.04) in the vitD-treated group, particularly in the subgroup of subjects with normal body mass index (P for interaction = 0.03) [14]. Aligned with these results, the first randomised trial, conducted in patients with advanced colorectal neoplasia undergoing chemotherapy, found a trend in favour of high doses of $vitD_3$ over standard doses, with an advantage of approximately 2 months in median progression-free survival (PFS; 13 vs 11 months, log-rank P = 0.07) and an HR in multivariate analysis of 0.64 (1-sided 95% CI, 0-0.90; P = 0.02) [15].

Finally, the far from negligible impact of vitD supplementation in the context of prevention of skeletal complications and palliation of symptoms in advanced stages of disease should also be mentioned [16,17].

Despite the interest and considerable number of both preclinical and clinical studies reported to date, important gaps remain in the knowledge regarding the potential effect of vitD in reducing tumour progression and cancer mortality [18].

From a preclinical point of view, recent literature has revealed an important role of vitD in reversing multidrug resistance, through interference with epithelial mesenchymal transition mechanisms (EMT), which support drug resistance and favour metastatic spread, and through modulation of specific miRNAs linked to neoplastic progression, thus suggesting its use in the context of advanced disease and in combination with other therapeutic strategies. From a clinical point of view, however, further studies are

TABLE 1.
Main meta-analyses conducted on the impact of vitD on cancer incidence and mortality in recent years

Incidence

Author	No. Trials	No. Patients	Cases (VitD)	Cases (cont)	RR	95% IC	P	Heterogeneity	Ref.
Zhang et al.	10	81.362	3716 (9,16%)	3799 (9,26%)	0,99	0,94-1,03	0,532	No	6
Keum et al.	10	-	6.537		0,98	0,93-1,03	0,420	No	7
Goulão et al.	24	18.440	540 (5,66%)	521 (5,85%)	1,03	0,91-1,15	n.s.	No	8

Mortality

Author	N. trial	No. Patients	Cases (VitD)	Cases (cont)	RR	95% IC	P	Heterogeneity	Ref.
Zhang et al.	7	77.653	821 (2,11%)	942 (2,43%)	0,87	0,79-0,95	0,003	No	6
Keum et al.	5	-	1,591		0,87	0,79-0,96	0,005	No	7
Goulão et al.	7	11.202	150 (2,67%)	170 (3,04%)	0,88	0,70-1,09	n.s.	No	8
Zhang et al.	5	39.197	397 (2,02%)	468 (2,39%)	0,85	0,74-0,97	0,01	No	13

needed not only to confirm the effect of reducing cancer mortality, but above all to clarify the potential role of vitD in tumours of specific anatomical districts, the possible interactions with specific tumour driver genetic alterations, the possible modulation of protective effects in specific individual genetic contexts (e.g. VDR polymorphisms) [18], in order to relate the therapeutic or preventive use of vitD to a context of precision Oncological treatment.

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