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EDITORIAL

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In this issue we encompass topics from dementia to pollution. We can do this discussing the subject of vitamin D because there are recent findings of a possible role here too.

The expert authors to whom we assigned the detailed study of the possible relationship between vitamin D deficiency and dementia have pointed out that studies in the field of neurological physiopathology indicate that vitamin D can exert numerous actions in the central and peripheral nervous system. These can be summarised in four main effects: neurotrophic support, neurotransmission, neuroprotection, and neuroplasticity. Moreover, epidemiological data available on the relationship between vitamin D status and degenerative neurological diseases, such as dementia, seem to support evidence described in animal models since they have generally described inverse relationships, including a dose-response relationship, between serum 25(OH)D levels and the risk of dementia. Nevertheless, the authors admit that there is currently no solid evidence that supports a preventive or otherwise positive effect of vitamin D supplementation in this field. Although given the important and varied limitations of the studies conducted so far, this effect cannot be ruled out. Still, they have wisely concluded that, since these are generally elderly subjects, supplementation should in any case be viewed as justified, considering the acknowledged benefits for the bones, which are certainly greater than the costs and risks of undesirable effects, and, I might add, the known inability of the elderly to naturally produce a daily dose of vitamin D.

On the other hand, the authors of the second article, have provided us with an original contribution, which is also based on their recent research on the possible correlation between pollution and alterations in vitamin D metabolism. Specifically, they have observed that per and polyfluoroalkyl substances (PFAS), which are mainly used to make different types of materials such as fabrics, carpets, upholstery, etc., resistant to oils and water, and which are the cause of widespread and worrying food contamination, especially in some areas of the Veneto region, may interfere with vitamin D receptors because of their similarity with steroid hormones. The result appears to be a reduced response of bone cells to vitamin D, which is manifested by lower bone mineralisation and an altered response of vitamin D-sensitive genes. Clinically, this would be corroborated by a higher prevalence of osteoporosis in populations exposed to PFAS and findings of higher average serum levels of parathormone, which is an expression of functional hypovitaminosis D. On the other hand, you can also see why subclinical and widespread vitamin D deficiency, which characterises our population, might represent a susceptibility factor to the negative health effects of PFAS exposure. These issues were the subject of a recent Commission of the Superior Council of Health, in which I had the pleasure of participating, which produced a document of specific recommendations that should be published soon on the Ministry of Health website. These include, among populations exposed to PFAS contamination, the recommendation to promote the dosage of circulating levels of 25(OH)D, its main metabolites and of biomarkers of their functionality in phosphocalcic and bone metabolism. In addition, densitometric screening and assessment of the incidence of fragility fractures and extra-skeletal diseases that may also be correlated with absolute or functional vitamin D deficiency (particularly cardiovascular and immunological diseases, whose prevalence seems to actually be

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increasing in individuals exposed to PFAS), and the possible implementation of studies involving vitamin D supplementation.
The problem of pollution, as you know, apart from being currently topical, is of great concern for future generations. Our school recently observed a correlation between air pollution (particularly particulate matter) and the prevalence of osteoporosis [1] or reactivation of disease [2] and a poorer response to treatment in patients suffering from rheumatoid arthritis [3]. Does absolute or functional vitamin D deficiency also play a role in explaining these correlations?
Merry Christmas and Happy New Year.

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Vitamin D and dementia

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Summary

Many findings arising from experimental studies, mostly using in vitro or animal models, seem to indicate that vitamin D plays a role in nervous system physiology and physiopathology with the potential for also determining the pathogenesis of certain degenerative diseases, such as dementia. It appears that vitamin D exerts neurotrophic, neuroprotective and neuroplastic effects, whilst also being involved in the synthesis of certain neurotransmitters. Data drawn from prospective observational studies have clearly confirmed the experimental observations by showing an inverse association between vitamin D status (25-hydroxyvitamin D concentration) and the incidence of dementia, with a dose-response relationship. To date, interventional studies using cholecalciferol to reduce the risk of dementia have not had positive results. This has been mainly due to significant limitations in terms of experimental design, treatment regimens, test population size and follow-up duration. Ad hoc and methodologically more appropriate study designs are needed to define the potential beneficial effect of cholecalciferol in preventing the risk of dementia.

VITAMIN D AND THE CENTRAL AND PERIPHERAL NERVOUS SYSTEM

A large body of scientific evidence has suggested that vitamin D plays a role in the physiology and physiopathology of the central (CNS) and peripheral nervous systems. Indeed, it has been advanced that vitamin D deficiency might play a role in the pathogenesis of some neurodegenerative diseases, including dementia, Parkinson's disease, multiple sclerosis and amyotrophic lateral sclerosis [1-3]. Many observations support vitamin D's involvement in CNS physiological and physiopathological processes. The vitamin D receptor (VDR) is ubiquitously distributed in the CNS and peripheral nervous system [1]. In fact, the topography of VDR distribution, initially defined in rats and hamsters, was subsequently confirmed and detailed in humans as well [1]. VDR is thought to be expressed in neurons and glia in several areas of the nervous system, including the cortex (e.g., temporal, frontal, parietal), the cerebellum, the spinal cord and in the basal ganglia [1]. 25-hydroxylase and 1α-hydroxylase activity has also been identified in the CNS, which is indicative of paracrine production of 1,25-dihydroxyvitamin D [1,25(OH)$_2$D] [1-3]. The same vitamin D metabolites have been identified in cerebrospinal fluid [1-3]. Finally, further evidence for the existence of paracrine production activity of 1,25(OH)$_2$D in the nervous system comes from the observation that the concentration of 1,25(OH)$_2$D in the CNS is in positive correlation with the plasma concentration of 25-hydroxyvitamin D [25(OH)D], whereas it does not correlate with the plasma concentration of 1,25(OH)$_2$D [1-3].

Based on this and other findings, it has therefore been hypothesised that vitamin D may exert several actions in the CNS and peripheral nervous system, which can be summarised in four main effects: neurotrophic support, neurotransmission, neuroprotection and neuroplasticity. [1-3].

It is believed that Vitamin D exerts neurotrophic functions related to neuronal differentiation, maturation and growth, through for example stimulating the synthesis of neurotrophic factors such as nerve growth factor (NGF), glial cell line-derived neurotrophic factor (GDNF) or neurotrophin 3 (NT-3) [1-3]. Similar significance is thought to be attached to the effect on levels of *neurotrophin 4* (NT-4) (*downregulation*) and on the regulation of gene expression of the neurotrophic low-affinity NGF receptor

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(p75NTR) [1-3]. Sustaining these hypotheses as well as the neurotrophic role of vitamin D, morphological studies in healthy elderly people or those suffering from various degrees of cognitive impairment have shown a correlation between vitamin D status (defined by plasma 25(OH)D concentration) and/or vitamin D deficiency, and the volume of grey matter and the hippocampus [4,5].

It also appears that Vitamin D and its metabolites mediate the synthesis of a variety of neurotransmitters, including acetylcholine, catecholamines, serotonin and dopamine [1]. This effect of vitamin D appears to be persistent over time and above all transgenerational. Indeed, early exposure to insufficient or deficient levels of vitamin D seems to induce epigenetic changes, which in turn are believed to influence gene expression and increase susceptibility to many neurodegenerative diseases over time (*metabolic imprinting*) [1,6-10].

Vitamin D's neuroprotective effect has been the subject of many experimental studies in animal models, where the administration of vitamin D or its metabolites has been shown to exert a protective effect on neurons by reducing cell damage and neurotoxicity mediated by certain substances known to be neurotoxic [1,11,12].

In one *in vitro* study, conducted on cortical neuronal cell cultures, Annweiler et al. showed that the combination of memantine (a drug used in the treatment of cognitive impairment) and vitamin D (but also vitamin D alone) was able to attenuate and prevent axonal degeneration produced by beta-amyloid and glutamate [11]. The mechanisms underlying vitamin D's neuroprotective effect have only been partially clarified and are still under discussion (e.g., regulation of calcium flow, anti-inflammatory effect and anti-oxidant effect) [11,12].

It seems that Vitamin D is able to influence neuroplasticity by regulating genes that have a significant impact on neuronal development and many neuronal functions (most likely already during pregnancy) [1,13]. For example, it appears that Vitamin D deficiency might alter the transcriptional profile of genes involved in cytoskeleton maintenance, mitochondrial function, neuronal plasticity, cell proliferation and growth [1]. Furthermore, as already detailed, during certain stages of pregnancy, vitamin D deficiency may lead to alterations in the regulation of neuronal function (on a molecular basis), which may influence susceptibility to certain degenerative diseases in adulthood [13].

IS THERE A RELATIONSHIP BETWEEN 25(OH)D CONCENTRATION AND THE RISK OF DEMENTIA?

Epidemiological data on the relationship between vitamin D status and neurodegenerative diseases, in particular dementia, seem to fully support the animal model findings described. A recent *overview* (Table I) analysed the results of the main review/meta-analysis studies on the relationship between vitamin D status and the risk of dementia and/or Alzheimer's disease (AD) [3]. Although the results of the different studies taken into consideration were not always easy to interpret, mainly due to the lack of standardisation of serological and clinical evaluations, overall, two important points emerged with significant uniformity and consistency [3,14-19]:

- there is an inverse relationship between the concentration of 25(OH)D and the risk of dementia or AD.
- It appears that this inverse relationship between 25(OH)D concentration and the risk of dementia or AD follows the "dose-response" principle.

For example, Chen et al. [14], undertook a meta-analysis of 10 cohort studies that included approximately 28,000 patients. The authors identified an inverse correlation between 25(OH)D concentration and the risk of dementia [relative risk 0.72 comparing the category with the highest 25(OH)D concentration with the category with the lowest 25(OH)D concentration] and AD [relative risk 0.78, comparing the category with the highest 25(OH)D concentration with the category with the lowest 25(OH)D concentration]. Furthermore, by analysing the dose effect [25(OH)D concentration] response, the authors also showed that the risk of dementia or AD decreased by 5 and 7% respectively for each 10 nmol/L increase in 25(OH)D concentration [14].

Consistent with the findings of Chen et al. and other similar studies (Table I) [14-19], a

TABLE I. Meta-analysis of cohort studies 14-19 that investigated the relationship between vitamin D status [defined by serum 25(OH)D concentration] and cognitive decline (from Maretzke et al., 2020, mod.) [3].

Reference	Studies included	No. of patients (age)	Cut-off 25(OH)D (nmol/L)	Outcome	Main results (95% CI)
Chen (2018) [14]	10 prospective	28,640 (56-85 y.o.)	High *vs* low concentration 25(OH)D	Dementia and AD	RR Dementia 0.72 (0.59-0.88) RR AD 0.78 (0.60-1.00)
Jayedi (2018) [15]	7 prospective + 1 retrospective	28,354 (≥ 18 y.o.)	Insufficiency: 25-50 Deficiency: < 25	Dementia and AD	HR Dementia due to deficiency 1.33 (1.08-1.58) HR AD due to deficiency 1.31 (0.98-1.65)
Goodwill (2017) [16]	14 prospective	30,000 (≥ 18 y.o.)	High *vs* low concentration 25(OH)D	Cognitive decline	OR cognitive decline 1.14 (1.06-1.23)
Cao (2016) [17]	3 prospective	12,702 (≥ 20 y.o.)	High *vs* low concentration 25(OH)D	Cognitive decline	RR cognitive decline 1.52 (1.17-1.98)
Shen (2015) [18]	2 prospective	8,086 (mean 74 y.o.)	Deficiency: < 50	Dementia and AD	OR Dementia 1.63 (1.09-2.16) or AD 1.21 (1.01-1.40)
Annweiler (2013) [19]	3 prospective	4095 (mean 75 y.o.)	High *vs* low concentration 25(OH)D	Executive functions	OR due to incident decline 1.25 (1.05-1.48)

RR: relative risk; 95% CI: 95% confidence interval; HR: hazard ratio; OR: odd ratio; 25(OH)D: Serum 25-hydroxyvitamin D; AD: Alzheimer's disease; y.o.: years old.

TABLE II. Incidence of dementia or Alzheimer's disease as a function of baseline 25(OH)D concentration (from Littlejohns et al., 2014, mod.) [20].

Dementia	No. Participants	No. cases	Serum 25(OH)D (nmol/L)			P
			≥ 50	≥ 25 - < 50 HR (95% CI)	< 25 HR (95% CI)	
Dementia (any type)						
Model A*	1658	171	1	1,51 (1,06-2,16)	2,22 (1,23-4,02)	,002
Model B**	1615	168	1	1,53 (1,06-2,21)	2,25 (1,23-4,13)	,002
Alzheimer's Disease						
Model A*	1589	102	1	1,67 (1,06-2,62)	2,27 (1,06-4,84)	,006
Model B**	1547	100	1	1,69 (1,06-2,69)	2,22 (1,02-4,83)	,008

25(OH)D: serum 25-hydroxyvitamin D; HR: hazard ratio; 95% CI: 95% confidence interval.
* Model A: correction (C*ox proportional hazards regression model*) by age and season in which 25(OH)D was dosed. ** Model B: correction (Cox proportional hazards regression model) by age and season in which 25(OH)D was dosed, schooling, gender, body mass index, smoking, alcohol consumption and depressive symptoms.

less recent longitudinal study (Table II) [20], which considered 1,658 elderly outpatients who did not have (at the time of enrolment) dementia, cardiovascular disease or cerebrovascular disease and who showed a higher incidence of dementia and/or AD (during a mean observation period of 5.6 years, range 0.1-8.4 years) in subjects with deficient (< 50 nmol/L) or severely deficient (< 25 nmol/L) vitamin D status at the time of enrolment, compared to subjects deemed to have 25(OH)D concentration in the sufficiency range [20]. Other studies have confirmed these findings, showing consistent results especially for 25(OH)D values < 25 nmol/L (severe vitamin D deficiency) [3]. For levels above this cut-off (e.g. between 25 nmol/L and 50 nmol/L) the results in favour of 25(OH)D seem to be less uniform and consistent.

CHOLECALCIFEROL SUPPLEMENTATION

In light of the experimental data (animal models) and data from epidemiological studies, the role, obviously not primary, of cholecalciferol supplementation in the prevention of neurodegenerative diseases especially dementia [3]. Neither did the randomised controlled trials, pre-post observational studies nor their meta-analyses demonstrate that cholecalciferol supplementation significantly affected the main cognitive parameters examined. Nevertheless, these studies presented significant and determining limitations in the interpretation of the results [3]. Both the randomised controlled trials and the pre-post observational studies were extremely heterogeneous in terms of experimental design and the treatment regimen used: the cholecalciferol dosages used varied from 400 IU per day (a dosage that is probably too low) to 5,000 IU per day, even with boluses of 600,000 IU (inappropriate). Most of these studies had rather short supplementation durations and follow-up periods. They were not at all sufficient in view of the complexity of the physiopathology of dementia/AD and therefore inappropriate for testing the potential protective effect of cholecalciferol on the risk of dementia/AD.

Lastly, in some trials the small number of patients was inadequate to test the hypothesis under study.

In view of the foregoing limitations, there is currently no solid evidence to support a preventive effect, or in any event the beneficial effect of cholecalciferol supplementation in dementia/AD. This potential benefit should not be completely ruled out. *Ad hoc* designed randomised controlled trials will be needed in future to clarify the potential of cholecalciferol supplementation in neurodegenerative diseases and especially in dementia.

In conclusion, one last consideration deserves to be emphasised: elderly patients are at the highest risk of cognitive impairment/dementia and are also the population with the highest prevalence of hypovitaminosis D. Therefore, this category of frail patients are always worth treating with cholecalciferol in view of its low cost, total safety and tolerability, and of its great efficacy in preventing falls and fractures, besides its potential, but likely, extra-skeletal benefits. A daily maintenance dose of 1,000 IU or 2,000 IU of cholecalciferol preceded, where indicated, by a loading dose, would appear to be the most natural strategy for optimising the skeletal and extra-skeletal effects of cholecalciferol [21].

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PFAS and correlation with vitamin D metabolism

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Summary

Perfluoroalkyl and polyfluoroalkyl substances (PFAS) are a class of compounds widely used in industry and consumer products. They are resistant to degradation and tend to accumulate in the environment and in living beings with possible toxic effects. These contaminants are a public health problem, especially in some areas of the Veneto Region, but have recently been isolated in waters from other parts of Italy. Perfluorooctanoic acid (PFOA) is the predominant form in human samples and has been shown to induce serious health consequences, such as neonatal alterations, neurotoxicity and immunotoxicity. Toxicological studies indicate that PFAS accumulate in bone tissue and alter bone development. Epidemiological studies have reported an inverse relationship between PFAS blood levels and bone health, especially in terms of bone mineral density (BMD). Osteopenia and osteoporosis have been shown in several cohorts, ranging from post-menopausal women to young men. Since the interaction between this class of compounds and certain nuclear hormone receptors (such as the thyroid hormone receptor and the androgen receptor) has already been demonstrated, an interaction with the vitamin D receptor has also been hypothesised, which is essential for the proper regulation of phosphocalcic metabolism, the main determinant of bone density. This study summarises the experimental and clinical evidence supporting the interference of PFOA with the vitamin D signalling pathway.

INTRODUCTION

Perfluoroalkyl and polyfluoroalkyl substances are molecules that can interfere with the endocrine system, i.e., they belong to the category of EDs (*endocrine disruptors*). An endocrine disruptor is defined as any chemical entity or mixture of compounds that is capable of interfering with any aspect of hormonal action and that is therefore responsible for altering hormone homoeostasis [1]. EDs exert their toxicity by promoting tissue growth and development. The mechanism of interaction with the reproductive system through the binding of these substances to the androgen receptor (AR) and the oestrogen receptor (ER) is well known. Binding of the endocrine disruptor to the receptor will result in a receptor agonist or receptor antagonist response, manifested by an increase or decrease in the cellular response to the physiological hormone stimulus [2].

PERFLUOROALKYL SUBSTANCES

Perfluoroalkyl and substances (PFAS) are a very broad class of organic molecules that belong to the category of polyfluorinated compounds. They are artificially produced molecules that do not occur naturally. The structure of PFAS has a hydrocarbon skeleton where all hydrogen atoms are replaced by fluorine atoms. The presence of fluorine enables the molecules to acquire special physical and chemical characteristics, above all amphiphilicity: they have an apolar and a polar side, being at the same time both hydrocarbons and strong acids. The polar side contains the functional group, which may be a carboxyl, a sulphuric group, an alcohol group or several others. The polar functional group and the length of the fluorocarbon chain define the individual PFAS. For example, the two most frequently studied compounds, because they occur most frequently in polluted areas, are perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). The main sources of exposure can be ingestion of contaminated drinking water or food with high levels of these compounds (e.g., fish and seafood). Several perfluoroalkyl compounds have

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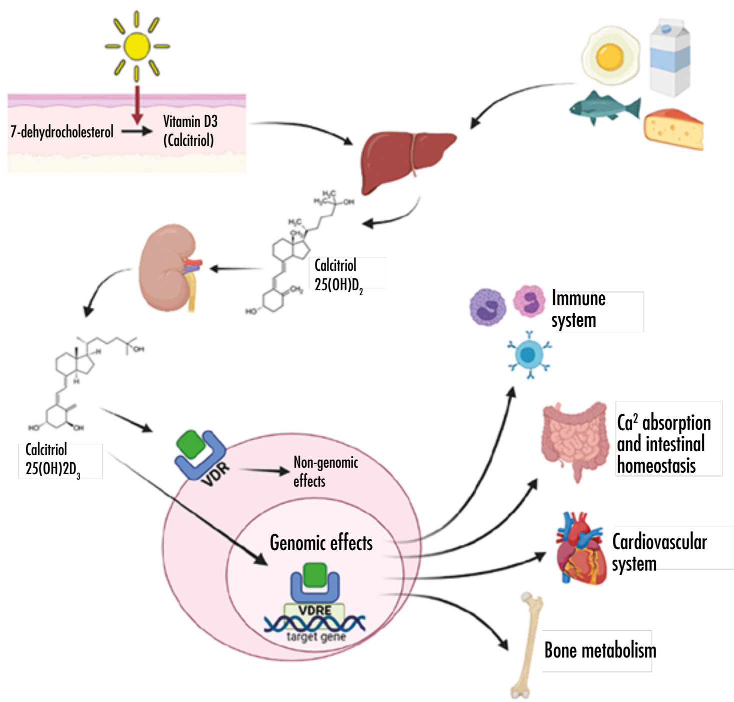

FIGURE 1.
Vitamin D metabolism: Vitamin D is produced endogenously by the skin following exposure to sunlight or exogenously through dietary ingestion. The vitamin D precursor (cholecalciferol) is then converted to 25-hydroxyvitamin D (25(OH)D, calcifediol) by the 25-hydroxylase enzyme in the liver. In its turn, 25(OH)D is hydroxylated in the kidney at the 1-α position in its biologically active metabolite, 1,25-hydroxyvitamin D (1,25(OH)D. Calcitriol performs its biological functions in different organs by acting on its nuclear receptor, which, following binding to the agonist, migrates into the nucleus and binds to specific recognition sequences (VDREs) at the level of target gene promoters.

been found in body fluids: serum, seminal fluid, breast milk and even in the umbilical cord, suggesting that exposure to these compounds is life-long from its inception. PFOA and PFOS induce serious human health consequences such as neonatal mortality, neurotoxicity and immunotoxicity. Health surveillance data in the USA have shown that PFAS are detectable in the serum of 95% of the population [3].
In humans, PFAS serum levels vary depending on the level of exposure. In the general population that has not been exposed, average concentrations of 5.5 ng/mL have been found for PFOA and 2.1 ng/mL for PFOS [4]. In the population living in the polluted areas of the Veneto region, PFOA concentrations range from 54 to 540 ng/mL [5].

SKELETAL TOXICITY

For about a decade, it has been known that the risk of osteoporosis and pathological fractures is associated with exposure to some environmental pollutants (lead, cadmium and mercury). PFAS are among the pollutants that interact with bone metabolism. Today, few studies are available on the interaction of these substances with bone metabolism. Foetal bone malformations have been reported in rodents with prenatal exposure to PFOS and, still in mice, environmental exposure to PFOS results in rapid accumulation in bone tissue. In humans, the presence of perfluoroalkyl compounds (predominantly PFOS) in bone has been demonstrated by analysis of skeletal findings from autopsies of subjects exposed to the contamination [6].
The most recent analyses concern those carried out by the two studies on the health of the American population, which highlighted the correlation between high serum levels of PFAS in contaminated areas and reduced bone mineral density, which varied based on the type of perfluoroalkyl substance considered. Looking more specifically at individual PFAS, there is a higher prevalence of osteoporosis and lower bone density in the tibia and femur and a high prevalence of osteoporosis among women associated with PFOA, PFNA, and PFHxS can be noted. More recently, these findings have been confirmed in other studies on adolescents or young adults [7,8].
These latter data were confirmed in a cohort of young men (aged 18 to 21) from polluted areas in the Veneto Region. In exposed subjects, the association between exposure to PFAS and the risk of fracture was demonstrated [9].

VITAMIN D METABOLISM

Most vitamin D3 is produced in the skin from provitamin 7-dehydrocholesterol through a photochemical reaction involving the UV component of solar radiation. Once synthesised in the skin, or absorbed from the intestine, vitamin D is found in the circulation bound to its serum *D binding protein* (DBP), an α-globulin synthesised by the liver. To be biologically active, vitamin D3 requires a double hydroxylation (Fig. 1).
The first hydroxylation, at position 25, is carried out by mitochondrial and microsomal enzymes that belong to the cytochrome P450 superfamily. The second hydroxylation is carried out in the kidney by an enzyme present in the proximal convoluted tubule, the 1α-hydroxylase, which also belongs to the cytochrome P450 family (CYP27B1). Unlike the previous one, this hydroxylase is strongly regulated: parathormone and hypophosphoremia activate it, while calcium, phosphate, FGF-23 and calcitriol (negative feedback mechanism) inhibit it. This results in 1,25-dihydroxy-cholecalciferol, or calcitriol, the true vitamin D derived hormone.
The vitamin D receptor (VDR) is expressed in numerous cell types and tissues. Calcitriol, the active form of vitamin D, directly or indirectly regulates more than 200 genes involved in cell proliferation, differentiation, apoptosis and neoangiogenesis. It is convenient to divide the biological effects of vitamin D into skeletal, i.e. those that affect phospho-calcium and extra-skeletal metabolism [10] (Fig. 1).

PFAS INTERFERENCE WITH VITAMIN D METABOLISM

Although the number of epidemiological studies confirming a negative effect of these substances on skeletal metabolism is increasing, the mechanisms that may induce this association have not yet been fully demonstrated. Vitamin D, a steroid hormone, which acts by stimulating intestinal reabsorption of calcium in favour of an anabolic action on bone is a key hormone in skeletal development. Several exogenous factors, such as obesity, diet and pollution, are known to influence circulating vitamin D levels. Vitamin D homoeostasis may also be affected by endocrine disruptors, since the biologically active metabolite, 1,25-hydroxyvitamin D, is very similar in its structure to classic steroid hormones. Its nuclear receptor is also comparable to receptors for thyroid or steroid hormones. For example, an inverse association between bisphenol A and phthalates and vitamin D levels has been reported in two epidemiological studies [11]. Given the similarity between steroid hormones, in particular testosterone, and vitamin D, and between the respective steroid receptors, such as the androgen receptors, in particular the androgen receptor, and the vitamin D receptor, it can be assumed that the reported interference of PFAS with steroid hormone function can also be extended to vitamin D metabolism.

This mechanism could demonstrate the previously reported associations between PFAS exposure and impaired skeletal development and osteoporosis. On the basis of this evidence, a role for PFAS in altering vitamin D metabolism has been hypothesised. This mechanism could be one of the possible modes of skeletal alteration induced by these substances. Vitamin D homoeostasis could be affected by endocrine disruptors, as this hormone is steroidal in origin and endocrine interference of PFAS with steroid receptors, such as the androgen receptor, has already been demonstrated [12].

A very recent study by Prof. Foresta's Group [13] has shown that PFAS interfere with the vitamin D receptor, inducing a reduced response of skeletal cells to vitamin D, which manifests itself in reduced bone mineralisation (Fig. 2). Firstly, PFOA competes with calcitriol at the same vitamin D receptor (VDR) binding site, leading to an alteration in the receptor's structural flexibility. Secondly, this interference leads to an altered response of vitamin D-sensitive genes in two cell populations targeted by this hormone, osteoblasts and colorectal epithelial cells. Third, mineralisation in human osteoblasts is reduced when coincubated with PFOA and calcitriol. Finally, in a cohort of healthy young males, vitamin D was not decreased in the exposed group, but PTH levels were higher in association with PFAS exposure, suggesting a compensatory mechanism in response to functional D hypovitaminosis. Overall, this finding shows an important pathophysiological involvement in vitamin D deficiency associated with environmental exposure to endocrine disrupting chemicals and could explain the epidemiological observations of reduced bone mass in this context. These results, in addition to clarifying the mechanisms by which PFAS interfere with the activity of this important hormone, suggest a possible role for these pollutants in the pathogenesis of osteoporosis, the main pathology related to reduced vitamin D levels.

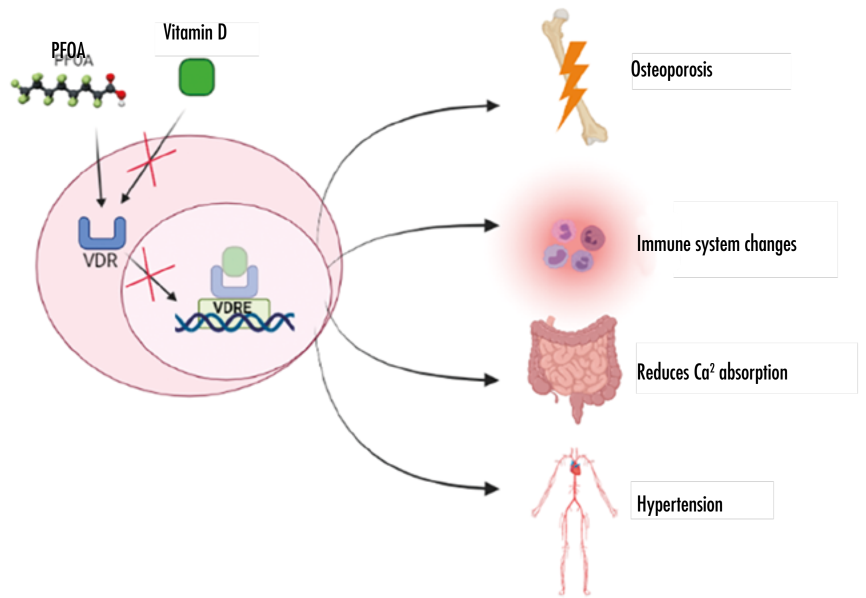

FIGURE 2.
PFAS' endocrine disruption mechanism on vitamin D: PFAS inhibit vitamin D from binding to its receptor (VDR) and preventing it from binding to the target gene promoters. This interference leads to a state of functional hypovitaminosis D in which, even with normal levels of vitamin D, it is unable to perform its biological function in the target cells. This mechanism may explain the several clinical manifestations observed in populations exposed to PFAS pollution and related to vitamin D activity, such as osteoporosis, reduced immune response, reduced calcium absorption and cardiovascular conditions.

CONCLUSIONS

Epidemiological and experimental evidence shows that PFAS alter vitamin D homoeostasis and therefore represent a risk factor for bone tissue in all age groups, from developmental age (growth phase) to post-menopause, the high-risk phase for osteoporosis. Monitoring of vitamin D status and skeletal health is highly recommended in exposed populations. At the same time, subclinical vitamin D deficiency (a widespread problem in Western societies) is a susceptibility factor to the effects of PFAS exposure. Therefore, it is particularly important to develop non-pharmacological awareness and prevention campaigns in exposed populations, based on the promotion of physical activity, proper exposure to sunlight and nutrition.

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