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EDITORIAL

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Dear Colleagues,
In this issue you will find some updates on the possible role of vitamin D in chronic kidney disease and certain gynaecological conditions. Note that in both articles the expert authors start out by highlighting how common it is to also find vitamin D deficiency in these pathological conditions. In the case of chronic kidney disease, they attribute this deficit to reduced nutritional intake secondary to typical dietary restrictions, frequent associated gastrointestinal disorders, and reduced exposure to sunlight, secondary to the disability.
It is also pointed out that in this condition, native vitamin D deficiency is compounded by impaired calcitriol synthesis, resulting in altered mineral and bone metabolism (Chronic Kidney Disease-Mineral Bone Disorder, CKD-MBD) characterised by a state of secondary hyperparathyroidism, which, though initially "adaptive" subsequently becomes "maladaptive" if not corrected by adequate vitamin D supplementation.
The observation that, even in patients with advanced chronic kidney disease, requiring dialysis, cholecalciferol administration is associated with an increase in calcitriol synthesis, is interesting, since it demonstrates that there is also extrarenal production of calcitriol, even at the level of the same parathyroid glands.
Although the subject has been debated, current guidelines suggest that supplementation with native vitamin D (cholecalciferol or ergocalciferol) be used, especially to prevent the onset or progression of hyperparathyroidism, perhaps achieving serum 25(OH)D levels well above 30 ng/mL in these patients, which would be preferable.
Active vitamin D metabolites should be reserved for the more advanced stages of chronic kidney disease, when there are high serum parathormone levels, despite adequate 25(OH) D levels. It should not be forgotten that the use of these metabolites may be associated with hypercalcaemia, hyperphosphatemia, altered FGF-23 levels and excessive reduction in PTH levels such that the risk of adynamic bone disease is increased.
Even when active vitamin D metabolites should be used, it would be wise, in any case, to ensure supplementation with native vitamin D, given its extrarenal physiological effects and presumed extra-skeletal benefits.

For example, how about receptors, vitamin D-modulated genes and vitamin D-activating enzymes in different tissues, including those of the reproductive tract?
Have you noticed how much new literature there is in our usual bibliographic update on obstetrics and gynaecology?
The authors of the other article in this issue point out that genetic polymorphisms of the specific vitamin D receptor (VDR) have been associated with different levels of sex hormones and that the addition of vitamin D to granulosa cells is able to increase their synthesis. This could well justify correlations observed between vitamin D deficiency and menstrual cycle disorders or polycystic ovary syndrome, characterised by oligo-anovulation, clinical and/or biochemical

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signs of hyperandrogenism and polycystic ovary morphology.
This could also justify the positive effects observed with supplementation, especially if daily, of patients with ovarian polycystosis, in terms of infertility and the correction of certain typical associated metabolic alterations, including hyperinsulinism, dyslipidaemia and chronic inflammatory status.
All good reasons not to neglect a vitamin D status assessment and a potential opportunity for supplementation in these patients as well.
What are your thoughts? Happy reading!

Vitamin D deficiency in gynaecological diseases

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The role of vitamin D (VitD) as a significant element in the pathophysiology of gynaecological diseases has been growing in recent years, with laboratory data intersecting with clinical data in indicating the role, or possible roles, that this vitamin may play in the field of gynaecology.

The production and metabolism of VitD originates from the stimulus exerted by ultraviolet rays on the skin, with the transformation of 7-dehydrocholesterol into cholecalciferol, which, in turn, is metabolised in the liver by a 25-hydroxylase. 25(OH)D is converted at the level of the kidneys by 1-alpha-hydroxylase into $1,25(OH)_2D$ or calcitriol, the active metabolite. Again in the kidney, $1,24,25(OH)_3D$, which is a biologically inactive compound, is formed by 24-hydroxylase (Fig. 1).

VitD, which should more properly be referred to as D-hormone, through its specific receptor (Vitamin D Receptor, VDR), is able to modulate the activity of approximately 3,000 genes located in different areas of the human body, including the tissues of the female reproductive tract (ovary, uterus, vagina). Genetic polymorphisms of VDR have been associated with different levels of luteinising hormone (LH), Sex Hormone Binding Globulin (SHBG), testosterone and insulin [1].

In particular, as far as the reproductive tract is concerned, VitD may exert control over ovarian follicle development and the luteal phase through an interaction with anti-Müllerian hormone (AMH) signalling and follicle-stimulating hormone (FSH) [2] sensitivity pathways.

Furthermore, and interestingly, the addition of VitD to human granulosa cells in a culture medium has also been shown to increase the production of certain hormones, which are critical for ovarian activity, compared to the non-addition of VitD, such as progesterone (to the extent of 13%; $p < 0.001$), oestradiol (to the extent of 9%; $p < 0.02$), estrone (to the extent of 21%; $p < 0.002$), again due to the presence of VDR in these cells, where it mediates such VitD-stimulating activity on ovarian activity [3].

In a 2018 study [4], which assessed the relationship between VitD status and the menstrual cycle in women who did not have a diagnosis of Polycystic Ovary Syndrome (PCOS), where there were 60 women with low VitD levels (< 30 ng/mL) and 17 with normal VitD levels (> 30 ng/mL ≤ 80 ng/mL), it was shown that in the group with low VitD levels there were 40% of subjects with irregular cycles, 27% with oligomenorrhea and 13% with amenorrhoea. Instead, in the group with normal VitD levels, only 12% of the women had menstrual cycle disorders, 6% had oligomenorrhea and 6% had amenorrhoea. Furthermore, belonging to the group with low VitD increased the likelihood of having an irregular menstrual cycle by a factor of 5 compared with those women in the group with normal VitD levels [OR = 5; (CI 95%: 1.047-23.87), p = 0.04]. Thus, even in women without hormonal disorders, VitD can contribute to the

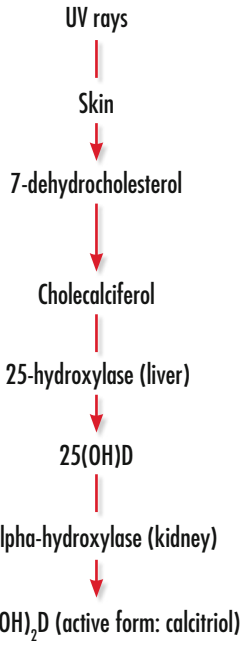

FIGURE 1.
Vitamin D: synthesis and metabolism

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regularity of the menstrual cycle rhythm by modulating ovarian activity. Furthermore, it is worth mentioning how VDR is present at the level of the endometrium [5] and how, also at the endometrial level, enzymatic activities are present, such as 1-alpha-hydroxylase, which are fundamental for VitD metabolism and the production of its active metabolite, $1,25(OH)_2D$ or calcitriol [6].

VITAMIN D AND POLYCYSTIC OVARY SYNDROME

PCOS is the most frequent hormonal disorder among females. It occurs in approximately the following percentages in association with different conditions: 20% of fertile healthy women, 75% of women with ovulatory infertility, 80% of women with oligomenorrhea, 80% of women with hypertrichosis and regular menstrual cycle, 30% of women with secondary amenorrhoea, 80% of women with severe acne [7].

Wanting to consider the implications of VitD in certain pathophysiological conditions of gynaecology, one cannot but consider PCOS, which is the most frequent hormonal disorder in women and whose diagnosis is based on the finding of two of the following three parameters: oligo-anovulation, clinical and/or biochemical signs of hyperandrogenism, and polycystic morphology of the ovary on ultrasound examination. In addition to this, the role of hyperinsulinemia as a factor that dysregulates ovarian activity and the production and action of androgenic hormones [8] must be mentioned from a pathophysiological point of view. Clinically, aside from menstrual cycle disorders, the dermatological manifestations associated with PCOS are frequently the reason for affected patients seeking medical advice. Specifically, PCOS can be associated with seborrhoea (oily skin), acne, hirsutism, and androgenetic alopecia. Besides this, hyperinsulinemia, which is frequently associated with PCOS, can cause a characteristic skin manifestation such as acanthosis nigricans, which is a condition that causes areas of dark, thick velvety skin to form in body folds and creases.

VitD deficiency can affect fertility among women with PCOS. As pointed out above, VDRs are present at various levels, such as in granulosa cells of ovarian follicles, in the pituitary gland and in the endometrium. The AMH promoter gene also contains VitD response elements [9].

As far as the association between VitD and PCOS is concerned, it is important to recall how lower VitD levels are often found in patients with PCOS and that an association between low VitD levels and insulin resistance (resulting in hyperinsulinemia) has been described in PCOS. Finally, low VitD levels are frequently found in obese PCOS patients [10,11].

Indeed, several studies have shown low levels of VitD in the PCOS population, with average 25(OH)D levels ranging from 11 to 31 ng/mL, although the majority of patients have levels <20 ng/mL (67-85%) [10].

A particularly interesting aspect is the relationship between VitD and glucose homoeostasis, which appears to be based on the presence of VDR in the beta cells of the pancreas and skeletal muscle, cells where the 1-alpha-hydroxylase enzyme is found, which catalyses the conversion of 25(OH)D to $1,25(OH)_2D$. Furthermore, there are response elements for VitD in the human insulin gene promoter [12]. First of all, it should be recalled that elevated calcium levels at the intracellular level may alter the post-receptor effects of insulin binding to its receptor, such as glycogen synthase dephosphorylation and Glucose Transporter-4 (GLUT-4) activation. Therefore, VitD deficiency could lead to a secondary increase in parathormone levels (secondary hyperparathyroidism), with increased intracellular calcium levels, thus reducing the response of target cells to insulin action (glucose transport). The prevalence of VitD deficiency in PCOS patients is approximately 67-85%, with serum levels of 25(OH)D < 20 ng/mL [13]. As such, the endocrine-metabolic consequences of VitD deficiency may be important in the pathogenesis of PCOS, as well as in its clinical expressivity (Fig. 2). With regard to the relationship between VitD levels and metabolic profiles in PCOS, reduced VitD levels are associated with insulin resistance, regardless of body mass index (BMI) or waist-to-hip ratio (WHR) in women with PCOS. Interestingly, there is an increase in insulin levels in women without PCOS but who are VitD deficient, whilst HDL cholesterol (high density lipoprotein) correlates positively with VitD levels regardless of BMI or WHR [14].

A cross-sectional study explored the association between VitD status and the diagnosis of ovulatory disorder/PCOS in a population of 67 infertile women in good general health. As a result, reduced VitD values (normalised for other confounding factors) were found in women with ovulation disorder and PCOS when compared to women with infertility from other causes. However, each unit increase in VitD levels (normalised for BMI) reduced the probability of being diagnosed with PCOS by 96% ($p = 0.015$), whilst none of the patients with both ovulation disorder and PCOS had normal VitD levels, with 39% of women with ovulation disorder and 38% of women with PCOS had serum levels <15 ng/mL indicating a vitamin Deficiency [15].

From the standpoint of metabolic impact, VitD status correlates with insulin resistance markers in PCOS (correlation between VitD deficiency and HOMA-IR with $p = 0.0001$ and with fasting blood glucose $p = 0.047$) [16].

A Chinese cross-sectional study[17], conducted on 169 women with PCOS and 114 controls, found lower VitD levels in PCOS patients compared to the controls (11.6 ± 7.2 *vs* 18.9 ± 8.4 ng/mL; $p < 0.05$) and lower VitD levels in PCOS patients with obesity or insulin resistance compared to women without obesity or insulin resistance (8.9 ± 3.7 *vs* 13.6 ± 5.3 ng/mL, $p < 0.05$; 7.2 ± 2.9 *vs* 15.8 ± 4.9 ng/mL, $p < 0.01$). Other metabolic and inflammatory parameters also correlated significantly with baseline VitD levels (Tab. I).

The first study, which evaluated the effect of VitD supplementation in the management of PCOS, was carried out by Thys-Jacobs et al. in 1999. In this study, 13 women with PCOS were treated with 50,000 IU ergocalciferol weekly or every 2 weeks to achieve a serum VitD level of 75-100 nmol/L. An improvement in menstrual regularity was reported within two months [18].

In a 2012 study [19], 12 women with PCOS, who were both overweight and VitD-deficient, received VitD supplements for 3 months (daily dose of 3,533 units, increased to 8,533 units after the first 5 participants) along with 530 mg calcium. After 3 months there was a reduction in total testosterone ($p = 0.036$) and androstenedione levels (although this reduction was not significant). A randomised, placebo-controlled trial [20] conducted on 70 women with PCOS and VitD deficiency (< 20 ng/mL) (aged between 18 and 40 years), studied two groups of patients. One group was treated with 50,000 units of VitD every 2 weeks for 3 months and the other with placebo. The results showed a statistically significant difference in fasting blood glucose levels (-3.1 ± 7.3 *vs* + 0.5 ± 6.3 mg/dL,

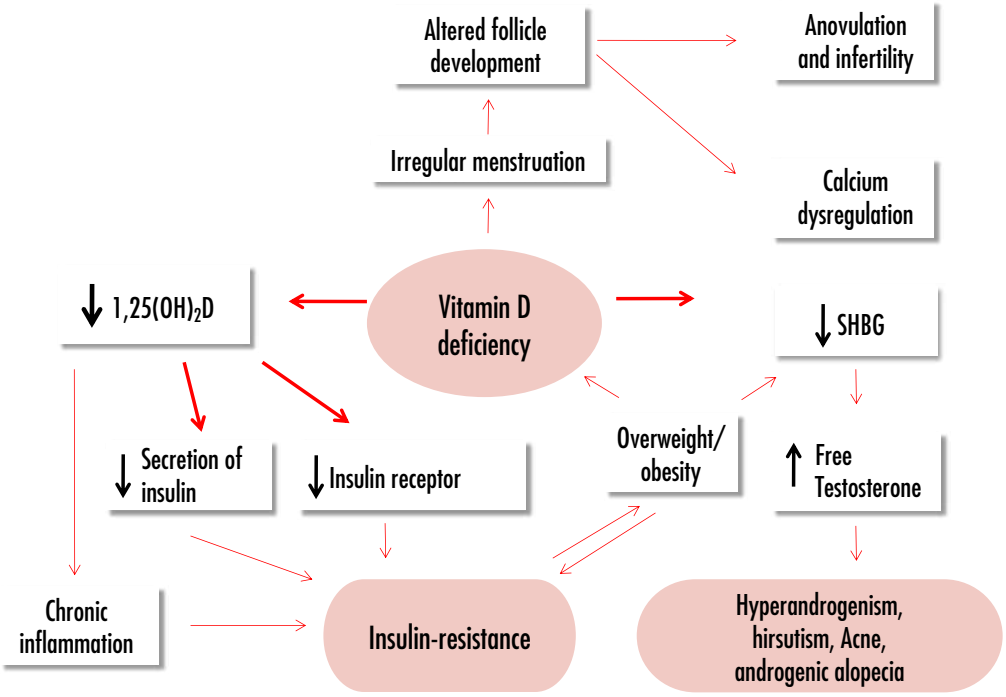

FIGURE 2.
Possible role of vitamin D in the pathogenesis of PCOS (from Thomson et al., 2012, mod.) [13].

p = 0.02), in baseline insulin levels (-1.4 ± 3.6 *vs* +2.6 ± 7.0 µIU/mL, p = 0.004) and in HOMA-IR levels (-0.3 ± 0.8 *vs* + 0.6 ± 1.6, p = 0.003).
Furthermore, hs-CRP levels were also found to be significantly lower (-0.7 ± 1.4 *vs* + 0.5 ± 2.1 µg/mL; p = 0.009), as were malondialdehyde levels (-0.1 ± 0.5 *vs* + 0.9 ± 2.1 µmol/L, p = 0.01).
A 2020 meta-analysis, published by Miao et al.[21], looked at 11 studies (= 483 subjects): of the 11 studies considered, 7 reported PCOS diagnosis and VitD deficiency as inclusion criteria. This meta-analysis showed VitD supplementation to be associated with a reduction in total testosterone (mean difference: -0.10; CI 95%: -0.18, -0.02; p = 0.02), reduction in HOMA-IR (mean difference: -0.44, CI 95%: -0.86, -0.03, p = 0.04), reduction in total cholesterol levels (mean difference:-11.90, CI 95%: -15.67, 8.13, p < 0.01), reduction in LDL-cholesterol levels (mean difference: -4.54, CI 95%: -7.29, -1.80, p = 0.001).
In another meta-analysis published in 2021, conducted considering 18 randomised, placebo-controlled trials (= 1,060 subjects, all with mean VitD values at baseline < 30 ng/mL), Zhao et al. [22] showed that VitD supplementation had a positive impact on the hormonal, oxidative and inflammatory profile in PCOS. Indeed, there was a reduction in total testosterone levels (CI 95%: -0.40, -0.07; p = 0.006), reduced levels of high-sensitivity C-reactive protein (hs CRP) (CI 95%: -0.73, -0.38; p < 0.00001), reduction in malondialdehyde levels (CI 95%: -0.90, -0.54; p < 0.0001), increased levels of total antioxidant capacity (CI 95%: 0.01, 0.83; p = 0.04). Again, in this meta-analysis, it was shown that the most appropriate supplementation scheme to achieve these results is daily supplementation with doses ≤ 1,000 U/day, which appeared to be better than weekly administration, with a suitable duration appearing to be at least 12 weeks.
A recent systematic review with methanolysis 23, was conducted considering 9 RCTs (randomised controlled trials) (n = 1677)

TABLE I
Vitamin D status and metabolic factors in PCOS (Wang et al., 2020, mod.).[17]

	25(OH)D < 20 ng/mL (deficiency)	25(OH)D ≥20 ≤30 ng/mL (deficiency)	25(OH)D >30 ng/mL (normal level)	p*
BMI	27.3 ± 9.2	25.4 ± 8.1	23.5 ± 9.3	0.029
WHR	1.0 ± 0.4	0.9 ± 0.5	0.8 ± 0.3	0.036
Insulin (mIU/L)	39.6 ± 10.7	33.5 ± 9.9	26.8 ± 8.5	0.012
HOMA-IR	8.9 ± 3.7	7.3 ± 2.8	5.7 ± 2.1	0.009
Total Cholesterol(mmol/L)	6.1 ± 1.7	5.5 ± 1.6	4.2 ± 1.4	0.03
hs-CRP (mg/L)	2.4 ± 0.9	1.9 ± 0.6	1.4 ± 0.3	0.017
HDL(mmol/L)	1.3 ± 0.6	1.4 ± 0.7	1.8 ± 0.6	0.03

* Analysis of variance. BMI: Body mass index; WHR: Waist/Hip Ratio; HOMA-IR: Homeostatic Model Assessment for Insulin Resistance; hs-CRP: high sensitivity C-reactive protein; HDL: high density lipoprotein.

and 3 cohort studies (n = 675), on infertile patients with VitD deficiency, that evaluated the influence of VitD supplementation on reproductive outcome, starting from the fact that a low VitD level is associated with an increased risk of infertility. So, VitD treatment significantly increased the clinical pregnancy rate compared to the control group (OR: 1.70, CI 95%: 1.24-2.34; p = 0.001). The improvement in the pregnancy rate was influenced by the patients' VitD level, the type of preparation administered, the total dosage administered, the duration of treatment, the frequency of administration, and the daily administration of VitD supplementation. Infertile women (with VitD levels <30 ng/mL) treated with multicomponent preparations with VitD or with 1,000-10,000 units of VitD per day for 30-60 days could have had a better pregnancy outcome.

CONCLUSIONS

VitD plays a physiological role in female reproductive function. Specifically, it is important to maintain an adequate vitamin D status, both under normal physiological conditions and in women with gynaecological conditions (e.g., PCOS). Assessment of vitamin D status in women's health and, if necessary, supplementation can be very important for clinical practice.

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The use of vitamin D in chronic kidney disease

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INTRODUCTION

The term "vitamin D" denotes a group of steroid, fat-soluble compounds that are essential for the regulation of calcium and phosphorous metabolism, mediated mainly through intestinal absorption.[1]

The two most important isoforms, referred to cumulatively as "native vitamin D", are ergocalciferol (vitamin D_2) and cholecalciferol (vitamin D_3). Ergocalciferol, which is only synthesised by plants and fungi, is introduced through the diet, whereas cholecalciferol, on the other hand, is both exogenously and endogenously synthesised and is derived from the photolysis of 7-dehydrocholesterol, mediated by UVB radiation affecting the skin [15].

Ergocalciferol and cholecalciferol represent the two inactive forms of vitamin D. Their transformation into the biologically active form, calcitriol [1,25(OH)$_2$D], requires a hydroxylation process that takes place in two consecutive steps. The first step takes place in the liver, where, vitamins D_2 and D_3 are hydroxylated at the C25 position by vitamin D 25-hydroxylase and converted to 25-hydroxy-vitamin D [25(OH)D or calcifediol], which is the quantifiable form primarily used to determine serum vitamin D levels. The second step takes place at the level of the proximal tubule of the kidney by way of 1-alpha-hydroxylase, where, 25(OH)D is hydroxylated at C1 to form 1,25-dihydroxyvitamin D, also known as 1,25(OH)$_2$D or calcitriol [1]. Nevertheless, it is also known that 1-alpha-hydroxylase activity (which represents the ability to produce 1,25-dihydroxyvitamin D) can also be found in activated macrophages, osteoblasts, and keratinocytes, whilst its presence has also been documented in the prostate, colon and breast. Furthermore, 1-alpha-hydroxylase is capable of activating nutritional and prohormonal forms of vitamin D.

1,25(OH)$_2$D is the "active" form of vitamin D. Its serum quantification, although important in some diseases, provides little information on vitamin D status, which can usually be normal or even elevated when hyperparathyroidism is associated with vitamin D deficiency [1].

Upon reaching its target organs, 1,25(OH)$_2$D, which is delivered into the bloodstream by a circulating vitamin D binding protein (VDBP), binds with the vitamin D receptor (VDR). The VDR, which can boast an almost ubiquitous and tissue-dependent expression in nucleated cells, belongs to a large group of ligand-activated nuclear transcription factors. This explains how vitamin D, in addition to regulating intestinal absorption and the mobilisation of calcium and phosphorous, also exerts several functions pertaining to mineral metabolism, aside from its osteogenic effects. Vitamin D responsive elements (VDRE) mediate the effects of vitamin D and lead to changes in the expression of several genes [2] (Fig. 1).

The morphological and functional integrity of bone tissue reflects the regulation and maintenance of bone remodelling. This latter is the expression of the activity of osteoblasts, which control bone neoformation, and osteoclasts, which have the ability to resorb mineralised bone, an activity that is modulated by osteoblasts through the RANK-RANKL-OPG system. The RANK ligand, which is secreted by osteoblasts, and binds to a receptor (RANK) present on the surface of pre-osteoclasts, stimulates their differentiation into active (mature) osteoclasts, whilst OPG, which is also secreted by osteoblasts, prevents RANK ligand binding to its receptor, thus inhibiting osteoclastic activation.

These complex interactions are regulated by local and systemic hormones such as PTH, Wnt signalling pathways, FGF23 and precisely 1,25(OH)$_2$D, which plays a key role in the regulation of bone remodelling [3].

The primary "endocrine" effect that follows vitamin D receptor (VDR) activation is the regulation of mineral and bone homoeostasis. VDR activation controls calcium and phosphate ab-

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Conflict of interest
The author states that there are no conflicts of interest.

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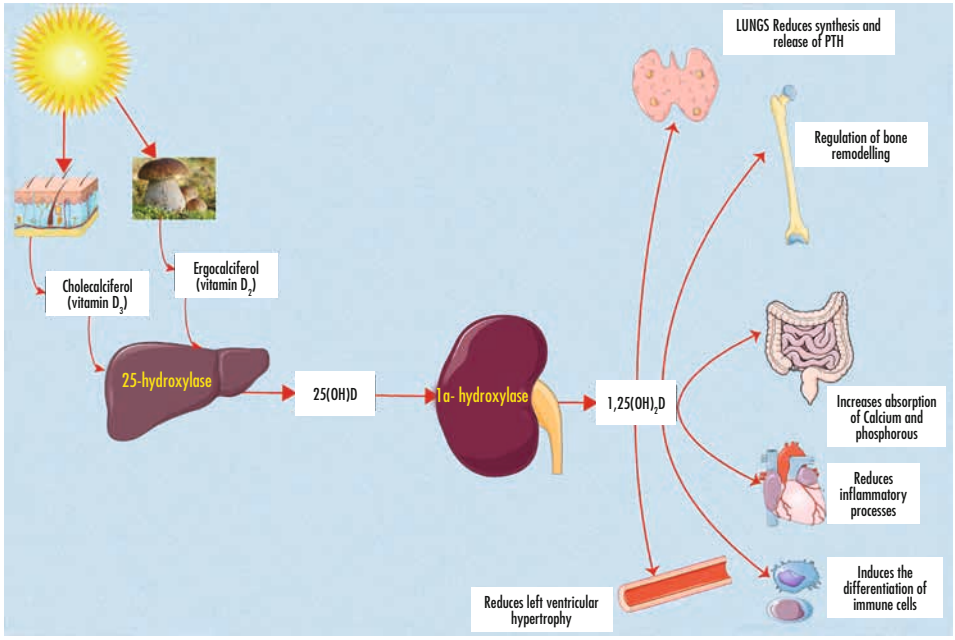

FIGURE 1.
Vitamin D metabolism and its effects.

sorption in the intestine, tubular calcium reabsorption in the kidney and the activity and vitality of bone cells. At the level of osteoblasts, 1,25$(OH)_2$D is able to increase the expression of the Runt-related transcription factor (RUNX2) [2], of osterix (OSX) and of alkaline phosphatase, which are molecules involved in osteoblastic differentiation and mineralisation in several ways. Furthermore, the expression of the Wingless-type (Wnt beta catenin pathway), an important regulator of osteoblast differentiation and function, is increased by 1,25$(OH)_2$D [4].
In addition to stimulating bone formation, calcitriol also promotes bone resorption by increasing the number and activity of osteoclasts. The effects may be mediated by VDRs and alpha-hydroxylase, which are also expressed in osteoclasts, macrophage colony-stimulating factor (m-CSF) and by the receptor activator of nuclear factor kappa-B ligand (RANKL) [5].

VITAMIN D IN CHRONIC KIDNEY DISEASE

Native vitamin D deficiency, which is extremely common in patients with chronic kidney disease (CKD), is attributable to several conditions, such as reduced nutritional intake secondary to dietary restrictions to which kidney patients are frequently subjected (low-protein and low-phosphate diet), reduced appetite and gastrointestinal symptoms, diminished UVB exposure related to reduced mobility and frequent hospitalisations [6]. Then, progressive decline in eGFR has been associated with an increase in the prevalence of vitamin D deficiency. A cross-sectional study conducted on 825 dialysis patients showed that 78% of those patients had a vitamin D deficiency with values < 30 ng/mL and 18% of those patients had severe deficiency with values <10 ng/ mL. That study also showed that low vitamin D values were associated with an increased risk of early mortality.[7] In addition to a native vitamin D deficiency, there is also reduced calcitriol synthesis in CKD. In fact, the progressive loss of renal function is frequently associated with reduced 1-alpha-hydroxylase activity and consequently reduced production of 1,25$(OH)_2$D [2]. In CKD, vitamin D deficiency should be seen in a broader context because it underlies (even though it cannot be seen as the only causative factor) the alterations in calcium, phosphorus and PTH. The development of secondary hyperparathyroidism typically follows the onset of these alterations, which is a clinical and laboratory situation that is peculiar to CKD. Furthermore, among these patients, the altered homoeostasis of mineral metabolism not only affects the skeletal system, but is also closely associated with other important alterations, such as the development of vascular calcifications and, above all, the progression of cardiovascular disease [4].

VITAMIN D IN MINERAL METABOLISM DISORDERS INDUCED BY CKD

CKD is closely associated with the presence of alterations in bone metabolism including dysregulation of calcium and phosphorus metabolism as well as the pathophysiological axis represented by vitamin D-PTH-FGF23. In 2006, the KDIGO (Kidney Disease Improving Global Outcomes) guidelines coined the definition of CKD-MBD (Chronic Kidney Disease-Mineral Bone Disorder) to describe alterations in mineral metabolism and the resulting diseases, such as bone and cardiovascular disorders, associated with increased fracture and cardiovascular risk [8]. These alterations have been found to already be present in approximately 40 to 80% of patients with stage 3 or 4 CKD [9].
Although the exact chronological sequence of the pathophysiological steps is not completely known, it is believed that the increased serum phosphate levels resulting from reduced kidney function stimulate the synthesis and release by osteoblasts and osteocytes of fibroblast growth factor 23 (FGF23), which inhibits PTH synthesis, whist it also inhibits 1-alpha-hydroxylase in the kidney, resulting in reduced calcitriol levels and increased PTH synthesis. Constant stimulation of the parathyroid cells and the failure to correct modifiable factors, such as vitamin D deficiency and hyperphosphatemia, induce a response that is initially "adaptive" but which later becomes, if not corrected by appropriate dietary and pharmacological intervention, "maladaptive", which is characterised by polyclonal hyperplasia of the parathyroid cells. The transition of polyclonal hyperplasia to a "nodular" form of hyperplasia leads to a further progression of secondary hyperparathyroidism, which is characterised, at the level of the parathyroid, by a series of morphological and functional adaptations (reduced expression of the VDR). All of this tends to make the clinical situation poorly responsive to pharmacological therapy, thereby making recourse to surgical treatment necessary (parathyroidectomy) [10].
Therefore, vitamin D does indeed play a key role in the genesis and progression of secondary hyperparathyroidism. Actually, physiological concentrations of 1,25(OH)D have an inhibitory effect on PTH transcription. Moreover, faced with a low affinity for VDR, high serum levels of 25(OH)D have been shown to activate VDR, thus mimicking the effect of 1,25$(OH)_2$D. Besides, 1-alpha-hy-

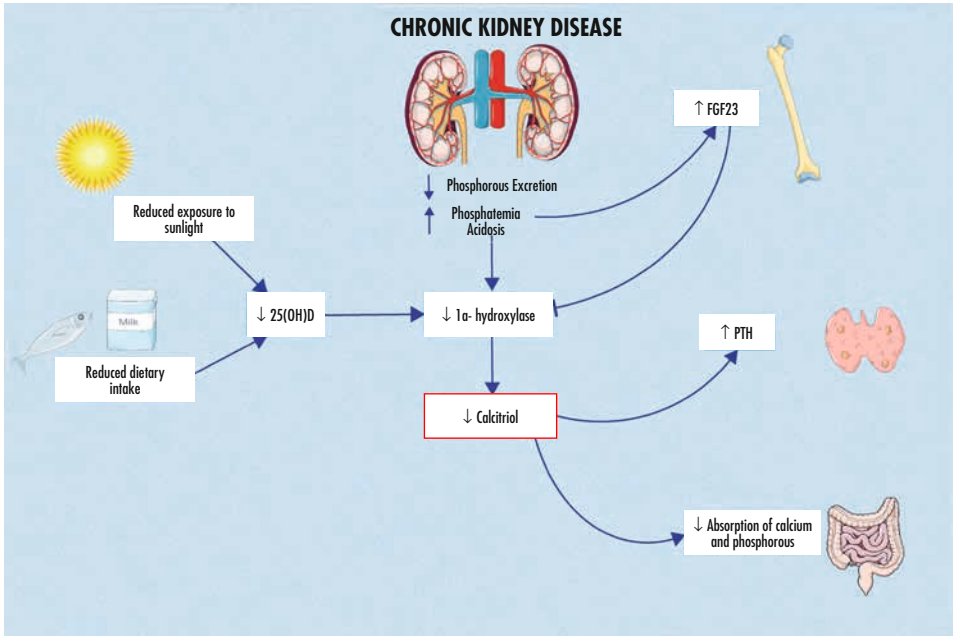

FIGURE 2.
Vitamin D and mineral metabolism disorders in CKD

droxylase, a key enzyme in the conversion of calcifediol to calcitriol, is found in the parathyroid glands and in many other extrarenal tissues, presumably for local production of the hormone.
In this regard, it has been shown that serum levels of 25(OH)D and $1,25(OH)_2D$ increase in response to the administration of nutritional vitamin D (cholecalciferol and ergocalciferol) in dialysed patients. Clearly, this suggests that 1-alpha-hydroxylase activity is also present in extrarenal tissues in CKD, which, in the presence of high levels of 25-hydroxyvitamin D, is able to allow sufficient extrarenal production of 1,25-dihydroxyvitamin D for PTH control.
Since the parathyroid glands express 1-alpha-hydroxylase, a possible autocrine mechanism through which nutritional vitamin D supplementation is able to reduce PTH production should be considered.

The latest KDIGO Guidelines (2017) for the management of CKD-MBD indicate the importance of monitoring serum levels of calcium, phosphate and PTH at the onset of stage G3a CKD and of assessing their trends over time, as well as suggesting that 25(OH)D levels be measured to diagnose vitamin D deficiency (Fig. 2) [8].
With regard to vitamin D values in the general population, reference is made to the recommendations of the Endocrine Society, which establish deficiency with 25(OH)D concentrations at < 20 ng/mL, insufficiency with concentrations between 21 and 29 ng/mL and normal levels or sufficiency with serum levels that are >30 ng/mL [12].
Several guidelines have been formulated over the years concerning the population with CKD that have made different claims regarding the diagnosis and treatment of vitamin D deficiency.
The most recent indications from the National Kidney Foundation, have established that 25(OH)D concentrations at >20 ng/mL can be considered "adequate", whereas concentrations at <15 ng/mL should be treated. For 25(OH)D levels between 15 and 20 ng/mL, consideration should also be given to PTH levels and the counter-regulatory activity of vitamin D on this hormone [13].
Vitamin D supplementation in patients with CKD is still a much debated topic. The KDIGO guidelines suggest supplementation with nutritional vitamin D (cholecalciferol and ergocalciferol), as for the general population, to improve the deficiency status and prevent the onset and progression of secondary hyperparathyroidism [8]. However, there are no conclusive studies currently available on the effect of native vitamin D supplementation on PTH values, although those that are available show no alterations in calcium and phosphorus levels nor any adverse events. It has been hypothesised that nutritional vitamin D supplementation tends to be more effective in preventing the onset and progression of hyperparathyroidism rather than actually reducing PTH values when these have already become elevated in the advanced stages of the disease.
Secondary hyperparathyroidism is a process whose onset begins slowly from the earliest stages of CKD (conservative phase), whilst its onset and/or progression may be prevented by correcting the vitamin D deficiency with early and adequate supplementation treatment. For 25(OH)D levels between 15 and 20 ng/mL, PTH levels should also be considered, whilst the activity of nutritional vitamin D, may reduce the negative effects of secondary hyperparathyroidism on bone remodelling [14], whereas supplementation may also reduce the risk of having PTH levels above the target ranges recommended by KDIGO along with the need for increased drug prescriptions during the subsequent dialysis phase [15].
Furthermore, in light of preclinical and clinical studies, it is likely that in the pathophysiological context of CKD, the antagonising action of the nutritional vitamin on the onset of secondary hyperparathyroidism would be expressed in the presence of higher serum levels of 25(OH) [> 40 ng/mL] than those considered "effective" for the general population [16]. All of this suggests that within a specific pathophysiological context, such as CKD, the currently recommended 25(OH)D levels (> 30 ng/mL) may not be effective, i.e., sufficient for the treatment of SPHT.
Both the KDIGO guidelines and the recommendations of the National Kidney Foundation submit that supplementation with nutritional vitamin D (ergocalciferol, cholecalciferol) should be prioritised first, and only afterwards should active vitamin D compounds (vitamin D receptor activators: VDRA) be introduced, reserving the latter for more advanced stages of CKD and for cases of severe hyperparathyroidism that cannot be controlled by nutritional vitamin D alone. Moreover, treatment with VDRAs should be undertaken when the stage of CKD is advanced, when high PTH values associated with adequate 25(OH)D levels are present and in the absence of elevated levels of calcaemia or phosphoremia [8,17].
In fact, VDRAs should be used with caution since cases of hypercalcaemia and hyperphosphoremia have been reported. Furthermore, their ability to induce excessive PTH depletion may increase the risk of adynamic

bone disease, whilst the increase in FGF-23 levels [17], which in itself is a negative effect, should always be taken into account.

In conclusion, clearly, vitamin D plays a key role in CKD considering that, in light of the ubiquity of vitamin D receptors, its role is crucial for the body's homoeostasis in general and its action cannot be reduced to bone metabolism alone. Therefore, vitamin D deficiency should be diagnosed and treated promptly, even more so in patients with CKD in light of its important impact on hyperparathyroidism and on the regulation of bone metabolism.

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