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The role of vitamin D in female reproductive health - a literature review.

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ABSTRACT

Vitamin D, traditionally associated with the regulation of calcium–phosphate homeostasis, also plays a key role in women's reproductive health. The presence of the vitamin D receptor (VDR) and its metabolic enzymes in the ovaries, endometrium, and placenta indicates its direct involvement in folliculogenesis, steroidogenesis, embryo implantation, and the maintenance of pregnancy. Evidence suggests that vitamin D deficiency may be linked to ovulatory disorders, menstrual irregularities, and reduced oocyte quality, as well as to the pathogenesis of polycystic ovary syndrome (PCOS), endometriosis, and infertility. Although vitamin D supplementation shows potential in improving metabolic and reproductive parameters, clinical findings remain inconsistent, particularly regarding the effectiveness of assisted reproductive technologies. During pregnancy, adequate vitamin D status is associated with a lower risk of preeclampsia, gestational diabetes, and with beneficial effects on fetal skeletal and immune development. Given the high prevalence of deficiency and the variability in individual responses to supplementation, further high-quality studies are needed to precisely determine its clinical significance in women's reproductive health.

Introduction

Vitamin D, traditionally classified as a vitamin regulating calcium–phosphate homeostasis, has in recent decades gained recognition as a steroid hormone with a broad spectrum of biological activity. Beyond its classical role in bone metabolism, an increasing number of studies highlight its importance in the regulation

of immunological, endocrine, and reproductive processes in women. The presence of the vitamin D receptor (VDR) and its metabolic enzymes in reproductive tissues such as the ovaries, endometrium, and placenta suggests a direct influence on the functioning of the human reproductive axis [1].

Women's reproductive health encompasses complex physiological processes, including ovarian follicle maturation, ovulation, embryo implantation, and the maintenance of a healthy pregnancy. Disruptions in these mechanisms may lead to infertility or obstetric complications. In this context, vitamin D has been examined as a potential modulator of reproductive function. Studies have demonstrated its impact on ovarian steroidogenesis, gonadotropin receptor expression, and oocyte quality [2]. The literature increasingly emphasizes the association between vitamin D deficiency and fertility disorders, including polycystic ovary syndrome (PCOS), endometriosis, and implantation failure in assisted reproductive technologies (ART). Recent meta-analyses suggest that adequate vitamin D levels may improve ovulatory parameters and increase pregnancy rates in women with PCOS [5], as well as influence infertility treatment outcomes in in vitro fertilization procedures [3], [6].

At the same time, clinical findings remain inconsistent. For example, a meta-analysis of randomized trials indicated that vitamin D supplementation may increase biochemical pregnancy rates in IVF, yet this does not consistently translate into higher clinical pregnancy rates [3]. This highlights the need for further studies with greater statistical power. Additionally, the role of vitamin D during pregnancy remains a significant area of research. Its deficiency has been linked to an increased risk of preeclampsia, gestational diabetes, and impaired fetal development [4]. Consequently, the importance of optimal vitamin D status is increasingly emphasized even in the preconception period.

The aim of this review is to present the current state of knowledge regarding the role of vitamin D in women's reproductive health, with particular focus on its effects on ovarian function, fertility, embryo implantation, and pregnancy outcomes.

Characteristics of vitamin D

Vitamin D belongs to the group of secosteroids, compounds with a steroid-like structure in which the B-ring is broken. It occurs in two main biological forms: vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol). Vitamin D_2 is derived primarily from plant and fungal sources, whereas vitamin D_3 is synthesized in human skin under UVB radiation and obtained from animal-based foods [7]. Both forms of vitamin D are biologically inactive and require further metabolic activation. They differ in biological efficiency - vitamin D_3 is more effective at increasing serum 25(OH)D concentrations and has a longer half-life in the body [8]. The synthesis of vitamin D_3 begins in the skin, where UVB radiation (290-315 nm) converts 7-dehydrocholesterol into pre-vitamin D_3, which then isomerizes into cholecalciferol [9]. This process depends on several factors, including skin pigmentation, geographic latitude, season, and sun exposure.

Vitamin D from both cutaneous synthesis and dietary intake is transported to the liver, where it undergoes the first hydroxylation to 25-hydroxyvitamin D [25(OH)D], catalyzed by cytochrome P450 enzymes (mainly CYP2R1). This is the main circulating form of vitamin D and the standard marker of vitamin D status [10]. Next, 25(OH)D is transported to the kidneys, where it undergoes a second hydroxylation to the biologically active form - 1,25-dihydroxyvitamin D [$1,25(OH)_2D$], known as calcitriol. This process is catalyzed by the enzyme CYP27B1 and is tightly regulated by hormones such as parathyroid hormone (PTH), phosphate levels, and FGF23 [11]. Calcitriol is the final active form of vitamin D and is responsible for most of its biological effects in the body [9], [11].

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The biological activity of vitamin D is mediated through two primary signaling pathways, genomic and nongenomic. The genomic mechanism involves the regulation of gene expression via the VDR-RXR complex, which directly modulates DNA transcription. Although these effects are characterized by a slower onset, they are persistent and encompass the regulation of cell proliferation, differentiation, and the modulation of immunological and metabolic processes [12], [13]. Simultaneously, vitamin D exerts rapid nongenomic actions that occur independently of DNA transcription. This pathway includes the modulation of ion channels, calcium signaling, and mitochondrial function, which may be mediated by membrane-associated forms of the VDR or other specific membrane proteins [14]. The synergistic interaction between these two pathways facilitates the broad spectrum of multifaceted biological effects attributed to vitamin D in the human body [12], [14].

Vitamin D and the female reproductive system

An increasing body of evidence indicates that vitamin D plays an important role in the functioning of the female reproductive system, extending beyond its classical involvement in calcium–phosphate homeostasis. The expression of the vitamin D receptor (VDR) and enzymes responsible for its metabolism in reproductive tissues suggests its participation in the regulation of ovarian, endometrial, and placental processes [15], [16]. VDR and activating enzymes (including CYP27B1) have been identified in multiple tissues of the female reproductive system, confirming the possibility of local vitamin D action [15].

In the ovaries, VDR and vitamin D-metabolizing enzymes are present in granulosa and theca cells, suggesting a role for vitamin D in follicular maturation and steroidogenesis [17]. Growing evidence indicates that vitamin D may act locally within the ovary, influencing its autocrine and paracrine functions [17], [18]. VDR expression has also been demonstrated in the endometrium, with its activity varying according to the phase of the menstrual cycle. Vitamin D may influence endometrial cell proliferation and modulate immune responses, which is relevant for embryo implantation [16], [19]. In the placenta, the presence of VDR and vitamin D-activating enzymes suggests a role in maintaining pregnancy. Vitamin D participates in the regulation of immune processes and angiogenesis within the placenta [16], and may contribute to maternal-fetal immune tolerance [20]. Vitamin D is involved in the regulation of follicular growth and maturation. Studies have shown that it can influence the expression of genes related to oocyte development and oocyte quality [18], [21]. The presence of VDR in ovarian follicles suggests a direct effect of vitamin D on folliculogenesis [18].

Increasing evidence indicates that vitamin D may modulate ovulation through its effects on steroidogenesis and gonadotropin responsiveness. Observational studies have demonstrated an association between vitamin D deficiency and ovulatory disorders, particularly in women with PCOS [22]. Vitamin D affects the synthesis of ovarian steroid hormones, including estrogens and progesterone. This mechanism is linked to the regulation of steroidogenic enzymes and the expression of FSH and LH receptors [17], [22]. Vitamin D also participates in the modulation of the hypothalamic-pituitary-ovarian axis. It influences the secretion of gonadotropic and steroid hormones, which may contribute to menstrual cycle stability [16]. Additionally, it affects insulin sensitivity, which is relevant in hormonal disorders such as PCOS [22]. Clinical studies suggest that adequate vitamin D levels may be associated with more regular menstrual cycles and improved ovulation. Women with vitamin D deficiency more frequently experience menstrual irregularities [22]. Meta-analyses have shown that vitamin D supplementation may improve cycle regularity in women with ovulatory disorders [22].

Vitamin D and selected reproductive disorders

An increasing body of evidence indicates that vitamin D plays a significant role in the pathophysiology of

several reproductive disorders in women. Its deficiency is frequently observed in patients with infertility, polycystic ovary syndrome (PCOS), and endometriosis, suggesting a potential involvement in the development of these conditions [23].

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age and is often associated with insulin resistance and metabolic abnormalities. Insulin resistance plays a key role in the pathogenesis of PCOS by exacerbating hyperandrogenism and ovulatory dysfunction. Vitamin D may influence tissue sensitivity to insulin by modulating insulin receptor expression and affecting low-grade inflammation [24], [25]. Studies have shown that vitamin D deficiency correlates with increased insulin resistance and a poorer metabolic profile in women with PCOS [26]. Meta-analyses indicate that vitamin D supplementation in women with PCOS may improve metabolic parameters, regulate menstrual cycles, and increase ovulation frequency [26], [27]. A reduction in testosterone levels and improved ovarian function have also been observed following supplementation [27]. However, study results remain inconsistent and depend on dosage and baseline 25(OH)D levels [24].

Endometriosis is a chronic hormone-dependent inflammatory disease characterized by the presence of endometrial tissue outside the uterine cavity. Immune dysregulation plays a crucial role in its pathogenesis, including abnormal inflammatory responses and altered activity of NK cells and macrophages. Vitamin D exhibits immunomodulatory effects by influencing the production of pro-inflammatory cytokines and regulating immune responses [23]. Studies suggest that vitamin D may inhibit the proliferation of endometrial cells and reduce inflammation within endometriotic lesions. Systematic reviews also indicate a possible association between vitamin D deficiency and increased pain severity and infertility in women with endometriosis [28]. Despite promising findings, the effectiveness of supplementation in treating endometriosis has not been conclusively confirmed [28].

Infertility is a multifactorial condition in which the role of vitamin D as a modulator of reproductive function is increasingly explored. Vitamin D affects the endometrium by regulating the expression of genes related to endometrial receptivity and implantation. The presence of VDR in the endometrium suggests a direct role in preparing the uterine lining for embryo implantation [23], [29]. Studies have shown that adequate vitamin D levels may improve markers of endometrial receptivity [29]. In the context of assisted reproductive technologies (IVF/ICSI), research findings are inconsistent. Meta-analyses indicate that vitamin D supplementation may increase biochemical pregnancy rates, although this does not consistently translate into higher clinical pregnancy rates. At the same time, vitamin D deficiency has been associated with poorer oocyte quality and reduced implantation success [30].

Vitamin D in pregnancy

Vitamin D plays an important role during pregnancy, influencing both maternal health and fetal development. Increasing evidence shows that vitamin D deficiency during pregnancy is associated with a higher risk of obstetric complications and abnormal intrauterine development [31], [32].

Preeclampsia is one of the most severe pregnancy complications, characterized by hypertension and organ dysfunction. Numerous meta-analyses show that vitamin D deficiency significantly increases the risk of preeclampsia [33]. Vitamin D supplementation may reduce this risk by approximately 40–45% compared with placebo [34], [35]. This effect is linked to vitamin D’s influence on endothelial function, immune regulation, and the renin–angiotensin system [33].

Vitamin D is also involved in glucose metabolism and insulin sensitivity. Its deficiency correlates with an increased risk of gestational diabetes mellitus (GDM), as confirmed by multiple systematic reviews [32], [36]. Meta-analyses of RCTs indicate that vitamin D supplementation may improve glycemic control and

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reduce the risk of GDM, particularly in women with baseline deficiency [34], [36].
Vitamin D is essential for fetal bone mineralization, as it regulates calcium-phosphate homeostasis and placental calcium transport. Cohort studies and meta-analyses show that vitamin D supplementation during pregnancy may increase bone mass and skeletal mineralization in offspring. Long-term observations also suggest beneficial effects on bone mineral density in childhood [37]. Vitamin D plays a key role in the maturation and regulation of the fetal immune system. It influences T-cell differentiation and modulates inflammatory responses, which may affect the later risk of allergic and autoimmune diseases [31], [32]. Meta-analyses indicate that adequate maternal vitamin D status may reduce the risk of certain immune-related disorders in children [31].

Vitamin D supplementation

Vitamin D supplementation is a key element in the prevention and treatment of deficiency in the general population and in high-risk groups, including women of reproductive age and pregnant women. Due to limited dietary intake and variable cutaneous synthesis, supplementation is often necessary to maintain adequate serum 25(OH)D levels [38], [39].

The primary source of vitamin D in humans is cutaneous synthesis under UVB radiation. In the skin, 7-dehydrocholesterol is converted into cholecalciferol (vitamin D_3), which subsequently undergoes metabolic activation [40]. The efficiency of this process depends on several factors, including geographic latitude, season, skin pigmentation, use of UV filters, and age [40]. In Central Europe, cutaneous synthesis is insufficient for much of the year, particularly from October to March [38]. Natural dietary sources of vitamin D are limited. The richest sources include fatty marine fish (salmon, mackerel, sardines), cod liver oil, egg yolks, and fortified foods such as milk and margarine [39]. However, diet alone is estimated to cover only a small proportion of the body's vitamin D requirements [40].

The most commonly used form of supplementation is cholecalciferol (vitamin D_3), with ergocalciferol (D_2) used less frequently. Guidelines indicate that vitamin D_3 is more effective in raising and maintaining serum 25(OH)D concentrations [38], [41]. Current recommendations emphasize the need to individualize supplementation based on age, body weight, sun exposure, and baseline 25(OH)D levels. According to current guidelines, the standard prophylactic dose ranges from 600 to 2000 IU/day, while individuals with obesity or limited sun exposure may require higher doses [42]. For pregnant women, international guidelines (including WHO and the Endocrine Society) typically recommend 1500-2000 IU/day. Some clinical trials have used doses up to 4000 IU/day, which remain safe and effective in achieving optimal 25(OH)D levels [42], [43]. Supplementation has been shown to reduce the risk of pregnancy complications such as preeclampsia and gestational diabetes [43].

Vitamin D toxicity is rare and usually results from excessive long-term supplementation. The primary mechanism of toxicity is hypercalcemia. Serum 25(OH)D concentrations above 100 ng/mL (250 nmol/L), accompanied by hypercalcemia and hypercalciuria, are considered potentially toxic. Symptoms may include nausea, weakness, cardiac arrhythmias, and kidney damage [39].

The most reliable marker of vitamin D status is serum 25(OH)D concentration. According to European and Polish guidelines:
<20 ng/mL - deficiency
20–30 ng/mL - suboptimal level
30-50 ng/mL - optimal level
50 ng/mL - high level

100 ng/mL - toxicity risk [38].
Monitoring is particularly recommended in individuals at risk of deficiency, during high-dose supplementation, and in pregnancy or chronic disease [38,42].

Conclusions

The analysis of the available literature indicates that vitamin D is a biologically versatile compound whose significance extends far beyond its classical role in calcium-phosphate homeostasis. The presence of VDR receptors and vitamin D-metabolizing enzymes in female reproductive tissues confirms its important involvement in the regulation of ovarian, endometrial, and placental processes, supporting its role as a modulator of reproductive function.

Vitamin D participates in the regulation of key reproductive processes, including folliculogenesis, steroidogenesis, and the preparation of the endometrium for embryo implantation. In this way, it influences ovulation quality, ovarian function, and overall reproductive potential, which is relevant both for natural fertility and assisted reproductive technologies. Current scientific evidence demonstrates an association between vitamin D deficiency and reproductive disorders such as polycystic ovary syndrome, endometriosis, and infertility. At the same time, findings regarding the effectiveness of vitamin D supplementation in improving reproductive parameters and infertility treatment outcomes remain inconsistent, suggesting the complexity of its mechanisms and the influence of multiple coexisting factors.
During pregnancy, adequate vitamin D status is associated with a more favorable course, including a reduced risk of preeclampsia and gestational diabetes, as well as beneficial effects on fetal skeletal and immune development. This highlights its importance not only during the reproductive years but also throughout pregnancy and prenatal development.

Vitamin D supplementation is an effective and safe method of correcting deficiency. However, it requires individualization based on serum 25(OH)D levels and specific risk factors. Monitoring vitamin D status is an essential component of clinical management, particularly in high-risk groups and during pregnancy.
In summary, vitamin D plays a significant and multifaceted role in women's reproductive health, although its full clinical relevance has not yet been clearly defined. Therefore, further research, especially high-quality randomized clinical trials, is needed to precisely determine its impact on fertility, pregnancy outcomes, and the effectiveness of infertility treatment.

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