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Review

Clinical Implications and Preventive Strategies for Neonatal and Infant Hypovitaminosis D: Analysis and Comparison of Current Evidence

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Abstract: Background: Vitamin D is essential for neonatal health, with maternal vitamin D status crucial in fetal development and neonatal outcomes. During pregnancy, vitamin D is transferred to the fetus via the placenta, forming an initial reserve. Postnatally, neonates rely on maternal levels and supplementation due to limited sunlight exposure and immature skin synthesis. **Objectives:** This review evaluates neonatal vitamin D deficiency's causes and clinical consequences, emphasizing its impact on newborn and infant health. **Results:** Maternal vitamin D levels strongly correlate with neonatal 25(OH)D concentrations, influencing birth weight, bone development, and overall health. Supplementation during pregnancy reduces the risk of severe deficiencies and rickets, particularly in exclusively breastfed infants who require daily supplementation of 400 IU. Formula-fed infants typically meet requirements through fortified formulas. Preterm infants are at a higher risk of complications like osteopenia and rickets, with mixed evidence on the effectiveness of higher supplementation doses. Vitamin D is critical in skeletal development, immune function, and protection against respiratory infections such as bronchiolitis and pneumonia. Deficiency is associated with respiratory distress syndrome (RDS), atopic dermatitis, and impaired bone mineralization due to reduced placental calcium transport. **Conclusions:** Vitamin D deficiency during pregnancy and infancy has significant clinical implications, including impaired skeletal and immune development. Maternal and neonatal supplementations are critical to prevent deficiencies, particularly in high-risk groups such as preterm and breastfed infants. Targeted strategies are essential to improve neonatal health outcomes and prevent complications.

Keywords: vitamin D; hypovitaminosis D; preventive strategies; 25-hydroxyvitamin D; childhood rickets; maternal vitamin D

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1. Introduction

Vitamin D is essential for regulating calcium and phosphorus metabolism and is crucial for bone mineralization and overall skeletal health. Beyond supporting bones,

it also plays a role in various biological processes. Vitamin D exists in two primary forms: ergocalciferol (vitamin D2), from plant sources, and cholecalciferol (vitamin D3), produced in the skin when exposed to UVB light (wavelengths 290–315 nm). Vitamin D3 synthesis begins when UVB radiation transforms 7-dehydrocholesterol in the skin into cholecalciferol. Once formed or ingested, vitamin D is activated through two hydroxylation steps. The first occurs in the liver, where 25-hydroxylase converts it into 25-hydroxyvitamin D (25(OH)D), the primary circulating form and a marker of vitamin D status. The second step happens in the kidneys, where 1α-hydroxylase produces the active form, 1,25-dihydroxyvitamin D [1,25(OH)2D], which supports calcium and phosphate absorption. The enzyme 24-hydroxylase regulates excess vitamin D (Figure 1).

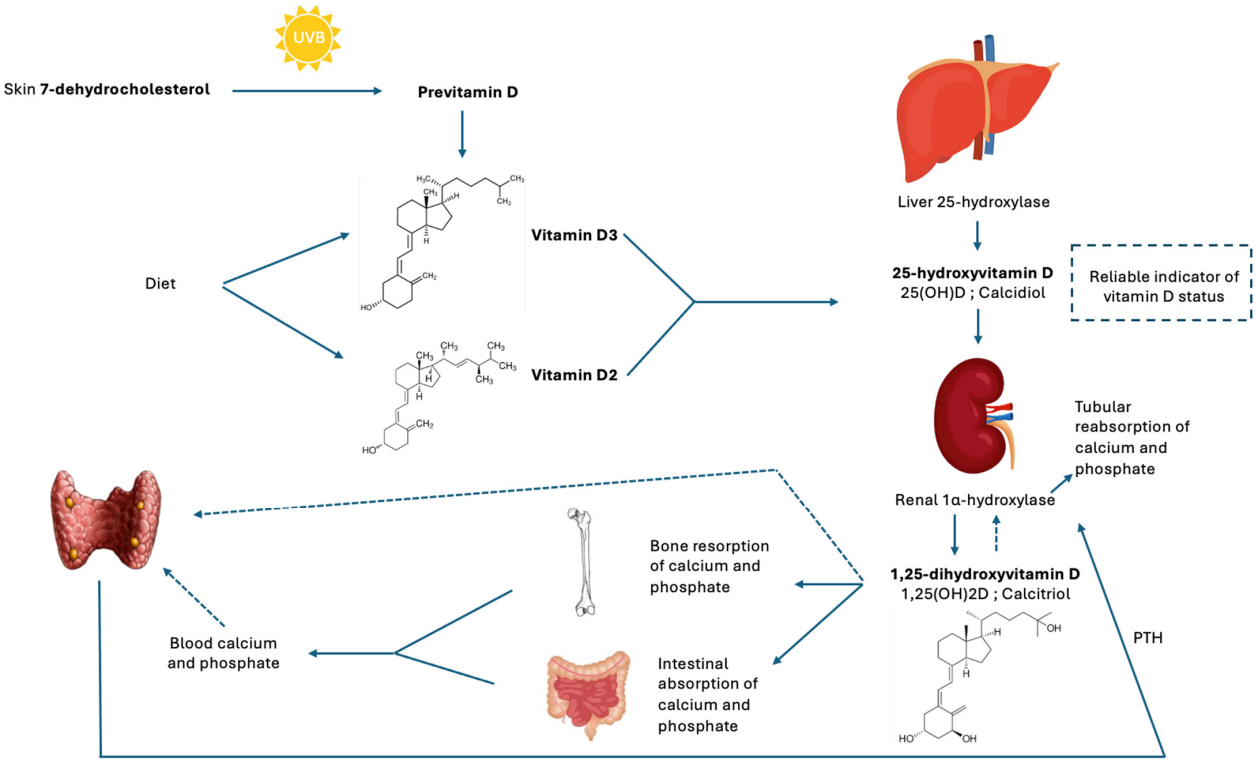

Figure 1. Vitamin D metabolism and regulation: Previtamin D is formed from the skin precursor 7-dehydrocholesterol through the action of UVB radiation. This intermediate spontaneously converts within 48 h into a thermodynamically more stable compound called vitamin D3 or cholecalciferol. Following two subsequent hydroxylations, which occur sequentially in the liver and then in the kidney, calcitriol is formed, a metabolically active compound that promotes the reabsorption of calcium and phosphate at three levels: in the bone, intestine, and kidney. The parathyroids play a key role in regulating calcium–phosphorus balance as, following the detection of low plasma calcium levels, they actively secrete parathormone (PTH), increasing the activity of renal 1α-hydroxylase. In contrast, increased 1,25(OH)2D serum concentrations inhibit parathyroid PTH secretion via negative feedback.

In neonates, maternal vitamin D levels during pregnancy heavily influence vitamin D status. The transfer of vitamin D from mother to fetus occurs primarily in the form of 25(OH)D, which readily crosses the placenta. As a result, fetal vitamin D levels typically reflect approximately 80% of maternal levels at birth [1]. This highlights the critical role of maternal vitamin D sufficiency during pregnancy in ensuring adequate neonatal stores. Deficiency in maternal vitamin D can lead to neonatal hypovitaminosis D, which underscores the importance of adequate maternal intake through diet, supplementation, or controlled sun exposure during pregnancy [2]. After birth, vitamin D intake becomes equally crucial, as neonates must acquire sufficient levels to support rapid growth and development.

Due to human breast milk's low vitamin D content, supplementation is advised for both breastfed and formula-fed infants to prevent deficiencies [3]. Epidemiological studies have demonstrated an inverse correlation between 25(OH)D levels and various diseases, though establishing causality remains a challenge [4,5]. Vitamin D deficiency is widespread globally, and studies have documented it across all trimesters of pregnancy [6–8]. While the optimal levels of 25(OH)D remain debated, some authors advocate maintaining serum levels above 40 ng/mL for better health outcomes [9,10].

The consequences of vitamin D deficiency during pregnancy and infancy can be profound, leading to conditions such as nutritional rickets and impaired immune function. Emerging evidence has also linked vitamin D deficiency to an increased risk of respiratory conditions like asthma [11]. Additionally, vitamin D may protect against autoimmune diseases, such as type 1 diabetes, by modulating immune responses and reducing pancreatic beta-cell destruction [12]. Furthermore, vitamin D enhances innate immunity by stimulating the production of antimicrobial peptides, which help the body combat infections [13]. Table 1 outlines the plasma vitamin D thresholds recommended by the latest updates in the literature, reflecting the diversity in guideline definitions.

Table 1. Threshold values for plasma 25(OH)D indicate deficiency, insufficiency, and sufficiency according to international guidelines.

Society	Authors (Year)	Severe Deficit	Deficit	Insufficiency	Sufficiency
Lawson Wilkins Pediatric Endocrine Society	Misra M (2008) [14]	<5 ng/mL	5–14 ng/mL	15–19 ng/mL	≥20 ng/mL
Endocrine Society	Holick MF (2011) [10]	-	<20 ng/mL	20–29 ng/mL	≥30 ng/mL
ESPHGAN	Braegger C (2013) [15]	<10 ng/mL	<20 ng/mL	-	≥20 ng/mL
Australian and New Zealand Bone and Mineral Society	Paxton GA (2013) [16]	<5 ng/mL	5–11 ng/mL	12–19 ng/mL	≥20 ng/mL
American Academy of Pediatrics	Golden NH (2014) [3]	-	<20 ng/mL	-	≥20 ng/mL
Italian Pediatric Society and the Italian Society of Preventive and Social Pediatrics	Saggese G (2018) [17]	<10 ng/mL	10–20 ng/mL	20–29 ng/mL	≥30 ng/mL

This review investigates the causes of neonatal vitamin D deficiency, highlighting its clinical significance and the influence of maternal vitamin D status on fetal development. It further examines the subsequent effects of vitamin D levels on children's growth and overall health. Moreover, the review evaluates and compares current evidence on supplementation strategies and therapeutic interventions for neonates and infants.

2. The Role of Maternal Vitamin D in Neonatal and Infant Growth

In newborns, vitamin D is primarily acquired through maternal transfer during pregnancy and postnatal supplementation. During pregnancy, the placenta facilitates maternal-to-fetal vitamin D transfer, creating an initial reserve for the neonate. However, neonates rely heavily on maternal vitamin D status and postnatal supplementation to maintain adequate serum levels due to their limited sunlight exposure and immature skin synthesis capabilities. An intriguing approach could involve utilizing the newborn screening test performed on dried blood spot (DBS) samples at birth to determine vitamin D levels. This could include both targeted genetic testing, such as identifying pathogenic variants in the

CYP27B1 gene indicative of vitamin D-dependent rickets type 1A, and direct biochemical assays to measure vitamin D concentrations [18,19]. Newborn screening, widely used for detecting various metabolic disorders, is universally recognized as a powerful diagnostic tool for preventing harm in the crucial early days of life and minimizing the risk of long-term complications [20]. A British study using vitamin D assays conducted on dried blood spot samples revealed that the current prenatal supplementation program in the UK falls short of adequately protecting infants from vitamin D deficiency, particularly among higher-risk ethnic minority groups. Nearly 70% of all infants, and 85% of those born in winter, had 25OHD concentrations below 20 ng/mL [21].

The most recent evidence in the literature highlights 25(OH)D as a key marker for assessing vitamin D status, reflecting total body stores. Studies underscore a strong correlation between maternal and neonatal 25(OH)D levels, illustrating the significant impact of maternal vitamin D status on fetal and neonatal health outcomes [22,23]. For example, Singh et al. [24] observed strong links between maternal 25(OH)D levels, neonatal birth weight, and maternal and neonatal vitamin D levels. Meanwhile, Kokkinari et al. [25] reported that maternal vitamin D deficiency at the end of pregnancy correlated with low neonatal birth weight despite supplementation, without affecting neonatal height or head circumference.

Contrasting findings exist regarding the influence of maternal deficiency on other birth parameters. Some studies found no significant effect on gestational length or neonatal BMI at later ages [26,27], while Zhao et al. [28] demonstrated a positive association between higher cord blood vitamin D levels and BMI-for-age growth during infancy. Chen et al. [29] reported that sufficient maternal 25(OH)D levels in the third trimester reduced the risk of excessive weight gain in children up to 4 years. In contrast, Mori et al. [30] suggested a minimal influence of maternal vitamin D levels on long-term growth outcomes.

Emerging research also investigates maternal lipid profiles and their role in fetal growth. Zheng et al. [31] revealed that adequate maternal 25(OH)D levels could mitigate risks of infants being born large (LGA) or small (SGA) for gestational age, potentially through the modulation of maternal lipid influences.

Recent intervention studies have further underscored the benefits of maternal vitamin D supplementation. For instance, Prabhakar et al. [32] demonstrated that third-trimester supplementation significantly increased neonatal and infant 25(OH)D levels, preventing severe deficiencies and rickets in exclusively breastfed infants at 6 months.

Therefore, while maternal vitamin D status plays a pivotal role in shaping neonatal and infant health, particularly concerning bone development and birth parameters, according to some authors, its long-term impact on growth and anthropometric outcomes remains a subject of ongoing research.

3. Clinical Implications of Vitamin D Deficiency in Neonates and Infants

Vitamin D deficiency in newborns significantly contributes to various early-life health issues. This condition is associated with a range of complications across different body systems. Examples include respiratory disorders such as respiratory distress syndrome and lower respiratory infections, hormonal imbalances during mini-puberty, a greater likelihood of developing atopic dermatitis, neurological impairments like hypoxic–ischemic encephalopathy, an increased vulnerability to early-onset sepsis, and skeletal problems like rickets and osteopenia. These findings emphasize the critical role of maintaining adequate vitamin D levels in mothers and infants to prevent such outcomes, highlighting the value of strategic supplementation during pregnancy and infancy.

3.1. Respiratory Distress Syndrome (RDS)

A systematic review and meta-analysis examined the relationship between serum vitamin D levels in mothers and infants and the risk of RDS in newborns. It found a significant association between low vitamin D levels and an increased risk of RDS and transient tachypnea of the newborn (TTN). Maternal supplementation with 50,000 IU of vitamin D before childbirth reduced the likelihood of RDS in newborns by 64% [33].

Maternal 25(OH)D status may impact fetal lung development. When comparing fetal pulmonary artery Doppler indices with maternal 25(OH)D levels, low maternal 25(OH)D appears to be associated with a higher fetal pulmonary artery pulsatility index (PI) and peak systolic velocity (PSV), as well as a lower acceleration time-to-ejection time (AT/ET) ratio. Neonates with RDS exhibited a higher PI and lower PSV and AT/ET ratios than those without RDS [34].

Lower respiratory tract infections (LRTIs), primarily pneumonia and bronchiolitis, are significant causes of morbidity and mortality in early childhood. Vitamin D enhances innate immunity by regulating antimicrobial peptide production [13]. Its active form, calcitriol, is produced locally in the lung epithelium, playing a role in host defense against respiratory pathogens [35].

A prospective study in the Netherlands found that newborns with 25(OH)D < 20 ng/mL had a higher incidence of respiratory syncytial virus-associated LRTI compared with those with 25(OH)D > 30 ng/mL [36].

Other studies also reported lower 25(OH)D levels in infants with bronchiolitis, with an inverse correlation between serum 25(OH)D levels and disease severity [37,38].

3.2. Mini-Puberty

Some authors have studied the relationship between 25(OH)D levels and gonadal hormones during mini-puberty, a critical period for future gonadal function. Kılınç et al. [39] observed that term, appropriate-for-gestational-age girls with 25(OH)D deficiency had higher total testosterone levels (0.52 ± 0.32 ng/mL vs. 0.26 ± 0.2 ng/mL; p: 0.008) and lower inhibin B (21.2 ± 15.71 pg/mL vs. 53.25 ± 47.25 pg/mL; p: 0.021) compared with those with sufficient 25(OH)D, suggesting that 25(OH)D status may modestly affect gonadal function during mini-puberty.

Daughters of women with 25(OH)D deficiency show higher and more sustained elevation in hormone levels (follicle-stimulating hormone, luteinizing hormone, and estradiol) during their first 18 months of life, along with a larger ovarian volume, uterine length, and breast diameter, compared to daughters of mothers with normal 25(OH)D levels during pregnancy. These findings suggest that low maternal vitamin D status leads to a more pronounced and prolonged activation of the reproductive axis, with more remarkable sexual organ development depending on the severity of the 25(OH)D deficiency [40].

3.3. Atopic Dermatitis

The potential protective role of 25(OH)D against atopic eczema has been suggested by several studies. Liu et al. [41] concluded that sufficient 25(OH)D levels in umbilical cord blood ($\geq$30 ng/mL) are associated with a lower risk of eczema in infants up to one year of age.

Zhang et al. [42] demonstrated that infants born to mothers with 25(OH)D deficiency in the first trimester had a higher risk of atopic dermatitis (RR: 1.77), whereas the risk appeared to decrease in infants whose mothers took 25(OH)D supplements during pregnancy (RR: 0.79).

El-Heis et al. [43] examined the effect of maternal 25(OH)D supplementation during pregnancy on the risk of atopic eczema in children. This study, part of the UK MAVIDOS

trial, involved pregnant women randomly assigned to receive either 1000 IU of cholecalciferol (vitamin D) daily or a placebo, starting at 14 weeks of gestation until delivery. The results showed that at 12 months of life, children whose mothers had taken cholecalciferol were significantly less likely to develop atopic eczema than those whose mothers had received the placebo. However, this protective effect weakened over time and was no longer significant by 48 months. The study also highlighted the role of breastfeeding duration in the effectiveness of the supplementation. Infants breastfed for at least one month had a reduced risk of eczema if their mothers took vitamin D, while no such benefit was seen in those breastfed for less than a month.

3.4. Hypoxic–Ischemic Encephalopathy (HIE)

The role of vitamin D as an adjunct therapy for HIE was investigated by Hagag et al. [44]. The study involved 60 neonates with grade II HIE who were subdivided into two groups. Group I (30 neonates) received a daily oral dose of vitamin D3 (1000 IU) for 2 weeks, along with erythropoietin and magnesium sulfate; group II (30 neonates) received only erythropoietin and magnesium sulfate, without vitamin D. Both groups were compared to a control group of 30 healthy neonates. The results showed no significant differences in pH, PO2, and PCO2 levels between the two groups before treatment. However, after 2 weeks, group I had significantly higher pH levels than group II. Before treatment, serum calcium-binding protein B (S100-B) levels were higher in both groups compared with the control group; after therapy, group I showed a significant reduction in S100-B levels.

A recent study comparing maternal and neonatal 25(OH)D levels in neonates with and without perinatal asphyxia found that both maternal and neonatal 25(OH)D levels were significantly lower in the asphyxia group, suggesting the potential benefits of 25(OH)D supplementation during pregnancy to reduce this risk [45].

25(OH)D deficiency in full-term neonates with HIE has been studied as a predictive factor for mortality, abnormal neurological outcomes, and developmental delays at 12 weeks. A recent study [46] highlighted that, using a threshold of <12 ng/mL, 25(OH)D deficiency predicted mortality with 100% sensitivity and 17% specificity and poor developmental outcomes with 100% sensitivity and 50% specificity. Therefore, 25(OH)D deficiency at birth can serve as an effective screening tool and prognostic marker for severe perinatal depression in neonates.

3.5. Early Onset Sepsis (EOS)

Several studies have investigated the relationship between maternal and neonatal 25(OH)D levels and EOS. In the study by Cetinkaya et al. [47], maternal and neonatal 25(OH)D levels were found to be significantly lower in the EOS patient group compared with the control group (22.2/8.6 ng/mL vs. 36.2/19 ng/mL, respectively; $p < 0.001$)

Also, Mohamed et al. [48] observed that neonates with EOS had significantly lower vitamin D levels than healthy controls. They identified that a 25(OH)D level below 19.7 ng/mL was associated with a higher risk of EOS (area under the ROC curve = 0.76).

The therapeutic role of 25(OH)D supplementation in EOS has also been studied. Hagag et al. [49] conducted a study that included 60 neonates with sepsis, divided into two groups: group I (30 neonates) received antibiotics only, while group II (30 neonates) received both antibiotics and vitamin D. The study also included 30 healthy neonates as a control group. Group II showed significant improvements in sepsis score and C-reactive protein (CRP) levels compared to group I ($p < 0.05$). A negative correlation was observed between CRP and serum 25(OH)D levels in group II at the start and after 2 weeks of treatment. In predicting EOS, the ROC curve for serum 25(OH)D levels showed a cut-off value of 20 ng/mL, with 100% sensitivity, 73% specificity, and 87% accuracy. Therefore,

vitamin D supplementation improves sepsis scores and reduces CRP levels, indicating its potential as an adjunct therapy for neonatal sepsis.

Other studies also support this relationship between 25(OH)D levels and sepsis markers [50,51].

3.6. Retinopathy of Prematurity (RoP)

Some studies have investigated the relationship between 25(OH)D plasma levels and the risk of developing RoP.

A case–control study compared serum vitamin D levels in VLBW infants with and without RoP. Infants with RoP showed significantly lower neonatal and maternal vitamin D levels, as well as lower Apgar scores and birth weight compared with those without RoP. The study also observed that as the stages of RoP worsened, progressively lower levels of vitamin D were associated with it [52].

25(OH)D levels show a positive correlation with vascular endothelial growth factor (VEGF) levels in early stages of RoP and a negative correlation in later stages, suggesting distinct signaling interactions throughout the disease. This may imply that 25(OH)D supplementation could potentially influence the progression and outcome of RoP [53].

3.7. Osteopenia and Rickets

Vitamin D is critical in fetal skeletal growth and mineralization, with 80% of skeletal mineralization occurring during the third trimester [54]. Plasma membrane calcium-dependent ATPase regulates placental calcium transport (PMCA 1–4) gene expression [55], and PMCA3 mRNA levels have been shown to predict neonatal whole-body bone mineral content (WB-BMC) at birth [56]. Evidence also suggests that 1,25(OH)2D may influence PMCA gene expression [57], indicating that vitamin D may support skeletal development by enhancing placental calcium transport and its availability to the fetus [7].

Recent research studies has linked maternal vitamin D status during pregnancy or in cord blood to offspring bone parameters, including bone mass, quality, and size, assessed by techniques like X-ray densitometry (DXA) and ultrasound [58,59]. Neonates with 25(OH)D levels below 13.2 ng/mL had lower WB-BMC compared with those with higher levels [60], and those with 25(OH)D above 20.8 ng/mL exhibited higher bone mineral density and content compared with those with levels below 14.5 ng/mL [61]. The long-term effects of maternal vitamin D deficiency have also been explored. For instance, children of mothers with 25(OH)D levels below 11 ng/mL during pregnancy had significantly lower WB-BMC at nine years of age compared to those whose mothers had sufficient vitamin D levels [7]. However, more extensive cohort studies have reported no significant association between maternal vitamin D levels and childhood bone mass [62,63].

Studies on vitamin D supplementation during pregnancy provide further insights. The MAVIDOS trial showed that supplementation with vitamin D3 (1000 IU/day) increased offspring bone mass, particularly in babies born during winter and early spring [64]. Similarly, the COPSAC2010 study found that higher doses of vitamin D (2800 IU/day) improved offspring bone mineral content and density, especially in children of mothers with insufficient vitamin D levels [65].

Maternal vitamin D status is also a critical factor in preventing rickets. Lautatzis et al. [66] investigated the effects of maternal vitamin D supplementation in low-income countries lacking routine infant supplementation using data from a randomized, placebo-controlled trial in Bangladesh. Pregnant women were divided into five groups, receiving either a placebo or varying weekly doses of vitamin D (4200 IU, 16,800 IU, or 28,000 IU) during the second trimester until delivery. Among the 790 infants screened, 4.9% had biochemical rickets. The highest prevalence was in the placebo group (7.8%), while combined

prenatal and postpartum supplementation at 28,000 IU/week significantly reduced the risk (1.3%; RR: 0.16; 95% CI: 0.03–0.72). Prenatal-only supplementation at any dose did not significantly lower the risk. These findings suggest that maternal vitamin D supplementation at 28,000 IU/week during late pregnancy and lactation can reduce the risk of infantile rickets.

Rickets is primarily diagnosed through radiological imaging rather than laboratory tests. Although biochemical abnormalities are often present, they are not definitive markers for the condition, and no specific biochemical values are consistently linked. In neonatal intensive care units, serum total alkaline phosphatase activity (APA) is the most commonly used screening tool. Elevated APA levels are frequently observed in very preterm infants, even without rickets. However, significantly high levels (particularly above 800 IU/L) are more likely to indicate rickets, especially when paired with low serum phosphorus and a history of delayed enteral feeding or necrotizing enterocolitis [67]. Routine screening in VLBW infants often includes total serum APA measurement, though bone-specific APA is less widely available and lacks standardized thresholds for abnormal values.

Treatment focuses on optimizing bone mineral intake. This includes adding calcium and phosphorus to the infant's diet through fortifiers or high-mineral formulas. Supplementation may be required if vitamin D levels are inadequate, though it has a limited impact unless serum 25(OH)D levels are below 30 nmol/L. Oral 1,25-dihydroxyvitamin D (calcitriol) may be considered. Table 2 summarises the neonatal complications of vitamin D deficiency.

Table 2. Complications of neonatal vitamin D deficiency divided by systems involved.

System Involved	Type of Complication	Main Clinical Consequences
Respiratory system	Respiratory Distress Syndrome (RDS), Transient Tachypnea of the Newborn (TTN), Lower Respiratory Tract Infections (LRTIs)	Increased risk of RDS and TTN [33]; impaired fetal lung function (Doppler indices) [34]; higher incidence of bronchiolitis and pneumonia [35].
Reproductive system	Alterations During Mini-Puberty	Higher testosterone and reduced inhibin B in girls [39]; prolonged activation of reproductive axis in children of mothers with vitamin D deficiency during pregnancy [40].
Immune system	Atopic Dermatitis	Higher risk of atopic eczema in infants with low cord blood vitamin D levels [41,42]; vitamin D supplementation during pregnancy could reduce the risk [42,43].
Central nervous system	Hypoxic–Ischemic Encephalopathy (HIE)	Predictor of poorer neurological outcomes and higher neonatal mortality in newborns with HIE [46]; possible reduction in S100-B levels and improvement in blood parameters in case of vitamin D supplementation [44]; 25(OH)D supplementation during pregnancy could reduce risk of asphyxia [45].
Hematologic/ immune system	Early-Onset Sepsis (EOS)	Increased risk of EOS [48]; improved sepsis scores and reduced CRP levels in case of vitamin D supplementation [49].
Visual system	Retinopathy of Prematurity (RoP)	Higher risk and severity of ROP [52]; potential influence of supplementation on disease progression [53].
Skeletal system	Osteopenia and Rickets	Higher prevalence of biochemical rickets in infants of mothers with vitamin D deficiency [7]; improved bone mineralization in case of supplementation during pregnancy and lactation [68].

4. Optimizing Vitamin D Levels During Pregnancy and in Neonates and Infants

The American Academy of Pediatrics [69] suggests that obstetricians should consider monitoring maternal vitamin D status by measuring its concentrations in pregnant women.

Different international societies have issued recommendations on the appropriate vitamin D dosage during pregnancy, as summarized in Table 3.

Various researchers have suggested different supplemental doses of vitamin D: Dawson-Hughes et al. [68] recommend a daily supplemental intake of 1000 to 1600 IU (25 to 40 mcg/day). Similarly, other studies advocate for doses of approximately 1000 IU/day

to maintain a blood concentration of 25(OH)D above 50 nmol/L (20 ng/mL) [70–72]. In addition, alternative regimens are proposed, such as weekly doses of 5000 IU (125 mcg/week) [73] or a single high dose of 200,000 IU (5 mg) or more [74].

Table 3. Recommended vitamin D intake during pregnancy.

Institution/Scientific Society	Authors (Year)	Recommended Vitamin D Intake During Pregnancy
Recommended Nutrient Intake (RNI) by WHO/U.N. FAO	WHO (2004) [70]	200 IU/day
Expert Panel in Central Europe	Płudowski (2013) [75]	1500 to 2000 IU/day
The Royal College of Obstetricians and Gynaecologists	RCOG (2014) [76]	400 IU/day to 1000 IU/day for high-risk women. In addition, for women with pre-eclampsia, min. 800 IU/day combined with calcium
European Food Safety Authority (EFSA) and USA Institute of Medicine	EFSA (2016) [77]	600 IU/day

Neonatal vitamin D supplementation differs significantly between term and preterm infants, reflecting their distinct physiological needs and the evolving guidelines over the years. Breast milk contains only trace amounts of vitamin D, necessitating supplementations for all breastfed infants. Formula-fed infants typically meet their vitamin D needs through fortified formulas containing 400 IU of vitamin D per liter. However, supplementation may still be required for those consuming less than 1 L daily. Supplementation is even more significant in preventing vitamin D deficiency in regions or populations with limited sunlight exposure.

Standardized vitamin D dosing is typical for breastfed neonates and infants, regardless of plasma 25(OH)D levels. Research indicates that neonates with deficient cord blood 25(OH)D levels (<20 ng/mL) are less responsive to standard-dose supplementation, often displaying lower serum levels at one month and higher rates of complications such as respiratory distress, sepsis, and prolonged hospital stays. Individualized dosing based on initial plasma levels could potentially mitigate these risks and improve health outcomes [78].

While the optimal vitamin D level remains debated, most U.S. authorities consider 20 ng/mL a target value [79,80].

Preterm infants face unique challenges, necessitating tailored supplementation strategies. Due to their reduced stores at birth and limited capacity for passive calcium absorption, many guidelines propose higher doses of vitamin D. U.S. guidelines from the American Academy of Pediatrics (AAP) recommend weight-specific dosing, with 400 IU/day for infants over 1500 g and an initial dose of 200 IU/day for those under 1500 g, increasing to 400 IU/day upon reaching 1500 g and tolerating enteral feeding [79]. European guidelines recommend higher doses, with ESPGHAN advising 800–1000 IU/day during the early months of life [81]. Although doses of 800–1000 IU/day can raise serum 25(OH)D levels more rapidly than 400 IU/day, there is limited evidence of meaningful clinical benefits and some concern about the risk of excess vitamin D, particularly in the smallest VLBW infants [82–84]. Nevertheless, supplementing with 800–1000 IU/day is unlikely to raise significant toxicity concerns or necessitate intensive monitoring, as doses up to 1000 IU/day have been safely used in preterm infants in many countries over time. Italian recommendations suggest 400–800 IU/day for infants weighing ≥1500 g and 200–400 IU/day for those below this threshold [17]. Australian guidelines adopt a weight-based approach, recommending 200 IU/kg/day with a 400 IU/day cap for preterm infants [16]. Recent recommendations by Pludowski et al. [85] differentiate preterm infants based on gestational age and suggest the following: for infants born at <32 weeks gestation, if enteral nutrition is possible, 800 IU/day of cholecalciferol should be administered from the first

days of life, regardless of the feeding method, during the first month; for infants born at 33–36 weeks gestation, 400 IU/day of cholecalciferol should be given from the first days of life, irrespective of the feeding method.

In preterm infants, the extent to which calcium absorption depends on vitamin D after birth remains uncertain. Infants born to mothers with lower vitamin D levels may take longer to reach these benchmarks. Still, no evidence supports the routine administration of higher doses for clinical benefit [86]. Furthermore, the high mineral concentrations in preterm formulas and human milk fortifiers may result in a predominance of passive, non-vitamin D-dependent absorption during the first weeks of life [87]. The routine monitoring of 25(OH)D is generally unnecessary for most preterm infants, except in specific cases such as rickets or renal or hepatic insufficiency, where higher doses of vitamin D and closer monitoring may be required. The timing of initiating oral vitamin D supplementation in preterm infants varies across neonatal units, but typically, supplementation begins when full enteral feeding is achieved [86].

Most international guidelines recommend standardized vitamin D dosing for full-term infants for breastfed neonates and infants, regardless of plasma 25(OH)D levels. The AAP advises supplementing all breastfed or partially breastfed infants with 400 IU/day of vitamin D until weaning [3], similarly to recommendations from the Endocrine Society [10] and the Canadian Paediatric Society [88]. These doses are consistent with the recommendations by ESPGHAN [15], which advise 400 IU/day for all term infants during their first year, regardless of feeding method. The Australian and New Zealand Bone and Mineral Society emphasizes the importance of identifying risk factors and administering 400 IU/day of vitamin D to infants born to vitamin D-deficient mothers or exposed to reduced sunlight [16]. Italian guidelines go a step further, recommending 400 IU/day for all newborns regardless of feeding type [17]. The most recent European guidelines, originating from Poland, recommend the following daily intake of cholecalciferol: for infants aged 0–6 months, 400 IU/day (10 µg/day) starting from the first days of life, regardless of the feeding method; and for infants aged 6–12 months, 400–600 IU/day (10–15 µg/day), depending on the daily intake of vitamin D through meals [85].

A 2020 Cochrane review confirmed that providing vitamin D via a supplement drop to breastfed infants increases serum 25(OH)D concentrations [89]. Alternatively, high-dose maternal supplementation of 6000 IU/day or more during lactation achieves a similar effect on infant vitamin D status [90]. A recent Canadian study found no impact on bone mineralization at 3 years of age when breastfed infants received more than 400 IU/day despite observing higher 25(OH)D levels with larger dose [91].

For mixed-fed infants, supplementation requirements depend on formula intake. Modern formulas, designed to meet vitamin D needs at lower consumption volumes (~800 mL/day), have reduced the necessity for additional supplementation. Exclusively formula-fed infants generally do not require extra vitamin D, whereas mixed-fed infants often need supplementation to meet the 400 IU/day target [86]. The routine monitoring of 25(OH)D levels is unnecessary for healthy, full-term infants, regardless of their feeding method.

While standardized vitamin D supplementation is broadly effective, tailored approaches for preterm newborns may enhance outcomes. Ensuring adequate vitamin D intake through direct infant supplementation or maternal dosing remains a cornerstone of neonatal and infant health. Tables 4 and 5 summarize the recommendations from the leading international scientific societies on vitamin D prophylaxis in preterm and term infants.

Table 4. Main international recommendations for vitamin D supplementation in children born preterm.

Scientific Society/Nation	Authors (Year)	Recommendation
ESPGHAN	Agostoni (2010) [81]	800–1000 IU/day (avoiding a per kg dosage) for the first months of life.
Australian and New Zealand Bone and Mineral Society	Paxton GA (2013) [16]	200 IU/kg/day, with a maximum of 400 IU/day.
American Academy of Pediatrics	Abrams SA (2013) [92]	Newborns with birth weights more than 1500 g should take 400 IU/day. Newborns with birth weights less than 1500 g should take 200–400 IU/day, which should be increased to 400 IU/day when infants reach a weight of 1500 g and tolerate total enteral feeding.
Italian Pediatric Society and the Italian Society of Preventive and Social Pediatrics	Saggese G (2018) [17]	400–800 IU/day for preterm infants with a weight $\geq$ 1500 g. 200–400 IU/day by enteral feeding in preterm infants with a weight < 1500 g.
Poland	Płudowski P (2023) [85]	Neonates born at <32 weeks of gestation: if enteral nutrition is possible, 800 IU/day of cholecalciferol from the first days of life, regardless of the feeding method, during the first month of life. The intake from a diet should be calculated from the second month of life. Neonates born at 33–36 weeks of gestation: 400 IU/day of cholecalciferol from the first days of life, regardless of the feeding method.

Table 5. Main recommendations for vitamin D prophylaxis in babies born at term.

Scientific Society/Nation	Authors (Year)	Recommendation
Endocrine Society	Holick MF (2011) [10]	400–1000 IU/day.
Australian and New Zealand Bone and Mineral Society	Paxton GA (2013) [16]	Children at risk of vitamin D deficiency should receive supplementation with 400 IU/day for at least the first year of life. Children at risk: children born to mothers with vitamin D deficiency and fed with exclusive breastfeeding and with at least one other risk factor among reduced sun exposure, dark skin (phototypes V and VI), and disease or medication that interferes with vitamin D metabolism.
ESPGHAN	Braegger C (2013) [15]	400 IU/day throughout the first year of life in all children.
Canadian Paediatric Society	Critch JN (2014) [88]	0–6 months: 400 IU/day of vitamin D in breastfed infants. Children who are not breastfed do not require vitamin D prophylaxis. Partially breastfed infants should receive 400 IU/day, regardless of the amount of formula milk taken. 6–12 months: Continued supplementation with 400 IU/day in children who are still exclusively breastfed or who are taking breast milk.
American Academy of Pediatrics	Golden NH (2014) [3]	Breastfed or partially breastfed infants should be supplemented with 400 IU/day until at least the time of weaning.
Italian Pediatric Society and the Italian Society of Preventive and Social Pediatrics	Saggese G (2018) [17]	400 IU/day in all newborns independent of the type of feeding.
Poland	Płudowski P (2023) [85]	0–6 months: 400 IU/day (10 µg/day) of cholecalciferol from first days of life, regardless of the feeding method. 6–12 months: 400–600 IU/day (10–15 µg/day) of cholecalciferol, depending on the daily amount of vitamin D consumed with meals.

5. Management of Neonatal Hypovitaminosis D

Vitamin D therapy is essential for infants who exhibit clinical signs of hypocalcemia resulting from vitamin D deficiency or rickets and when vitamin D levels fall within the deficiency range [3,14,85]. In such cases, prompt and appropriate treatment is crucial to restore vitamin D levels, address the symptoms, and prevent potential complications related to bone health and calcium metabolism. Despite the importance of standardized treatment protocols, no international consensus on the plasma 25(OH)D threshold defines deficiency. Different guidelines propose varying plasma vitamin D levels to identify deficiency, creating potential disparities in diagnosis and management strategies.

The decision to use a therapeutic rather than prophylactic dosage depends on identifying a clear 25(OH)D deficiency rather than insufficiency. This distinction is critical, as it may influence the clinical approach for individual patients. International recommendations for neonatal vitamin D deficiency therapy differ in their approaches and have evolved.

For example, the Australian and New Zealand Bone and Mineral Society [16] advise 400 IU/day for infants with 25(OH)D levels between 12 and 20 ng/mL and 1000 IU/day for those below 12 ng/mL, with a three-month treatment period followed by maintenance of 400 IU/day. In contrast, Polish guidelines [85] recommend a simplified regimen of 2000 IU/day for infants aged 0–12 months, explicitly advising against using calcifediol. Similarly, the American Academy of Pediatrics [3] supports administering 2000 IU/day for six weeks or a weekly dose of 50,000 IU, followed by maintenance therapy with 400–1000 IU/day. The Endocrine Society [10] aligns with these recommendations but also offers the option of using either vitamin D2 or D3, emphasizing the importance of a maintenance dose after six weeks of therapy. Lastly, the Italian Pediatric Society [17] advocates for 2000 IU/day or 50,000 IU/week for 6–8 weeks in asymptomatic infants with plasma vitamin D levels below 20 ng/mL, recommending the reevaluation of serum levels post-treatment. They also stress continuing age-appropriate supplementation once levels stabilize above 30 ng/mL. Table 6 summarizes the main international guidelines for treating overt vitamin D deficiency, illustrating the nuanced differences and shared goals in managing this condition.

Table 6. Main international recommendations for the treatment of overt vitamin D deficiency.

Scientific Society/Nation	Authors (Year)	Recommendation
Endocrine Society	Holick MF (2011) [10]	2000 IU/d of vitamin D2 or vitamin D3 or 50,000 IU of vitamin D2 or vitamin D3 once weekly for 6 weeks followed by maintenance therapy with 400–1000 IU/day.
Australian and New Zealand Bone and Mineral Society	Paxton GA (2013) [16]	Between 0 and 3 months: if 25(OH)D levels are 12–20 ng/mL, 400 IU/day for 3 months; if 25(OH)D levels < 12 ng/mL, 1000 IU/day for 3 months. Between 3 and 12 months: if levels of 25(OH)D are 12–20 ng/mL, 400 IU/day for 3 months; if levels of 25(OH)D < 12 ng/mL, 1000 IU/day for 3 months or a bolus of 50,000 IU (consider repeating another dose after one month). Thereafter, maintenance with 400 IU/day.
American Academy of Pediatrics	Golden NH (2014) [3]	2000 IU/day for 6 weeks or 50,000 IU per week for 6 weeks; subsequent maintenance with 400–1000 IU/day.
Canadian Paediatric Society	Critch JN (2014) [88]	0–6 months: 400 IU/day of vitamin D in breastfed infants. Children who are not breastfed do not require vitamin D prophylaxis. Partially breastfed infants should receive 400 IU/day, regardless of the amount of formula milk taken. 6–12 months: Continued supplementation with 400 IU/day in children who are still exclusively breastfed or who are taking breast milk.
Italian Pediatric Society and the Italian Society of Preventive and Social Pediatrics	Saggese G (2018) [17]	In the case of asymptomatic vitamin D deficiency [25(OH)D < 20 ng/mL]: 2000 IU/day or 50,000 IU/week of vitamin D2 or D3 for 6–8 weeks). After the completion of treatment, serum concentrations should be reevaluated. In the presence of an adequate vitamin D status [25(OH)D ≥ 30 ng/mL], continue vitamin D supplementation as recommended for the age.
Poland	Płudowski P (2023) [85]	0–12 months: 2000 IU/day. Calcifediol is not recommended.

6. Conclusions

Maternal vitamin D status plays a crucial role in shaping the health outcomes of both neonates and infants, particularly in areas such as bone development, immune function, and overall growth. The research highlights the significant influence of maternal vitamin D levels on neonatal 25(OH)D concentrations, with a direct correlation with birth weight, respiratory health, and the risk of developing conditions such as atopic dermatitis, hypoxic–ischemic encephalopathy, and early-onset sepsis. While the long-term effects of maternal

vitamin D on growth and developmental outcomes remain an area of ongoing investigation, it is clear that ensuring adequate maternal vitamin D levels through supplementation during pregnancy is essential for optimal neonatal health.

Vitamin D deficiency in newborns is associated with numerous clinical concerns, ranging from respiratory issues like respiratory distress syndrome to skeletal problems such as rickets. Given these risks, healthcare providers must prioritize vitamin D supplementation for both mothers and infants, particularly in populations at higher risk, such as those with limited sunlight exposure or darker skin tones. The importance of 25(OH)D supplementation seems even more crucial for pregnant women with obesity, as the incidence of vitamin D deficiency appears to be high in this group [93].

Intervention studies have demonstrated that vitamin D supplementation can significantly improve neonatal vitamin D levels, reducing the risk of rickets and preventing long-term complications. These findings underscore the importance of early, targeted supplementation strategies to ensure that infants, especially those exclusively breastfed, receive adequate vitamin D during the early months of life. This is particularly important as breast milk contains only trace amounts of vitamin D, necessitating supplemental doses for breastfed infants.

While the optimal vitamin D dosing guidelines for neonates and infants vary across regions, most international recommendations advocate a standardized daily intake of 400 IU for full-term infants. Preterm infants, however, require more individualized and often higher doses, reflecting their unique needs and vulnerability to vitamin D deficiency due to lower birth stores and altered calcium absorption capacity.

Furthermore, vitamin D therapy in cases of clinical deficiency is crucial for preventing and managing complications such as hypocalcemia, rickets, and poor bone mineralization. A variety of therapeutic regimens exist, with international guidelines providing varying approaches for the treatment of hypovitaminosis D in infants. However, despite the lack of a universal consensus on the exact threshold for deficiency, early detection and appropriate management are vital to improving neonatal health and preventing long-term developmental issues.

Despite progress in understanding the role of maternal vitamin D in neonatal health, several key questions remain unanswered. Longitudinal studies are essential to determine the long-term impact of neonatal 25(OH)D levels on children's skeletal, immune, and neurological development. A universal consensus on optimal neonatal 25(OH)D thresholds for defining deficiency is still lacking, making it challenging to standardize clinical guidelines. Furthermore, the impact of maternal obesity, skin phototype, and geographic sunlight exposure on vitamin D bioavailability in newborns requires further investigation, particularly regarding long-term consequences.

In conclusion, ensuring optimal vitamin D levels in neonates and infants is a vital component of preventive healthcare, with maternal supplementation playing a crucial role in supporting both short-term and long-term health outcomes. However, significant knowledge gaps remain. To address these uncertainties, further rigorous research and evidence-based recommendations are essential for refining clinical practices and protecting infant health from the very beginning of life.

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Abbreviations

The following abbreviations are used in this manuscript:

1,25(OH)2D	1,25-Dihydroxyvitamin D
25(OH)D	25-Hydroxyvitamin D
APA	Acceleration Time-to-Ejection Time Ratio
AT/ET	Alkaline Phosphatase Activity
BMI	Body Mass Index
CYP27B1	Cytochrome P450 Family 27 Subfamily B Member
DBS	Dried Blood Spot
EOS	Early-Onset Sepsis
HIE	Hypoxic–Ischemic Encephalopathy
LGA	Large for Gestational Age
LRTIs	Lower Respiratory Tract Infections
PTH	Parathyroid Hormone
PI	Pulsatility Index
PMCA	Plasma Membrane Calcium-Dependent ATPase
PSV	Peak Systolic Velocity
RDS	Respiratory Distress Syndrome
RoP	Retinopathy of Prematurity
ROC	Receiver Operating Characteristic
SGA	Small for Gestational Age
TPN	Total Parenteral Nutrition
TTN	Transient Tachypnea of the Newborn
UVB	Ultraviolet B
VEGF	Vascular Endothelial Growth Factor
WB-BMC	Whole-Body Bone Mineral Content

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