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A Review Article on Impact of Vitamin D Deficiency on Maternal and Fetal Health During Pregnancy

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ABSTRACT

Vitamin D plays a central role in maternal–fetal physiology, influencing placental function, calcium homeostasis, immune regulation, neurodevelopment, and fetal metabolic programming. Deficiency during pregnancy disrupts several tightly coordinated biological processes, ultimately predisposing the fetus to structural, functional, and developmental abnormalities. Vitamin D is added to milk, plant-based milks, cereals, fat spreads, and orange juice in several nations.: In addition to influencing trophoblast invasion, immunological modulation, and angiogenesis in the placenta, vitamin D aids in controlling placental calcium transport, which is crucial for the mineralisation of foetal bones. Clinical research indicates that even at modest vitamin D levels, women with specific vitamin D receptor gene polymorphisms may have increased rates of gestational diabetes and unfavourable pregnancy outcomes. Reduced bone mineral density, craniotabes, and rickets in infancy are examples of poor foetal and neonatal bone growth. Increased incidence of seizures and hypocalcaemia in newborns (in extremely deficient conditions. Heightened vulnerability to infections throughout childhood and potential long-term consequences for the health of kids, such as a higher incidence of asthma, type 1 diabetes, and neurodevelopmental impairments. These studies are primarily useful in developing evidence based prenatal care guidelines to lower morbidity and protect the mother's & child's long-term health.

Keywords: Vitamin D deficiency, maternal health, fetal health, pregnancy

1. INTRODUCTION

A fat-soluble vitamin, vitamin D, functions in the body similarly to a hormone (1,5,4). Its well-established major function is to promote bone health by improving calcium and phosphorus absorption and preserving the mineralization of teeth and bone (1,4). The need for vitamin D rises during pregnancy in order to promote foetal development and maternal health (5,12). The main source of vitamin D Sunlight and skin synthesis: The most organic source skin produces vitamin D3 (cholecalciferol) when exposed to ultraviolet B (UVB) radiation from sunshine (2). Latitude, season, skin pigmentation, amount of skin exposed, time spent outside, and use of sunscreen all affect how much is produced (5,11). Sun exposure is good, but getting vitamin D must be weighed against the danger of skin damage or cancer (2).

Dietary sources: There aren't many foods that are naturally high in vitamin D (11). Among the reliable sources are: Fish high in

fat, such as sardines, mackerel, and salmon liver oil from cod (extremely high), Liver, egg yolks, and some mushrooms (particularly those exposed to UV light) Fortified foods: Vitamin D is added to milk, plant-based milks, cereals, fat spreads, and orange juice in several nations (11). Despite its significance, vitamin D deficiency is very common among pregnant women globally, especially in areas with little sun exposure, cultural clothing customs, darker skin tones, or inadequate consumption of foods high in vitamin D (5,11,24).

Mechanism: In addition to influencing trophoblast invasion, immunological modulation, and angiogenesis in the placenta, vitamin D aids in controlling placental calcium transport, which is crucial for the mineralization of foetal bones (29). For its storage, the foetus is totally reliant on the mother's supply of vitamin D and Its metabolites (1,29).

Associated Risks: Numerous issues have been linked to maternal vitamin D deficiency, though it's crucial to remember that many of these data are observational and do not always demonstrate causation (27,28).

For the mother: Preeclampsia, or pregnancy-related hypertension, is more likely to occur (5,12,27). Increased prevalence of GDM (gestational diabetes mellitus). A higher chance of a caesarean birth (12,23). Maternal bone loss, myopathy (weakness of the muscles), or poor calcium metabolism are possible (1).

For the Foetus & Infant: elevated risk of low birthweight babies and premature birth (less than 37 weeks) (5,27). Reduced bone mineral density, craniotabes, and rickets in infancy are examples of poor foetal and neonatal bone growth (10). Increased incidence of seizures and hypocalcaemia in newborns (in extreme deficient conditions) (10). Heightened vulnerability to infections throughout childhood and potential long-term consequences for the health of kids, such as a higher incidence of asthma, type 1 diabetes, and neurodevelopmental impairments (16,24).

2. AIM:

To determine associated maternal risk factors and determine the prevalence of vitamin D insufficiency among pregnant women.

3. Mechanistic Pathways Linking Maternal Vitamin D Deficiency to Fetal Damage

Vitamin D plays a central role in maternal–fetal physiology, influencing placental function, calcium homeostasis, immune regulation, neurodevelopment, and fetal metabolic programming. Deficiency during pregnancy disrupts several tightly coordinated biological processes, ultimately predisposing the fetus to structural, functional, and developmental abnormalities.

3.1. Disruption of Placental Development and Function (29)

3.1.1 Impaired trophoblast invasion and vascular remodeling

Vitamin D facilitates differentiation and invasion of extravillous trophoblasts, which are essential for remodeling maternal spiral arteries. Deficiency reduces trophoblast motility and depth of invasion, leading to shallow placentation. Inadequate conversion of spiral arteries results in suboptimal uteroplacental perfusion, causing chronic fetal hypoxia and contributing to fetal growth restriction (FGR).

3.1.2 Enhanced placental inflammatory signaling

Vitamin D acts as an immunomodulator, suppressing pro-inflammatory cytokines such as IL-6, IL-1β, and TNF-α. In deficiency states, this regulatory balance is lost, leading to upregulated inflammatory cascades within the placenta. Persistent inflammation disturbs nutrient exchange, increases oxidative stress, and contributes to placental insufficiency, elevating risks of FGR and preeclampsia (19).

3.1.3 Impaired trans-placental nutrient transfer (7)

Vitamin D regulates the expression of calcium transport proteins (e.g., TRPV6, PMCA3) in syncytiotrophoblast cells. Deficiency reduces their activity, resulting in diminished maternal-to-fetal calcium flux, a critical determinant of fetal skeletal mineralization.

3.2. Abnormal Fetal Skeletal Development (8,9)

3.2.1 Calcium homeostasis disturbances

Low maternal Vitamin D reduces intestinal calcium absorption and disrupts parathyroid hormone (PTH) balance. Maternal hypocalcemia limits the calcium pool available for placental transfer, causing reduced fetal serum calcium.

3.2.2 Impaired endochondral ossification

Calcium deficiency impedes the mineralization of the cartilage matrix and delays conversion of cartilage to bone, resulting in:

- Fetal osteopenia
- Craniotabes
- Widened growth plates
- Risk of neonatal rickets

Thus, inadequate maternal Vitamin D directly compromises foetal skeletal integrity.

3.3. Altered Immune System Development

Vitamin D influences the maturation of both innate and adaptive immunity.

3.3.1 Impaired innate immune defense

Vitamin D triggers transcription of antimicrobial peptides such as cathelicidin and defensins. Deficiency reduces their synthesis in the fetus, weakening first-line immunity and increasing susceptibility to neonatal infections (21).

3.3.2 Dysregulated adaptive immune maturation

Vitamin D aids the development of T-regulatory cells and maintains Th1/Th2 immune balance. Deficiency results in immune dysregulation, which increases the likelihood of allergic disorders and autoimmune tendencies later in life.

3.4. Neurodevelopmental Impairment (3,4)

Vitamin D acts as a neurosteroid during fetal brain development.

3.4.1 Disrupted neuronal differentiation and synaptogenesis (19)

Vitamin D regulates genes involved in axonal growth, neurotransmitter synthesis, and synaptic formation. Deficiency interferes with cortical layering, neuronal maturation, and dendritic growth.

3.4.2 Oxidative and inflammatory stress in developing brain

Reduced Vitamin D availability increases oxidative stress and inflammatory mediators in neural tissue, potentially leading to altered cortical architecture (9).

3.4.3 Functional consequences

This disruption is associated with:

- delayed speech and cognitive development
- impaired psychomotor outcomes
- behavioral symptoms and heightened risk for neurodevelopmental disorder.

3.5. Increased Risk of Preterm Birth and Fetal Distress

Vitamin D deficiency amplifies maternal and placental inflammatory responses and weakens defenses against genitourinary infections. Chronic inflammation increases susceptibility to preterm premature rupture of membranes and preterm labor, exposing the fetus to complications of prematurity and intra-uterine stress (19)

3.6. Altered Metabolic Programming

Vitamin D influences the expression of genes involved in insulin secretion, adipogenesis, and glucose metabolism.

3.6.1 Pancreatic β-cell development

Vitamin D deficiency affects fetal pancreatic β-cell differentiation and insulin synthesis pathways, which may predispose the infant to dysglycemia and increase the risk of childhood type 1 or type 2 diabetes (11,22).

3.6.2 Impact on adipose tissue and metabolic risk

Disturbances in Vitamin D–dependent gene expression led to altered adipocyte formation and metabolic signaling, increasing vulnerability to childhood obesity, metabolic syndrome, and insulin resistance.

4. DISCUSSION

Vitamin D deficiency is a relatively prevalent issue among pregnant women globally due to a number of factors, such as limited sun exposure, cultural clothing conventions, darker skin colour, and inadequate food intake (2,5). Vitamin D, a fat-soluble vitamin that acts like a hormone to assist both baby growth and maternal health, is more necessary during pregnancy (1,5). Angiogenesis, immunological modulation, and placental calcium transport regulation all depend on it (29). The vitamin's primary recognized function is to promote bone health by enhancing the absorption of calcium and phosphorus and maintaining bone mineralization (4,20).

The literature shows a clear correlation between low maternal vitamin D level and an increased risk of various adverse pregnancy and newborn outcomes (12,27). Specifically, preeclampsia (pregnancy-associated hypertension illness) and gestational diabetes mellitus (GDM) are linked to maternal insufficiency (18,25). Additionally, a shortfall is associated with adverse foetal and neonatal outcomes, such as an increased risk of preterm birth (less than 37 weeks) and low birth weight (LBW) or Small for Gestational Age (SGA), since the foetus depends entirely on the mother's supply for its reserves (17,26). Low status can also lead to poor prenatal bone growth and neonatal hypocalcaemia (10,17).

Vitamin D supplementation reliably increases 25-OH-D concentrations in mothers and newborns, with a clear dose-response effect observed in certain groups, according to several research and reviews (13,15). Although the quality of the evidence varies across clinical endpoints, pooled trials suggest that supplementation may reduce the incidence of pre-eclampsia and preterm birth (PTB) (27,28). However, there is still conflicting or inconsistent data regarding definitive clinical benefits across all outcomes, such as boosting offspring bone mass (14,16).

Additionally, recent research indicates that the timing of vitamin D deficit during pregnancy may affect the severity of results; preterm delivery is most strongly associated with vitamin D deficiency in the first trimester (17). Additionally, research has shown that even in cases where other maternal nutritional parameters are normal, vitamin D insufficiency raises the risk of delivering small-for-gestational-age infants (7).

REASONS	RESULT	REFERENCE ARTICLES.
Supplementation Efficacy	Efficiency of Supplements maternal and/or neonatal serum 25(OH)D concentrations are effectively increased by vitamin D administration.	De-Regil et al. (2016), Chawes et al. (2016), Enkhmaa et al. (2018), Rostami et al. (2018), Özdemir et al. (2018), Palacios et al. (2019), Enkhmaa et al. (2019), Gallo et al. (2020), Sass et al. (2020)
Clinical Benefit (RCTs/MA)	Preterm delivery and/or pre-eclampsia are less likely when supplements are used.	De-Regil et al. (2016), Chien et al. (2024), Moghib et al. (2024)
Observational Risk	There is observational evidence linking low maternal vitamin D status to higher risks of preterm birth, GDM, pre-eclampsia, and/or low birth weight.	Agarwal et al. (2017), Gernand et al. (2017), Lo et al. (2019), Danese (2020), Zhang H et al. (2022), Karpova N, et al. (2022), Tammo Ö (2022), Tahsin et al. (2023), Lee et al. (2023)

SGA/LBW Link	Low vitamin D status is linked to increased risk of low birth weight or small for gestational age (SGA).	De-Regil et al. (2016), Agarwal et al. (2017), Hu et al. (2018), Lo et al. (2019), Zhang H et al. (2022), Chien et al. (2024)
GDM Link	A increased incidence of gestational diabetes mellitus (GDM) is linked to low maternal vitamin D status.	Agarwal et al. (2017), Lo et al. (2019), Danese (2020), Milajerdi A, et al. (2021), Zhang H et al. (2022), Milan et al. (2023)

According to certain studies, it is possible to more successfully reach ideal 25-OH-D concentrations by adjusting supplementation schedules based on baseline serum levels (20). Moreover, high-dose supplementation during pregnancy may benefit the skeletal growth and bone density of the fetus (14). Additionally, research suggests that better neurodevelopmental outcomes in early childhood are associated with appropriate prenatal vitamin D status (16). Clinical research indicates that even at modest vitamin D levels, women with specific vitamin D receptor gene polymorphisms may have increased rates of gestational diabetes and unfavorable pregnancy outcomes (25). Additionally, broader population-based research demonstrates that vitamin D is an essential part of all-encompassing prenatal care, especially in areas where exposure to sunshine is insufficient to meet physiological demands (22).

5. CONCLUSION

Vitamin D deficiency is a Serious and common pregnancy issue .Due to Insufficient Sun exposure and food, which is necessary for both foetal growth and the health of mother's bones .Low Vitamin D levels has been observed to enhance the mother's risk of pre-eclampsia and gestational diabetes mellitus (GDM) .Deficiency in which the foetus is totally dependent on mother's supply, has been in seen to be linked to unfavourable outcomes for newborn such as preterm birth, low birth weight.While Vitamin D directly controls calcium status, other Critical, frequently concomitant deficiencies like iron which Causes anemia) and folic acid also contribute to serious maternal & foetal issues. Universal Screening and targeted Supplementing which has been demonstrated to successfully Increase maternal and newborn 25(OH)D levels, care essential for Eradication. To Show conclusive therapeutic Efficacy, future research must prioritise high quality (RCT), particularly in lowering the risk of premature birth and preeclampsia. These studies are primarily useful in developing evidence based prenatal care guidelines to lower morbidity and protect the mother's & child's long-term health.

Declaration by Authors

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