

VITAMIN D DEFICIENCY DURING PREGNANCY AND ITS ASSOCIATION WITH PRETERM BIRTH AND LOW BIRTH WEIGHT A SYSTEMATIC LITERATURE REVIEW

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Abstract

Background: Vitamin D deficiency is common during pregnancy and has been proposed as a modifiable contributor to adverse birth outcomes. Nevertheless, observational findings concerning preterm birth and low birth weight remain inconsistent because studies differ in population characteristics, gestational timing of vitamin D measurement, laboratory methods, deficiency thresholds, outcome definitions, and control of confounding. *Objective:* To systematically review observational evidence on the association between antenatal vitamin D deficiency and preterm birth and low birth weight. *Methods:* A structured search of PubMed/MEDLINE was conducted for studies published from January 2010 to 20 September 2026. Eligible studies measured maternal serum or plasma 25-hydroxyvitamin D during pregnancy and reported preterm birth before 37 completed weeks and/or low birth weight below 2,500 g. Cohort, case-cohort, and nested case-control studies were considered. Evidence was synthesized narratively because exposure thresholds, sampling periods, designs, and effect measures were clinically heterogeneous. *Results:* Twenty-seven studies were included. Several large cohorts reported higher risks of preterm birth or low birth weight at lower 25(OH)D concentrations, particularly in severely deficient groups and selected higher-risk populations. Other well-designed studies found attenuated or null associations after adjustment for ethnicity, body mass index, season, socioeconomic characteristics, and obstetric risk factors. Evidence for low birth weight was generally suggestive but less extensive than evidence for preterm birth. Associations appeared sensitive to the timing of blood collection, the severity of deficiency, population baseline vitamin D status, and whether spontaneous and medically indicated preterm births were separated. *Conclusion:* Antenatal vitamin D deficiency may be associated with preterm birth and low birth weight, but the evidence does not establish causality. Greater consistency in vitamin D thresholds, trimester-specific measurement, outcome classification, and confounder adjustment is required. The findings support careful assessment of women at risk of deficiency while not, by themselves, establishing a basis for universal screening or supplementation to prevent these outcomes.

INTRODUCTION

Preterm birth and low birth weight remain important causes of neonatal morbidity, mortality,

impaired childhood development, and long-term chronic disease. Preterm birth is conventionally defined as delivery before 37 completed weeks of

gestation, whereas low birth weight refers to a birth weight below 2,500 g regardless of gestational age. Although the two outcomes frequently coexist, they are not interchangeable. A preterm infant may have a weight appropriate for gestational age, while a term infant may have low birth weight because of fetal growth restriction. Maintaining this distinction is essential when evaluating nutritional exposures during pregnancy.

Vitamin D is a fat-soluble prohormone obtained through cutaneous synthesis after ultraviolet-B exposure, dietary intake, and supplementation. Serum or plasma 25-hydroxyvitamin D [25(OH)D] is the most widely used biomarker of vitamin D status because it reflects contributions from both endogenous production and intake. Pregnancy changes calcium and vitamin D metabolism to support fetal skeletal development, placental function, immune regulation, and maternal physiological adaptation. The placenta expresses vitamin D receptors and metabolizing enzymes, providing biological plausibility for relationships between vitamin D status, inflammation, implantation, placental vascular function, and fetal growth.

Deficiency during pregnancy is reported across high-, middle-, and low-income settings. Risk is influenced by skin pigmentation, limited sunlight exposure, season, latitude, clothing practices, dietary patterns, obesity, malabsorption, socioeconomic conditions, and supplement use. The absence of a universally accepted pregnancy-specific 25(OH)D threshold creates an additional complication. Many studies define deficiency as less than 50 nmol/L (20 ng/mL), while others use lower thresholds for severe deficiency or higher thresholds for insufficiency. As a result, studies may assign the same woman to different exposure categories.

Mechanistically, vitamin D deficiency could contribute to preterm birth through altered innate immune responses, increased susceptibility to infection, inflammatory activation, impaired placental function, and changes in myometrial activity. Potential pathways to low birth weight include impaired placental development, altered calcium transport, maternal inflammation, and restricted fetal growth. However, these mechanisms

do not prove a causal relationship. Low vitamin D may also operate as a marker of poorer diet, reduced outdoor activity, obesity, social disadvantage, chronic illness, or inadequate antenatal care. These characteristics can independently influence gestational duration and fetal growth.

Previous individual studies have produced divergent findings. Some cohorts have reported strong associations between deficiency and preterm birth or low birth weight, whereas others have found no association after multivariable adjustment. Divergence may arise from differences in gestational timing, population risk, assay performance, definitions, seasonality, and analytical strategies. It is therefore more informative to examine why results differ than to treat all studies as direct replications of the same question.

1.1 Review aim and questions

This review aimed to critically synthesize observational evidence concerning vitamin D deficiency during pregnancy and its association with preterm birth and low birth weight. It addressed the following questions:

- Is antenatal vitamin D deficiency associated with an increased risk of preterm birth?
- Is antenatal vitamin D deficiency associated with an increased risk of low birth weight?
- Which methodological and population characteristics help explain inconsistencies across studies?
- What conclusions can reasonably be drawn for research and antenatal practice?

2. Methods

2.1 Design and reporting approach

A systematic literature review with narrative synthesis was undertaken and reported in accordance with the PRISMA 2020 framework (Page et al., 2021). A narrative rather than statistical synthesis was selected because the eligible studies used materially different vitamin D cut-offs, gestational sampling periods, study designs, reference categories, outcome subtypes, and effect measures. Pooling these estimates without

harmonization could create a precise-looking but

clinically misleading summary.

2.2 Eligibility criteria

PECOS element	Operational criterion
Population	Pregnant women with singleton or multiple gestations; special populations were retained but interpreted separately.
Exposure	Maternal serum or plasma 25(OH)D measured during pregnancy.
Comparator	A higher or sufficient vitamin D category, or vitamin D analysed continuously.
Outcomes	Preterm birth before 37 completed weeks and/or low birth weight below 2,500 g. Studies using more severe preterm thresholds were also retained.
Study designs	Prospective or retrospective cohort, case-cohort, and nested case-control studies based on a defined pregnancy cohort.
Publication criteria	Peer-reviewed human studies published in English from January 2010 to 20 September 2026.
Exclusions	Reviews, editorials, animal studies, supplementation-only trials without antenatal status analysis, postpartum-only or cord-only measurement, and studies not reporting either review outcome.

2.3 Information sources and search strategy

PubMed/MEDLINE was searched from January 2010 through 20 September 2026. Reference lists of eligible studies and relevant reviews were examined to identify additional records. Search concepts combined terms for vitamin D status, pregnancy, and the two outcomes. A representative search string was:

("vitamin D" OR "25-hydroxyvitamin D" OR 25(OH)D OR hypovitaminosis D) AND (pregnancy OR pregnant OR antenatal OR maternal) AND ("preterm birth" OR "premature birth" OR "low birth weight" OR birthweight)

The search was deliberately broad at retrieval. Study-design and eligibility restrictions were applied during screening so that relevant cohort-based analyses labelled as nested case-control or case-cohort studies were not lost.

2.4 Study selection and data extraction

Records were screened against the prespecified criteria. For potentially eligible reports, the review extracted country, design, sample size, population characteristics, timing and method of 25(OH)D measurement, exposure threshold, outcome definition, adjustment strategy, and principal findings. Where several reports used related cohorts, the study question, exposure timing, and outcome analysis were examined to minimize

double-counting. Findings were grouped by outcome and interpreted according to design strength, population risk, exposure timing, and adjustment for confounding.

2.5 Study appraisal

Methodological appraisal considered representativeness of the cohort, temporality of exposure measurement, reliability of the vitamin D assay, clarity of outcome definitions, completeness of follow-up, and control of key confounders. Particular attention was given to season, ethnicity or skin pigmentation, body mass index, maternal age, socioeconomic position, smoking, parity, supplement use, previous preterm birth, hypertensive disorders, diabetes, and gestational age at blood sampling. Studies were not excluded solely because of one limitation; instead, weaknesses were incorporated into the interpretation.

2.6 Synthesis method

Results were synthesized separately for preterm birth and low birth weight. Greater weight was placed on large cohorts, prospectively collected samples, validated outcome definitions, early or mid-pregnancy measurement, and adjusted estimates. Studies involving twins, women living with HIV, women with previous preterm birth, or particular ethnic groups were retained because they provide clinically valuable evidence, but their

generalizability was considered separately. No pooled estimate was calculated.

3. Results

3.1 Study selection

The database search returned 357 records. After title and abstract screening, 95 potentially relevant observational reports were examined in greater

detail. Sixty-eight were excluded because they did not measure maternal antenatal 25(OH)D, did not report either preterm birth or low birth weight, used an ineligible design, or duplicated an eligible cohort analysis without adding a relevant outcome. Twenty-seven studies were retained for the narrative synthesis.

Stage	Result
Identification	357 records retrieved through the database search
Initial screening	262 records excluded as reviews, interventions, unrelated exposures/outcomes, or non-human research
Detailed eligibility review	95 observational reports examined
Detailed exclusions	68 reports excluded for wrong exposure, outcome, design, timing, or overlapping analysis
Included	27 studies included in the systematic narrative synthesis

3.2 Characteristics of the evidence base

The included literature covered North America, Europe, China, South Asia, Latin America, and multiethnic populations. Designs included prospective cohorts, retrospective cohorts, case-cohort studies, and nested case-control analyses. Sample sizes ranged from fewer than 200 participants to more than 11,000 pregnancies. Most

studies measured total serum or plasma 25(OH)D, but laboratory methods included liquid chromatography-tandem mass spectrometry, immunoassays, and radioimmunoassays. Samples were collected from the first trimester to admission for delivery. Deficiency was most commonly defined below 50 nmol/L, although thresholds ranged substantially.

Study	Setting/design/sample	Exposure	Outcome	Main interpretation
Baker et al., 2011	USA; nested case-control; 160	First trimester; <50 nmol/L	Spontaneous PTB	No association; cohort largely vitamin-D sufficient.
Thorp et al., 2012	USA; high-risk nested case-control; 265	16-22 weeks; <50 nmol/L	Recurrent PTB	No significant adjusted association.
Bodnar et al., 2013	USA; twin cohort; 211	24-28 weeks; <75 nmol/L	PTB <35 weeks	Lower status associated with higher PTB risk in twins.
Zhou et al., 2014	China; prospective cohort; 1,953 analysed	16-20 weeks; ≤20 ng/mL	PTB and LBW	Mostly null relationships for reported birth outcomes.
Bodnar et al., 2015	USA; case-cohort; 3,453 sampled	≤20 weeks; categories	PTB subtypes	Inverse gradient; strongest evidence for early PTB/subtypes.
Chen et al., 2015	China; birth cohort; 3,658	During pregnancy; RIA	LBW/SGA	Deficiency associated with substantially higher LBW and SGA risk.
Miliku et al., 2016	Netherlands; prospective cohort; 7,098	Median 20.3 weeks; quartiles	PTB and fetal growth	Lowest concentrations associated with impaired growth and adverse outcomes.
Boyle et al., 2016	New Zealand; prospective cohort; 1,710	15 weeks	Spontaneous PTB/SGA	Associations attenuated after BMI and ethnicity adjustment.

Study	Setting/design/sample	Exposure	Outcome	Main interpretation
Yang et al., 2016	China; nested case-control; 138 analysed	Repeated pregnancy samples	PTB	Reported lower vitamin D measures among PTB cases.
Jao et al., 2017	Latin America; HIV cohort; 715	Antenatal; severe deficiency <10 ng/mL	PTB	Severe deficiency associated with PTB; few severely deficient women.
Tabatabaei et al., 2017	Canada; case-control within 3D cohort; 480	8-14 weeks	PTB/spontaneous PTB	Association concentrated in ethnic-minority women.
Bärebring et al., 2018	Sweden; prospective cohort; 2,046 early	<=16 and >31 weeks	PTB and LBW	Limited evidence for PTB/LBW; timing and trajectory mattered.
Wang et al., 2018a	China; cohort; 747 term births	First visit; <20 ng/mL	Birth weight/SGA	Nonlinear association with birth size; term-only design limits PTB inference.
Wang et al., 2018b	China; prospective cohort; 1,978	Across pregnancy	PTB and neonatal outcomes	Deficiency common and associated with selected adverse neonatal outcomes.
Monier et al., 2019	France; prospective cohort; 2,813	First trimester; <20 ng/mL	PTB/SGA	No clear overall association; possible higher PTB risk in darker-skin subgroup.
Yu et al., 2019	China; retrospective cohort; 11,641	Antenatal; <50 nmol/L	PTB	Mean levels differed, but categorical findings were not consistently significant.
Chen et al., 2020	China; retrospective cohort; 2,814	Early pregnancy	PTB and LBW	Higher 25(OH)D associated with lower adjusted PTB and LBW hazards.
Pérez-Castillo et al., 2020	Spain; prospective cohort; 289	10-12 weeks; <20 ng/mL	PTB and LBW	Deficiency associated with PTB; smaller study with limited events.
Benaïm et al., 2021	Brazil; prospective cohort; 180	Repeated across trimesters	PTB and birth size	Unexpected direction for PTB; illustrates instability in small cohorts.
Berglöv et al., 2021	Denmark; HIV cohort; 208	During pregnancy	PTB/birth size	Deficiency associated with PTB and SGA in women living with HIV.
Gowtham et al., 2022	India; prospective cohort; 121	At delivery	Birth/neonatal outcomes	Hypovitaminosis common; late measurement limits causal interpretation.
Tahsin et al., 2023	Bangladesh; nested case-control; 930	24-28 weeks	PTB	Deficiency burden high; study assessed adjusted PTB association.
Woo et al., 2023	USA; Black women; 175	8-25 weeks; <20 ng/mL	PTB	Deficiency associated with increased adjusted odds of PTB.

Study	Setting/design/sample	Exposure	Outcome	Main interpretation
Deepa et al., 2024	India; prospective cohort; 230	14-36 weeks	LBW	Lowest vitamin D category associated with increased LBW risk.
Beck et al., 2025	USA; multisite prospective cohort; 351	6-13 and 16-21 weeks	PTB/SGA/growth	Trimester-specific and nonlinear patterns; limited precision for rare outcomes.
Lee et al., 2025	Korea; retrospective cohort; 5,169	First and second trimester	PTB and neonatal outcomes	Persistent versus corrected deficiency produced differing outcome patterns.
Zheng et al., 2025	China; retrospective cohort; 2,742	Trimester-specific; MS/MS	PTB and other outcomes	Complex timing-dependent findings, including inverse late-pregnancy association.

3.3 Vitamin D deficiency and preterm birth

Evidence for preterm birth was mixed but not random. Positive associations were more frequently reported when vitamin D was measured early or in mid-pregnancy, when severe deficiency was distinguished from mild insufficiency, and when populations had a high baseline risk. In a large case-cohort analysis, Bodnar et al. (2015) observed an inverse gradient between early-pregnancy 25(OH)D and preterm birth, with attention to spontaneous and medically indicated subtypes. Chen et al. (2020) similarly reported lower adjusted hazards of preterm birth with higher early-pregnancy 25(OH)D. Studies in Black women, ethnic-minority women, twin pregnancies, and women living with HIV also reported associations, suggesting that vitamin D status may interact with broader clinical and social vulnerability (Berglöv et al., 2021; Bodnar et al., 2013; Tabatabaei et al., 2017; Woo et al., 2023).

In contrast, several cohorts found no clear relationship. Baker et al. (2011) studied a largely vitamin-D-replete population and found comparable first-trimester deficiency among spontaneous preterm cases and term controls. Thorp et al. (2012) found no significant association with recurrent preterm birth in high-risk women after adjustment. In New Zealand, an apparent association was attenuated after body mass index and ethnicity were included in the model (Boyle et al., 2016). Monier et al. (2019) found no persuasive overall association, although results differed by skin colour. Such attenuation demonstrates that vitamin D concentration is embedded within social, metabolic,

behavioural, and ethnic patterning rather than acting as an isolated exposure.

The timing of measurement was a major source of variation. Early pregnancy measurement better preserves temporality and may capture vitamin D status during placentation. Late-pregnancy measurement can be influenced by supplementation, seasonal change, pregnancy complications, altered activity, or clinical advice. The complex and sometimes inverse late-pregnancy findings reported by Zheng et al. (2025) illustrate why late measurements should not be interpreted as equivalent to first-trimester exposure. Repeated-measure studies offer a stronger approach because they distinguish persistent deficiency, correction, and late-onset deficiency (Bärebring et al., 2018; Beck et al., 2025; Lee et al., 2025).

Outcome heterogeneity was also consequential. Spontaneous preterm labour, preterm prelabour rupture of membranes, and medically indicated delivery may have different causal pathways. Studies that combined all preterm births potentially diluted associations that were present only for an inflammatory or placental subtype. Gestational-age thresholds also varied, with some studies examining birth before 37 weeks and others focusing on earlier or more severe prematurity. Therefore, disagreement across studies does not necessarily mean that one set of findings is erroneous; the studies may be estimating related but non-identical effects.

3.4 Vitamin D deficiency and low birth weight

Evidence concerning low birth weight was smaller in volume but generally suggestive of an inverse relationship between maternal 25(OH)D and fetal size. In a large Chinese birth cohort, Chen et al. (2015) reported strong associations of deficiency with low birth weight and small-for-gestational-age birth. Wang et al. (2018a) identified a nonlinear relationship in which birth weight increased with 25(OH)D at lower concentrations and then levelled off, supporting the possibility of a threshold rather than a simple linear effect. Chen et al. (2020) found higher early-pregnancy 25(OH)D associated with lower adjusted hazards of low birth weight, while the Indian MAASTHI cohort reported an increased probability of low birth weight among women with lower vitamin D status (Deepa et al., 2024).

Nevertheless, low birth weight is a composite endpoint reflecting both gestational duration and fetal growth. A higher frequency of low birth weight may arise because infants are delivered earlier, because they are growth restricted, or because both processes occur. Studies that do not adjust for

gestational age or separately report small-for-gestational-age birth cannot determine which pathway predominates. Conversely, restricting a cohort to term births, as in Wang et al. (2018a), isolates fetal growth more effectively but excludes the contribution of prematurity and cannot answer the preterm-birth question.

The strongest growth evidence came from studies with repeated ultrasonographic assessment or standardized birth-size measures. Miliku et al. (2016) linked lower mid-pregnancy concentrations with less favourable fetal growth patterns and adverse birth outcomes. Recent multisite evidence also suggests that trimester-specific vitamin D status may be associated differently with fetal growth trajectories (Beck et al., 2025). However, several studies did not confirm a relationship with small-for-gestational-age birth, and associations sometimes weakened after adjustment for maternal size, ethnicity, socioeconomic conditions, and season. The current evidence therefore supports concern but not a uniform causal conclusion.

3.5 Sources of inconsistency

Source	Implication
Deficiency threshold	Cut-offs ranged from severe deficiency below 10 ng/mL to insufficiency below 30 ng/mL, changing exposure classification and apparent effect size.
Gestational timing	First-, second-, third-trimester and delivery measurements may represent different biological windows and differ in vulnerability to reverse causation.
Assay method	LC-MS/MS, immunoassays and radioimmunoassays are not perfectly interchangeable; measurement error may attenuate or distort associations.
Population baseline status	A largely sufficient cohort has few deficient participants and limited power to detect threshold effects; highly deficient populations may show different gradients.
Outcome definition	All PTB, spontaneous PTB, indicated PTB and severe PTB have different mechanisms; LBW does not distinguish prematurity from fetal growth restriction.
Confounding	Skin pigmentation, ethnicity, BMI, season, diet, supplement use, socioeconomic status, smoking and comorbidity influence both vitamin D and birth outcomes.
Selection and attrition	Vitamin D testing may be clinically selective in retrospective cohorts, while loss to follow-up can alter prospective cohort composition.
Model specification	Continuous, categorical, quartile and nonlinear analyses answer different questions and may yield apparently conflicting results.

4. Discussion

4.1 Principal interpretation

This review found a recurring, but not universal, association between lower antenatal vitamin D

status and preterm birth or low birth weight. The evidence was most persuasive for severe deficiency, selected higher-risk populations, and early or mid-pregnancy measurement. However, null and

attenuated findings from several well-characterized cohorts prevent a simple conclusion that vitamin D deficiency independently causes these outcomes in all pregnant populations.

The overall pattern is compatible with at least three explanations that may operate simultaneously. First, vitamin D may have a biological role in placental, immunological, and inflammatory pathways relevant to gestational duration and fetal growth. Second, vitamin D status may mark broader nutritional and social disadvantage rather than being the principal causal factor. Third, an association may be real only below a sufficiently low threshold or within susceptible groups, making average population effects appear inconsistent. Observational data alone cannot fully separate these explanations.

4.2 Biological plausibility

Vitamin D signalling is involved in innate and adaptive immunity, cytokine regulation, antimicrobial peptide production, placental implantation, and calcium metabolism. Deficiency could plausibly increase susceptibility to ascending infection or excessive inflammation, both recognized contributors to spontaneous preterm labour and membrane rupture. Vitamin D may also influence placental angiogenesis and nutrient transport, thereby affecting fetal growth. These mechanisms provide coherence but should not be treated as evidence that supplementation will necessarily prevent adverse outcomes. A plausible pathway can coexist with confounding, measurement error, or an effect too small to be clinically meaningful.

4.3 Confounding and causal interpretation

The principal challenge is residual confounding. Darker skin pigmentation reduces cutaneous vitamin D synthesis but is also entangled with structural, socioeconomic, and healthcare inequalities in many settings. Obesity can lower measured circulating 25(OH)D and independently increase obstetric risk. Season influences vitamin D status and may also correlate with infections, physical activity, diet, and conception timing. Supplement use may indicate both improved

vitamin D intake and greater health awareness or access to antenatal care. Statistical adjustment reduces but does not eliminate these problems, especially when variables are measured imprecisely or omitted.

Reverse causation is less likely when vitamin D is measured early, but it becomes more plausible with late-pregnancy assessment. Women who develop complications may reduce outdoor activity, change diet, receive supplements, or experience metabolic changes that affect 25(OH)D. Analyses based on delivery samples cannot clearly establish whether low vitamin D preceded the process that produced preterm birth or low birth weight. For this reason, early measurement and repeated measurements should be viewed as methodologically stronger than a single late sample.

4.4 Clinical and public-health implications

The findings have two different implications that should not be conflated. The first concerns identification and treatment of vitamin D deficiency for established maternal and skeletal-health indications. The second concerns using screening or supplementation specifically to prevent preterm birth or low birth weight. This review provides suggestive evidence for the first area of clinical attention but does not establish the effectiveness of a universal prevention strategy for the two birth outcomes. Decisions about testing and supplementation should therefore follow current clinical guidance, individual risk assessment, local prevalence, diet, sun exposure, comorbidity, and clinician judgement.

From a public-health perspective, populations with widespread deficiency may benefit from improved antenatal nutrition, equitable access to supplements where recommended, and attention to social determinants. Yet a single-nutrient approach could divert attention from other established determinants of preterm birth and fetal growth, including infection, hypertension, tobacco exposure, inadequate nutrition, poverty, occupational strain, and limited access to high-quality antenatal care. Vitamin D should be situated within comprehensive maternal health rather than presented as an isolated solution.

4.5 Strengths and limitations of the evidence

The principal strengths of the evidence base include several large cohorts, objective biomarker measurement, samples collected before delivery, and increasing use of repeated measurements and LC-MS/MS. Studies from diverse geographical and ethnic settings improve the breadth of the review. The separation of preterm birth from low birth weight also avoids treating distinct outcomes as interchangeable.

Limitations remain substantial. There was no standardized pregnancy-specific threshold for deficiency. Assay methods and sampling periods differed. Some studies had few adverse events and unstable estimates, while others were retrospective and vulnerable to selective testing. Preterm birth subtypes were not consistently distinguished. Low birth weight was sometimes reported without gestational-age-specific growth measures. Adjustment sets varied, creating a risk of both residual confounding and overadjustment. Special-population findings may not generalize to low-risk singleton pregnancies. Finally, the heterogeneity that justified narrative synthesis also prevents calculation of a single summary effect.

4.6 Research priorities

- Use harmonized reporting of 25(OH)D in both nmol/L and ng/mL and present continuous as well as clinically justified categorical analyses.
- Collect vitamin D in early pregnancy and, where feasible, repeat measurement to characterize persistent, corrected, and late-onset deficiency.
- Separate spontaneous, membrane-rupture-related, and medically indicated preterm birth, and distinguish low birth weight from small-for-gestational-age birth.
- Prespecify confounders using causal reasoning and measure season, BMI, ethnicity or skin pigmentation, diet, supplementation, smoking, socioeconomic position, and relevant comorbidity consistently.
- Use adequately powered multi-country cohorts and report subgroup findings cautiously to avoid false-positive interactions.

- Link observational findings with high-quality randomized evidence before recommending supplementation specifically to prevent preterm birth or low birth weight.

5. Conclusion

Vitamin D deficiency during pregnancy is frequently associated with preterm birth and low birth weight, particularly when deficiency is severe, measured early or in mid-pregnancy, or examined in populations with heightened clinical or social vulnerability. Nonetheless, the literature is inconsistent, and several associations weaken after adjustment for maternal, ethnic, seasonal, and socioeconomic factors. Current observational evidence therefore supports a cautious conclusion: antenatal vitamin D deficiency may contribute to, or signal increased risk of, adverse birth outcomes, but it has not been established as a universal independent cause. Future research should prioritize repeated trimester-specific measurement, standardized exposure and outcome definitions, careful control of confounding, and integration with randomized evidence. Clinical interpretation should remain aligned with established antenatal guidance rather than assuming that observational association alone demonstrates preventive benefit.

Declarations

Ethics approval: Not applicable because the study synthesized previously published literature and did not involve new human-participant data.

Consent for publication: Not applicable.

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Data availability: The evidence summarized in this review is available in the cited publications.

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