

Assessing the Impact of Maternal Vitamin D Deficiency During Pregnancy on Obstetric Outcomes and Neonatal Health: A Retrospective Cohort Study

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Received: 02-10-2025 / Revised: 20-10-2025 / Accepted: 10-11-2025

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Conflict of interest: Nil

Abstract:

Background: Vitamin D is essential for the health of both mother and fetus and plays important roles in placental ability, fetal growth, and immunity. Maternal vitamin D deficiency during pregnancy has been linked to negative obstetrical and neonatal outcomes.

Objective: To investigate the relationship between maternal vitamin D deficiency and pregnancy complications and neonatal outcomes.

Methods: A retrospective cohort study was performed among 450 singleton pregnancies at Nalanda Medical College & Hospital, Patna, Bihar, India. Participants were classified based on serum 25(OH)D levels as deficient (DD, <20 ng/mL), insufficient (DS, 20–30 ng/mL), or sufficient (SS, ≥30 ng/mL). Obstetric and neonatal outcomes were examined using ANOVA, Chi-square, and multivariable logistic regression.

Results: Vitamin D deficiency was associated with an increased rate of preterm births (<34 weeks; DD 6.7%, DS 2.3%; adjusted OR = 2.56, 95% CI 1.02–6.42) and lower mean birth weight (2850 ± 590 g vs. 2975 ± 520 g; p = 0.041). Mothers who were vitamin D deficient had higher rates of necrotizing enterocolitis (DD 3.3% vs. SS 0.3%; OR = 12.5) and developmental delay at one year (DD 8.9% vs. SS 2.0%; OR = 3.94).

Conclusion: Maternal vitamin D deficiency is linked to preterm birth, low birth weight, and higher rates of neonatal morbidity and developmental delay. Adequate vitamin D status during pregnancy is important for optimal maternal and neonatal health outcomes.

Keywords: Vitamin D Deficiency, Pregnancy, Neonatal Outcomes, Preterm Birth, Developmental Delay, Retrospective Cohort Study.

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Introduction

Vitamin D is a hormone that is fundamental for maintaining calcium and phosphorus homeostasis and has numerous essential physiological roles [1]. In addition to its role in the traditional area of bone metabolism, the multisystem roles of vitamin D involving cellular proliferation, immune modulation, and regulation of gene expression in relation to maternal and fetal health is the focus of much recent literature [2]. Vitamin D status during pregnancy affects fetal skeletal development but is also critically important for placental function and maternal immune competence. A recent review indicated that maternal serum 25-hydroxyvitamin D [25(OH)D] levels should be at least 40 ng/mL to optimize maternal and fetal well-being. While the specific cutoff value for vitamin D deficiency will differ depending

on health status, age, ethnicity, and lifestyle, many expert organizations, including WHO, would generally define vitamin D deficiency as a serum 25(OH)D concentration of less than 20 ng/mL [3].

The most recent research has highlighted issues related to maternal vitamin D deficiency and maternal and neonatal effects. Several studies have suggested that maternal vitamin D deficiency is associated with a higher risk of gestational diabetes mellitus (GDM), gestational hypertension, and pre-eclampsia [4]. These complications may compromise maternal health and could lead to potential long-term issues for the child, such as a higher risk of metabolic disorders as they mature. Furthermore, in addition to obstetric complications, maternal vitamin D deficiency is associated with preterm birth and

small for gestational age (SGA) infants, rising the possibility of fetal skeletal mineralization impairment which could result in congenital rickets or poor neonatal bone health. Low D during pregnancy is also linked to adverse neonatal results, including respiratory distress syndrome (RDS) and neonatal hypocalcemia, suggesting D plays a role in pulmonary and endocrine maturation during pregnancy [5].

New evidence also indicates that the offspring of vitamin D deficiency mothers may experience poor neurodevelopmental outcomes. Maternal vitamin D deficiency has been shown in observational studies to significantly increase the risk of neurocognitive delay and behavioral issues in offspring, while also being associated with neuropsychiatric disorders, such as autism spectrum disorder and schizophrenia [6]. The data provided suggest that maternal vitamin D status has implications for the developing brain and suggests a possible fetal neuroprotective role of vitamin D through neurotrophic and anti-inflammatory effects.

The worldwide incidence of maternal vitamin D deficiency continues to be disturbingly high, and represents not only geographically, but lifestyle variations. In 2016, a systematic review estimated maternal vitamin D deficiency rates ranged from 64% in the Americas, to 57% in Europe, 46% in the Eastern Mediterranean, to 87% in Southeast Asia, and 83% in the Western Pacific. In South Korea, it is concerning that an overwhelming number of women (80-86%) of reproductive age had circulating levels of vitamin D that were insufficient. Studies suggest a public health significance to the issue of maternal vitamin D insufficiency, not only among women in different countries but also across ethnic groups. However, despite a high prevalence, the actual implications of maternal vitamin D insufficiency for obstetric complications and neonatal and child health are only beginning to be clarified. The variability in the literature may be attributed to variations in research design, population characteristics, and timing of analyses of vitamin D. This warrants further rigorously designed studies that could help us home in on causality.

There are several barriers to the effective management of vitamin D deficiency in pregnant women. There is limited awareness of the need to ensure adequate vitamin D status among healthcare providers and pregnant women. Routine screening of maternal vitamin D is still not widely integrated into antenatal care in most areas of the world; therefore, it remains undiagnosed and undertreated. In addition, vitamin D can be synthesized by the skin as long as it is exposed to UVB rays, yet its production is often very low due to multiple environmental and cultural factors that restrict direct sunlight exposure. Urbanization, an indoor lifestyle, and air pollution all impact on the cutaneous synthesis of vitamin D. Cultural

factors, such as dressing conservatively and avoiding sunlight, also contribute to the problem in some cultures. While dietary sources and fortified foods may help contribute to overall vitamin D status, they do not, on their own, provide enough for optimal serum concentration especially during pregnancy when requirements are increased [8].”

Existing research demonstrates that it is paramount that adequate vitamin D status is achieved during gestation, but there continue to be gaps in literature. Specifically, the period of gestation when inadequate vitamin D levels would have the most acute impact on maternal and fetal outcomes is currently ambiguous. Additionally, it remains unknown whether optimization of vitamin D status must be initiated prior to conception or if interventions begun in early pregnancy are sufficient to prevent associated risks. There is some evidence to suggest that maternal vitamin D status early in pregnancy may have very long-term implications for fetal growth and development; however, few studies have thoroughly explored changes in vitamin D status across the gestation period and their corresponding relation to outcomes.

In this respect, the current retrospective cohort study will look at the effect of vitamin D deficiency in pregnant mothers on obstetric outcomes and neonatal health. More precisely, the study examines how different maternal levels of vitamin D at the beginning and midpoint of pregnancy influence outcomes of birth, such as preterm birth, gestational diabetes, hypertensive disorders, and neonatal complications. It also examines if the normalization of vitamin D deficiency through intervention during pregnancy can reduce these risks. This will help explain the time-course relationships for vitamin D in pregnancy and inform strategies for the optimization of maternal nutrition to improve outcomes for both mothers and babies.

Materials and Methods

Study Design: This is a retrospective cohort study to assess the association of maternal vitamin D deficiency in pregnancy with obstetric and neonatal outcomes. The medical records of pregnant women who delivered at Nalanda Medical College & Hospital, Patna, Bihar, India.

Study Area: The research was done in the Department of Obstetrics and Gynecology, Nalanda Medical College & Hospital, Patna, Bihar, India for six months.

Study Population: The study participants were pregnant women in singleton pregnancies attending antenatal clinics and delivering at NMCH during the study period, for whom maternal serum 25-hydroxyvitamin D [25(OH)D] levels were available both during the first and second trimesters.

Sample Size: A total number of 450 pregnant women who fulfilled the inclusion criteria were enrolled in the study. The sample size was determined based on the availability of complete medical records and vitamin D assay data within the study period.

Inclusion Criteria

- Women with singleton pregnancies.
- Women who had serum 25(OH)D levels measured in both the first (≤ 13 weeks) and second (14–27 weeks) trimesters.
- Women who delivered at NMCH between January 2018 and December 2024.
- Availability of complete obstetric and neonatal records.

Exclusion Criteria

- Multifetal pregnancies.
- Women with pre-existing chronic medical or surgical conditions (e.g., diabetes mellitus, renal disease, thyroid disorders, hypertension prior to conception).
- Pregnancies with known fetal chromosomal or congenital abnormalities.
- Incomplete medical records or missing vitamin D data.

Data Collection: Data was collected retrospectively from medical records and the obstetrics/gynecology electronic records. Our variables of interest were variables such as maternal demographics as well as clinical characteristics, including maternal age, parity, pre-pregnancy BMI and mode of conception. Review of medical records included adverse pregnancy outcomes gestational diabetes mellitus (gestational diabetes), gestational hypertension (preeclampsia/eclampsia), placenta previa and preeclampsia. Laboratory data included serum 25(OH)D levels from samples taken during the first and second trimesters. Assessment of obstetric outcomes included gestational age at delivery, mode of delivery, preterm delivery, postpartum hemorrhage, and other complications. The assessment of neonatal outcomes included birth weight, APGAR scoring (1 and 5 min), NICU admission, and diagnoses including respiratory distress syndrome, jaundice, sepsis, necrotizing enterocolitis, and intraventricular hemorrhage (if applicable). Long-term outcomes, including developmental delay at one year, were obtained when the children were followed up.

Grouping Based on Vitamin D Levels: Maternal serum 25(OH)D levels were defined based on the following standard clinical thresholds: deficient (<10 ng/mL), insufficient (10–20 ng/mL), and sufficient (≥ 20 ng/mL). maternal vitamin D status varying across trimesters was defined categorically as: consistently deficient (DD) - deficient in both trimesters; deficient-sufficient (DS) - deficient in the first trimester but sufficient in the second trimester

due to counseling and/or supplementation; consistently sufficient (SS) - sufficient throughout pregnancy. Categorization allows analysis of the outcomes of maternal deficiency or sufficiency.

Procedure: At NMCH, maternal vitamin D testing is routinely conducted during antenatal visits as part of normal biochemical evaluation. After first trimester testing of vitamin D, women were identified as low in serum 25(OH)D and were provided counseling from their obstetricians about diet and lifestyle. Women were encouraged to increase dietary intake of vitamin D rich foods, namely fish, eggs, and dairy products, to obtain sun exposure appropriately, and to take vitamin D supplements (approximately 1000 IU per day) when diet and sun exposure were inadequate. Vitamin D was re-addressed in the second Trimester to assess serum concentration and to stratify women by their vitamin D status. The measure of serum 25(OH)D was conducted by chemiluminescence immunoassay (LIAISON 25 OH Vitamin D TOTAL Assay, REF 310600) as per manufacture guidelines.

Statistical Analysis: Statistical analysis was conducted using SPSS version 28.0 (IBM Corp., Chicago, IL, USA). Continuous variables were shown as mean $\pm$ standard deviation (SD); while categorical variables were shown as frequencies and percentages. Group differences were calculated using one-way analysis of variance (ANOVA) for continuous variables, and Chi-square test or Fisher's exact test for categorical variables as appropriate. Bonferroni correction was applied as appropriate for post-hoc comparisons. To determine independent predictors for obstetric and neonatal adverse outcomes, multivariate logistic regression analysis was applied to control potential confounding variables that included maternal age, pre-pregnancy BMI, parity, and mode of conception. The adjusted odds ratios (aOR) (95% confidence intervals [CI]) are presented, with $p < 0.05$ defined as statistical significance".

Result

Table 1 illustrates the baseline clinical characteristics of 450 study participants categorized into three groups based on their vitamin D status during pregnancy: DD - deficient during pregnancy; DS - deficient during first trimester and sufficient during second trimester; and SS - sufficient during pregnancy. Maternal age showed a statistically significant difference between groups ($p = 0.032$), with the oldest participants in the SS group. Pre-pregnancy BMI did not differ significantly between groups ($p = 0.684$). Nulliparity was more common in the DS group (83.3%) compared to the DD (80.0%) and SS (72.0%) groups ($p = 0.049$). Prior preterm birth was not significantly different between the groups ($p = 0.531$). The method of conception differed significantly between the groups ($p < 0.001$), with

spontaneous conception being most common in the DS group and IVF being more common in the SS group. As expected, serum 25(OH)D levels differed

significantly ($p < 0.001$) between groups in both the first and second trimesters, confirming we effectively stratified the groups by vitamin D status.

Table 1: Clinical Characteristics of the Study Population (N = 450)

Variable	DD Group (n = 90)	DS Group (n = 60)	SS Group (n = 300)	p-value
Maternal age (years, mean $\pm$ SD)	27.9 $\pm$ 4.3	28.6 $\pm$ 4.1	29.4 $\pm$ 4.2	0.032
Pre-pregnancy BMI (kg/m ² , mean $\pm$ SD)	22.4 $\pm$ 3.6	22.3 $\pm$ 3.4	22.1 $\pm$ 3.5	0.684
Nulliparity, n (%)	72 (80.0)	50 (83.3)	216 (72.0)	0.049
Prior preterm birth, n (%)	2 (2.2)	0 (0.0)	5 (1.7)	0.531
Method of conception, n (%)				<0.001
– Spontaneous	58 (64.4)	45 (75.0)	170 (56.7)	
– Ovarian stimulation	7 (7.8)	3 (5.0)	14 (4.7)	
– In vitro fertilization	25 (27.8)	12 (20.0)	116 (38.6)	
25(OH)D level – 1st trimester (ng/mL, mean $\pm$ SD)	8.1 $\pm$ 1.7	8.3 $\pm$ 1.6	26.2 $\pm$ 7.2	<0.001
25(OH)D level – 2nd trimester (ng/mL, mean $\pm$ SD)	7.9 $\pm$ 2.1	24.8 $\pm$ 5.6	27.1 $\pm$ 6.5	<0.001

Table 2 outlines obstetric outcomes by maternal vitamin D status group. Rates of preeclampsia and gestational diabetes mellitus (GDM) were similar by group and did not differ statistically ($p = 0.745$ and $p = 0.892$, respectively). Occurrence of placenta previa was rare and did not differ by group. The incidence of cesarean delivery was significantly lower for the DD (53.3%) and DS (45.0%) groups compared to the SS group (66.0%) ($p = 0.001$); adjusted for other potential confounders, the odds of cesarean delivery was lower for the DD compared with SS group (odds ratio = 0.68 (95% CI: 0.45–0.99)). Mean

gestational age at delivery was slightly but statistically significantly lower for the DD group (37.8 $\pm$ 2.6) compared with the other groups ($p = 0.047$). Notably, rates of preterm birth <34 weeks and <37 weeks were elevated among mothers with vitamin D deficiency (6.7% and 11.1%, respectively) compared with the SS group (2.3% and 6.3%, respectively), and both differences were statistically significant ($p = 0.018$ and $p = 0.048$, respectively). The adjusted odds of preterm birth <34 weeks were more than double for the DD group (odds ratio = 2.56 (95% CI: 1.02–6.42)).

Table 2: Obstetric Outcomes According to Maternal Vitamin D Status

Variable	DD Group (n = 90)	DS Group (n = 60)	SS Group (n = 300)	p-value	Adjusted OR (95% CI)* (DD vs SS)
Preeclampsia, n (%)	5 (5.6)	2 (3.3)	17 (5.7)	0.745	0.91 (0.33–2.48)
Gestational diabetes mellitus, n (%)	6 (6.7)	5 (8.3)	20 (6.7)	0.892	0.96 (0.37–2.48)
Placenta previa, n (%)	1 (1.1)	0 (0.0)	4 (1.3)	0.821	—
Cesarean delivery, n (%)	48 (53.3)	27 (45.0)	198 (66.0)	0.001	0.68 (0.45–0.99)
Mean GA at delivery (weeks, mean $\pm$ SD)	37.8 $\pm$ 2.6	38.3 $\pm$ 1.9	38.1 $\pm$ 1.8	0.047	—
Preterm birth <34 weeks, n (%)	6 (6.7)	2 (3.3)	7 (2.3)	0.018	2.56 (1.02–6.42)
Preterm birth <37 weeks, n (%)	10 (11.1)	3 (5.0)	19 (6.3)	0.048	1.89 (0.88–4.05)

Table 3 indicates that maternal vitamin D level has a statistically significant impact on neonatal outcomes. Infants born to mothers with vitamin D deficiency (DD group) had lower mean birth weight (2850 $\pm$ 590 g) compared to infants in the vitamin D supplemented (DS) and vitamin D sufficient (SS) cohorts (3010 $\pm$ 470 g and 2975 $\pm$ 520 g, $p = 0.041$). While there was a similar proportion of low 5-minute Apgar scores (<7) and NICU admits across groups, infants in the DD group had a slightly elevated NICU admission rate (20%) and longer length of stay in the NICU (16.8 $\pm$ 20.1 days), but this was not statistically significant. Most respiratory morbidities were slightly higher among infants in the

DD group, such as transient tachypnea (4.4%) and respiratory distress syndrome (5.6%), whereas rates of jaundice among neonates were similar across groups. Although rates were low in all cohorts, serious complications were higher in the DD group: necrotizing enterocolitis (3.3% in DD vs 0.3% in SS; $p = 0.004$; OR = 12.5) and developmental delay at one year (8.9% in DD vs 2.0% in SS; $p = 0.006$; OR = 3.94). Findings support maternal vitamin D deficiency is related to lower neonatal birth weight and higher neonatal morbidity, highlighting the importance of maternal vitamin D for neonatal outcomes.

Table 3: Neonatal Outcomes and Morbidities

Variable	DD Group (n = 90)	DS Group (n = 60)	SS Group (n = 300)	p-value	Adjusted OR (95% CI)* (DD vs SS)
Birth weight (g, mean $\pm$ SD)	2850 $\pm$ 590	3010 $\pm$ 470	2975 $\pm$ 520	0.041	—
5-min Apgar score <7 , n (%)	2 (2.2)	1 (1.7)	5 (1.7)	0.931	—
NICU admission, n (%)	18 (20.0)	6 (10.0)	58 (19.3)	0.127	0.96 (0.55–1.67)
NICU stay (days, mean $\pm$ SD)	16.8 $\pm$ 20.1	13.4 $\pm$ 15.2	15.9 $\pm$ 17.5	0.312	—
Transient tachypnea (TTN), n (%)	4 (4.4)	1 (1.7)	23 (7.7)	0.091	0.54 (0.22–1.29)
Respiratory distress syndrome (RDS), n (%)	5 (5.6)	1 (1.7)	11 (3.7)	0.221	1.48 (0.61–3.60)
Neonatal jaundice, n (%)	21 (23.3)	9 (15.0)	63 (21.0)	0.318	1.19 (0.75–1.91)
Necrotizing enterocolitis (NEC), n (%)	3 (3.3)	0 (0.0)	1 (0.3)	0.004	12.5 (1.85–84.6)
Intraventricular hemorrhage (IVH), n (%)	1 (1.1)	0 (0.0)	1 (0.3)	0.493	—
Developmental delay at 1 year, n (%)	8 (8.9)	3 (5.0)	6 (2.0)	0.006	3.94 (1.42–10.9)

Discussion

Our results from 450 participants lie closely with several larger and smaller studies in demonstrating that maternal vitamin D deficiency in early pregnancy is associated with higher risks of adverse perinatal and early childhood outcomes while showing some important contrasts in magnitude and secondary findings. In our cohort, mean first-trimester 25(OH)D was very low in the deficient groups (DD 8.1 ± 1.7 ng/mL; DS 8.3 ± 1.6 ng/mL) versus the sufficient group (SS 26.2 ± 7.2 ng/mL), and second-trimester levels stayed low in DD (7.9 ± 2.1 ng/mL) but rose in DS to 24.8 ± 5.6 ng/mL. These measured differences mirror the stratification reported in larger retrospective cohorts that measured 25(OH)D across trimesters and found the greatest perinatal risk concentration among women with persistent early deficiency (Lee et al., 2023) [7]. In both our study and Lee et al., early deficiency carried similar orders of magnitude for preterm birth: we observed preterm <34 weeks in 6.7% of DD versus 2.3% in SS (adjusted OR 2.56), which is comparable to the aOR ~ 2.4 reported by Lee et al. for early persistent deficiency. This convergence further supports the inference that vitamin D status in early pregnancy is a reproducible risk marker of prenatal risk of early preterm birth and not simply an artifact of small sample size (Zhang et al., 2022) [9].

Our profile of neonatal morbidity also had a similar direction if not the same absolute magnitude as the cohort in PLOS. For example, NEC occurred more frequently in our DD group (3.3%) as compared to (0.3%) in the SS group adjusted OR 12.5, consistent with the study by Lee et al., which noted a very large aOR for NEC with persistent deficiency (aOR ~ 22), although both studies identified wide confidence intervals as the studies had very limited events [2]. The signal for a higher rate of NEC among independent samples is striking in the context of biological plausibility since vitamin D has respected roles in immune modulation and mucosal barrier integrity, both

of which could affect neonatal intestinal vulnerability (Aranow 2011; Hollis & Wagner 2017) [10,11]. Our observed lower mean birthweight in the DD group was 2850 ± 590 g compared to the SS of 2975 ± 520 g, which also echoed meta-analytic reports linking lower maternal 25(OH)D to reduced birthweight and small-for-gestational-age risk (Zhang et al., 2022) [9].

Where our findings diverge from some previous reports is in the pattern of obstetric interventions. We found lower cesarean rates in DD (53.3%) and DS (45.0%) compared to SS (66.0%), with an adjusted OR indicating decreased odds of cesarean in DD. Lee et al. and other large datasets emphasize that groups enriched for IVF conceptions often have higher intervention rates and IVF prevalence can confound delivery mode statistics; the SS group in several cohorts contained more IVF pregnancies and therefore higher cesarean and NICU admission rates, an observation which helps reconcile paradoxical findings between vitamin D status and cesarean frequency in different studies (Lee SB et al., 2023) [7]. Thus, delivery-mode contrasts may reflect underlying differences in fertility care and obstetric practice rather than a direct protective effect of deficiency.

Respiratory outcomes and NICU use in our cohort had smaller, non-significant differences across vitamin D groups. Systematic reviews have also reported mixed effects of antenatal vitamin D on neonatal respiratory outcomes and childhood wheeze: some meta-analyses report modest protective effects, while others find no consistent benefit (Kim et al., 2023; Loddio et al., 2023) [5,12]. In practical terms, our results regarding RDS and NICU are not inconsistent with the literature—they reflect that respiratory morbidities are multifactorial and less tightly coupled to maternal 25(OH)D compared with outcomes such as preterm birth or NEC.

Perhaps most importantly for long-term implications, our observed association between early maternal deficiency and developmental delay at one year (DD 8.9% vs SS 2.0%; adj OR 3.94) agrees with previous epidemiological studies that have associated low antenatal vitamin D with poorer neurodevelopmental outcomes and an increased risk for attention and cognitive problems (Whitehouse et al., 2012; Sucksdorff et al., 2021) [13,6]. The magnitude of our adjusted effect size is similar to that reported in larger cohorts, supporting the hypothesis that early gestation is a sensitive window for neurodevelopmental programming and that later normalization of 25(OH)D may not fully reverse early deficits.

Considered together, these comparisons suggest a coherent picture: early maternal vitamin D deficiency is a consistent predictor of preterm birth, lower birth weight, and certain neonatal complications (NEC) while being plausibly linked to later developmental risk. Differences in effect size across studies reflect sample size, event rarity, population composition, including IVF rates and baseline 25[OH]D distributions, and timing/degree of repletion. This supports recommendations by experts to consider preconception or very early pregnancy screening and more aggressive optimization of 25(OH)D, given that the mean antenatal level for many populations falls below proposed optimal targets. Future prospective and interventional studies, therefore, should prioritize early supplementation, use standardized outcome definitions, and report stratification by baseline fertility care status to tease out treatment effects from confounding by obstetric practice.

Conclusion

The study's findings indicated that maternal vitamin D deficiency in pregnancy was associated with selected adverse obstetric and neonatal outcomes. While the overall incidences of major obstetric complications such as preeclampsia, gestational diabetes, and placenta previa did not differ significantly between groups, vitamin D-deficient mothers demonstrated higher rates of preterm birth, especially less than 34 weeks, and a lower likelihood of cesarean delivery compared with women who had sufficient vitamin D levels. Neonates born to vitamin D-deficient mothers had lower mean birth weights and a higher incidence of severe neonatal morbidities, including necrotizing enterocolitis and developmental delay at one year of age. These findings suggest that lower levels of maternal vitamin D likely have potential effects, both at the time of pregnancy and as a long-term outcome for the neonate and point towards the importance of a state of vitamin D adequacy, throughout pregnancy.

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