Vitamin D insufficiency in pregnant and nonpregnant women of childbearing age in the United States

Adit A. Ginde, MD, MPH, **Ashley F. Sullivan, MS, MPH**, **Jonathan M. Mansbach, MD**, and **Carlos A. Camargo Jr., MD, DrPH**

Department of Emergency Medicine (Dr Ginde), University of Colorado, Denver, School of Medicine, Aurora, CO, and the Department of Emergency Medicine (Ms Sullivan and Dr Camargo), Massachusetts General Hospital, and the Department of Medicine (Dr Mansbach), Children's Hospital Boston, Harvard Medical School, Boston, MA.

Abstract

OBJECTIVE—We evaluated vitamin D insufficiency in a nationally representative sample of women and assessed the role of vitamin supplementation.

STUDY DESIGN—We conducted secondary analysis of 928 pregnant and 5173 nonpregnant women aged 13–44 years from the National Health and Nutrition Examination Survey 2001–2006.

RESULTS—The mean 25-hydroxyvitamin D (25[OH]D) level was 65 nmol/L for pregnant women and 59 nmol/L for nonpregnant women. The prevalence of 25(OH)D <75 nmol/L was 69% and 78%, respectively. Pregnant women in the first trimester had similar 25(OH)D levels as nonpregnant women (55 vs 59 nmol/L), despite a higher proportion taking vitamin D supplementation (61% vs 32%). However, first-trimester women had lower 25(OH)D levels than third-trimester women (80 nmol/L), likely from shorter duration of supplement use.

CONCLUSION—Adolescent and adult women of childbearing age have a high prevalence of vitamin D insufficiency. Current prenatal multivitamins (400 IU vitamin D) helped to raise serum 25(OH)D levels, but higher doses and longer duration may be required.

Keywords

epidemiology; nutrition; pregnancy; supplementation; vitamin D

Over the past few decades, vitamin D has reemerged as an important nutritional factor in maternal and infant health. The resurgence of childhood rickets and other problems of bone mineralization associated with severe vitamin D deficiency appears to represent only part of the story for vitamin D and health.[1,2] Indeed, vitamin D regulates >1000 human genes, and vitamin D receptors are found in most tissues and cells throughout the body.[3] Accordingly, in utero or early life vitamin D insufficiency has been linked to increased risk of childhood wheezing,[4–6] respiratory infection,[6–8] type 1 diabetes,[9] multiple sclerosis,[10] schizophrenia,[11] and even placental development and function.[12] Vitamin D insufficiency in adults has also been linked to cardiovascular disease,[13] infection,[14] cancer,[15] and even mortality.[16] While relatively small amounts of vitamin D prevent nutritional rickets, larger doses and higher serum 25-hydroxyvitamin D (25[OH]D) levels appear necessary for optimal general health outcomes.[17]

Individuals with darker skin pigment, those who cover their skin with clothing for religious or cultural reasons, and those living further away from the equator during the winter season are at particular risk for vitamin D insufficiency.[18,19] Additionally, successful campaigns to control sun exposure through avoidance and sun protection,[20] coupled with decreased outdoor physical activity,[21] have likely led to rising prevalence of vitamin D insufficiency in the general population.[22] Until recently, serum 25(OH)D levels   50 nmol/L (to convert to ng/mL divide by 2.496) appeared adequate based on improved skeletal outcomes,[23] but increasing evidence suggests that 75 nmol/L or even 100 nmol/L may be required for optimum health.[24] Indeed, current recommended doses of vitamin D supplementation, 200–600 IU daily,[25] may be inadequate to achieve these higher serum 25(OH)D levels on a population level.[17,18,24]

Since 25(OH)D readily crosses the placenta,[26] fetal and newborn vitamin D status is almost entirely dependent on vitamin D from the mother.[27] Not surprisingly, cord blood 25(OH)D levels are strongly correlated with maternal vitamin D status.[23] Additionally, because vitamin D secretion in breast milk is limited,[28] lactating women require robust serum 25(OH)D levels to support vitamin D status in nursing infants.[29] A high prevalence of vitamin D insufficiency has been documented in pregnant and lactating women, particularly in highrisk cohorts.[30–32] These studies have raised awareness of higher doses of vitamin D supplementation that may be required to improve maternal and infant skeletal and general health outcomes. For instance, the Canadian Pediatric Society recently recommended 2000 IU daily of vitamin D supplementation, and higher target serum 25(OH)D levels for pregnant and lactating women.[33]

In this study, we sought to measure vitamin D insufficiency in a nationally representative sample of adolescent and adult women of childbearing age in the United States and to evaluate the role of vitamin D supplementation and outdoor activity in prevention of vitamin D insufficiency.

Materials and Methods

Study design and participants

Annually, the National Center for Health Statistics conducts the National Health and Nutrition Examination Survey (NHANES), a nationally representative probability sample of the noninstitutionalized US civilian population. Because the data are publicly available and deidentified, we received a waiver from our institutional review board to analyze the 3 most recent cycles of NHANES data (ie,January 2001 through December 2006).

Details of survey methodology are described elsewhere.[34] During 2001–2006, NHANES collected household interview data for 7932 (83%) of 9521 invited adolescent and adult women aged 12–49 years. Subsequently, 7642 (96%) received physical and laboratory examination in a mobile examination center, of which 7026 had serum 25(OH)D testing (616 missing). Pregnancy status was determined by self-report, supplemented by urine human chorionic gonadotropin testing for most women. The NHANES data included an adjudicated pregnancy status variable that was used for analysis in this study, as recommended by the NHANES instructions for data analysis.[34] Since the age range for pregnant women in NHANES was 13–44 years, our analysis included 928 pregnant and 5173 nonpregnant women aged 13–44 with serum 25(OH)D levels recorded. This represents approximately 62 million US women.

Data collection

Throughout the NHANES years, strategies for sampling and methodologies for data collection were very similar to maintain consistency and facilitate comparisons. To avoid

weather issues and improve response, the NHANES mobile examination centers preferentially scheduled data collection in the lower latitudes (further south) during winter months, and higher latitudes (further north) during the summer months. Since month of data collection was not reported in the NHANES 2001– 2006 data set (to protect participant confidentiality), we were unable to record or control for season.

Demographic data (age, sex, race/ethnicity) and current breast-feeding were recorded based on self-report. The poverty-income ratio is provided in the NHANES data set and was used to control for socioeconomic status; values < 1 were considered low socioeconomic status. Physical activity was measured by a query of 48 common leisure-time activities over the past month. We categorized activities as outdoor (eg, walking, gardening), indoor (eg, aerobics, weightlifting), or indeterminate (eg, basketball, swimming), as previously described.[21] We then classified leisure-time activities during the past month for each study participant as: none; at least 1 outdoor activity; or indoor/indeterminate activities only. For pregnant women, month of pregnancy was recorded based on last menstrual period (if known), and we stratified the estimated gestational ages into trimesters or unknown gestational age. For the vitamin supplement data, the interviewer identified all of the vitamin/ mineral supplements being used and duration of use.

Blood samples for serum 25(OH)D testing collected during the examination were centrifuged, aliquoted, and frozen to –70°C until analysis. Serum 25(OH)D levels were measured by a radioimmunoassay kit after extraction with acetonitrile (DiaSorin, Stillwater, MN) by the National Center for Environmental Health (Atlanta, GA).

Data analysis

We performed statistical analyses using software (Stata 9.0; StataCorp, College Station, TX). We applied the recommended subsample weights for the interview plus examination data to account for unequal probabilities of selection and to provide population estimates of 25(OH)D levels. Although all results are presented as weighted values, we also provide the raw numbers to allow for insight about the sample size. We calculated variance based on NHANES-provided masked variance units, using Taylor Series linearization method. We reported mean serum 25(OH)D levels and further stratified results by 2 common definitions of vitamin D insufficiency, <50 nmol/L, and <75 nmol/L.[22] Primary analysis is descriptive with 95% confidence intervals (CIs).

We made univariate statistical comparisons using χ^2 test or analysis of variance, as appropriate. All P values are 2-tailed with $P < .05$ considered statistically significant. To evaluate the independent association between covariates and serum 25(OH)D levels, we created multivariable linear and logistic regression models for mean 25(OH)D levels and dichotomous outcomes of vitamin D insufficiency, respectively. We included vitamin D supplementation dose, but not duration of vitamin D supplements, due to colinearity. We reported β coefficients and odds ratios with 95% CI for variables in the multivariable models.

Results

In NHANES 2001–2006, there were 928 pregnant women and 5173 nonpregnant women of childbearing age who were eligible for inclusion, which represents 4 million (7%) and 58 million (93%) women, respectively. Demographic and clinical characteristics of the study population are presented in Table 1.

Overall, the mean serum 25(OH)D level among all US women aged 13–44 years was 59 nmol/L (95% CI, 57–61). Table 2 displays the mean 25(OH)D levels of participants,

stratified by characteristics. Serum 25(OH)D levels were higher among pregnant compared to nonpregnant women (65 vs 59 nmol/L; $P < .001$). Currently breast-feeding women (n = 139) had similar 25(OH)D levels as other nonpregnant women (60 vs 59 nmol/L; $P = .40$); thus these groups were combined in the analysis.

The largest magnitude of association was observed between race/ethnicity and 25(OH)D levels, with non-Hispanic whites have much higher 25(OH)D levels than racial/ethnic minorities in both pregnant and nonpregnant groups. Higher socioeconomic status, later trimester, vitamin D supplementation dose, duration of vitamin D supplementation use, and outdoor physical activity were all associated with higher 25(OH)D levels. Older age was also associated with higher 25(OH)D levels in pregnant but not nonpregnant women. However, pregnant adolescent women aged 13–19 years were more likely to be nonwhite than pregnant women aged 35–44 years (57% vs 28%, $P < .001$). When stratified into individual race/ethnicity groups, there was no significant association between age and 25(OH)D level (data not shown).

Women in the first trimester of pregnancy had lower 25(OH)D levels than third-trimester women (55 vs 80 nmol/L; $P < .001$). Non-Hispanic white women in the third trimester had higher 25(OH)D levels (93 nmol/L) than non-Hispanic black (45 nmol/L) or Hispanic (69 nmol/L) women ($P < .001$). However, distribution of race/ethnicity was similar across trimesters and thus, does not likely explain increasing 25(OH)D levels with later trimester. Rather, pregnant women were more likely to be taking any vitamin D supplementation with increasing trimester (61%, 72%, and 86%, respectively; P for trend = .002). Additionally, mean 25(OH)D levels by increasing trimester were 46, 46, and 54 nmol/L, respectively, among women not taking vitamin D-containing supplements (P for trend = .27), compared to 61, 69, and 84 nmol/L among those taking supplements (P for trend < .001). Moreover, later trimester was associated with longer duration of vitamin D supplement use (among those taking vitamin D supplements, median duration was 61, 122, and 304 days, respectively; P for trend <.001).

A higher proportion of pregnant women in the first trimester were taking vitamin D supplementation compared to nonpregnant women (61% vs 32%; $P < .001$), but they had similar 25(OH)D levels (55 vs 59 nmol/L; $P = .16$). One possible explanation is that women in the first trimester had shorter duration of vitamin D supplement use, compared to nonpregnant women (among those taking vitamin D supplements, median: 61 vs 365 days; $P < .001$). Younger age, minority race/ethnicity, lower socioeconomic status, pregnancy, and later trimester were independently associated with nonuse of any vitamin D supplementation (Table 3). Among those taking vitamin D supplementation, the median vitamin D dose was 400 IU daily for both pregnant and nonpregnant women. Among pregnant women taking vitamin D supplementation, 83% were taking 400 IU daily, and 7% took >400 IU daily, compared to 77% and 13%, respectively, for nonpregnant women.

Women in their third trimester of pregnancy had much higher 25(OH)D levels than women who were currently breast-feeding (80 vs 60 nmol/L, $P < .001$). Indeed, use of any vitamin D supplementation was higher in third trimester of pregnancy than while breast-feeding (86% vs 65%; $P < .001$).

Overall, 41% (95% confidence interval [CI], 38–45%) of women had serum 25(OH)D levels <50 nmol/L and 78% (95% CI, 75–80%) had levels <75 nmol/L. Results stratified by pregnancy status and characteristics for these 2 thresholds are presented in Table 4. Additionally, 7% (95% CI, 4–10) of pregnant women and 10% (95% CI, 8–12) of nonpregnant women had 25(OH)D levels <25 nmol/L.

Multivariable predictors of 25(OH)D levels (continuous) for pregnant and nonpregnant women are presented in Table 5. There was no material difference in multivariable logistic regression results for the dichotomous outcomes of 25(OH)D levels <50 nmol/L and <75 nmol/L (data not shown). Race and vitamin D supplementation dose, but not age and socioeconomic status, were strongly associated with 25(OH)D levels. Outdoor physical activity was associated with significantly higher 25(OH)D levels in nonpregnant but not pregnant women. For pregnant women, later trimester was independently associated with higher 25(OH)D levels, even after controlling for other covariates including demographics, vitamin D supplement use, and outdoor activity.

Comment

The present study reports the vitamin D status of a nationally representative sample of adolescent and adult women of childbearing age. We confirm prior studies that found a high prevalence of vitamin D insufficiency within[30,31] and outside[32] the United States. As previously reported, serum 25(OH)D levels for black women were nearly half those of white women.[30] In the present study, we also report a novel finding that Hispanic women had prevalence of 25(OH)D <75 nmol/L that is similar to non-Hispanic blacks. The absorption of ultraviolet B radiation by skin melanin and consequent reduction of vitamin D synthesis[35] is likely the major cause of the observed differences by race/ethnicity.

Maternal vitamin D deficiency during pregnancy not only has adverse health effects on the mother,[28] but also means the fetus develops in a low vitamin D state,[27] because 25(OH)D readily crosses the placenta.[26] Indeed, maternal 25(OH)D levels are directly correlated to cord blood levels.[23] However, the serum 25(OH)D levels associated with optimal maternal, fetal, and infant general health remain unknown. Gordon et al[36] found that one third of infants and toddlers with a serum 25(OH)D level <50 nmol/L were noted to have some evidence of bone demineralization. In addition, rickets has been found in infants with serum 25(OH)D levels close to 50 nmol/L.[1,2,23] Most clinicians and investigators would agree that, at minimum, 25(OH)D levels 50 nmol//L are needed for health benefits in children and adults, and recent evidence suggests that at least 75 nmol/L may be required, particularly for nonskeletal outcomes.[17,37].

Camargo et al[6] found that risk of respiratory infection and wheezing was lowest for children with cord blood 25(OH)D 75 nmol/L in a prospective birth cohort, which was confirmed in US adolescents and adults.[14] Looking beyond respiratory infections, a systematic review of the association between vitamin D and several nonskeletal outcomes in adults concluded that "the most advantageous target concentration of 25(OH)D begins at 75 nmol/L,"[24] and there is mounting evidence that 25(OH)D levels 75 nmol/L might also be required for pregnant women and infants.[4–6,27] Accordingly, the Canadian Pediatric Society adopted 75 nmol/L as "sufficient" for pregnant and lactating women and young children.[33]

The present results support prior reports that 400 IU daily may be inadequate for raising maternal or cord blood 25(OH)D levels to 75 nmol/L,[29,38–40] particularly for higher-risk women, such as racial/ethnic minorities, women living at northern latitudes, and during the winter season. Pilot studies have suggested 1000 IU,[39–43] 2000 IU,[44] 4000 IU,[29] or even 6400 IU[45] daily would help to eliminate vitamin D insufficiency without apparent toxicity in pregnant women and their infants. Although the safe Tolerable Upper Intake Level for vitamin D was set at 2000 IU daily in 1997,[25] recent analyses suggest that this could be raised to 10,000 IU daily for most adults.[46] The Tolerable Upper Intake Level of fetuses and infants is less clear, and some investigators have raised concerns that excessive vitamin D may increase risk of allergic conditions.[47]

The optimal dose of a vitamin D supplement depends on many factors, including race/ethnicity, sunlight exposure, dietary intake, absorption, and metabolism. For example, white women in San Diego, CA, during the summer will require significantly lower doses than black women in Seattle, WA, during the winter. As the proposed dose of vitamin D supplement in pregnancy increases (eg, to 4000 IU daily),[29] the importance of these individual factors rises. Indeed, we found that white women in the third trimester had a mean 25(OH)D level of 93 nmol/L, indicating that some individuals, particularly in the summer season, may not need higher doses than 400 IU daily. Additionally, we found that women of younger age, minority race/ ethnicity, and lower socioeconomic status were more likely to forgo any vitamin D supplementation; particular attention should be given to these groups in clinical practice. Ultimately, measurement of serum 25(OH)D levels during pregnancy and early childhood may prove essential for high-quality preventive care.

We were surprised that serum 25(OH)D levels were higher with later trimester, since prior opinion suggests that 25(OH)D levels should be equal or even decrease during pregnancy.[26] However, Bodnar et al[30] also reported higher 25(OH)D levels in late, as compared to early, pregnancy. In the present study, longer duration of vitamin D supplementation was likely responsible for the higher levels later in pregnancy. For instance, we found that among pregnant women not taking vitamin D supplements, 25(OH)D levels were similar throughout pregnancy.

Although 61% of pregnant women in the first trimester were taking vitamin D-containing supplements, it appears that many had only recently initiated vitamin D-containing supplements (median duration: 61 days). In contrast, 86% of pregnant women during the third trimester were taking vitamin D-containing supplements for a median duration of 304 days; thus, even though the vitamin D doses were similar, the 25(OH)D levels were approximately 25 nmol/L higher in third-trimester compared to first-trimester women. These data suggest that 25(OH)D levels could be improved during early pregnancy by initiating vitamin D supplementation before pregnancy (eg, to increase supplement duration).

Although 25(OH)D passes poorly in breast milk,[28] breast milk concentrations are related to maternal serum levels.[40] We found that women who were currently breast-feeding had much lower 25(OH)D levels than in third-trimester pregnancy and similar to other nonpregnant women. Increased compliance and possibly higher maternal doses of vitamin D supplements during lactation (eg, at least 1000 IU daily) may be required to adequately raise 25(OH)D levels in lactating mothers, breast milk, and nursing infants.[29] Further, a pilot study found that high-dose supplementation (6400 IU daily) doubled 25(OH)D levels in serum and milk, but vitamin D levels (ie, the parent compound), which transfers efficiently in breast milk, increased 10-fold.[45]

This study has some potential limitations. We have based our serum 25(OH)D thresholds on prior outcome studies; no outcomes were assessed in this analysis. Serum was preferentially collected in northern states in the summer and southern states in the winter. As a result, the data presented most likely represent the best-case scenario; random sampling across all seasons should yield an even higher prevalence of vitamin D insufficiency. Latitude and season, which are important determinants of serum 25(OH)D levels, were not available for analysis. However, since determinants of vitamin D insufficiency have been well described, the primary purpose of our analysis was to examine the prevalence of vitamin D insufficiency.

We conclude that many adolescent and adult women of childbearing age have 25(OH)D levels <75 nmol/L. Although the optimal dosage of vitamin D supplementation depends on a variety of factors, current US vitamin D supplementation guidelines of 200–400 IU daily for

these women[25,37] may be inadequate for many women and their infants. While the current dose of vitamin D in prenatal multivitamins (400 IU) helps to raise 25(OH)D levels, nearly half of pregnant women in the third trimester still had 25(OH)D levels <75 nmol/L. Additionally, because duration of supplementation appeared important, women should ideally start vitamin D supplementation a few months before becoming pregnant. Ongoing clinical trials are looking at different regimens to establish the optimal dose, duration, and safety of even higher vitamin D supplementation during pregnancy (www.clinicaltrials.gov, #R01HD043921). Further study of vitamin D-related outcomes in pregnant women and young children will help to inform recommendations for vitamin D supplementation so that both mothers and children achieve serum 25(OH)D levels that optimize their skeletal and general health.

Acknowledgments

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TABLE 1

United States women of childbearing age, 2001–2006

Characteristic	Pregnant (n = 928) (95% CI)	Nonpregnant (n = 5173) (95% CI)	P value
Age, y			< .001
13–19 (n = 2710)	9% (7–12)	21% (20–23)	
20–34 (n = 2203)	78% (73–82)	44% (41–46)	
35–44 (n = 1188)	13% (10–18)	35% (32–37)	
Race			< .001
NH white (n = 2296)	56% (50–62)	65% (62–69)	
NH black (n = 1572)	16% (12–22)	13% (11–16)	
Hispanic (n = 1969)	20% (16–26)	16% (13–18)	
Other (n = 264)	8% (6–10)	6% (4–7)	
Poverty-income ratio			.03
1 (n = 1650)	25% (20–30)	18% (17–20)	
>1 (n = 4182)	71% (65–76)	78% (76–79)	
Unknown (n = 269)	4% (2–8)	4% (3–5)	
Gestational age			-
First trimester (n = 198)	29% (24–34)	-	
Second trimester (n = 331)	31% (26–36)	-	
Third trimester (n = 312)	30% (25–36)	-	
Unknown (n = 87)	10% (7–15)	-	
Vitamin D supplement dose (IU)			< .001
0 (n = 4268)	28% (22–34)	68% (66–70)	
1–399 (n = 157)	7% (5–10)	3% (2–4)	
400(n = 1676)	66% (59–71)	29% (27–31)	
Duration of vitamin D supplement use (d)[a]			< .001
0 (n = 4269)	28% (22–34)	68% (66–70)	
1–30 (n = 251)	12% (8–17)	4% (3–5)	
31–180 (n = 423)	21% (16–26)	4% (4–5)	
181 (n = 1152)	29% (33–45)	24% (22–26)	
Physical activity in past 30 d			< .001
None(n = 1784)	39% (32–46)	26% (24–28)	
Outdoor (n = 3516)	53% (45–60)	62% (59–64)	
Indoor/indeterminate only (n = 801)	8% (5–12)	12% (11–14)	

CI, confidence interval; *NH*, non-Hispanic

Weighted to represent total 4 million pregnant and 58 million nonpregnant women in US population.

[a]Based on longest duration of use for any current vitamin D-containing supplement; 6 missing/unknown.

TABLE 2

Mean serum 25-hydroxyvitamin D levels

Variable	Pregnant (n = 928) nmol/L (95% CI)	Nonpregnant (n = 5173) nmol/L (95% CI)
Total (n =6101)	65(61–68)	59(57–61)
Age, y		
13–19 (n = 2710)	57 (50–64)	59(56–61)
20–34 (n = 2203)	64(61–68)	60 (57–63)
35–44 (n = 1188)	71 (64–79)	57 (54–60)
Race		
NH white (n = 2296)	77 (73–80)	67 (65–70)
NH black (n = 1572)	39 (35–42)	33 (32–35)
Hispanic (n = 1969)	56(51–62)	48 (46–49)
Other (n = 264)	54 (46–62)	46 (42–50)
Poverty-income ratio		
1 (n = 1650)	56 (50–63)	53 (49–56)
>1 (n = 4182)	68 (64–72)	60 (58–62)
Unknown (n = 269)	53 (48–59)	60 (53–66)
Gestational age		
First trimester (n = 198)	55 (50–60)	–
Second trimester (n = 331)	62 (62–57)	–
Third trimester (n = 312)	80 (73–87)	–
Unknown (n = 87)	53(45–61)	–
Vitamin D supplement dose (IU)		
0 (n = 4268)	46(41–50)	55 (53–57)
1–399 (n = 157)	68 (59–77)	65 (59–71)
400(n = 1676)	72 (69–76)	67 (65–69)
Duration of vitamin D supplement use, d[a]		
0 (n = 4269)	46(41–50)	55 (53–57)
1–30 (n = 251)	62 (55–68)	62 (55–68)
31–180 (n =423)	68 (62–73)	62 (57–66)
181 (n = 1152)	77 (72–82)	68 (66–70)
Physical activity in past 30 d		
None(n = 1784)	56(51–61)	50 (47–52)
Outdoor (n = 3516)	72 (67–76)	63 (61–65)
Indoor/indeterminate only (n = 801)	62 (54–71)	56 (53–59)

CI, confidence interval; *NH*, non-Hispanic.

[a]Based on longest duration of use for any current vitamin D-containing supplement; 6 missing/unknown.

TABLE 3

Nonuse of vitamin D-containing supplements

Variable	(95% CI)[a]	OR (95% CI)[b]
Age, y		
13–19	81% (78–83)	2.3(1.8–3.0)
20–34	62% (58–65)	Referent
35–44	61% (58–65)	0.9(0.8–1.1)
Race		
NH white	59% (56–62)	Referent
NH black	80% (76–83)	2.7(2.1–3.6)
Hispanic	80% (77–82)	2.8 (2.3–3.5)
Poverty-income ratio		
1	77% (73–81)	1.8 (1.4–2.3)
>1	62% (65–79)	Referent
Pregnancy		
None	68% (66–70)	Referent
First trimester (n =198)	39% (28–51)	0.3(0.1–0.5)
Second trimester (n = 331)	28% (20–37)	0.1 (0.1–0.2)
Third trimester (n = 312)	14% (8–24)	0.1 (0–0.1)

CI, confidence interval; *NH*, non-Hispanic; *OR*, odds ratio.

[a]Proportion of participants reporting no use of vitamin D-containing supplements in past month (χ^2 *P*< .001 for all categories compared)

[b]Multivariable logistic regression model for outcome of nonuse of any vitamin D-containing supplements.

TABLE 4

Vitamin D insufficiency in study sample

Variable	Pregnant (n = 928)		Nonpregnant (n = 5173)	
	<50 nmol/L (n = 309) (95% CI)	<75 nmol/L (n = 643) (95% CI)	<50 nmol/L (n = 2904) (95% CI)	<75 nmol/L (n = 4477) (95% CI)
Total (n = 6101)	33% (28–40)	69% (63–75)	42% (38–46)	78% (76–80)
Age, y				
13–19 (n = 2710)	51% (41–61)[a]	74% (61–84)	40% (36–44)	81% (77–84)[b]
20–34 (n = 2203)	34% (27–41)[a]	70% (64–74)	42% (38–46)	75% (72–79)[b]
35–44 (n = 1188)	20% (12–32)[a]	65% (43–82)	44% (39–49)	80% (77–83)[b]
Race				
NH white (n = 2296)	13% (8–19)[a]	54% (46–63)[a]	26% (23–30)[a]	69% (66–72)[a]
NH black (n = 1572)	80% (68–88)[a]	95% (89–98)[a]	86% (83–89)[a]	99% (98–99)[a]
Hispanic (n = 1969)	45% (36–55)[a]	83% (75–89)[a]	60% (56–63)[a]	93% (90–95)[a]
Poverty-income ratio				
1 (n = 1650)	46% (37–56)[a]	81% (72–88)[a]	53% (47–59)[a]	83% (78–87)[b]
>1 (n = 4182)	29% (22–37)[a]	64% (57–70)[a]	39% (36–43)[a]	77% (75–80)[b]
Gestational age				
First trimester (n = 198)	46% (32–61)[a]	83% (70–91)[a]	–	–
Second trimester (n = 331)	32% (23–43)[a]	75% (67–82)[a]	–	–
Third trimester (n = 312)	18% (11–27)[a]	47% (36–58)[a]	–	–
Vitamin D supplement dose (IU)				
0 (n = 4268)	66% (55–76)[a]	92% (86–95)[a]	49% (45–53)[a]	82% (79–84)[a]
1–399 (n = 157)	19% (8–39)[a]	67% (40–87)[a]	26% (15–40)[a]	71% (56–83)[a]
400(n = 1676)	21% (16–27)[a]	60% (54–66)[a]	27% (23–32)[a]	71% (68–73)[a]
Duration of vitamin D supplement use (d)				
0 (n = 4269)	66% (55–76)[a]	92% (86–95)[a]	49% (45–53)[a]	82% (79–84)[a]
1–30 (n = 251)	37% (22–56)[a]	83% (69–91)[a]	38% (26–51)[a]	79% (67–87)[a]

Variable	Pregnant (n = 928)		Nonpregnant (n = 5173)	
	<50 nmol/L (n = 309) (95% CI)	<75 nmol/L (n = 643) (95% CI)	<50 nmol/L (n = 2904) (95% CI)	<75 nmol/L (n = 4477) (95% CI)
31–180 (n =423)	23% (15–33)[a]	66% (54–75)[a]	35% (27–45)[a]	75% (66–83)[a]
181 (n = 1152)	15% (9–23)[a]	52% (43–61)[a]	24% (20–28)[a]	69% (66–71)[a]
Physical activity in past 30 d				
None(n = 1784)	48% (37–58)[a]	81% (73–87)[a]	56% (50–61)[a]	86% (83–89)[a]
Outdoor (n = 3516)	24% (18–30)[a]	60% (52–68)[a]	35% (31–38)[a]	74% (71–77)[a]
Indoor/indeterminate only (n = 801)	31% (15–53)[a]	73% (53–87)[a]	50% (44–56)[a]	81% (77–85)[a]

CI, confidence interval; NH, non-Hispanic.

Row percentages provided to facilitate comparisons, and P values denote differences in prevalence for each covariate.

[a] P< .01

[b] P< .05.

TABLE 5

Multivariable models for serum 25-hydroxyvitamin D levels

Variable	Pregnant (n = 928)		Nonpregnant (n = 5173)	
	Model 1 (95% CI)	Model 2 (95% CI)	Model 1 (95% CI)	Model 2 (95% CI)
Age/y	0.1 (−0.2 to 0.4)	0 (−0.4 to 0.3)	−0.1 (−0.2 to−0.04)	−0.1 (−0.2 to−0.04)
Race				
NH white	Referent	Referent	Referent	Referent
NH black	−37 (−42 to−32)	−32 (−38 to−27)	−34 (−37 to− −32)	−32 (−34 to−29)
Hispanic	−20 (−26 to−14)	−16 (−23 to−9.1)	−20 (−23 to− −17)	−17 (−20 to−15)
Poverty-income ratio				
1	0.2 (−5.8 to 6.2)	0.4 (−5.2 to 6.1)	−1.0 (−3.6 to 1.6)	0.7 (−1.8 to 3.3)
>1	Referent	Referent	Referent	Referent
Gestational age				
First trimester	−25 (−32 to−17)	−21 (−28 to−14)	–	–
Second trimester	−14 (−20 to−7.5)	−12 (−18 to−6.3)	–	–
Third trimester	Referent	Referent	–	–
Vitamin D supplement dose (IU)				
0	–	−12 (−16 to−8.6)	–	−7.5 (−9.4 to−5.6)
1–399	–	−3.5 (−12 to 5.1)	–	−2.3 (−7.1 to 2.4)
400	–	Referent	–	Referent
Physical activity in past 30 d				
None	–	−3.5 (−7.7 to 0.8)	–	−7.5 (−9.3 to−5.6)
Outdoor	–	Referent	–	Referent
Indoor/indeterminate only	–	−2.3 (−10 to 5.7)	–	−3.2 (−5.8 to −0.5)

CI, confidence interval; *NH*, non-Hispanic.