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Determination of vitamin D in relation to body mass index and race in a defined population of black and white women

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Abstract

Objective—To examine the contributions of obesity and race to levels of 25-hydroxyvitamin D [25(OH)D] and parathyroid hormone (PTH) in a defined cohort of black and white women.

Methods—An interventional study was conducted from October 2004 to March 2008, among 219 healthy female volunteers. Serum 25(OH)D and PTH levels were determined in 117 African American women and 102 white women and the results were compared with body mass index (BMI), percentage body fat, serum lipids, and PTH levels.

Results—Black women had lower median levels of 25(OH)D compared with white women (27.3 nmol/L vs 52.4 nmol/L; $P<0.001$). Serum levels of 25(OH)D below 50 nmol/L were found in 98% of black women and 45% of white women ($P<0.001$). The differences between the racial groups in the levels of 25(OH)D persisted despite adjustments for body weight, percentage body fat, and BMI. Black women had higher median serum levels of PTH than white women (31.9 pg/mL vs 22.3 pg/mL; $P<0.01$).

Conclusion—African American women are at significant risk for low vitamin D levels. Studies are needed to determine if low vitamin D status in young African American women is associated with a greater risk for vitamin D-related chronic diseases that can be reduced with vitamin D supplementation.

Keywords

25-hydroxyvitamin D; Obesity; Parathyroid hormone; Race; Vitamin D

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Synopsis: Young, African American women are at significant risk for low 25-hydroxyvitamin D levels despite adjustments for percentage body fat and body mass index.

Conflict of interest

The authors have no conflicts of interest to declare.

1. Introduction

Vitamin D is an important vitamin because of the critical role it plays in calcium homeostasis and bone metabolism [1]. In recent years, interest in low vitamin D levels has intensified as the role of vitamin D has been linked to important physiologic actions including neuromuscular function, inflammation, and the action of genes involved in the regulation of cell proliferation, differentiation, and apoptosis [2]. Vitamin D insufficiency has been associated with heart disease, hypertension, autoimmune disease, and certain cancers [3,4]. With improved methods for measuring 25-hydroxyvitamin D [25(OH)D], a higher prevalence of vitamin D insufficiency has been shown to exist across all ages and ethnic groups [5]. A number of risk factors have been associated with low vitamin D levels, including inadequate dietary intake, limited exposure to sunshine, old age, obesity, and the increased use of sun blocking agents that compromise cholesterol metabolism in the skin. In addition, any compromise in parathyroid hormone (PTH) synthesis and/or release can have direct effects on the bioavailability of the most active form of vitamin D. Recent reports have drawn attention to low vitamin D levels in a number of vulnerable populations. These include women of reproductive age, infants, African Americans, the elderly, and patients with diabetes mellitus [3-14].

A recent study reported that body mass index (BMI) is inversely related to circulating levels of 25(OH)D [6]. Hyppönen and Power [11], in a British birth cohort study of 25(OH)D and glucose homeostasis, noted that body size was a strong determinant of 25(OH)D levels with concentrations suboptimal in obese patients, and suggested an association between vitamin D status and diabetes. Other reports have noted decreased bioavailability of 25(OH)D in obesity [12].

The aim of the present study was to determine vitamin D status in relation to BMI and race in a well-defined cohort of healthy African American and white premenopausal women. In addition, given the suggested association of vitamin D and diabetes, the homeostasis model of assessment-insulin resistance (HOMA-IR) ratio for these 2 populations was determined as a biomarker of insulin sensitivity.

2. Materials and methods

An interventional study was conducted from October 1, 2004, to March 1, 2008, among 219 healthy female volunteers, aged 18-45 years, at 2 different institutions from 2 different geographical locations in the USA. Racial group was self-reported. The African American study population (n=117) came from a site in Nashville, Tennessee, a southern US state, while the white study population (n=102) came from a site in Hershey, Pennsylvania, a northern US state.

The Institutional Review Boards at both sites approved all study procedures. All participants provided written informed consent to participate in the study. Participants with known chronic diseases including diabetes, heart disease, and endocrine disorders were excluded. Smoking, pregnancy, and the use of hormonal therapy were also criteria for exclusion.

Each study participant underwent a physical examination with measurements taken of weight and height to the nearest 0.1 kg and 0.1 cm, respectively. BMI was calculated as body weight in kilograms divided by height in meters squared. Body fat measurements were obtained by dual-energy X-ray absorptiometry using a Hologic QDR-4500W (Hologic Corporation, Waltham, MA, USA).

A fasting blood sample was obtained at the first visit prior to any intervention. Serum samples were stored frozen at -80°C until analyzed for 25(OH)D, PTH, sex hormone binding globulin (SHBG), glucose, insulin, and lipid studies.

Serum 25(OH)D levels were measured by radioimmunoassay (Immunodiagnostic Systems Limited, Boldon, UK) using an ^{125}I -labeled 25(OH)D based competitive radioimmunoassay.

Consistent with expert opinion, 25(OH)D deficiency was defined as a level of less than 50 nmol/L ($<20\text{ ng/mL}$) and vitamin D insufficiency was defined as 50-75 nmol/L (20-30 ng/mL) [12,13]. Results greater than 75 nmol/L are considered optimal to maximize normal intestinal calcium absorption [12,13]. The 25(OH)D immunoassay had an interassay precision of 8.2% at a mean of 19.6 nmol/L.

Intact serum PTH levels were measured with the IMMULITE 1000 (Siemens Diagnostics, Los Angeles, CA, USA) using a chemiluminescence-based assay [14]. Interassay coefficient of variation (CV) was less than 6% at a mean concentration of approximately 45 pg/mL. The reference range for PTH in the study laboratory is 12-65 pg/mL.

Insulin was determined with the IMMULITE 1000 (Siemens Diagnostics) using a chemiluminescence-based assay. Interassay CVs were less than 5% in a concentration range of 7-26 $\mu\text{IU/mL}$. The insulin method had a reference range of 9-30 $\mu\text{IU/mL}$.

Glucose was determined using a glucose hexokinase method. SHBG was determined with an immunoradiometric assay (IRMA) reagents obtained from Siemens Diagnostics. Interassay CVs average 7.9% at a concentration of 29 nmol/L. The reference range for this assay in nonpregnant females is 16-120 nmol/L.

Lipid studies including cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides were determined using standard colorimetric methods.

Results were reported as mean $\pm$ SD or median (25th, 75th percentiles) depending on the normality of the distribution. Skewed data were transformed using the natural logarithm prior to analysis. The 2-sample *t* test was used to compare the 2 populations for continuous demographic and metabolic measures, as well as for 25(OH)D and PTH levels. Categorical data are reported as frequencies and percentages. The Fisher exact test was used to compare the study populations for binary variables. Analysis of variance (ANOVA) models were fit to 25(OH)D and PTH levels to assess differences between the study populations with respect to season, categorized BMI, or dichotomized percentage body fat. Back-transformation from the logarithm scale in the ANOVA models results in the data being reported as a ratio of black to white. To account for multiple comparisons testing within each ANOVA model, *P* values and 95% confidence intervals (CIs) were adjusted using the Bonferroni method.

Regression analyses were performed to assess the effect of race on the association between 25(OH)D levels and indices of obesity (BMI, weight, and percentage body fat), as well as the HOMA-IR ratio as a marker of insulin resistance [15]. Multivariable regression models were fit to 25(OH)D levels. The pool of potential predictors included race, BMI, HOMA-IR ratio, and polycystic ovary syndrome (PCOS) diagnosis. All possible combinations of these predictors were examined. Assessment of Mallows Cp statistic, Akaike information criterion, and the Schwarz Bayesian criterion were used to determine the best multivariable regression model.

All hypothesis tests were 2-sided and $P < 0.05$ was considered significant. SAS software, version 9.1 (SAS Institute Inc., Cary, NC, USA) or S-plus software, version 8.0 (Insightful, Seattle, WA, USA) were used for analyses.

3. Results

The study sample comprised 219 women: 117 (53.4%) self-designated black women and 102 (46.6%) self-designated white women. Combining both racial groups, 86 (39.3%) women had normal menstrual cycles, while 95 (43.4%) had clinical evidence of PCOS. Table 1 shows the age and degree of obesity in the participants by race. Although white women had a slightly greater body fat percentage ($P = 0.036$), the groups were not significantly different for both weight and BMI ($P > 0.05$). Despite similar indices of obesity, the HOMA-IR ratio was significantly higher in black women than in white women (2.1 vs 1.4; $P < 0.001$). This difference persisted even after women with documented PCOS were excluded from the analysis (data not shown). There was a significant difference in both HDL cholesterol and triglyceride levels between the 2 populations. HDL cholesterol was significantly higher in black women than in white women (52.2 ± 13.1 mg/dL vs 32.7 ± 12.3 mg/dL; $P < 0.001$), while triglyceride levels were significantly higher in white women compared with black women (117.3 ± 76.2 mg/dL vs 73.6 ± 41.0 mg/dL; $P < 0.001$).

Serum levels of 25(OH)D were significantly lower in black women than in white women (27.3 nmol/L vs 52.4 nmol/L; $P < 0.001$) (Table 2). Furthermore, almost all of the black women (97.5%) had levels of 25(OH)D less than 50 nmol/L, whereas less than half of the white women (44.7%) had levels below this threshold ($P < 0.001$). The majority of black women (61.0%) had 25(OH)D levels below 30 nmol/L, while only 6.8% of white women had levels below this threshold ($P < 0.001$). Although the median PTH level for black women was higher than that for white women (31.9 pg/mL vs 22.3 pg/mL; $P < 0.001$), few women from either group had PTH levels greater than 65 pg/mL—the upper cutoff level for a normal reference population.

Indices of obesity, whether measured as BMI, weight, or percentage body fat, were negatively correlated with serum 25(OH)D levels, as shown in Figure 1 (all $P < 0.01$). Furthermore, the effect of race was maintained in each of these regression models (all $P < 0.001$). Differences between racial groups persisted for all classes of BMI and for women categorized by percentage body fat ($P < 0.001$; Table 3). Calculated differences between racial groups were present upon regression analysis of fasting HOMA-IR ratios and the serum levels of 25(OH)D (data not shown). The effects of race and HOMA-IR ratios on 25(OH)D levels persisted after women with PCOS were removed from the analysis (data not shown). As expected, the levels of 25(OH)D were highest during the summer season and lowest during the winter season in both groups (Table 4). However, racial differences in 25(OH)D levels were observed in all 4 seasons ($P < 0.001$). In contrast, racial differences in serum PTH levels were observed only in the summer season ($P < 0.001$) and, regardless of racial group, did not show a seasonal trend, as did serum 25(OH)D levels ($P_{\text{trend}} = 0.60$) (Table 4).

Using race, BMI, glucose to insulin (G:I) ratio, and PCOS status as a group of potential confounding variables, the best predictor of the 25(OH)D level was a multivariable regression model ($R^2 = 0.49$) consisting of only race ($P < 0.001$) and BMI ($P < 0.001$). Polycystic ovary syndrome and G:I ratio had little predictive value for the 25(OH)D levels when evaluating all combinations of these 4 candidate predictors.

4. Discussion

The study found that serum levels of 25(OH)D were significantly lower in black women compared with the levels in white women [6,8]. It was also observed that vitamin D status in black women was negatively correlated with BMI. The levels of 25(OH)D in these 2 populations varied with the season of the year, as expected, but did not differ dramatically in either group by season. Although the HOMA-IR ratio in these women showed a correlation with the 25(OH)D levels, this effect disappeared when adjusted for BMI and race. In addition, no association was found between PCOS and 25(OH)D levels after controlling for BMI and race.

Vitamin D has been recognized as an important determinant of health and well-being, with low vitamin D levels linked to a number of chronic disease states. In a cross-sectional survey, Scragg et al. [18] found significantly lower levels of serum 25(OH)D in newly detected cases of diabetes with abnormal impaired glucose tolerance presentation in male and female Polynesian patients in New Zealand, aged 40-64 years. The present study found differences in the HOMA-IR ratio between black and white participants. Whether these differences are linked to the differences noted in 25(OH)D remains to be determined.

Other studies from the Third National Health and Nutrition Examination Survey (1988-1994) involving 6228 participants reported an inverse association between vitamin D status and diabetes, and a possible link to insulin resistance in the non-Hispanic white population and Mexican Americans, but not in the non-Hispanic black population [19]. Further support for the importance of vitamin D in health and disease was noted in a study demonstrating that hyperparathyroidism may be a contributing factor to abnormal glucose metabolism, development of the metabolic syndrome, and the onset of type 2 diabetes [20]. The association of PTH with impaired glucose tolerance is part of a newly proposed mechanism underlying the development of the metabolic syndrome [20]. Other studies have also suggested that individuals with hyperparathyroidism have an increased risk of developing type 2 diabetes [21]. Whether the presence of low vitamin D levels is a contributing factor to the basic pathogenesis of the metabolic syndrome remains to be determined.

Studies examining the relationship between BMI and 25(OH)D levels in black versus white women have at times produced inconsistent findings. Nesby-O'Dell et al. [6], in a large and representative cohort of African American and white women, found that African American women with a BMI of greater than 30 were not more likely to have low vitamin D levels (25[OH]D <37.5 nmol/L) compared with women with a normal BMI; in contrast, obese white women in the same study were more likely to have vitamin D insufficiency than white women of normal weight (OR 3.3). In another study, Egan et al. [22] found an inverse correlation between BMI and 25(OH)D levels in 99 black women aged 40-79 years—a correlation that was not observed in 98 white women evaluated in the same study. The results of the present study demonstrate a marked racial disparity in 25(OH)D levels in the 2 populations of women and a strong and consistent inverse correlation with measures of obesity in very young women. These findings suggest the need to examine the long-term effects of 25(OH)D deficiency on both health and disease in black and white women across the entire adult age spectrum.

It is notable that the results show that the majority of the participants in both racial groups did not have secondary hyperparathyroidism defined by either threshold, despite the observed prevalence of low serum levels of 25(OH)D. However, the median serum level of PTH was shown to be higher in black women than in white women, further reinforcing the conclusion that a racial disparity in vitamin D levels does exist. The clinical importance of

the minor racial differences in PTH levels albeit within the normal range remains to be elucidated.

Finally, the study examined the interrelationship between vitamin D status, BMI and race. Race was a strong determinant for 25(OH)D status, with blood levels suboptimal (deficient or insufficient) in virtually all of the African American participants. The prevalence of a very low serum level (<75 nmol/L) of 25(OH)D was 61% among African American women in contrast to only 7% for white women. 25(OH)D results were also negatively correlated with measures of obesity in black women including BMI, body weight, and percentage body fat. In addition, significant differences were noted in both HDL cholesterol and triglycerides between the 2 populations. These differences may be related to the nutritional status of the 2 populations.

The levels of 25(OH)D were not associated with seasonal changes in serum PTH levels. There was also no association between PCOS and 25(OH)D levels after controlling for BMI and race.

The present study confirms that young and middle-aged women, particularly black women, have a high prevalence of low 25(OH)D levels. This may reflect either a modern form of vitamin D malnutrition, a significant genetic variation in vitamin D synthesis and/or metabolism, or is a result of limited sun exposure in young African American women. If a normal serum level of 25(OH)D is an important determinant of overall health and well-being, there needs to be renewed attention paid to simple measures of enhanced vitamin D supplementation in this population that could prove to be of benefit to long-term health. Clearly, further research is needed to understand the consequences of chronic low 25(OH)D levels in black populations of different ages and its effect on the development of certain chronic and/or immune diseases that might be prevented by vitamin D supplementation.

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Figure 1.
Negative correlation of 25-hydroxyvitamin D levels with body mass index, weight, and percentage body fat.

Table 1Clinical data from 219 healthy female participants aged 18–45 years.^a

Characteristics	Racial group		P value
	Black (n=117)	White (n=102)	
Age, y ^b	27.3 ± 6.5	29.6 ± 6.3	0.009
Site			
Meharry	114 (97.4)	18 (17.6)	<0.001
PSU	4 (3.4)	85 (83.3)	
Diagnosis			
Normal	58 (49.6)	28 (27.4)	<0.001
Leiomyomas	23 (19.7)	17 (16.7)	
PCOS	37 (31.6)	58 (56.9)	
Weight, kg ^b	85.6 ± 24.0 (n=115)	87.8 ± 24.5 (n=101)	0.515
BMI ^b	31.7 ± 8.6 (n=115)	32.4 ± 8.5 (n=101)	0.567
Body fat, % ^b	35.5 ± 8.3 (n=107)	37.9 ± 7.7 (n=98)	0.036
HOMA-IR ratio ^c	2.1 [1.3, 3.3]	1.4 [0.8, 2.3]	<0.001
SHBG, nmol/L ^c	34.7 [24.9, 49.2]	30.4 [18.8, 51.9]	0.339
Cholesterol, mg/dL	170.1 ± 40.1	171.2 ± 34.3 (n=99)	0.822
HDL cholesterol	52.2 ± 13.1	32.7 ± 12.3	<0.001
LDL cholesterol	103.1 ± 35.6	114.7 ± 29.0	0.012
Triglycerides	73.6 ± 41.0	117.3 ± 76.2	<0.001

Abbreviations: PSU, Pennsylvania State University; PCOS, polycystic ovary syndrome; BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); HOMA-IR, homeostasis model of assessment-insulin resistance; SHBG, sex hormone binding globulin; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

^aValues are given as mean ± SD, number (percentage), and median [25th, 75th percentile].

^bTwo-sample *t* test.

^cData were log-transformed for 2-sample *t* test.

Table 2Comparison of 25-hydroxyvitamin D and parathyroid hormone levels.^a

Variables	Racial group		P value
	Black	White	
25 (OH)D, nmol/L ^b	27.3 [22.1, 35.9] (n=118)	52.4 [41.3, 64.9] (n=103)	<0.001
Vitamin D deficiency ^c			
<50 nmol/L (<20 ng/mL)	115 (97.5)	46 (44.7)	<0.001
Vitamin D insufficiency ^c			
<75 nmol/L (<30 ng/mL)	72 (61.0%)	7 (6.8%)	<0.001
PTH, pg/mL ^b	31.9 [20.3, 42.5] (n=92)	22.3 [11.3, 37.5] (n=86)	<0.001
> 65 pg/mL ^c	9 (9.8)	6 (7.0)	0.594
> 46 pg/mL ^c	21 (22.8)	10 (11.6)	0.074

^aValues are given as median [25th, 75th percentile] and number (percentage).^bData were log-transformed for 2-sample *t* test.^cFisher exact test.

Table 3

Comparisons between body mass index and body fat as determined by dual-energy X-ray absorptiometry in study participants.^a

	Black	White	Bonferroni-adjusted model based statistics	P value
	25 (OH)D, nmol/L	25 (OH)D, nmol/L	Ratio of Black:White (95% CI) ^b	
BMI				
<25	32.1 [24.1, 39.7]	59.3 [47.5, 69.1]	0.54 (0.43-0.67)	<0.001
>25-<30	27.1 [22.0, 32.9]	54.8 [40.8, 64.2]	0.54 (0.41-0.70)	<0.001
>30-<40	26.3 [20.6, 32.6]	51.6 [45.2, 59.5]	0.48 (0.40-0.58)	<0.001
>40-60	24.7 [21.2, 32.2]	37.0 [32.7, 55.6]	0.64 (0.49-0.83)	<0.001
% body fat				
<30	31.6 [23.2, 40.3]	58.1 [49.7, 71.6]	0.50 (0.39-0.64)	<0.001
>30	27.1 [21.8, 34.2]	50.4 [40.3, 63.3]	0.54 (0.48-0.61)	<0.001

^aValues are given as median [25th, 75th percentile] unless otherwise indicated.

^bANOVA model fit to log-transformed 25 (OH)D. Back transformation from the log scale results in ratio of black to white.

Table 4Median serum levels of 25-hydroxyvitamin D and parathyroid hormone by season.^a

Level by season	Black	White	Bonferroni-adjusted model based statistics	P value
Ratio of Black:White (95% CI) ^b				
25(OH)D, nmol/L				
Spring	28.4 [23.7, 37.1]	49.7 [42.6, 64.7]	0.57 (0.45-0.73)	<0.001
Summer	29.2 [24.5, 38.2]	62.7 [48.1, 73.1]	0.51 (0.40-0.65)	<0.001
Autumn	27.2 [22.9, 33.3]	52.0 [40.5, 63.0]	0.57 (0.47-0.69)	<0.001
Winter	24.3 [18.9, 30.0]	48.2 [40.2, 55.4]	0.53 (0.42-0.67)	<0.001
PTH, pg/mL				
Spring	33.2 [19.7, 39.0]	19.5 [9.3, 33.7]	2.01 (0.98-4.12)	0.06
Summer	32.8 [26.2, 46.0]	14.1 [9.6, 26.6]	2.45 (1.36-4.41)	<0.001
Autumn	37.5 [24.1, 46.6]	31.0 [14.6, 39.5]	1.29 (0.78-2.13)	0.80
Winter	25.0 [15.3, 41.4]	24.4 [11.7, 40.1]	1.16 (0.56-2.40)	1.00

Abbreviations: 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone.

^aValues are given as median [25th, 75th percentile] unless otherwise indicated.^bANOVA model fit to log-transformed analytes. Back-transformation from the log scale results in a ratio of black to white.