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Relationship of vitamin D levels to blood pressure in a biethnic population

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Abstract

Background and aims—Accumulating epidemiological and clinical studies have suggested that vitamin D insufficiency may be associated with hypertension. Blacks tend to have lower vitamin D levels than Whites, but it is unclear whether this difference explains the higher blood pressure (BP) observed in Blacks in a population with healthy lifestyle practices.

Methods and results—We examined cross-sectional data in the Adventist Health Study-2 (AHS-2), a cohort of non-smoking, mostly non-drinking men and women following a range of diets from vegan to non-vegetarian. Each participant provided dietary, demographic, lifestyle and medical history data. Measurements of weight, height, waist circumference, percent body fat and blood pressure and fasting blood samples were obtained from a randomly selected non-diabetic sample of 284 Blacks and 284 Whites aged 30–95 years. Multiple regression analyses were used to assess independent relationships between blood pressure and 25(OH)D levels. Levels of 25(OH)D were inversely associated with systolic BP in Whites after control for age, gender, BMI, and use of BP-lowering medications (β -coefficient -0.23 [95% CI, $-0.43, -0.03$; $p = 0.02$]). This relationship was not seen in Blacks (β -coefficient 0.08 [95% CI, $-0.14, 0.30$; $p = 0.4$]). Results were similar when controlling for waist circumference or percentage body fat instead of BMI. No relationship between serum 25(OH)D and diastolic BP was seen.

Conclusion—Systolic BP is inversely associated with 25(OH)D levels in Whites but not in Blacks. Vitamin D may not be a major contributor to the White-Black differential in BP.

Keywords

Serum hydroxyvitamin D; Blood pressure; Diet; Ethnic groups

Introduction

Concern for vitamin D deficiency is growing worldwide [1,2] and researchers have found vitamin D insufficiency or deficiency to be related to hypertension [3–5]. Reduced synthesis of vitamin D or hypovitaminosis D may contribute to the development of hypertension through the inhibition of the renin-angiotensin system [6,7] which compromises vasculature and promotes the pathogenesis of diabetes, metabolic syndrome, obesity and cardiovascular

diseases [8–11]. Further, when serum 25(OH)D falls below 30 ng/ml, secondary hyperparathyroidism can occur which promotes vascular calcification [12]. Vitamin D deficiency is more prevalent among populations with darker skin pigmentation notably in Black individuals who are also well-documented to have higher prevalence of hypertension [13,14]. The Joint National Commission on Prevention, Detection, Evaluation and Treatment (JNC 7) states that Blacks tend to have uncontrolled and complicated hypertension and respond differently to customary management [15]. Knowledge of adequate vitamin D levels for Blacks may contribute to treatment of hypertension in this population.

Vitamin D synthesis is a multi-factorial process affected by many factors in addition to skin pigmentation, including sun exposure and dietary intake of vitamin D rich foods, or supplementation. It is widely known that dietary intake of vitamin D foods or supplementation has less effect on the body's ability to synthesize serum 25(OH)D than cutaneous synthesis [16,17]. A cross-sectional study showed vegan or vegetarian diets may contain suboptimal amounts of vitamin D [18], though another study found that serum 25(OH)D levels were not associated with vegan, lacto-ovo or non-vegetarian status [19].

The relationship of vitamin D with blood pressure (BP) remains conflicting because it is largely based on data with observational designs, particularly pertaining to differences among the Blacks and Whites. We sought to establish whether vitamin D levels of individuals with a wide range of dietary patterns are associated with BP in Blacks or non-Hispanic Whites. Seventh-day Adventists practice a wide range of dietary patterns including vegetarian and are mostly non-drinkers of alcohol and non-smokers. The Adventist Health Study-2 (AHS-2) is a prospective, ongoing epidemiological cohort study of 96,592 adult church members (aged ≥ 30 years) living in United States and Canada. The purpose of this study was to determine the relationship between serum and BP in Whites and Blacks in this population with generally healthy lifestyle habits.

Methods

Design and subjects

This cross-sectional study enrolled randomly selected participants of the Adventist Health Study-2 (AHS-2) cohort who were asked to attend a clinic visit for biological measurements. Recruitment and enrollment methods of this study are described elsewhere [20,21].

Subjects who reported diabetes at baseline or had blood glucose ≥ 126 mg/dl were excluded. The present study consisted of 568 subjects (284 Blacks and 284 Whites) from a calibration study of 939 subjects who were randomly selected from the parent cohort by church, then subjects-within-church with equal representation of Blacks and Whites. We excluded those without serum ($n = 291$), no BP measurement ($n = 2$), and those diagnosed with diabetes mellitus type 2 ($n = 78$). “Blacks” indicated self-reported Black/African Americans or West Indian/Caribbean ($n = 26$, 4.6%) or other Black ethnicities, whereas “Whites” in this study included non-Hispanic Whites. The study was approved by the Loma Linda University Human Subjects Committee Institutional Review Board.

Materials and procedure

Each participant completed a 50-page written questionnaire regarding demographics, lifestyle, and medical illnesses. From November 2003 to May 2007 subjects provided fasting blood samples and anthropometric measurements including height and weight, waist circumference and percent body fat using the Tanita scale (Tanita Corp, Arlington Heights, IL). Dietary intake was assessed using a validated food frequency questionnaire (FFQ) that was completed within 1–3 months of blood sample collection [20]. In addition, information

regarding use of BP-lowering medications was obtained. All pharmacological treatment of hypertension was registered and a cardiologist designated medications known to reduce BP.

BP was measured using an Omron (Omron Healthcare, Inc. Bannockburn, IL) automated sphygmomanometer [22] after subjects were seated in a quiet room at a comfortable temperature for approximately 10 min. Readings were taken three times, 5 min apart, and the mean of the second and third readings was used except when there was a difference of 5 mmHg in the three readings, in which case, the mean of all three readings was used.

Laboratory methods

After fasting blood was drawn, plasma and cells were separated immediately by centrifuge and transported on frozen gel packs within 30 h of sample collection, and stored in liquid nitrogen. Laboratory assays for serum 25(OH)D were done at the Reproductive Endocrine Research Laboratory, Department of Obstetrics and Gynecology, University of Southern California Keck School of Medicine batch-wise in August 2005, September 2005 and March 2007 (total $n = 408$) and November 2010 ($n = 160$) using DiaSorin radio-immunoassays (DiaSorin, Stillwater, MN). The intra-assay and inter-assay coefficient variations (CVs) were 10% and 16%, respectively. The lowest level detected was 5.45 ng/ml and highest at 73.80 ng/ml. Quality control (QC) samples showed mean levels of 14.2 ng/ml for low (QC 9.3 ng/ml–19.7 ng/ml) and 51 ng/ml for high (QC 32.5 ng/ml–68.9 ng/ml) levels. All samples were in this range with coefficients of variation of 3.37% and 7.63% for low and high levels respectively. To ensure consistency between the batches, 20 specimens from previous batches were reanalyzed together with the latest November 2010 batch and showed intra-class correlation coefficient (ICC) = 0.75 and Pearson's $r = 0.85$. No subjects had unmeasurable levels. Serum 25(OH)D levels were classified as follows: deficient (< 20 ng/ml), insufficient (21–29 ng/ml), and sufficient (≥ 30 ng/ml) [23,24].

Lifestyle measures

Diet: Dietary status was categorized as vegan (animal products including red meat, poultry, fish, eggs, milk and dairy products consumed < 1 time/month); lacto-ovo vegetarian (dairy products and/or eggs consumed ≥ 1 time/month but red meat, poultry and fish consumed < 1 time/month); pesco vegetarians (fish consumed ≥ 1 time/month and dairy products and/or eggs but red meat and poultry consumed < 1 time/month); semi-vegetarians (dairy products and/or eggs and red meat and poultry consumed ≥ 1 time/month but < 1 time/week); while non-vegetarians consumed animal products (red meat, poultry, fish, eggs, milk and dairy products > 1 time/week). Dietary vitamin D intake was adjusted for energy intake using the residual method [25]. The total dietary vitamin D intake was calculated as the sum of the energy-adjusted dietary vitamin D intake and supplemental intake.

Physical activity: Participants were categorized as no regular exercise, low exercise (vigorous exercise < 105 min/week or jogging/running < 105 min/week or < 3 miles/week), moderate exercise (vigorous exercise 105–175 min/week or jogging/running 105–175 min/week or 3–9 miles/week) and high exercise level if activities exceeded the moderate level.

UV season

The seasons of the year when blood was drawn were categorized according to months that had similar patterns of noontime erythral radiation strength. Noon erythral doses are varied and highly dependent on number of hours and percentage of body parts exposed to the sun, latitude, longitude, presence and amount of cloud and ozone or real-time atmospheric conditions [26–28]. Measurements of the UV season and sun exposure factors in this study were discussed in our previous study [29].

Statistical analyses

For continuous variables, distributions were reviewed for normality and outliers checked by scatterplot. Simple and multiple correlational analyses were performed to determine relationships between variables. Independent *t*-tests or Mann–Whitney tests were performed to determine differences in participants' characteristics for continuous variables according to the distribution of the data, and chi-square was used for categorical variables. Missing values for variables being tested were not substituted.

Multiple regression analyses were conducted to evaluate relationships between SBP or DBP and serum 25(OH)D levels. The regression model included serum 25(OH)D levels, age, gender, ethnicity, and use of BP medications as independent variables and either SBP or DBP was used as the dependent variable. When a cross-product interaction term between ethnicity and serum 25(OH)D levels was added into the model, it showed statistically significant, or near statistically significant *p*-values (0.05–0.09), so analysis was stratified by ethnicity. In the ethnicity-stratified model, BMI, waist circumference, and body fat percentage were sequentially added. We further explored controlling for dietary patterns, vitamin D intake and supplementation, and UV season and other demographic variables including education, marital status, exercise, smoking and alcohol use. None of these variables contributed importantly to the variance in serum 25(OH)D levels (data not shown). All statistical analyses were performed using SAS statistical software (SAS, version 9.2, SAS Institute Inc., Cary, NC) and SPSS version 17, Chicago, IL. Using the G-power 3.2.1 computerized software for multiple regression (post hoc, two-tailed test), with power of 0.80 and α of 0.05 and effect size of 0.15 (medium effect), a sample size of 284 per group attained 99% actual power.

Results

Whites and Blacks were equally represented ($n=284$ each) (Table 1). The mean age and mean BMI were ~60 years and 27.4 kg/m² respectively. BMI and percent body fat were higher among Blacks than Whites. Systolic and diastolic BP and pulse rate were higher among Blacks than Whites, but mean age was 5.5 years lower in Blacks. Serum 25(OH)D levels were higher among Whites than Blacks and Blacks had a higher prevalence of vitamin D deficiency individuals than Whites. The prevalence of vitamin D deficiency among Blacks was 55% versus 15% among Whites. The proportion of cigarette smokers in Blacks was 1.4%, and in Whites, 0.7%; of Blacks, 5.5% reported current use of alcohol, while among Whites, 3.2% reported current use of alcohol. Report of physical activity in Blacks was 81%, consisting of 24.3%, 34.5%, and 22% for low, moderate and vigorous levels respectively. Among Whites, 79% were active at 20.3% low, 34.4% moderate and 24.0% vigorous levels. Dietary patterns differed in that Blacks had higher proportion of nonvegetarians (46%) versus Whites (31%). Blacks consumed more of meat products while Whites consumed more dairy products.

Mean BPs for both Whites and Blacks across increasing serum 25(OH)D classification group adjusted for age, BMI, and use of BP medications are shown in Table 2. Whites had somewhat lower systolic and diastolic BP than Blacks. In Whites, those with deficient levels of vitamin D had higher SBP compared to those who had sufficient levels, in contrast to findings in Blacks. In the absence of important associations between DBP and serum levels, further analyses focused on SBP.

The regression model for Whites (Table 3) showed an inverse relationship between 25(OH)D levels and SBP across all three sequential models. Adjustment for season of blood draw or calcium intake did not change the results (data not shown). The regression models for Blacks (Table 4) showed non-significant relationships between 25(OH)D levels and SBP

across the three models. Age, use of BP medications, BMI, waist circumference, and percent body fat were significantly related with SBP but being male versus female showed a significant relationship only when percent body fat was included in model three. For all subjects serum 25(OH)D was not significantly related to diastolic BP and no interaction with ethnicity was seen (Table 5). Among Whites, we repeated the multiple regression among subjects who were not treated for hypertension and found the same tendency for an inverse relationship between serum 25(OH)D and SBP as observed in the whole group, while for those who were treated for hypertension we did not find a significant association (data not shown), however, power may be an issue as we are selecting the high end of a normal distribution by doing this analysis. In Blacks, there were no significant relationships between serum 25(OH)D and SBP in those treated and not treated for hypertension consistent with results for the whole group (data not shown).

Discussion

Findings from this study showed that in a population with generally healthy lifestyles, there was an inverse relationship between serum vitamin D levels and systolic BP in Whites but not in Blacks after controlling for age, gender, use of BP meds, and BMI, waist circumference, or percentage body fat. There were no significant associations between DBP and serum 25(OH)D levels. While the adjusted mean systolic BP difference of 2.5 mmHg between Whites with sufficient versus deficient levels may seem small, the public health benefit of adequate serum 25(OH)D levels may be large because such small differences are associated with 10–15% reductions in cardiovascular mortality [5]. We controlled for waist circumference and percentage body fat in addition to controlling for BMI in separate models, but this did not change the results. Dietary patterns differed between Blacks and Whites in that Blacks tended to be more omnivorous than Whites which may have explained their significant differences in serum 25(OH)D levels. However vegetarianism, as in a similar study in this population [19], did not show association with vitamin D levels.

The inverse association between serum 25(OH)D and systolic BP in Whites is consistent with findings of Judd et al., [4], and Scragg et al., [5]. Judd et al., reported that among non-hypertensive Whites, vitamin D sufficiency attenuated the predicted age-related rise in systolic BP by 20% after adjustment for age, sex, BMI, and physical activity. The majority of participants in the survey (NHANES data) were <50 years of age and 92% of Blacks had 25(OH)D levels <32 ng/ml. In another study with the same NHANES III participants, Scragg, et al. reported that Blacks had the lowest mean serum levels (19.6 ng/ml) versus that of Whites (31.6 ng/ml). Our study showed a similar inverse relationship between vitamin D levels and systolic BP among Whites despite our subjects being older than those taking part in the NHANES survey. Miljkovic et al., [30], reported a low prevalence of vitamin D deficiency (2.8%) among older Afro-Caribbean subjects while our study showed seven subjects or 26% had deficient levels, a proportion lower than seen in non-Afro Caribbean Blacks. We also included hypertensives and non-hypertensives, but adjusted for use of anti-hypertensive medications.

Scragg et al., [5], found an inverse relationship between 25(OH)D and systolic BP both in Whites and Blacks. This group reported a difference of 3.0 mmHg in systolic BP and 1.6 mmHg in diastolic BP in the highest versus the lowest quintiles of 25(OH)D when all ethnicities were included. The age adjusted mean difference of 3.5 mmHg in systolic BP seen among Blacks versus Whites in the Scragg et al., study is quite similar to the adjusted mean difference of 4.0 mm Hg systolic BP we noted among Blacks versus Whites in those with insufficient or deficient levels (Table 2). Scragg et al found that vitamin D insufficiency explained 50% of the higher prevalence of hypertension among Blacks than in Whites. In contrast, in the current study, Blacks with deficient vitamin D levels had lower

systolic BP than Blacks with sufficient vitamin D levels, though the difference was not statistically significant. While mean 25(OH)D levels were similar in the current study (20.6 ng/ml) compared to the study by Scragg et al (19.6 ng/ml), other differences in study participants may explain the diverging results.

Another recent study Fiscella et al., [31], attributed 26% of the Black-White disparity in systolic BP to differences in vitamin D levels. This study was also based on NHANES data (2001–2006) and included a large number of participants ($n=7,140$). However, participant characteristics in that study contrasted with those in the current study. In the NHANES database there were higher percentages of individuals with cardiovascular risk factors such as cigarette smoking and obesity. For example, smoking rates among White individuals were 26.2% versus 0.7% in the current study. Furthermore, 30.2% were obese compared to 23.6% in the current study. We also found that the proportion of Whites with sufficient 25(OH)D levels was 25.5% versus 46.4% in the current study. It is possible that the health characteristics of the current study's population explain this difference.

As in other studies that utilized NHANES data [4,5], we found that the proportion of Blacks with sufficient vitamin D levels was low (16.5%). There is little high quality evidence that low vitamin D concentrations in Blacks have negative consequences with respect to cardiovascular risks or diseases. In a cross-sectional study Reis et al. [32] found that about one-third of the increased risk of peripheral arterial disease (PAD) among Blacks was due to racial differences in vitamin D status. However, randomized controlled clinical trial data is sparse. The Women Health Initiative included only 9% Blacks and results of this study showed no effect of calcium with vitamin D supplementation on incident coronary heart disease, or stroke or other CVD risks among 36,282 postmenopausal women after 7 years of follow-up [33].

Strengths and limitations

One of the main strengths is that study participants were predominantly non-smokers, non-alcohol drinkers and mostly engaged in regular physical activity. This condition eliminated several of the common confounders in population based research. In addition, our participants provided a wide range of dietary patterns from pure vegan to omnivore. More importantly, we had a wide range of serum 25(OH)D levels (5–73 ng/ml) in equally represented Black and White individuals. We used multivariable analyses to account for covariates known to affect vitamin D levels. However, our study was cross-sectional which limits our ability to determine causality. We also did not measure serum calcium, phosphorus and parathyroid hormone concentrations to study their possible mediation between vitamin D concentrations and blood pressure.

Conclusion

This study showed an inverse relationship between serum and systolic BP in non-diabetic Whites but not in Blacks after adjustment for age, gender, use of BP-lowering medications, and BMI, waist circumference or percentage body fat. In this rather healthy population, vitamin D disparities did not seem to explain disparities in BP between Blacks and Whites.

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Table 1Means $\pm$ SD and proportions of selected baseline characteristics stratified by ethnicities.

	White (<i>n</i> = 284)	Black (<i>n</i> = 284)	<i>a</i> <i>p</i> -value
Age, mean $\pm$ SD	62.4 (13.8)	56.9 (13.4)	<0.0001
BMI, mean $\pm$ SD	26.6 (5.3)	28.1 (5.8)	0.002
Normal, <i>n</i> (%)	124 (43.8)	91 (32.0)	0.014
Overweight, <i>n</i> (%)	92 (32.5)	107 (37.6)	
Obese, <i>n</i> (%)	67 (23.6)	86 (30.2)	
Waist circumference (cm), mean $\pm$ SD	94.1 (17.9)	93.3 (14.3)	0.571
Percent body fat, mean $\pm$ SD	31.0 (9.4)	33.9 (10.2)	0.001
Female, <i>n</i> (%)	142 (50.0)	156 (54.9)	0.240
Systolic BP (mmHg), mean $\pm$ SD	124.1 (18.8)	127.8 (20.9)	0.027
Normal, <i>n</i> (%)	133 (46.8)	112 (39.4)	0.205
Pre-hypertension, <i>n</i> (%)	95 (33.4)	109 (38.3)	
Hypertension, <i>n</i> (%)	56 (19.7)	63 (22.1)	
Diastolic BP (mmHg)	74.7 (9.3)	78.9 (11.0)	<0.0001
Normal, <i>n</i> (%)	204 (71.8)	160 (56.3)	<0.0001
Pre-hypertension, <i>n</i> (%)	63 (22.1)	75 (26.4)	
Hypertension, <i>n</i> (%)	17 (5.9)	49 (17.2)	
Pulse rate, mean $\pm$ SD	64 (9)	67 (10)	<0.0001
Subjects with hypertension, <i>n</i> (%)	112 (39.4)	151 (53.1)	0.001
BP medication (% among hypertensives)	62 (55.3)	78 (51.6)	0.119
Serum 25(OH)D (ng/ml), mean $\pm$ SD	29.91 (9.98)	20.57 (10.35)	<0.0001
Deficiency (<20 ng/ml), <i>n</i> (%)	42 (14.7)	156 (54.9)	<0.0001
Insufficiency (20–30 ng/ml), <i>n</i> (%)	110 (38.7)	81 (28.5)	
Sufficiency (>30 ng/ml), <i>n</i> (%)	132 (46.4)	47 (16.5)	
UV season blood drawn, <i>n</i> (%)			
Spring (Nov–Mar)	87 (30.6)	59 (20.7)	<0.0001
Summer (Apr–Aug)	98 (34.5)	148 (52.1)	
Fall (Sep–Oct)	99 (34.8)	77 (27.1)	
Vitamin D intake (mcg/day)			

	White (<i>n</i> = 284)		Black (<i>n</i> = 284)		^a <i>p</i> -value
Total intake (energy-adjusted), mean ± SD	8.2	(7.3)	8.8	(8.3)	0.661
Dietary (energy-adjusted), mean ± SD	2.8	(2.4)	3.0	(2.8)	0.460
Supplemental, mean ± SD	5.3	(6.8)	5.8	(8.1)	0.787
No. missing	8		22		
Calcium intake (mg/day)					
Calcium total (energy-adjusted), mean ± SD	1251.2	542.8	1074.8	527.9	<0.0001
Total intake (energy-adjusted), mean ± SD	852.8	265.6	802.6	278.2	0.008
Supplemental, mean ± SD	398.3	464.8	272.1	426.3	0.002
Dietary pattern, <i>n</i> (%)					
Vegan	25	(9.0)	25	(9.2)	<0.0001
Lacto-ovo	118	(42.6)	49	(18.1)	
Semi/pesco	49	(17.6)	71	(26.3)	
Non-vegetarian	85	(30.6)	125	(46.3)	
No. missing	7		14		

^a *p*-values compare Black and White subjects.

Table 2Mean systolic and diastolic BP by serum categories^{*,a}.

	<u>Systolic BP (mmHg)</u>		<u>Diastolic BP (mmHg)</u>	
	Mean	*SE	Mean	*SE
Whites				
Deficiency (<20)	124.2	2.7	77.0	1.5
Insufficiency (20–30)	124.6	1.6	75.1	0.9
Sufficiency (>30)	121.7	1.5	74.7	0.8
<i>p</i> -value ^a	0.0259		0.1098	
Blacks				
Deficiency (<20)	128.2	1.4	79.6	0.8
Insufficiency (20–30)	128.8	1.9	77.0	1.0
Sufficiency (>30)	131.6	2.5	77.6	1.4
<i>p</i> -value ^a	0.6242		0.0973	

* Calculated SE reflects use of least square means.

^a Adjusted for age, BMI, and BP medication.

Table 3

Relationship models for serum 25(OH)D and systolic BP for Whites.

Variable	Model 1: Adjusting for age, gender, BP meds, and BMI				Model 2: Adjusting for age, gender, BP meds, and waist				Model 3: Adjusting for age, gender, BP meds, and %body fat			
	Beta coefficient	95% CI		p-value	Beta coefficient	95% CI		p-value	Beta coefficient	95% CI		p-value
Intercept	129.20	122.64	135.77	<0.0001	129.42	122.86	135.97	<0.0001	127.33	120.41	134.24	<0.0001
Serum 25(OH)D	-0.23	-0.43	-0.03	0.024	-0.24	-0.43	-0.04	0.019	-0.25	-0.44	-0.05	0.015
Age ^a	0.51	0.37	0.65	<0.0001	0.50	0.36	0.64	<0.0001	0.49	0.35	0.63	<0.0001
Gender												
Female	Reference				Reference				Reference			
Male	-1.08	-4.94	2.78	0.582	-2.13	-6.04	1.78	0.284	3.39	-1.85	8.63	0.204
BP medication	7.43	2.58	12.29	0.003	7.12	2.16	12.08	0.005	7.11	2.08	12.14	0.006
BMI ^a	0.43	0.06	0.81	0.022								
Waist circumference ^a					0.12	0.00	0.23	0.050				
Percent body fat ^a									0.31	0.03	0.59	0.028

^aCentered at its mean.

Table 4

Relationship models for serum 25(OH)D and systolic BP for Blacks.

Variable	Model 1: Adjusting for age, gender, BP meds, and BMI				Model 2: Adjusting for age, gender, BP meds, and waist				Model 3: Adjusting for age, gender, BP meds, and %body fat			
	Beta coefficient		95% CI		Beta coefficient		95% CI		Beta coefficient		95% CI	
Intercept	125.05		119.29	130.80	<0.0001	126.18	120.50	131.86	<0.0001	121.63	115.14	128.12
Serum 25(OH)D	0.08		-0.14	0.30	0.464	0.09	-0.13	0.31	0.419	0.08	-0.15	0.30
Age ^a	0.60		0.43	0.78	<0.0001	0.56	0.39	0.73	<0.0001	0.54	0.37	0.71
Gender												
Female	Reference					Reference				Reference		
Male	3.31		-1.10	7.73	0.141	0.56	-3.92	5.03	0.81	9.25	3.22	15.29
BP medication	5.81		0.46	11.17	0.034	6.02	0.74	11.30	0.026	7.57	2.20	12.94
BMI ^a	0.71		0.32	1.10	<0.000							
Waist circumference ^a						0.33	0.17	0.49	<0.0001			
Percent body fat ^a										0.51	0.21	0.80
												0.0018

^aCentered at its mean.

Table 5

Relationship models for serum 25(OH)D and diastolic blood pressure for all subjects.

Model 1: Adjusting for age, gender, BP meds, and BMI					Model 2: Adjusting for age, gender, BP meds, and waist fat					Model 3: Adjusting for age, gender, BP meds, and %body fat					
R-square: 0.14					R-square: 0.12					R-square: 0.14					
Variable	Beta coefficient	95% Conf interval			p-value	Beta coefficient	95% Conf interval			p-value	Beta coefficient	95% Conf interval			p-value
		Lower	Upper				Lower	Upper				Lower	Upper		
Intercept	76.58	72.81	80.35	<0.0001	76.73	72.92	80.53	<0.0001	75.19	71.29	79.10	<0.0001			
Serum 25(OH)D	-0.08	-0.19	0.04	0.190	-0.08	-0.20	0.03	0.160	-0.11	-0.23	0.01	0.0677			
Race															
White	Reference				Reference				Reference						
Black	2.71	-1.76	7.18	0.235	3.67	-0.85	8.19	0.112	1.94	-2.58	6.47	0.390			
Race × 25(OH)D interaction															
White	Reference														
Black	0.00	-0.16	0.16	0.969	-0.01	-0.18	0.15	0.856	0.01	-0.15	0.18	0.869			
Age ^a	0.01	-0.05	0.07	0.799	-0.01	-0.07	0.05	0.680	-0.03	-0.09	0.04	0.425			
Gender															
Female	Reference				Reference				Reference						
Male	1.59	-0.04	3.22	0.056	0.23	-1.44	1.91	0.786	5.79	3.57	8.00	<0.0001			
BP medication	1.88	-0.13	3.89	0.067	1.95	-0.09	3.99	0.062	2.28	0.24	4.32	0.029			
BMI ^a	0.51	0.36	0.66	<0.0001											
Waist circumference ^a					0.15	0.10	0.20	<0.0001							
Percent body fat ^a									0.37	0.25	0.48	<0.0001			

^aCentered at its mean.