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# Demographic, dietary, and serum factors and parathyroid hormone in the National Health and Nutrition Examination Survey

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## Abstract

**Summary—**Many determinants of parathyroid hormone (PTH) are unknown. In the National Health and Nutrition Examination Survey (NHANES), numerous factors not classically associated with calcium–phosphorus homeostasis, such as uric acid and smoking, are independently associated with PTH in adults without chronic kidney disease. Associations between serum phosphorus and PTH may vary by race.

**Introduction—**Although PTH may be an important biomarker for osteoporosis and cardiovascular disease, many determinants of PTH are unknown. We investigated associations between demographic, dietary, and serum factors and PTH level.

**Methods—**We studied 4,026 white, 1,792 black, and 1,834 Mexican-American adult participants without chronic kidney disease from the 2003–2004 and 2005–2006 NHANES.

**Results—**The mean serum PTH level was 38.3 pg/ml for whites, 42.6 pg/ml for blacks, and 41.3 pg/ml for Mexican-Americans. After adjusting for diet, body mass index, serum levels of calcium, phosphorus, 25-hydroxyvitamin D, creatinine, and other factors, smokers compared to non-smokers had lower PTH, ranging from −4.2 pg/ml (95% confidence interval (CI) −7.3 to −1.1) in Mexican-Americans to −6.1 pg/ml (95% CI −8.7 to −3.5) in blacks. After multivariate adjustment, PTH was higher in females compared to males, ranging from 1.1 pg/ml (95% CI −1.2 to 3.4) in Mexican-Americans to 4.5 pg/ml (95% CI 1.9 to 7.0) in blacks, and in older (>60 years) compared to younger participants (<30 years), ranging from 3.7 pg/ml (95% CI 1.3 to 6.1) in Mexican-Americans to 8.0 pg/ml (95% CI 5.4 to 10.7) in blacks. Higher uric acid was associated with



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**Conflicts of interest:** None.

higher PTH. In whites only, lower serum phosphorus and lower serum retinol were associated with higher PTH.

**Conclusions—**Numerous factors not classically associated with calcium–phosphorus homeostasis are independently associated with PTH and should be considered in future studies of PTH and chronic disease. Additional research is needed to elucidate mechanisms underlying identified associations with PTH and to explore possible racial differences in phosphorus handling.

### Keywords

CDC; Centers for Disease Control and Prevention; National Health and Nutrition Examination Survey; NHANES; Parathyroid hormone

## Introduction

The central role of parathyroid hormone (PTH) in calcium and phosphorus homeostasis, through effects on the bone, kidney, and intestines, is well recognized. However, a growing body of research suggests that PTH also is involved in the development of a variety of chronic diseases. Higher PTH has been implicated in hypertension [1, 2], coronary artery disease [3], heart failure [4], cardiovascular mortality [5], and poorer bone health [6, 7]. Hagström et al. found in a community-based prospective cohort study of 958 elderly men that higher PTH, even within the normal range, was associated with a 38% greater risk for cardiovascular mortality [5].

Although the effects of dietary and serum calcium, short-term phosphorus intake, and serum 25-hydroxyvitamin D (25(OH)D) on PTH have been well studied, less is known about other factors that impact PTH. In a recent study of 1,288 predominantly pre-menopausal young white women without chronic kidney disease from the Nurses' Health Study II [8], we found that after controlling for 25-hydroxyvitamin D level, calcium intake, and other factors, intact PTH was lower among current smokers, women with higher vitamin A intake, and women with higher plasma phosphorus level. In addition, although no participants with body mass index (BMI) ≥30 kg/$m^2$ were included in the study population, higher PTH was independently associated with BMI. Of note, we were able to account for only 12% of the variation in PTH in this study population.

Additional factors also may impact PTH levels. Prior research suggests that PTH varies by race [9–13], gender [14], age [15–17], and menopause [14, 18]. However, previous studies reporting associations between age and PTH and between gender and PTH were small [16, 17, 19, 20] or did not control for variables such as serum 25(OH)D [15, 21, 22]. In vitro studies found that retinol directly suppresses PTH secretion in parathyroid cells [23, 24], but no studies to date have examined the association between retinol and PTH in a human study population. In rats, uric acid suppresses the synthesis of dihydroxyvitamin D (1,25 $[OH]_2D$) [25]. Thus, uric acid also may affect PTH level.

We examined the cross-sectional associations between demographic, lifestyle, dietary, and serum factors and intact PTH in 4,026 white, 1,792 African-American, and 1,834 Mexican-American adult participants without chronic kidney disease in the 2003–2004 and 2005–2006 National Health and Nutrition Examination Surveys (NHANES). NHANES allowed us to expand our previous analyses of factors influencing PTH [8] to a larger sample size, to include non-whites, men, postmenopausal women, and a large number of smokers, and to study broader ranges of age and BMI. In addition, NHANES includes serum retinol and uric acid, which could be related to PTH.

# Methods

## Study population

We studied adult participants in NHANES 2003–2004 and 2005–2006. The design and operation of NHANES have been described previously (data downloaded from http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/nhanes03_04.htm and http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/nhanes05_06.htm) [26]. NHANES provides nationally representative cross-sectional data on the health status of the civilian, non-institutionalized US population. Participants, who are selected using a complex, multistage sampling design, are interviewed and examined. Participants over the age of 12 years without hemophilia or recent cancer chemotherapy were eligible for measurement of a biochemistry profile. Written informed consent was obtained from all adult participants. Current smokers were defined as participants who were currently smoking and had smoked ≥100 cigarettes in their life.

## Serum measurements

Serum PTH was measured by an electrochemiluminescence immunoassay on the Elecsys 1010 autoanalyzer (Roche Diagnostics, Indianapolis, IN). The coefficient of variation ranged between 2.3–4.8% and 2.5–5.3%. Serum 25(OH)D was measured using a Diasorin (formerly Incstar) 25(OH)D assay (Stillwater, MN). To account for drift in the 25(OH)D assay from NHANES 2003–2004 to 2005–2006, adjusted 25(OH)D levels were used in our analysis (http://www.cdc.gov/nchs/data/nhanes/nhanes3/VitaminD_analyticnote.pdf) [27]. Serum creatinine, total calcium, phosphorus, and uric acid were measured with a Beckman Synchron analyzer (Brea, CA). Serum retinol was measured using high performance liquid chromatography (Craft Technologies, Wilson, NC). Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease Study equation [28].

## Ascertainment of dietary intake

A dietary interview was used to estimate the daily intake of various dietary factors, including calcium, phosphorus, magnesium, and protein, on the basis of foods and beverages that participants consumed during the 24-h period preceding the interview (midnight to midnight).

## Ascertainment of demographic variables

Questionnaire information provided the participants' gender, age, race, and ethnicity. Race–ethnicity was predefined as non-Hispanic white, non-Hispanic black, Mexican-American, other Hispanic, and other race. We excluded participants who self-identified as other Hispanic or other race. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Season of blood draw was divided into 6-month blocks, either winter (November–April) or summer (May–October).

## Statistical analyses

Since older individuals, Mexican-Americans, and blacks were intentionally overrepresented in NHANES, appropriate sample weights were used to obtain weighted regression estimates so that the final results would be applicable to the general US population.

Our analyses were cross-sectional. We excluded adults with an eGFR <60 ml/min/1.73 $m^2$. To remove extreme values, we excluded participants in the top 5% of the distribution for intact PTH and serum calcium level greater than 10.3 mg/dl. The final range for intact PTH was 6 to 81 pg/ml. Because associations with PTH were similar in men and women, we combined both sexes in the multivariate analyses. We divided age into decades and BMI

into five categories (<21, 21–24, 25–29, 30–34, and ≥35 kg/m$^2$). Serum 25(OH)D was divided into race-specific quartiles due to the lack of overlap in 25(OH)D distributions across the three race categories (non-Hispanic whites, non-Hispanic blacks, and Mexican-Americans). To facilitate comparisons across races, quartiles were obtained for the other dietary and serum variables in non-Hispanic whites, and these cut points were used to demarcate the dietary and serum variable categories in the other two races (non-Hispanic blacks and Mexican-Americans). Smoking status was divided into never, past, and current smokers.

We created multivariable linear regression models with intact PTH as the dependent variable for each of the three race categories (non-Hispanic whites, non-Hispanic blacks, and Mexican-Americans). Multivariable linear regression models with intact PTH as the dependent variable included the following potential confounders plus factors with univariate associations: age; gender; BMI; physical activity; smoking status; intakes of calcium, protein, magnesium, alcohol, and caffeine; season of blood draw; serum levels of 25(OH)D, calcium, phosphorus, creatinine, uric acid, and retinol; and fasting status (defined as blood draw >9 h since last meal). The following factors were examined in women only: estrogen use, progesterone use, bisphosphonate use, birth control use, and menopause. Variables were excluded from the final multivariate model if they were not associated with the outcome or were not confounders (change in regression coefficient <10%). Due to high correlations between dietary calcium and dietary phosphorus, we did not include dietary phosphorus in the final multivariate models. Tests of trend were calculated for the categorical variables. We also performed stratified analysis by gender. We adjusted for gender in analyses that included both men and women.

Data analysis was performed using SAS version 9.1 (SAS Institute, Cary, NC, USA) and SUDAAN version 9.0 (Research Triangle Park, NC, USA). Protocols to recruit and study participants of NHANES 2003–2004 and NHANES 2005–2006 were reviewed and approved by the National Center for Health Statistics Institutional Review Board.

## Results

### Study population characteristics

The characteristics of the study population (*N*=7,652) are shown in Table 1. The study population included 4,026 non-Hispanic whites, 1,792 non-Hispanic blacks, and 1,834 Mexican-Americans. The mean serum PTH level was 38.3 pg/ml for whites, 42.6 pg/ml for blacks, and 41.3 pg/ml for Mexican-Americans. The mean serum 25 (OH)D level was 26.1 ng/ml for whites, 15.1 ng/ml for blacks, and 19.9 ng/ml for Mexican-Americans. Twenty-seven percent of whites, 26.6% of blacks, and 22.9% of Mexican-Americans were current smokers.

### Associations with intact parathyroid hormone levels

The univariate associations between age, BMI, dietary and serum variables, and PTH are listed in Table 2. Tables 3, 4, and 5 display multivariate-adjusted differences in intact PTH levels for non-Hispanic whites, non-Hispanic blacks, and Mexican-Americans. Because associations with PTH were similar in each gender, we combined men and women into a single population for each racial/ethnic group. The final multivariable regression model included the following variables: age, gender, BMI, smoking status, dietary calcium intake, dietary protein intake, season of blood draw, serum 25(OH)D, serum calcium, serum phosphorus, serum creatinine, serum uric acid, serum retinol, and fasting status.

After multivariate adjustment, the PTH level was lower in current smokers compared to non-smokers. Compared to non-smokers, current white smokers had a 5.3 pg/ml lower PTH

level (95% confidence interval (CI) −6.5 to −4.2), black smokers had a 6.1 pg/ml lower PTH level (95% CI −8.7 to −3.5), and Mexican-American smokers had a 4.2 pg/ml lower PTH level (95% CI −7.3 to −1.1).

Age and female gender were both positively associated with PTH level. After controlling for creatinine, serum 25 (OH)D level, and other factors, age >60 years compared to age <30 years was associated with a 7.0 pg/ml higher PTH level (95% CI 5.5 to 8.4) in whites, 8.0 pg/ml higher PTH level (95% CI 5.4 to 10.7) in blacks, and 3.7 pg/ml higher PTH level (95% CI 1.3 to 6.1) in Mexican-Americans. In analyses restricted to pre-menopausal women, older age was still associated with higher PTH level. Women had a higher PTH level compared to men: white females had a 2.2 pg/ml higher PTH level (95% CI 0.6 to 3.9), black females had a 4.5 pg/ml higher PTH level (95% CI 1.9 to 7.0), and Mexican-American females had a 1.1 pg/ml higher PTH level (95% CI −1.2 to 3.4) compared to their male counterparts.

We also found that higher serum uric acid was associated with higher PTH level. Compared to the lowest category of serum uric acid level, the highest uric acid level category had a 4.9 pg/ml higher PTH level (95% CI 2.7 to 7.0) in whites, 5.5 pg/ml higher PTH level (95% CI 3.0 to 8.1) in blacks, and 4.8 pg/ml higher PTH level (95% CI 1.5 to 8.1) in Mexican-Americans.

We observed differences in several associations by race. In whites, the highest compared to lowest quartile of serum retinol was associated with a 1.6 pg/ml lower PTH level (95% CI −3.4 to 0.2), and the highest compared to lowest quartile of serum phosphorus was associated with a 2.3 pg/ml lower PTH level (95% CI −4.0 to −0.6). No association was found between these factors and PTH level in blacks or Mexican-Americans. The interaction terms by race (white vs. black) for the association between serum phosphorus and PTH were statistically non-significant ($p$=0.18).

For the association between BMI and PTH, Mexican-Americans with BMI ≥35 kg/m$^2$ had a 3.5 pg/ml higher PTH level (95% CI 0.1 to 6.8) compared to Mexican-Americans with BMI 21–24 kg/m$^2$. Although the comparable associations in blacks and whites were statistically insignificant, the $p$ values for tests of trend for BMI were ≤0.05 in all three races ($p$=0.05 in whites, $p$=0.05 in blacks, and $p$=0.01 in Mexican-Americans). There were a limited number of participants with BMI >40 kg/m$^2$ (169 whites, 158 blacks, and 70 Mexican-Americans, 397 in total).

Dietary calcium intake, serum calcium, and serum 25 (OH)D were inversely associated with PTH level across all races. We found no significant associations between PTH and phosphorus intake, protein intake, magnesium intake, alcohol intake, caffeine consumption, physical activity, and season of blood draw. The following factors were examined in women only and no significant associations with PTH were found: estrogen use, progesterone use, bisphosphonate use, birth control use, and menopause.

## Discussion

In NHANES 2003–2004 and 2005–2006, we identified numerous factors that were independently associated with intact PTH levels in adult men and women of different races without chronic kidney disease. This is the first study to examine associations between dietary, demographic, and serum factors and parathyroid hormone levels in a large, racially diverse, nationally representative population.

We found consistently lower PTH levels in current smokers compared to non-smokers and past smokers, independent of 25(OH)D and other factors. The magnitude of the association

between smoking and serum PTH is similar to what we found in our previous study of 1,288 young Caucasian nurses [8], underscoring the robustness of the association. A major strength of this NHANES study is the greater number of current smokers included in the analysis (1,630 vs. 58 previously). The mechanism underlying the association between smoking and lower PTH remains unclear. Prior studies on female rats have found that nicotine either had no effect on PTH [29] or actually raised PTH level [30]. The association between smoking and PTH could be due to an unknown toxin found in cigarettes [31].

We identified a novel association between higher uric acid and higher PTH. One possible mechanism explaining this association could involve lower $1,25(OH)_2D$ levels. In rats, uric acid suppresses 1 alpha-hydroxylase activity and $1,25(OH)_2D$ synthesis [25], which in turn could raise PTH levels (since $1,25(OH)_2D$ suppresses PTH). While our analysis controlled for 25(OH)D, serum $1,25(OH)_2D$ level was not measured. Our findings suggest that studies examining PTH as a risk factor for coronary heart disease and hypertension should also consider uric acid since it is possible that positive associations observed between PTH and hypertension [1] and cardiovascular disease [3] could be related to uric acid [32–35].

We also found that older age was independently associated with higher PTH, even after controlling for serum creatinine and 25(OH)D. Previous studies reporting associations between age and PTH were small [16, 17, 19, 20] or did not control for variables such as serum 25(OH)D [15, 21, 22]. The mechanism underlying the association between older age and higher PTH remains unclear. Because we found that age was associated with higher PTH even after adjusting for serum 25(OH)D level, dietary calcium intake, and renal function, our findings do not support previous hypotheses that age-related changes in PTH level are due to changes in any of these factors [15, 16, 19, 21, 36, 37]. Two plausible hypotheses to explain the higher PTH level seen with aging are decreased intestinal calcium absorption or a higher set point for calcium-mediated PTH release from the parathyroid gland. Studies have shown that intestinal absorption of calcium decreases with age [22, 38–40], possibly due to intestinal resistance to $1,25(OH)_2D$ [20, 41]. Another explanation could be a higher set point for calcium-mediated PTH release from the parathyroid gland with aging [17] so that PTH concentrations are higher for any given calcium level.

Women had higher PTH levels compared to men. It is possible that estrogen has both direct and indirect effects on PTH level [14, 18, 42]. Estrogen has been found to directly increase PTH release [18, 42] and to inhibit the bone-resorbing effects of PTH [43], which could result in higher PTH levels. However, in stratified analysis with women only, we did not find any significant association between PTH and menopausal status, supplemental estrogen use, and birth control use.

We found that higher serum phosphorus was associated with lower PTH. Because acute phosphorus loading raises PTH levels [44, 45] and because serum phosphorus has a direct effect on PTH release and parathyroid cell proliferation [46, 47], we expected phosphorus to be positively associated with PTH. In contrast, the counterintuitive *inverse* association between serum phosphorus and PTH (independent of serum calcium or dietary calcium intake) suggests that additional serum factors involved in phosphorus metabolism affect PTH. Of note, higher phosphorus concentrations result in higher FGF-23 levels [48], and FGF-23 may suppress PTH [49, 50].

We observed independent associations between serum phosphorus and PTH levels only in whites. We speculate that these results may reflect racial differences in phosphorus handling, possibly due to end-organ resistance to the effects of phosphaturic hormones in blacks. Gutierrez et al. [51] reported that blacks had lower fractional excretion of urinary phosphate than whites despite similar levels of PTH and FGF-23, and a previous metabolic

trial demonstrated decreased serum bone resorption markers in blacks compared to whites after intravenous infusion of PTH [52]. If the association between higher serum phosphorus and lower PTH in whites is a result of phosphorus-mediated increases in the skeletal production and release of FGF-23, it follows that a relative skeletal "resistance" to phosphorus in blacks would result in smaller associations between serum phosphorus and PTH.

We also identified a novel association between serum retinol and PTH. The mechanism by which retinol affects PTH remains unclear, but it could be related to direct effects of retinoic acid on PTH, since retinoic acid directly suppresses PTH secretion in bovine parathyroid cells [23] and human cells [24, 53]. However, it is unclear why these associations were found only in whites. In our previous study of 1,288 nurses [8], we found that higher vitamin A intake, which includes carotenoids and preformed retinyl esters, was associated with lower PTH level. While serum retinol provides a more limited measure of long-term dietary vitamin A intake [54], dietary vitamin A intake data were unavailable in NHANES.

We found a positive association between BMI ≥35 kg/m$^2$ and PTH in Mexican-Americans only. However, the test for trend for BMI was statistically significant for all races. BMI has been correlated with PTH in several studies [55, 56] and also with primary hyperparathyroidism [57]. In our prior study, we found an independent association between PTH and BMI 27–29 kg/m$^2$ compared to BMI 21–22 kg/m$^2$. Of note, our previous study group did not include participants with BMI ≥30 kg/m$^2$. One possible explanation for these findings is that the relationship between BMI and PTH could be non-linear. There were only a small number of participants in this NHANES analysis with BMI >40 kg/m$^2$, and further research in very obese people may help elucidate the nature of the association between PTH and BMI.

There are several limitations to our study. First, our analysis was cross-sectional, which does not allow for inferences of causality. Second, 1,25$(OH)_2$D and FGF-23, both of which could be potential mediators in the association between PTH and other variables, were not measured in this study. Third, serum ionized calcium, which is a powerful regulator of PTH synthesis, secretion, and parathyroid gland growth, was not measured in this study. Fourth, only short-term (i.e., 24 h) diet was assessed. Fifth, we do not have data about time of blood draw available and PTH is known to have diurnal variation [58]. However, we would expect time of blood draw to be random with respect to many of the exposure variables. Lastly, although we found associations between serum phosphorus and PTH in whites but not blacks, the interaction term between race and serum phosphorus was statistically non-significant. We did not have large enough sample sizes to examine other race/ethnic groups.

In conclusion, numerous factors not classically associated with calcium–phosphorus homeostasis, such as uric acid and smoking, are independently associated with PTH in adults without chronic kidney disease across gender and race. Future population-based studies of PTH as a biomarker for chronic diseases, including coronary heart disease and hypertension, should include these factors, particularly if they impact the severity or development of the disease in question. Finally, our study should stimulate additional research to elucidate mechanisms underlying the independent associations we observed with PTH and to explore possible racial differences in phosphorus handling.

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## Table 1

Characteristics of the study population (*N*=7,652)

| Characteristic | Whites (*N*=4,026) | Blacks (*N*=1,792) | Mexican-Americans (*N*=1,834) |
|---|---|---|---|
| Age (years) | 45.6±0.5 | 40.5±0.6 | 37.4±0.7 |
| Body mass index (kg/m$^2$) | 27.9±0.2 | 30.1±0.3 | 28.6±0.2 |
| Female (%) | 50.3 | 53.5 | 49.0 |
| Dietary intake | | | |
| Calcium (mg/day) | 980.5±15.1 | 753.6±20.3 | 933.2±23.0 |
| Total protein (grams/day) | 86.4±1.0 | 81.1±1.7 | 89.7±1.8 |
| Supplement intake | | | |
| Calcium (%) | 13.1 | 6.4 | 6.7 |
| Smoking parameters | | | |
| Smoking status (%) | | | |
| Never smoker (%) | 45.1 | 57.2 | 57.4 |
| Past smoker (%) | 27.5 | 16.2 | 19.7 |
| Current smoker (%) | 27.3 | 26.6 | 22.9 |
| Number of cigarettes per day | 18.9±0.6 | 15.2±1.8 | 9.9±0.8 |
| Laboratory parameters | | | |
| 25 (OH)D (ng/ml) | 26.1±0.1 | 15.1±0.2 | 19.9±0.2 |
| Calcium (mg/dL) | 9.5±0.0 | 9.5±0.0 | 9.5±0.0 |
| Phosphorus (mg/dL) | 3.8±0.0 | 3.8±0.0 | 3.9±0.0 |
| Creatinine (mg/dL) | 0.9±0.0 | 0.9±0.0 | 0.8±0.0 |
| Uric acid (mg/dL) | 5.3±0.0 | 5.3±0.0 | 5.1±0.1 |
| Retinol (μg/dL) | 61.1±0.4 | 51.9±0.6 | 52.9±0.6 |
| Parathyroid hormone (pg/ml) | 38.3±0.5 | 42.6±0.5 | 41.3±0.5 |

Variables are expressed as the mean±standard error unless otherwise specified

**Table 2**

Correlation coefficients between dietary factors, serum factors, and intact parathyroid hormone in non-Hispanic whites

| | Age | BMI | Serum calcium | Serum 25(OH)D | Serum phosphorus | Serum retinol | Serum uric acid | Dietary calcium intake |
|---|---|---|---|---|---|---|---|---|
| Serum PTH | 0.34 (<0.01) | 0.18 (<0.01) | −0.13 (<0.01) | −0.30 (<0.01) | −0.15 (<0.01) | 0.03 (0.06) | 0.19 (<0.01) | −0.16 (<0.01) |
| Age | – | 0.13 (<0.01) | −0.05 (<0.01) | −0.14 (<0.01) | −0.14 (<0.01) | 0.21 (<0.01) | 0.09 (<0.01) | −0.15 (<0.01) |
| BMI | – | – | −0.14 (<0.01) | −0.22 (<0.01) | −0.08 (<0.01) | 0.03 (0.03) | 0.35 (<0.01) | −0.04 (<0.01) |
| Serum calcium | – | – | – | 0.06 (<0.01) | 0.20 (<0.01) | 0.22 (<0.01) | 0.17 (<0.01) | 0.03 (0.07) |
| Serum 25(OH)D | – | – | – | – | 0.05 (<0.01) | 0.14 (<0.01) | −0.12 (<0.01) | 0.12 (<0.01) |
| Serum phosphorus | – | – | – | – | – | 0.02 (0.20) | −0.08 (<0.01) | 0.09 (<0.01) |
| Serum retinol | – | – | – | – | – | – | 0.28 (<0.01) | 0.06 (<0.01) |
| Serum uric acid | – | – | – | – | – | – | – | −0.01 (0.59) |

Values are Spearman correlation coefficients. *p* values are in parentheses

## Table 3

Multivariate-adjusted differences in intact parathyroid hormone in non-Hispanic whites

| Variable | Number | Difference in PTH (pg/ml) | 95% CI | *p*, trend |
|---|---|---|---|---|
| Age (years) | | | | |
| <30 | 961 | Referent | | <0.01 |
| 30–39 | 643 | 2.6 | 1.3, 4.0 | |
| 40–49 | 639 | 5.0 | 3.2, 6.9 | |
| 50–59 | 583 | 5.9 | 4.0, 7.9 | |
| ≥60 | 1,200 | 7.0 | 5.5, 8.4 | |
| Gender | | | | |
| Male | 1,988 | Referent | | |
| Female | 2,038 | 2.2 | 0.6, 3.9 | |
| Body mass index (kg/m$^2$) | | | | |
| <21 | 389 | 0.5 | −1.6, 2.5 | 0.05 |
| 21–24 | 1,069 | Referent | | |
| 25–29 | 1,345 | −0.5 | −1.6, 0.7 | |
| 30–34 | 699 | 0.4 | −1.2, 2.0 | |
| ≥35 | 470 | 1.8 | −0.2, 3.8 | |
| Smoking | | | | |
| Never | 1,681 | Referent | | |
| Past | 1,126 | −0.4 | −1.4, 0.7 | |
| Current | 966 | −5.3 | −6.5, −4.2 | |
| Number of cigarettes per day[a] | | | | |
| <10 | 115 | Referent | | 0.01 |
| 10–19 | 247 | −2.8 | −6.6, 1.0 | |
| ≥20 | 484 | −4.2 | −7.6, −0.7 | |
| Dietary calcium intake (mg/day) | | | | |
| <558 | 1,006 | Referent | | 0.02 |
| 558–836 | 1,007 | −0.2 | −1.9, 1.5 | |
| 837–1,246 | 1,005 | −0.8 | −2.6, 1.0 | |
| ≥1,247 | 1,008 | −2.5 | −4.8, −0.2 | |
| Serum 25(OH)D (ng/mL) | | | | |
| <20 | 983 | Referent | | <0.01 |
| 20–25 | 1,099 | −3.3 | −5.0, −1.6 | |
| 26–30 | 964 | −5.8 | −7.0, −4.6 | |
| ≥31 | 1,124 | −7.9 | −9.8, −6.0 | |
| Serum calcium (mg/dL) | | | | |
| <9.3 | 852 | Referent | | <0.01 |
| 9.3–9.4 | 849 | −2.9 | −5.3, −0.5 | |
| 9.5–9.6 | 951 | −3.6 | −5.9, −1.2 | |
| ≥9.7 | 1,374 | −4.8 | −6.7, −2.8 | |
| Serum phosphorus (mg/dL) | | | | |

| Variable | Number | Difference in PTH (pg/ml) | 95% CI | *p*, trend |
|---|---|---|---|---|
| <3.5 | 957 | Referent | | 0.01 |
| 3.5–3.7 | 898 | −0.2 | −1.8, 1.4 | |
| 3.8–4.1 | 1,145 | −0.7 | −2.3, 0.9 | |
| ≥4.2 | 1,026 | −2.3 | −4.0, −0.6 | |
| Serum uric acid (mg/dL) | | | | |
| <4.3 | 984 | Referent | | <0.01 |
| 4.3–5.1 | 972 | 2.2 | 0.2, 4.1 | |
| 5.2–6.0 | 991 | 3.6 | 1.8, 5.5 | |
| ≥6.1 | 1,078 | 4.9 | 2.7, 7.0 | |
| Serum retinol (μg/dL) | | | | |
| <49.2 | 994 | Referent | | 0.06 |
| 49.2–58.7 | 1,004 | −0.2 | −1.9, 1.5 | |
| 58.8–69.5 | 997 | −0.2 | −1.8, 2.1 | |
| ≥69.6 | 1,005 | −1.6 | −3.4, 0.2 | |

Covariates in the multivariate regression model are adjusted for age, gender, body mass index, smoking status, dietary calcium intake, dietary protein intake, season of blood draw, serum 25(OH)D, serum calcium, serum phosphorus, serum creatinine, serum uric acid, serum retinol, and fasting status

[a]Model limited to participants who reported currently smoking

## Table 4

Multivariate-adjusted differences in intact parathyroid hormone in non-Hispanic blacks

| Variable | Number | Difference in PTH (pg/ml) | 95% CI | *p*, trend |
|---|---|---|---|---|
| Age (years) | | | | |
| <30 | 673 | Referent | | <0.01 |
| 30–39 | 281 | 3.5 | 0.6, 6.5 | |
| 40–49 | 307 | 5.0 | 1.9, 8.0 | |
| 50–59 | 199 | 6.6 | 3.7, 9.4 | |
| ≥60 | 332 | 8.0 | 5.4, 10.7 | |
| Gender | | | | |
| Male | 886 | Referent | | |
| Female | 906 | 4.5 | 1.9, 7.0 | |
| Body mass index (kg/m$^2$) | | | | |
| <21 | 164 | −4.0 | −7.9, 0.0 | 0.05 |
| 21–24 | 365 | Referent | | |
| 25–29 | 511 | 0.1 | −2.3, 2.5 | |
| 30–34 | 381 | −2.1 | −4.7, 0.5 | |
| ≥35 | 344 | 2.2 | −1.3, 5.6 | |
| Smoking | | | | |
| Never | 827 | Referent | | |
| Past | 262 | 0.4 | −2.4, 3.2 | |
| Current | 373 | −6.1 | −8.7, −3.5 | |
| Number of cigarettes per day[a] | | | | |
| <10 | 115 | Referent | | <0.01 |
| 10–19 | 104 | −3.2 | −8.5, 2.0 | |
| ≥20 | 97 | −6.0 | −9.4, −2.6 | |
| Dietary calcium intake (mg/day) | | | | |
| <558 | 765 | Referent | | 0.19 |
| 558–836 | 413 | −0.7 | −2.8, 1.4 | |
| 837–1,246 | 350 | −3.6 | −6.3, −0.8 | |
| ≥1,247 | 264 | −1.6 | −5.9, 2.7 | |
| Serum 25(OH) vitamin D (ng/mL) | | | | |
| <10 | 422 | Referent | | <0.01 |
| 10–13 | 477 | −3.5 | −6.4, −0.6 | |
| 14–18 | 479 | −4.0 | −7.3, −0.6 | |
| ≥19 | 522 | −8.8 | −11.3, −6.3 | |
| Serum calcium (mg/dL) | | | | |
| <9.3 | 340 | Referent | | <0.01 |
| 9.3–9.4 | 373 | 0.9 | −2.0, 3.8 | |
| 9.5–9.6 | 395 | −0.6 | −3.6, 2.4 | |
| ≥9.7 | 684 | −5.1 | −7.6, −2.6 | |
| Serum phosphorous (mg/dL) | | | | |

| Variable | Number | Difference in PTH (pg/ml) | 95% CI | *p*, trend |
|---|---|---|---|---|
| <3.5 | 431 | Referent | | 0.71 |
| 3.5–3.7 | 402 | 1.7 | −0.3, 3.6 | |
| 3.8–4.1 | 462 | 1.4 | −1.3, 4.2 | |
| ≥4.2 | 497 | 0.0 | −2.3, 2.4 | |
| Serum uric acid (mg/dL) | | | | |
| <4.3 | 418 | Referent | | <0.01 |
| 4.3–5.1 | 421 | 3.5 | 0.4, 6.7 | |
| 5.2–6.0 | 287 | 4.7 | 2.3, 7.2 | |
| ≥6.1 | 215 | 5.5 | 3.0, 8.1 | |
| Serum retinol (μg/dL) | | | | |
| <49.2 | 869 | Referent | | 0.65 |
| 49.2–58.7 | 412 | 1.5 | −0.5, 3.5 | |
| 58.8–69.5 | 287 | 0.0 | −3.0, 3.0 | |
| ≥69.6 | 215 | 1.9 | −1.4, 5.1 | |

Note: Covariates in the multivariate regression model are adjusted for age, gender, body mass index, smoking status, dietary calcium intake, dietary protein intake, season of blood draw, serum 25(OH)D serum calcium, serum phosphorus, serum creatinine, serum uric acid, serum retinol, and fasting status

[a]Model limited to participants who reported currently smoking

## Table 5

Multivariate-adjusted differences in intact parathyroid hormone in Mexican-Americans

| Variable | Number | Difference in PTH (pg/ml) | 95% CI | *p*, trend |
|---|---|---|---|---|
| Age (years) | | | | |
| <30 | 701 | Referent | | <0.01 |
| 30–39 | 297 | 0.9 | −2.0, 3.7 | |
| 40–49 | 278 | 3.8 | 1.2, 6.5 | |
| 50–59 | 129 | 2.0 | −2.3, 6.3 | |
| ≥60 | 429 | 3.7 | 1.3, 6.1 | |
| Gender | | | | |
| Male | 874 | Referent | | |
| Female | 960 | 1.1 | −1.2, 3.4 | |
| Body mass index (kg/m$^2$) | | | | |
| <21 | 122 | −5.0 | −9.9, −0.0 | 0.01 |
| 21–24 | 406 | Referent | | |
| 25–29 | 691 | −1.5 | −3.9, 0.9 | |
| 30–34 | 394 | 0.5 | −2.5, 3.5 | |
| ≥35 | 203 | 3.5 | 0.1, 6.8 | |
| Smoking | | | | |
| Never | 908 | Referent | | |
| Past | 350 | 0.3 | −2.0, 2.6 | |
| Current | 291 | −4.2 | −7.3, −1.1 | |
| Number of cigarettes per day[a] | | | | |
| <10 | 95 | Referent | | 0.80 |
| 10–19 | 48 | −6.6 | −14.5, 1.3 | |
| ≥20 | 37 | 0.1 | −7.2, 7.5 | |
| Dietary calcium intake (mg/day) | | | | |
| <558 | 545 | Referent | | 0.40 |
| 558–836 | 456 | −0.3 | −2.6, 1.9 | |
| 837–1,246 | 441 | 0.1 | −3.2, 3.3 | |
| ≥1,247 | 392 | −0.9 | −4.1, 2.2 | |
| Serum 25(OH)D (ng/mL) | | | | |
| <15 | 480 | Referent | | <0.01 |
| 15–18 | 363 | −4.1 | −7.4, −0.9 | |
| 19–24 | 583 | −5.3 | −7.8, −2.8 | |
| ≥5 | 494 | −5.4 | −8.9, −1.8 | |
| Serum calcium (mg/dL) | | | | |
| <9.3 | 509 | Referent | | <0.01 |
| 9.3–9.4 | 410 | −1.5 | −4.1, 1.1 | |
| 9 .5–9.6 | 408 | −3.5 | −6.4, −0.6 | |
| ≥9.7 | 507 | −5.6 | −8.9, −2.4 | |
| Serum phosphorus (mg/mL) | | | | |

| Variable | Number | Difference in PTH (pg/ml) | 95% CI | *p*, trend |
|---|---|---|---|---|
| <3.5 | 398 | Referent | | 0.37 |
| 3.5–3.7 | 359 | −0.5 | −3.3, 2.2 | |
| 3.8–4.1 | 548 | −1.0 | −4.0, 2.1 | |
| ≥4.2 | 529 | −1.3 | −4.1, 1.6 | |
| Serum uric acid (mg/dL) | | | | |
| <4.3 | 566 | Referent | | 0.02 |
| 4.3–5.1 | 491 | 1.4 | −2.3, 5.1 | |
| 5.2–6.0 | 387 | 4.6 | 1.2, 8.0 | |
| =6.1 | 390 | 4.8 | 1.5, 8.1 | |
| Serum retinol (μg/dL) | | | | |
| <49.2 | 832 | Referent | | 0.45 |
| 49.2–58.7 | 460 | −0.7 | −3.3, 2.0 | |
| 58.8–69.5 | 326 | 0.8 | −1.6, 3.1 | |
| ≥69.6 | 203 | −2.3 | −7.3, 2.6 | |

Covariates in the multivariate regression model are adjusted for age, gender, body mass index, smoking status, dietary calcium intake, dietary protein intake, season of blood draw, serum 25(OH)D, serum calcium, serum phosphorus, serum creatinine, serum uric acid, serum retinol, and fasting status

[a]Model limited to participants who reported currently smoking