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Association of Glucocorticoid Use and Low 25-Hydroxyvitamin D Levels: Results from the National Health and Nutrition Examination Survey (NHANES): 2001–2006

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Abstract

Context:

In many disorders requiring steroid therapy, there is substantial decrease in bone mineral density. The association between steroid use and 25-hydroxyvitamin D [25(OH)D] deficiency has not been confirmed in large population-based studies, and currently there are no specific vitamin D recommendations for steroid users.

Objective:

The aim of the study was to evaluate the association of serum 25(OH)D deficiency [defined as 25(OH)D <10 ng/ml] with oral steroid use.

Design:

Cross-sectional analysis was performed using NHANES 2001–2006.

Setting:

We analyzed a nationally representative sample of U.S. children and adults.

Participants:

The study sample consisted of children, adolescents, and adults from NHANES 2001–2006 (n = 22,650), representative of 286 million U.S. residents, with serum 25(OH)D levels and data on other potential confounders.

Main Outcome Measure:

We measured serum 25(OH)D levels below 10 ng/ml.

Results:

A total of 181 individuals (0.9% of the population) used steroids within the past 30 d. Overall, 5% of the population had 25(OH)D levels below 10 ng/ml. Among steroid users, 11% had 25(OH)D levels below 10 ng/ml, compared to 5% among steroid nonusers ($P = 0.009$). The odds of having 25(OH)D deficiency were 2-fold higher in those who reported steroid use compared to those without steroid use [odds ratio (OR), 2.36; 95% confidence interval (CI), 1.25, 4.45]. This association remained after multivariable adjustment (OR, 2.21; 95% CI, 1.01, 4.85) and in a multivariable model using NHANES III data (OR, 1.88; 95% CI, 1.01, 3.48).

Conclusion:

Steroid use is independently associated with 25(OH)D deficiency in this nationally representative cohort limited by cross-sectional data. It suggests the need for screening and repletion in patients on chronic steroids.

Vitamin D deficiency, variously defined as 25-hydroxyvitamin D [25(OH)D] levels below 20, 15, or 10 ng/ml, is common in the general population (1–3). Multiple studies have shown an association of low 25(OH)D with all-cause mortality, cardiovascular disease, autoimmune diseases, infectious diseases, and increased risk of fractures (2, 4–9). Glucocorticoid use has also been linked to many of these outcomes.

Glucocorticoid use is common in patients with chronic pulmonary, rheumatic, and kidney diseases, and in these patients glucocorticoid use is associated with osteoporosis and increased fractures. Glucocorticoids decrease intestinal calcium absorption and increase urinary excretion of calcium (10). Additionally, glucocorticoids are known to enhance bone resorption and decrease bone formation, thereby decreasing bone mass and increasing the risk of fractures (11). Laboratory studies reveal that steroids may increase 24-hydroxylase activity, thereby decreasing 25(OH)D levels (12–14). Recent literature in pulmonology, gastroenterology, and rheumatology has noted that patients treated with oral glucocorticoids have significantly lower levels of 25(OH)D (15–17).

This association between steroid use and 25(OH)D deficiency has not been confirmed in large population-based studies. In November 2010, the Institute of Medicine published new recommendations of 600 IU/d for children and adults (1–70 yr of age) and 800 IU/d for those more than 70 yr of age (18). There were no specific recommendations made for those who are treated with steroids.

The aim of the present study was to examine the association of serum 25(OH)D deficiency with oral steroid use in a large representative sample of U.S. residents using the National Health and Nutrition Examination Surveys (NHANES) from 2001–2006.

Subjects and Methods

Study population

NHANES is a nationally representative, cross-sectional survey designed to assess the health and nutritional status of civilian noninstitutionalized adults and children in the United States. Participants were selected through a complex, multistage probability design. All of the participants underwent standardized home interviews as well as physical examinations and laboratory testing at a mobile health center. The current study included all children, adolescents, and adults ages 1 yr and older with recorded 25(OH)D levels and data available on other covariates. A total of 31,509 participants were examined in NHANES 2001–2006, of which 8,859 participants were excluded from the analysis for having the following missing values: serum 25(OH)D (n = 3,417), poverty income ratio (PIR) (n = 1,913), obesity (n = 3,509), milk intake (n = 3), and days missed from school or work (n = 17). 25(OH)D levels were measured in all

participants at least 6 yr of age from 2001–2002 and in all participants at least 1 yr of age from 2003–2006. NHANES 2001–2006 was approved by the National Center for Health Statistics Institutional Review Board. All of the participants at least 18 yr old provided informed consent, participants 12 to 17 yr old and their parents provided informed consent, participants 7 to 11 yr old provided assent and parental consent, and parents provided informed consent for those less than 7 yr old.

Participants in NHANES III, performed between 1988–1994, were examined to evaluate the robustness of the associations. A total of 33,994 participants were examined in NHANES III, of which 18,620 participants were excluded from the analysis for having the following missing values: serum 25(OH)D (n = 8,331), PIR (n = 3,410), obesity (n = 5,122), and supplement use (n = 1,757).

Study variables

Demographic variables in the current analysis of NHANES 2001–2006 included age, sex, race/ethnicity, and the PIR. Participants over age 85 yr of age were reported by NHANES as being 85 yr old to maintain anonymity. Race/ethnicity was self-reported and categorized as non-Hispanic white, non-Hispanic black, Mexican-American, or other. Poverty status was defined using the PIR, an index calculated by dividing family income by a poverty threshold specific to the family size. We defined PIR of 1 or less as below the poverty threshold. PIR categories were: 5.00 or greater, above 1.0 to 4.99, and 1.00 or less.

Steroid use and the use of any medication data were acquired from the medication questionnaire and pill-bottle review. Participants were asked whether they used any medications within the past 30 d and were classified as “yes” or “no” to medication use. If yes, they were asked to report the medication(s) used. We coded positive steroid use for anyone who reported the use of prednisone, prednisolone, methylprednisolone, or methylprednisolone acetate within the past 30 d. Those who did not report use of any of these medications were recorded as no steroid use. Steroid use data from NHANES III were acquired from the medication questionnaire with an affirmative response for the prescription drug class category adrenal corticosteroids coded as positive steroid use. Milk consumption data were obtained from the diet, behavior, and nutrition section of the NHANES 2001–2006 Sample Person Questionnaire and were categorized as daily, more than once per week but less than daily, less than once per week, and never. Vitamin D supplementation use was assessed by participant questionnaire and pill-bottle review. Supplementation use was categorized as none, more than zero and less than 400 IU/d, or at least 400 IU/d. In NHANES III, individual vitamin D intake was not assessed; therefore, we used the use of a multivitamin supplement.

As part of the physical exam, each participant's height and weight were measured. Obesity was categorized by weight in 1 yr olds and by body mass index (BMI) in those at least 2 yr old. In 1 yr olds, obesity was defined as weight exceeding the 95th percentile for gender-specific weight curves. BMI was calculated as weight in kilograms divided by height in meters squared. In those from 2 to <18 yr of age, obesity was defined using age- and gender-specific percentile curves of BMI. In those at least 18 yr old, the widely used adult cutoff point of 30 kg/m^2 defined obesity.

Days missed from school or work were obtained from the medical conditions section of the NHANES 2001–2006 Sample Person Questionnaire. Those 6–19 yr of age were asked: how many days during the past 12 months did you miss from school because of injury or illness. Those 16 yr and older were asked: how many days during the past 12 months did you miss from work. Physical activity was assessed based on direct questioning of the participants regarding hours per day spent using computers, television, and video games.

25(OH)D was measured using the DiaSorin 25(OH)D assay (DiaSorin, Stillwater, MN). 25(OH)D samples from NHANES III were adjusted to make a valid comparison to the NHANES 2001–2006 survey years due to a reformulation of the DiaSorin RIA kit that resulted in a shift in assay results between the two time periods (19). Although there is no consensus as to the definition of 25(OH)D deficiency, we chose severe

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25(OH)D deficiency (<10 ng/ml) as the main outcome measure, a value associated with clinical myopathy, osteomalacia, and rickets (20). Given that far fewer participants in NHANES III had 25(OH)D levels below 10 ng/ml (1%), too few for adequate statistical inferences, we used a cutoff of below 15 ng/ml because the prevalence of 25(OH)D deficiency with this cutoff was closer to that using the below 10 ng/ml cutoff in 2001–2006, and thus appeared more directly comparable. Previous research has shown differences in vitamin D levels over time, most likely due to changes in sunscreen use, outside activity, and obesity prevalence (21).

Serum creatinine was measured using the Beckman Synchron LX20 method. Serum creatinine was recorded in all participants 12 yr of age and older for NHANES 2001–2006. Estimated glomerular filtration rate (eGFR) was then calculated using the Schwartz formula in those participants less than 18 yr of age and the CKD-EPI equation in those 18 yr of age and older (22, 23). PTH levels were measured in NHANES 2003–2006 using the ECL/Origen-Electrohemiluminescent method. Serum albumin was measured via a bichromatic digital endpoint method.

Statistical analysis

All analyses were performed using sample weights that account for unequal probability of selection, nonresponse, and planned oversampling of non-Hispanic blacks and Mexican-Americans. Survey analysis was performed by creating a subpopulation of all participants with a recorded vitamin D level and any value for the chosen study variables. Participants were categorized by 25(OH)D levels below 10 ng/ml and at least 10 ng/ml. Statistical significance of participant characteristics between the two 25(OH)D subgroups was determined by univariate linear and logistic regression for continuous and dichotomous variables, respectively. Participants were also categorized by those who reported and those who did not report steroid use. Statistical significance was again determined using univariate linear and logistic regression.

Univariate logistic regression was performed to analyze predictors of 25(OH)D deficiency. Those variables that were significant at $P < 0.25$ on univariate testing were included in the analysis. Multivariable logistic regression analysis was then used to investigate the association between glucocorticoid use and 25(OH)D levels below10 ng/ml in an unadjusted model. We then created a model including the variables age, sex, and race/ethnicity determined *a priori* to be clinically meaningful and to remain in the model regardless of statistical significance. In the third model, variables that were significant at $P < 0.25$ on univariate testing and variables that were felt to be *a priori* confounders of the association were also considered for inclusion in the final multivariable model. A backward selection method was used to create the final model, which ultimately included the variables age, sex, race/ethnicity, PIR, obesity, milk intake, supplement use, days missed from school or work, and the use of any medication. Although we lost 4421 participants when adjusting for eGFR, it was felt to be an important variable, and therefore the primary analysis is presented both with and without adjustment for eGFR.

Additional multivariable analyses were performed in prespecified subgroups including age, sex, race/ethnicity, and obesity. Sensitivity analyses were performed using different definitions of 25(OH)D deficiency and adjusting for additional potential confounders. Associations between serum albumin and mortality have been shown in many disease states (24). To assess for possible confounding due to nutritional/health status, we performed a sensitivity analysis with serum albumin. Low levels of physical activity have been associated with vitamin D insufficiency. We examined whether adjustment for physical activity altered the association between low vitamin D levels and steroid use. Data on physical activity was available on 10,605 participants. We also examined whether adjustment for PTH levels altered the observed associations. PTH values were available in 13,803 of the initial population. Separate models were rerun adding albumin, physical activity, and PTH as adjustors.

All statistical analyses were performed in STATA 11.1 (StataCorp LP, College Station, TX).

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Results

Low 25(OH)D levels

There were 22,650 children, adolescents, and adults in NHANES 2001–2006 with recorded 25(OH)D levels and complete data on other variables, representative of 286 million U.S. residents (Table 1). Overall, 32% of the population had levels less than 20 ng/ml, 15% less than 15 ng/ml, and 5% less than 10 ng/ml. Participants with 25(OH)D levels below 10 ng/ml were more likely to be female, non-Hispanic black, and obese; have lower PIR; and have lower levels of milk intake and supplement use. They were also less likely to use any medication but more likely to use steroids. On multivariable modeling, consistent with recent studies, females [odds ratio (OR), 1.71; 95% confidence interval (CI), 1.45, 2.02], non-Hispanic blacks (OR, 11.83; 95% CI, 9.58, 14.61), Mexican-Americans (OR, 2.49; 95% CI, 1.79, 4.54), obese individuals (OR, 1.87; 95% CI, 1.59, 2.20), and those who drank milk less than once per week or never (OR, 3.44; 95% CI, 2.73, 4.34; and OR, 4.66; 95% CI, 3.59, 6.05, respectively) were more likely to be 25(OH)D deficient, when controlling for steroid use, age, sex, race/ethnicity, PIR, obesity, milk intake, supplement use, days missed from school or work, and the use of any medication. Those who used either more than zero and less than 400 IU/d, or at least 400 IU/d of vitamin D supplementation per day were less likely to be 25(OH)D deficient (OR, 0.16; 95% CI, 0.07, 0.34; and OR, 0.25; 95% CI, 0.15, 0.43, respectively) compared with those who reported no vitamin D supplementation in the fully adjusted model. The use of any medication in the fully adjusted model was not statistically significant (OR, 0.92; 95% CI, 0.79, 1.07).

Steroid use

Overall, 0.9% of the population used steroids within the past 30 d. Among steroid users, 11% had 25(OH)D levels below 10 ng/ml, compared with 5% among steroid nonusers ($P = 0.009$) (Table 2). Steroid use compared with no steroid use was more common in those who were older, obese, and used less than 400 IU/d of vitamin D supplements (Table 2). Steroid use was more common (2%) in patients with 25(OH)D levels less than 10 ng/ml, compared with 0.9% in patients with 25(OH)D levels above 10 ng/ml ($P = 0.009$).

Association of steroid use and low 25(OH)D levels

The odds of having 25(OH)D deficiency were 2-fold higher in those who reported steroid use compared with those without steroid use (OR, 2.36; 95% CI, 1.25, 4.45). This association remained after multivariable adjustment for age, sex, and race/ethnicity (OR, 2.21; 95% CI, 1.06, 4.62) and in the fully adjusted multivariable model (OR, 2.21; 95% CI, 1.01, 4.85) (Table 3). When adding eGFR to the multivariable analysis, the OR for steroid use was 2.34 (95% CI, 1.02, 5.34).

Subgroup analysis

The association of steroid use with 25(OH)D deficiency was examined within categories of age, sex, race/ethnicity, and obesity. Among participants no older than 18 yr, the OR for steroid use was 14.05 (95% CI, 1.22, 161.98), whereas among participants more than 18 yr old, the OR was 2.13 (95% CI, 0.92, 4.90). Other groups are presented in Fig. 1.

Sensitivity analysis

Sensitivity analyses were performed to test whether adjustments for physical activity, PTH levels, or albumin levels altered the observed associations between steroid use and 25(OH)D deficiency. When adjusting the main analysis for physical activity, the OR for steroid use was 2.31 (95% CI, 0.66, 7.99). Adding PTH to the main multivariable model, the OR for steroid use was 3.32 (95% CI, 1.41, 7.82). When

adjusting the main multivariable model for serum albumin as a measure of overall nutritional/health status, the OR for steroid use was 2.18 (95% CI, 0.99, 4.80).

Several sensitivity analyses were also performed to explore the association of steroid use with other definitions of 25(OH)D deficiency (Table 3). When defining 25(OH)D deficiency as levels below 15 ng/ml, the unadjusted OR for steroid users was 1.43 (95% CI, 1.00, 2.04). The association lost significance when adjusting for other variables. When defining 25(OH)D deficiency as levels below 20 ng/ml, none of the models had statistically significant results.

A sensitivity analysis was performed using data from NHANES III, with the adjusted 25(OH)D assay values. We analyzed 15,374 participants representative of 166 million U.S. residents. Similar to NHANES 2001–2006, 1% of the population reported steroid use. Unlike NHANES 2001–2006, only 1% of the participants had 25(OH)D levels below 10 ng/ml; 9% had 25(OH)D levels below 15 ng/ml and 24% below 20 ng/ml. Among steroid users, 14% had 25(OH)D levels below 15 ng/ml, compared with 9% among steroid nonusers ($P = 0.04$). Steroid use was more common in participants with 25(OH)D levels less than 15 ng/ml, 2% compared with 1% in patients with 25(OH)D levels above 15 ng/ml ($P = 0.04$). The odds of having 25(OH)D deficiency, defined as 25(OH)D level below 15 ng/ml, were approximately 2-fold higher in those who reported steroid use compared with those without steroid use (OR, 1.75; 95% CI, 1.03, 3.00). This association remained after multivariable adjustment with age, sex, race/ethnicity, obesity, PIR, and supplement use (OR, 1.88; 95% CI, 1.01, 3.48).

Discussion

Recent research has confirmed that vitamin D deficiency is a global problem affecting an estimated 1 billion people worldwide (2, 4, 25–27). Multiple large-scale studies have estimated a prevalence of vitamin D deficiency, using various definitions of vitamin D deficiency, to vary between 24 and 70% in the general pediatric population and between 36 and 57% in an adult population (1–4, 25, 26, 28–31). Studies in vitamin D receptor (VDR) knockout mice demonstrate that VDR-deficient mice have increased sensitivity to autoimmune diseases, are more prone to oncogene-induced tumors, and develop high renin hypertension and cardiac hypertrophy (32, 33). In addition, recent studies in humans reveal that there are potential benefits of adequate vitamin D levels not only on bone, but also on blood pressure, rate of kidney disease progression, and prevention of cardiovascular disease, autoimmune diseases, cancer, and all-cause mortality (5, 6, 8, 9, 34–37).

This is the first study, to our knowledge, that shows a significant association between steroid use and vitamin D deficiency in a large, nationally representative sample of children and adults. In 2001–2006, approximately 0.9% (2.1 million) of U.S. children and adults reported steroid use within the past 30 d. Of those who used steroids, a statistically significant greater percentage had 25(OH)D levels less than 10 ng/ml compared with those who did not report steroid use.

The present findings are consistent with results from recent smaller studies in the fields of pulmonology, gastroenterology, and rheumatology that found an association between steroid use and low levels of 25(OH)D. In a cohort study of 124 women with systemic lupus erythematosus, Toloza *et al.* (17) demonstrated by multivariable logistic regression that cumulative glucocorticoid exposure was significantly associated with low levels of 25(OH)D ($P = 0.03$) when adjusting for ethnicity, season, and serum creatinine. Sentongo *et al.* (16) demonstrated that among 112 children, adolescents, and young adults with Crohn's disease, vitamin D deficiency was associated with winter season, African-American ethnicity, Crohn's disease confined to the upper gastrointestinal tract, and magnitude of lifetime exposure to glucocorticoid therapy (23.7 ± 13.5 compared with 17.5 ± 12.2 mg/d glucocorticoids; $P = 0.05$). In a study of 100 asthmatic children, the use of inhaled steroids ($P = 0.05$), oral steroids ($P = 0.02$), and total steroid dose ($P = 0.001$) all showed significant inverse correlations with serum vitamin D levels (15).

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The mechanism of action of glucocorticoids and its association with vitamin D deficiency is not completely understood. Glucocorticoids are known to enhance bone resorption, decrease bone formation, decrease intestinal calcium absorption, and increase urinary calcium excretion (2). The administration of glucocorticoids to vitamin D-deficient rats does not affect the rate of conversion of a physiological dose of 25(OH)D to 1,25-dihydroxyvitamin D [1,25$(OH)_2$D] (38, 39). In 2000, Akeno *et al.* (12) demonstrated that dexamethasone increased renal expression of vitamin D-24-hydroxylase, which in turn degrades vitamin D metabolites such as 25(OH)D and 1,25$(OH)_2$D. This same group then used an established renal cell line, LLC-PK_1 cells, and UMR-106 osteoblast-like cells, in which they demonstrated that cells treated with 1,25$(OH)_2$D expressed 24-hydroxylase mRNA and that treatment with dexamethasone for 24 h significantly enhanced the abundance of 24-hydroxylase mRNA (13). This increased glucocorticoid-stimulated expression of 24-hydroxylase mRNA was completely abolished with the addition of cycloheximide, a protein synthesis inhibitor (13). Most recently, in 2010, Dhawan and Christakos (14) demonstrated that via a novel mechanism of functional cooperation of glucocorticoid receptor, C/EBPβ, and VDR, glucocorticoids directly enhance 24-hydroxylase transcription. In summary, it appears that steroids may enhance inactivation of 25(OH)D by up-regulating 24-hydroxylase activity.

Extending current clinical and experimental data supporting an association between glucocorticoid use and vitamin D deficiency, we showed in a nationally representative sample of U.S. children and adults that there is a statistically significant association between the two. It is likely that those participants who reported steroid use suffered from chronic illness that may be associated with poor nutrition, decreased consumption of vitamin D and calcium-rich foods, as well as decreased sun exposure from lack of outside physical activity, all of which place these participants at increased risk for vitamin D deficiency. However, even when adjusting for days missed from school or work, use of any medications, and eGFR, glucocorticoid use remained independently associated with severe vitamin D deficiency.

Although the results of this study from the analysis of NHANES 2001–2006 and replicated in NHANES III, representative samples of the U.S. population, could have broad implications, our study has several important limitations. We are limited by a lack of information on several potential important confounders, including the season of measurement of 25(OH)D, the latitude of the participants' homes, the reason for glucocorticoid use, as well as the cumulative dose of glucocorticoid. Although NHANES 2001–2006 provides some of the best and most recent estimates of the prevalence of chronic disease, this study uses a cross-sectional study design, and therefore, caution should be taken when considering the direction of associations, and, as in any observational study, causality cannot be established. Despite the cross-sectional nature of the study, the sample size of the study cohort is larger than any previously reported study investigating steroid use and vitamin D levels. This allowed us to control for a variety of confounders including demographics, dietary factors, and obesity scores, which were all collected in a standardized fashion.

This current study indicates that steroid use is associated with severe 25(OH)D deficiency. It is generally recognized that serum 25(OH)D levels less than 10 ng/ml are associated with clinical myopathy, osteomalacia, and rickets (20). Therefore, it is prudent that we address the issue of vitamin D deficiency in the general population, but even more so in those who use glucocorticoids and are therefore at higher risk of vitamin D deficiency and its possible sequelae.

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Footnotes

Abbreviations:

BMI Body mass index
CI confidence interval
eGFR estimated glomerular filtration rate
OR odds ratio
$1,25(OH)_2D$ 1,25-dihydroxyvitamin D
25(OH)D 25-hydroxyvitamin D
PIR poverty income ratio
VDR vitamin D receptor.

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Figures and Tables

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Table 1.

Participant characteristics by 25(OH)D levels within children ages 1–18 yr and adults older than 18 yr from NHANES 2001–2006 (n = 22,650)

	Age 1–18 yr			Age >18 yr		
	<10 ng/ml (n = 536)	≥10 ng/ml (n = 8,926)	*P* value[b]	<10 ng/ml (n = 1,133)	≥10 ng/ml (n = 12,055)	*P* value[b]
Age (yr)[a]	15 ± 0.20	11 ± 0.10	<0.001	44 ± 0.77	46 ± 0.35	0.01
Female (%)	65	48	<0.001	64	51	<0.001
Race/ethnicity (%)						
Non-Hispanic white	8	62	<0.001	31	75	<0.001
Non-Hispanic black	69	13	<0.001	50	8	<0.001
Mexican-American	17	18	0.58	13	12	0.41
Other, multiracial	7	6	0.63	6	5	0.45
PIR (%)						
≥5	8	15	0.03	14	26	<0.001
>1–4.99	57	63	0.05	65	62	0.15
0–1	36	22	<0.001	21	12	<0.001
Obese (%)	34	18	<0.001	50	31	<0.001
Milk intake (%)						
≥1/d	39	78	<0.001	21	47	<0.001
≥1/wk but <1/d	33	14	<0.001	29	26	0.07
<1/wk or varied	16	5	<0.001	22	14	<0.001
Never	12	3	<0.001	28	13	<0.001
Supplement use (%)						
None	96	87	0.01	96	80	<0.001
<400 IU/d	2	3	0.41	1	6	<0.001
≥400 IU/d	2	10	<0.001	4	14	<0.001
Days missed from school or work[a]	3 ± 0.30	3 ± 0.12	0.89	5 ± 1.11	4 ± 0.26	0.26
eGFR (ml/min/1.73 m^2)[a,c]	134 ± 2.00	140 ± 0.77	0.02	101 ± 1.19	94 ± 0.52	<0.001
Any medications used (%)	19	27	0.02	51	55	0.03
Steroid use (%)	1	0.4	0.1	2	1	0.04

Age 1–18 yr | Age >18 yr
<10 ng/ml (n = 536) | ≥10 ng/ml (n = 8,926) | P value | <10 ng/ml (n = 1,133) | ≥10 ng/ml (n = 12,055) | P value

Participants over 85 yr of age are reported as being 85 yr old in order to protect anonymity.

[a]Values are expressed as mean ± SE.
[b]*P* value from linear or logistic regression.
[c]eGFR only available in participants at least 12 yr of age; number of observations = 18,229.

Table 2.

Participant characteristics by steroid use within the past 30 d of 22,650 children and adults aged 1 to 85 yr of age from NHANES 2001–2006

	Steroid use (n = 181)	No steroid use (n = 22,469)	*P* value[b]
25(OH)D levels (%)			
<10 ng/ml	11	5	0.009
<15 ng/ml	21	15	0.05
<20 ng/ml	36	32	0.39
<30 ng/ml	75	76	0.85
Age (yr)[a]	52 ± 1.98	37 ± 0.35	<0.001
Female (%)	46	51	0.32
Race/ethnicity (%)			
Non-Hispanic white	77	70	0.08
Non-Hispanic black	14	12	0.39
Mexican-American	7	13	0.01
Other, multiracial	2	5	0.18
PIR (%)			
≥5	22	23	0.94
>1–4.99	63	62	0.94
0–1	15	15	0.99
Obese (%)	38	29	0.04
Milk intake (%)			
≥1/d	50	53	0.44
≥1/wk but <1/d	27	23	0.26
<1/wk or varied	9	12	0.36
Never	14	12	0.54
Supplement use (%)			
None	77	82	0.09
<400 IU/d	11	5	0.002
≥400 IU/d	12	13	0.88
Days missed from school or work[a]	10 ± 4.90	4 ± 0.20	0.01
eGFR (ml/min/1.73m^2)[a,c]	76 ± 2.68	100 ± 0.56	<0.001
Any medications used (%)	100	48	[d]

Participants over 85 yr of age are reported as being 85 yr old in order to protect anonymity.

[a]Values are expressed as mean ± SE.
[b]*P* value from linear or logistic regression.
[c]eGFR was only available in participants ≥12 yr of age; number of observations = 18,229.
[d]Unable to calculate *P* value secondary to cell with no participants.

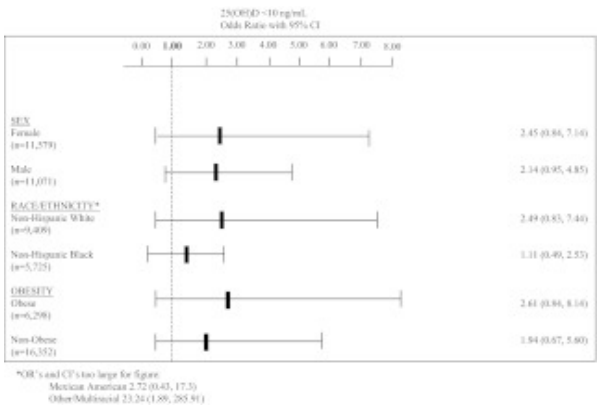
Table 3.

Logistic regression models, OR of low 25(OH)D associated with steroid use

	OR	95% CI
25(OH)D levels <10 ng/ml		
	OR	**95% CI**
Unadjusted model	2.36	1.25, 4.45
Partially adjusted model[a]	2.21	1.06, 4.62
Multivariable adjusted model[b]	2.21	1.01, 4.85
25(OH)D levels <15 ng/ml		
Unadjusted model	1.43	1.00, 2.04
Partially adjusted model[a]	1.23	0.86, 1.77
Multivariable adjusted model[b]	1.36	0.92, 2.00
25(OH)D levels <20 ng/ml		
Unadjusted model	1.17	0.82, 1.67
Partially adjusted model[a]	1.01	0.70, 1.45
Multivariable adjusted model[b]	1.18	0.78, 1.78

[a]Adjusted for age, sex, race/ethnicity.
[b]Adjusted for age, sex, race/ethnicity, PIR, obesity, milk intake, vitamin D supplementation use, any days missed from school or work, and any medication use.

Fig. 1.



Fully adjusted OR and 95% CI for subgroup analyses of sex, race/ethnicity, and obesity.